Form 22

CORONERS ACT (NORTHERN IRELAND) 1959

VERDICT ON INQUEST

On an inquest taken for our Sovereign Lady the Queen, at THE OLD TOWNHALL BUILDING, 80 VICTORIA STREET, BELFAST on THURSDAY the 4TH day of MAY 2006, before me MR J L LECKEY, Senior Coroner for the Coroners Service of Northern Ireland touching the death of CLAIRE MARGARET ROBERTS to inquire how, when and where the said CLAIRE MARGARET ROBERTS came to her death, the following matters were found:

1. Name and surname of deceased: CLAIRE MARGARET ROBERTS

2. Sex: FEMALE

3. Date of Death: 23 OCTOBER 1996

4. Place of Death: ROYAL BELFAST HOSPITAL for SICK CHILDREN

5. Usual Address: [redacted]

6. Marital Status: SINGLE

7. Date and Place of Birth: 10 JANUARY 1987 ULSTER HOSPITAL, DUNDONALD

8. Occupation: SCHOOLGIRL - DAUGHTER OF ALAN JOHN ROBERTS - [redacted]

9. Maiden Surname: N/A

10. Cause of Death: 1(a) CEREBRAL OEDEMA

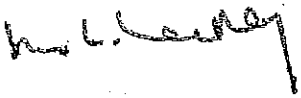
Due to

(b) MENINGO-ENCEPHALITIS, HYPONATRAEMIA DUE TO EXCESS ADH PRODUCTION AND STATUS EPILEPTICUS

Findings: On 21st October 1996 Claire was referred by her General Practitioner to the Accident and Emergency Department of the Royal Belfast Hospital for Sick Children. She was pale and lethargic, she had vomited three times and her pupils were reacting but she did not like light. She had a past history of moderate learning

difficulties and epilepsy. On examination at the Accident and Emergency Department that night she exhibited the same symptoms. Her neurological function was found to be abnormal and it was decided to admit her with a provisional diagnosis of an unspecified viral illness query encephalitis. On admission to the ward she was noted as not responding to her parents' voice though intermittently responding to deep pain. By midnight as she had become slightly more responsive it was decided to observe her overnight and re-assess her in the morning. Blood test results then available showed a low serum sodium of 132mmol/l. Though constituting hyponatraemia this reading was not sufficiently low to explain her presenting symptoms. Blood test results at 23.30 on the 22nd October and at 03.00 on the 23rd October showed that the serum sodium level had fallen to an extremely low level of 121 mmol/l. At about 03.00 Claire suffered a sudden respiratory arrest and her pupils became fixed and dilated. A CT scan showed severe swelling of the brain (cerebral oedema). She died later that day. The fall in the serum sodium level to 121 mmol/l was significant and I am satisfied from the evidence that hyponatraemia of this degree contributed to the development of the cerebral oedema that caused Claire's death. However, from the evidence before me I accept that it was not the only underlying cause of her death, the others being meningo-encephalitis and status epilepticus. Each of these three contributed, though in proportions that cannot be determined. I accept the evidence of Dr Heather Steen, Consultant Paediatrician, that the first blood test showing a serum sodium level of 121 mmol/l should have led to a clinical re-assessment of Claire. That blood test should have been repeated and at the same time there should have been a reduction in fluids. However, by then it was unlikely that Claire's condition was survivable even if prompt action had been taken. Dr Steen also stated that now the fluid management of Claire would have been different.

Witness my hand this 4TH day of May 2006.

Signature  Senior Coroner for the Coroners Service of Northern Ireland

Statement on Claire Roberts

The following statement is on my daughter Claire, her illness, subsequent treatment and care management at the Belfast Royal Hospital.

Claire attended school on Monday 21 October 1996 and her teacher reported that she had been sick in school before returning home at approximately 15.00. This sickness continued at home with Claire vomiting on two or three occasions. She also had one loose bowel movement at home but no continuous diarrhoea symptoms.

