STATEMENT OF: ANDREW SANDS, REGISTERED MEDICAL PRACTITIONER
Name *Rank*

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

NOT SIGNED IN POLICE OFFICER'S PRESENCE

TO BE COMPLETED WHEN THE STATEMENT HAS BEEN WRITTEN

I declare that this statement consisting of 1 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 10th day of November.

SIGNATURE OF MEMBER by whom statement was recorded or received

Andrew J Sands
SIGNATURE OF WITNESS

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

I am a registered medical practitioner and consultant in paediatric cardiology. I graduated from the Queen's University Belfast on 1st July 1992. My professional qualifications are MPhil, MB, BCh, BAO, MRCP.

H M Coroner has asked me to comment on Mr Alan Roberts' letter of 29th September 2005.

Clearly I am unable to respond to some of Mr Roberts' comments. However I should like to emphasize that I was very concerned regarding Claire's level of consciousness on the morning of 22/10/96. This prompted the urgent neurology referral. I have also stated that I thought a CT scan of brain may be appropriate. At the time this required the sanction of a consultant neurologist.

My immediate worries for Claire were probably allayed to some degree by Dr Webb's assessment. The initial serum sodium result did not seem out of keeping with Claire's presentation. Indeed this was and remains quite a common finding in many hospitalized children. We do not know at what time the second test of electrolytes was requested or taken. Claire had at least one further intravenous cannula inserted before 5pm. This is often when blood samples are taken in children (to avoid another needle). With hindsight, further investigations may well have drawn attention to sodium loss or fluid retention.

I have a clear recollection of quite lengthy discussions with Claire's mother on 22/10/05. Although this was as much to help me understand Claire's condition I believe that I also explained my concerns whilst avoiding alarm. I would have deferred to the senior doctor in attendance for more definitive counselling.

SIGNATURE OF WITNESS..... Andrew J Sands .....

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STATEMENT OF: ANDREW SANDS, REGISTERED MEDICAL PRACTITIONER
Name Rank

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

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Dated this Sixth day of July 2005

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 consultant in paediatric cardiology. I graduated from the Queen's University Belfast on 1st July 1992. My professional qualifications are MPhil, MB, BCh, BAO, MRCP.

May I first express my sympathy with Claire Robert's parents and wider family, having spoken quite recently with them I realize that the passage of time has done little to lessen their grief. At the time of Claire's admission I was employed by the Royal Group Hospitals Trust. I had commenced my first substantive post as a paediatric registrar on the 7th August 1996. Previously I had worked as a locum registrar in paediatric cardiology. I was based in Allen ward in the Royal Belfast Hospital for Sick Children.

I met Claire on the morning of 22nd October 1996. I was conducting a ward round with at least one senior house officer who recorded the ward round notes. It is likely also that there was a senior nurse in attendance. My recollection is that Claire's mother was also present. Claire had been admitted the previous night and the recorded notes suggested a short history of vomiting small quantities, increasing lethargy and impaired level of consciousness. As Claire was not drinking, intravenous fluids, started after admission were continued at maintenance dose. She was given (dextrose 4%/0.18% saline). This was standard fluid therapy at that time.

Claire's past history of seizures and developmental delay were noted as was her elevated white cell count (16.4 thousands/Ul) and slightly low serum sodium (132 mmol/l). On examination Claire's pupils reacted only sluggishly to light. She was largely unresponsive and appeared pale. She appeared to have bilateral upper motor neurone signs. I was very concerned that Claire had a major neurological problem and suspected she was in "non-fitting" status epilepticus. Other recorded differentials were encephalitis or encephalopathy. My recollection is that Claire's mother felt this was not Claire's usual condition, although when unwell she would commonly be lethargic and that she expected her to improve soon. However I (and the ward team) felt that she was really very unwell. A dose of diazepam was given rectally (5mg). I believe this was after contacting Dr. Webb (consultant paediatric neurologist). I recall spending quite some time with Claire and her mother trying to get a clear history and an idea of Claire's normal behaviour. We contacted the Ulster Hospital Dundonald and requested old notes to be faxed to assist with this. Hourly CNS observations were started.