Claire's GP Dr Savage (Castlereagh Medical Centre) was called for advice; she called to our home at approximately 18.00 to examine Claire. Dr Savage recommended that Claire be taken to Hospital. Claire was admitted to the Belfast Royal Hospital on Monday 21 October 1996 at 19.00. She was administered intravenous fluids on Allen Ward over the following hours and Doctors' advised my Wife and I that she had a viral infection. We asked about other illnesses and were relieved that Doctors did not think Claire was in danger from meningitis.

Claire appeared more settled after 21.00, was asleep so my wife and I left the hospital to prepare for Tuesday morning and our two sons schooling.

My wife and I arrived at the hospital on Tuesday morning and were pleased to be advised by nursing staff that Claire had been comfortable through the night. However Claire did not appear to be herself that morning and my wife and I expressed our concerns to Dr Sands about her lack of response.

My wife and I stayed with Claire for the rest of that morning and when both grandparents arrived aronnd 13.00 we went for lunch. We actually went into Belfast for some personal items for Claire, in the hope that her viral infection would pass and she would possibly be ready to leave hospital the next day.

On returning to hospital at around 14.00 grandparents informed me that a Doctor had examined Claire. I left the hospital at 15:00 to collect our two sons from school with my wife remaining in hospital with Claire.

I returned to hospital at approximately 18.30 with our two sons and my wife informed me that Doctor Webb had examined Claire at 16.00 and 17.00 with a different type of medication being administered. I assumed that his medication was counteracting any viral infection Claire had and was having a sedation effect. Like all children Claire over the years had had several childhood illness from measles to common cold which would have made her unwell for a few days before she would bonnce back into action.

Over the following hours to 21:15 Claire was reviewed by the ward nurse in a way that appeared as general observation and certainly without alarm or concern.

We left the hospital at 21:15 with as we thought Claire settled and asleep and a reassurance from nursing staff that Claire was comfortable. We informed the nursing staff that we would return to the hospital the following morning. Throughout Tuesday 22 October no Doctor, nurse or any medical staff indicated to my wife or I that Claire was in a serious condition or in any danger.

I received a call from the hospital at 3:45 Wednesday 23 October to say that Claire was having breathing difficulties and that my wife and I should make our way to the Hospital as soon as possible. On arrival Dr Steen and Dr Webb informed us that there was a build up of fluid around Claire's brain and pressure was being applied to her brain stem. Claire was being sent for a CT scan to confirm this.

Dr Steen and Dr Webb later advised us that the outcome of the CT scan confirmed severe fluid build up, that Claire was brain dead and that nothing could be done to save her. At 18:45 a decision was taken by my wife and I to discontinue Claire's life support.

Having reviewed all the reports and letters regarding Claire's diagnosis and treatment I would like to make the following comments:-

1. Claire's diagnosis on admission to hospital, during her treatment on Allen ward, at ICU and at subsequent meeting in 1996/1997 at the Belfast Royal Hospital was a viral infection.
   The post-mortem report (condensed and full versions) also refers to a viral infection.
   Subsequent meetings with Dr Steen at the Belfast Royal Hospital continued to state a viral infection.

   At no time was Hyponatraemia or falling sodium levels defined as a cause for the fluid build up.
   At a meeting on 7 December 2004 with medical staff from the Belfast Royal Hospital Professor Young stated that hyponatraemia (falling sodium) may have contributed to swelling of Claire's brain and therefore ultimately to her death.

2. At a meeting on 7 December 2004 Dr Steen and Dr Sands stated that Claire was very unwell.
   Why was this concern never expressed to my wife or I?
   Why was Claire not examined by a Doctor between 17:00 and 21:15 on Tuesday 22 October 1996 if she was so unwell?

   At 21:30 blood cultures were taken to check for 'viral infections' (requested at 17:00 by Dr Webb). Why was there a 4.5 hour delay before this blood test was taken, which actually amounts to a 6.5 hour delay between blood sample request and results being available?