SIGNATURE OF WITNESS.....

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STATEMENT OF: ANDREW SANDS CONTINUATION PAGE NO: 1

I personally went to talk to the consultant paediatric neurologist on call. The paediatric consultant under whom Claire was admitted was unavailable: although I believe she was kept informed by telephone. I described Claire's problem to the paediatric neurologist and told him I thought a CT scan of brain might be required. He came and assessed Claire in Allen ward. He also saw her once if not twice more during the afternoon and prescribed further treatment. I do not recall being present in the mid-afternoon. It may be that I had teaching or other duties. However, I did not feel that Claire's condition had changed. I did administer an intravenous dose of sodium valproate as requested by the neurologist, at 5.15pm. I do not recall if Claire's care had been formally taken over by the neurology team. I note that a further serum electrolyte result is recorded in the chart although it is not clear when this was requested or taken.

I was not on call that night but heard of Claire's sudden collapse subsequently. I was naturally very shocked and saddened. After her death I was asked by nursing staff to speak to Claire's mother and father on the ward. I did this on 11th November 1996 as recorded. I explained, as far as I was able, the course of events but said that I would ask Dr. Steen to discuss the post-mortem findings (of which I was not aware) as soon as possible.

SIGNATURE OF STATEMENT MAKER: Andrew J Sands

HMSO 8990140 C10,000 1/91 GP5560 59102

STATEMENT OF: HEATHER STEEN, REGISTRED MEDICAL PRACTITIONER
Name *Rank*

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

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TO BE COMPLETED WHEN THE STATEMENT HAS BEEN WRITTEN

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Dated this 16th March day of 2005

SIGNATURE OF MEMBER by whom statement was recorded or received

Heather J Steen
SIGNATURE OF WITNESS

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

I am a registered Paediatric Consultant, having qualified at Queen's University Belfast in 1978 with MB. BCH. BAO. I also have a Diploma in Child Health, am a Member of the Royal College of Physicians (Edin) and a Fellow of the Royal College of Paediatrics and Child Health.

This nine year old girl with a history of severe learning disability and previous history of epilepsy was referred to the Accident & Emergency Department of the Royal Belfast Hospital for Sick Children by her General Practitioner on the evening of the 21st October 1996 with a history of vomiting and lethargy since returning from school that day. She was triaged by the emergency nurse at 1903 hours and assessed by the emergency SHO at 1915 hours. The SHO being concerned that she may have been suffering form encephalitis, asked the paediatric registrar Dr. O'Hare to see and assess Claire. The history given at that time was that Claire had suffered loose motions some three days prior to her presentation at the Emergency Department. She had been slightly off form the previous day but had been well enough to go to school. However, on return from school she was drowsy with slurred speech. She had vomited every hour from 3pm and having been assessed by her GP was referred to the Emergency Department. It was noted that she previously had seizures in infancy requiring treatment with Sodium Valproate but anticonvulsant therapy had been discontinued in 1995 by the Ulster Hospital Dundonald Team without recurrence of her seizures to date.

On examination her temperature was 37°c and the only abnormalities noted were on examination of the neurological system. Although able to sit up in bed, she stared vacantly in front of her and only responded intermittently to her parent's voice. She did however respond to deep pain. Fundi were normal and it was noted that her discs were not blurred. Pupils were equal and reacting to light and accommodation. Cranial nerves, 7, 9 and 10 were intact. On examination of her peripheral nervous system it was noted that tone was increased in all four limbs but more markedly on the right. There were brisk reflexes present with bilateral clonus and down going planters. Fuller assessment of the central nervous system was not able to be carried out because of Claire's inability to co-operate. Dr. O'Hare felt that there was an underlying viral illness and had concerns that she may be also having seizure activity. Blood was taken at approximately 2230 hours for full blood picture, U&E, blood culture and nurses were requested to commence four hourly temperature pulse and respiration observations. She also advised that intravenous diazepam was given if there were seizure activity.

Form 38/36 (Plain)

SIGNATURE OF WITNESS Heather J Steen

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STATEMENT OF: HEATHER STEEN CONTINUATION PAGE NO: 1

Dr. O'Hare reassessed her at midnight and felt she was slightly more responsive with no signs of meningism and so advised to continue with observations. It is noted that at the blood results from the sample taken earlier were U&E - sodium of 132 mmol/L, potassium 3.8 mmol/L, urea 3.5, mmol/L glucose, 6.6 mmol/L, creatinine 36 umol/L, chloride 96 mmol/L. Her haemoglobin was 10.4 G/DL with a PCV of 0.35, white cell count 16.5 thousands/UL and a platelet count of 422 thousands/UL.

It was noted over night that she had numerous small vomits and at 11am that she had a loose motion and passed a large volume of urine. She was seen in and around 11 o'clock by Dr. Sands, Paediatric Registrar attached to Allen Ward at that time. Although no seizures had been noted, he was concerned at her continuing unresponsiveness and her overall general condition. He felt that the differential diagnosis should include non-fitting status epilepticus, encephalitis and encephalopathy. He advised that rectal diazepam 5mg be administered per rectum, that her previous notes should be accessed from the Ulster Hospital, Dundonald, where she had been attending for the previous few years and that a neurology opinion should be sought. She was seen around lunchtime by Dr. David Webb, Consultant Neurologist. Her parents were not present at the time but Dr. Webb was able to obtain a history from her grandmother. He noted that *"The picture is encephalopathy most probably postictal in nature. I note (N, biochemistry profile)"*

In view of her non fitting status he advised that she be loaded with intravenous phenytoin 18mg/kg stat followed by 2.5mg twelve hourly. He advised that the levels of phenytoin should be checked six hours after the loading dose. He requested that hourly CNS observations be carried out and that a CT scan be arranged for the following day if her condition did not improve. At 14.45 hours intravenous phenytoin was administered and she was reviewed again by Dr. Webb at approximately 15.10 hours. He felt that she continued to be in status epilepticus and advised commencement of Midazolam with a stat dose of 12mg intravenously infusion of 2mg/kg per minute. The dose being increased hourly to achieve a dose of 69 kg over 24 hours. Dr. Webb reviewed Claire again at 17.00 hours and her mother was in attendance at this time. He notes that Mrs. Robert was able to report that Claire had had contact with a cousin on Saturday who had a tummy upset but Claire then went on to develop loose motions on the Sunday with vomiting on the Monday and some vocal sounds on Monday with right sided stiffening. Although he did not think meningoencephalitis was likely he advised Cefuroxime and Acyclovir for 48 hours with further stool, blood and urine sent for viral cultures and that in view of her continuing non fitting status that intravenous Sodium Valproate be commenced with a loading dose of 20mg/kilo IV and then an infusion of 10mg/kilo over 12 hours. CNS observations continued to be carried out over this period of time and it was noted that Claire's Glasgow coma scale varied between 6 and 7.

At 1915 hours Claire clenched her teeth and groaned for approximately one minute. It was noted at 2100 hours that she passed urine but also had an episode of screaming and drawing up of her arms. Her pulse rate increased at that time to 165 b/min and her pupils were large but reacting to light. This episode lasted approximately 30 seconds and the doctor was informed of this. At approximately 2100 hours bloods were taken for phenytoin levels along with a repeat urea and electrolytes. Intravenous antibiotics were given and her drug card was rewritten by Dr. Hughes. It is noted at that time she had a low grade pyrexia with a Glasgow coma scale of 6.

SIGNATURE OF STATEMENT MAKER: Heather S Steen

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STATEMENT OF: HEATHER STEEN CONTINUATION PAGE NO: 2

At 2330 hours blood results were received from the sample takes at approx 2100 hours, showing a sodium of 121 mmol/L, potassium of 3.3 mmol/L, urea 2.9 mmol/L and creatinine 33 umol/L. Dr. Neil Stewart, Paediatric SHO, was informed of these results and discussed them with Dr. Brigitte Bartholome, the Paediatric Registrar, on call for the hospital. She advised that the N/5 saline be reduced to 2/3 of its present value i.e. 41 mls per hour and that urine be sent for osmolality.

At approximately 0230 hours a nurse in attendance with Claire noticed a slight tremor of her right hand lasting a few seconds and her breathing became laboured and grunted. Oxygen and suction were given and Dr. Bartholome was called to assess her breathing. She felt that she had Cheyne-Stoke breathing and required Paediatric Intensive Care. Claire was intubated by the anaesthetic Registrar and transferred to PICU. Dr. Seamus McKaigue, Consultant Paediatric Anaesthetist on-call for Paediatric Intensive Care, was contacted as was myself. We both attended and I noted at 0400 hours that Claire had been intubated and ventilated. She had had some Midazolam but it was no longer running. Her pupils, however, were fixed and dilated with bilateral papilloedema, more marked on the left fundus. She had no response to painful stimuli. Her blood pressure was maintained at 90/55 and her heart rate was 100b/min. I advised that an infusion of Manitol should be given along with a Dopamine infusion to maintain her blood pressure and an urgent CT scan be carried out. I also contacted Dr. Webb who attended at 0440 hours. The CT scan was performed at 0530 hours and showed severe diffuse hemispheric swelling with complete effacement of the basal cisterns, no focal abnormality was identified.

Dr. Webb and myself discussed Claire's condition with her parents, emphasising that we felt she had cerebral oedema as confirmed by her CT scan which had resulted in coning of her brain and brain stem death. We also discussed the possibility of organ donation.

At 0600 hours Dr. Webb and myself completed brain stem death protocol and blood which was drawn for U&E at that time showed a sodium of 129 mmol/L, potassium of 3.6 mmol/L, urea of 3.7 mmol/L and osmolality of 274 mmol/kg. At 0710 hours Dr. McKaigue summarised Claire's admission to Paediatric Intensive Care and highlighted his concern about deterioration in her arterial blood gases, which might have been in keeping with pulmonary aspiration or early neurogenic pulmonary oedema. At 0800 hours he advised that her maintenance fluids be changed to normal saline with urea and electrolyte levels checked two hourly.

At approximately 1000 hours Dr. Bob Taylor, Consultant Paediatric Anaesthetist in charge of Paediatric Intensive Care on the 23rd October noted that Claire had now become polyuric and hypotensive with a systolic blood pressure of 70. He advised a bolus of HPPF 500mls to be given along with an infusion of Desmopressin 4mcg in 39 mls of normal saline at 1ml per hour. A repeat urea and electrolyte carried out during the afternoon showed a sodium of 152 mmol/L, potassium of 2.8 mmol/L, urea 3.3 mmol/L, calcium 2.69 mmol/L and serum osmolality of 313 mmol/kg.

At 1825 hours Dr. Webb and myself completed the second part of the brain death protocol and following discussion with her parents there was agreement that ventilation should be withdrawn. Consent was also requested for post mortem and given for a limited post mortem only of her brain. Her ventilation was discontinued at 1845 hours and Claire died shortly afterwards.

Form 38/36 [a] Plain)

SIGNATURE OF STATEMENT MAKER: Heather S Steen

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STATEMENT OF: IAN YOUNG, CONSULTANT IN CLINICAL BIOCHEMISTRY
Name *Rank*

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

NOT SIGNED IN POLICE OFFICER'S PRESENCE

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Dated this day of

SIGNATURE OF MEMBER by whom statement was recorded or received

Ian Young
SIGNATURE OF WITNESS

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

I am a registered Consultant in Clinical Biochemistry, and qualified at Queen's University Belfast in 1985 with MB BCH BAO. I am Fellow of the Royal College of Physicians (London), Fellow of the Royal College of Physicians of Ireland and a Fellow of the Royal College of Pathologists.

I was asked to review the medical records of this 9-year-old girl by Dr Michael McBride, Medical Director of the Royal Group of Hospitals. I was asked to give my opinion on whether hyponatraemia may have contributed to Claire's death. This statement is based on my inspection of the medical and nursing notes relating to her hospital admission in 1996. In addition I spoke to Dr Heather Steen, Dr Andrew Sands, Dr Nichola Rooney and to Claire's parents. I have provided an honest and true opinion based on my reading of the notes. However, I did not have access to comments from all of the other medical practitioners involved in Claire's care.

Claire was referred to the Accident and Emergency Department of the Royal Belfast Hospital for Sick Children by her general practitioner on the evening of the 21st October 1996 with a history of vomiting and lethargy. Blood was taken at approximately 22.30 hours for an estimation of urea and electrolytes. It is noted that this revealed serum sodium of 132mmo/l. A "down arrow" is present beside the sodium of 132 mmol/l at 12 midnight on the 21st October, indicating that the sodium was noted to be below the lower reference limit. A subsequent note in the chart by Dr David Webb, Consultant Neurologist, from around lunchtime on the 22nd October 1996, states: "I note (N, biochemistry profile".

Claire received intravenous fluid replacement following admission and throughout the day of the 22nd October with predominantly 0.18% saline / 4% dextrose. There was a progressive deterioration in her clinical condition with evidence of status epilepticus. A record of fluid balance is present, but losses are not accurately recorded so that fluid balance cannot be judged.

A repeat blood sample was taken at around 9pm on the evening of the 22nd October. A note timed 23.30 on the 22nd October records serum sodium of 121mmol/l, and suggests that fluid overload with low sodium containing fluids or syndrome of inappropriate ADH production were considered as possible diagnoses. Intravenous fluid replacement was reduced to 2/3rds of previous values. A note was taken to send urine for osmolality although there is no record of a result.

Form 38/36 (Plain)

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STATEMENT OF: IAN YOUNG CONTINUATION PAGE NO: 1

At approximately 3am on 23rd October Claire suffered a respiratory arrest and was noted to have fixed dilated pupils. She was transferred to the Paediatric Intensive Care Unit. At 4am it is noted that pupils were fixed and dilated and there was bilateral papilloedema. A Note at 4.40am on the 23rd October form Dr David Webb indicated the likely diagnosis of syndrome of inappropriate ADH production with hyponatraemia, hypo-osmolality and cerebral oedema following prolonged epileptic seizures. Claire subsequently died on the 23rd October at 18.45 hours. A death certificate was issued indicating cerebral oedema secondary to status epilepticus.

I informed Dr Michael McBride, the Medical Director of the Trust that in my opinion hyponatraemia may have made a contribution to the development of cerebral oedema in Claire's case. I advised that it would be appropriate to consider discussing the case with the coroner for an independent external opinion with access to statements from all of the staff involved in Claire's care.

SIGNATURE OF STATEMENT MAKER: I A Young

STATEMENT OF: IAN YOUNG, CONSULTANT IN CLINICAL BIOCHEMISTRY
Name *Rank*

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

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Dated this day of

Ian Young

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 Consultant in Clinical Biochemistry, and qualified at Queen's University Belfast in 1985 with MB BCH BAO. I am Fellow of the Royal College of Physicians (London), Fellow of the Royal College of Physicians of Ireland and a Fellow of the Royal College of Pathologists.

In addition to my previous statement I have been asked to comment on the reports by Dr R M Bingham and Dr Maconochie and a response from Mr Alan Roberts.

In general, I agree with the conclusions which Dr Bingham has reached. However, I would like to make the following comments:

1) On page 3 of his statement, in paragraph 1, Dr Bingham interprets the written note from Dr Webb to say: 'I note no biochemistry profile'. In my earlier statement, I interpreted this note to mean: 'I note normal biochemistry profile', and having reviewed the chart I continue to interpret the note in this way. There is a biochemistry profile result recorded in the notes prior to Dr Webb's written note, and this seems inconsistent with Dr Bingham's interpretation of the comment.

2) On page 4, paragraph 1, Dr Bingham indicates that it is unlikely that the serum sodium on admission (132mmol/l) was the cause of Claire's presenting symptoms. I think that this is an important point, with which I agree. While Claire's sodium was low on admission, the degree of hyponatraemia was relatively minor and was unlikely to be making a significant contribution to her presentation.

3) As indicated by Dr Bingham, urine output from Claire was not measured. Dr Bingham believes that there is sufficient recorded information relating to wet nappies to conclude that urine output was reasonably high. I do not think that it is possible to reach any conclusion as to whether urine output was high or low.

4) Dr Bingham indicates that the intravenous fluid volume recorded in Claire's notes would not be sufficient to account for the fall in her serum sodium. In contrast, I do not think that it is possible to reach any firm conclusion on this matter in the absence of any record of urine volume or urinary sodium concentration. I believe that the changes in Claire's serum sodium are entirely consistent with the recorded intravenous fluid intake when possible urinary losses of water and sodium are taken into account.

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STATEMENT OF: IAN YOUNG CONTINUATION PAGE NO: 1

5) In his report, Dr Bingham raises the possibility that the serum sodium measurement of 121 mmol/l was wrong. The laboratory measurement of sodium is extremely accurate. Assuming that an appropriate sample was taken (and there is nothing in the notes to suggest that sample collection was difficult), I believe that the possibility of an inaccurate laboratory result is negligibly small.

In addition to the above comments, I would like to make one comment in response to the letter from Mr Alan Roberts dated 29th September '05. Mr Roberts refers to my earlier statement that: 'The practice at that time would be firstly, to restrict fluid intake and secondly, to consider administration of fluid with a high content of sodium, if symptoms attributable to hyponatraemia were present'. This statement was made in response to a question about the action taken when Claire's serum sodium was noted to be 121mmol/l. In my opinion, when Claire was initially admitted her serum sodium of 132mmol/l was unlikely to have made a significant contribution to her presenting symptoms, although serum sodium was slightly below the lower reference limit and therefore in the hyponatraemic range.

Form 38/36 [a] Plain)

SIGNATURE OF STATEMENT MAKER: Ian Young

HMSO 8990140 C10,000 1/91 GP5560 59102

STATEMENT OF: DAVID WEBB, CONSULTANT PAEDIATRIC NEUROLOGIST
Name *Rank*

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

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Dated this day of

SIGNATURE OF MEMBER by whom statement was recorded or received

DWMWebb
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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In August 1987 aged 7 months she was started on Carbamazepine (an anti-convulsant). Investigations at that stage including cererbral 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? Apyrexic (*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

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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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 am

The 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. Apyrexic (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. Apyrexic. 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 - morning

Paediatric 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. Apyrexic. 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.

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 - 2pm

Paediatric 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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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 - 8pm

Paediatric 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.

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 ? SIADH.
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 am

Doctor'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 - 4am

Paediatric 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 Acylovir 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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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

*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. Serium 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 CO_2 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.'*

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STATEMENT OF: DAVID WEBB CONTINUATION PAGE NO: 9

Review of Investigations

Blood results

	October 21	22nd	23rd
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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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

Time	Score	Response
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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STATEMENT OF: DAVID WEBB CONTINUATION PAGE NO: 11

Neurological Observations - Glasgow Coma Scale

Time	Score	Response
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.

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).

ML ① Urine output
Urea ↔ PU large → 1000 Nursing note.
HRC – "600 ml" round
– Physical impossibility of
1 Litre

② Impairment after Diazepam is an indicator of oxygen activity

HRC Read SHO admission note Imp.

Diazepam impaired
CSF shunt
Case book —
SIADH from infection
Blood & sample from SHO on duty

Fluids
Trigger for SIADH urea monitoring
Candidate for electrolyte imbalance

References

1. Al-Zahara Omar F, Al Bunyan M. Severe hyponatremia as poor prognostic factor in childhood neurological diseases. J Neuro Sci,1997:151(2);213-216
2. Handbook of Pediatrics, Hospital for Sick Children, Toronto, 9th Edition, 1997
3. Forfar and Arneil's – Textbook of Pediatrics, 5th Edition, 1998 (4th Edition 1992)
4. Moritz ML, Ayus JC. Prevention of hospital acquired hyponatremia: a case for using isotonic saline. Pediatrics, 2003;111(2);227-230