   At 23:30 blood tests revealed a sodium level of 121mmol/l (taken at 21:30) possibly dropping to 120 mmol/l or less by 23:30.
   Why was Claire's sodium allowed to drop to such a critical level without being monitored over a 27 hour period?
   Why was Claire not admitted to ICU at 23:30 when her condition became critical?
   Why were we not informed at 23:30 of this critical development, considering in Professor Young's opinion that at 23:30 Claire's condition was irreversible?
   Why were we allowed to leave hospital 2 hours earlier without any concerns being expressed by medical staff?
   What level of medical care was Claire given between 23:30 and 3:00 on Wednesday 23 October?
   Why was there a 4 hour delay between 23:30 and 3:45 before we were contacted?

3. Why were no urine tests carried out from Claire's admission to hospital until 23:30 on Tuesday 22 October?
   Was a urine test carried out at 23:30 and are the results available?
   Why were tests not carried out to check Claire's urine output?
   Would a urine test identify urine with a substantial sodium quantity?

4. Why was an inquest not held into Claire's death considering it was sudden, unexpected and without a clear diagnosis?
   Why did Dr Steen state to me on Wednesday 23 October 1996 at approximately 19:00 that there would be 'no need' for an inquest?

   Dr Steen highlighted that a post mortem may give answers to Claire's death and help prevent similar tragedies in the future. Was a report issued and did it define hyponatraemia?
   Why does the post mortem report not mention hyponatraemia?

I would like to make the following comments and highlight some points on Dr Bingham's report dated 14 April 2005.

Page 3 Para 8

'.....This difficulty may have contributed to the delay in recognition of the serious nature of her condition'

Page 4 Para 1

If hyponatraemia was not considered to be the cause of the presenting symptoms would it not have been essential to monitor a low sodium level of 132mmol/l which was falling to below 121mmol/l within 27 hours.

Page 4 Para 2

Claire was started on intravenous fluids, however in response to a question raised at a previous meeting on fluid administration Professor Young states that 'The practice at the time (October '96) would have been firstly, to restrict fluid intake and secondly to consider administration of fluid with a high content of sodium if symptoms attributed to hyponatraemia were present'.
This refers to sodium levels below 135mmol/l
(Reference Point 9 letter dated 12 January 2005 from Belfast Royal Hospital}

Page 4 Para 2

*'.....A record of poor urine output could confirm this but in fact there are several notes of her passing urine recorded on the fluid charts and on one occasion it was noted that there was a large volume'.*

Unfortunately no accurate records of urine output were taken. The records of urine output referred to on the fluid charts were observations of a damp nappy made by Claire's mum who was only concerned about Claire's comfort.
No accurate urine tests were carried out to check volume or composition of Claire's urine over a 27 hour period.

Page 4 Para 3

*'.....Another possibility is that she was passing urine with very high sodium content.....'.*

This highlights the fact that no urine tests were carried out which would have given critical information on Claire's fluid excretion loss/urine composition.

If Claire was passing urine with very high sodium content this highlights the importance of urine testing, none of which was carried out.

Page 4 Para 3

*'.....Finally it is possible that the result was inaccurate as the sodium levels in the ICU at 06.00 on 23 October 1996 were much higher (133mmol/l (blood gas analyser) or 129mmol/l (laboratory))'*

Laboratory results are more accurate than blood gas analyser as defined by Doctor in Adam Strain case. There is also a 9 hour gap between the 121mmol/l and 129 mmol/l reading over which time Claire was receiving a more appropriate fluid management albeit too late.

In ICU i.e. after 3:00 Claire was administered mannitol. Would this medication increase sodium levels?

Page 4 Para 4

*'.....It is likely this was the cause of deterioration in Claire's condition on the evening of 22 October 1996, a sodium of 121mmol/l is known to cause brain swelling and convulsions which can progress to respiratory arrest and death'.*

I believe this to be the true diagnosis for Claire i.e. hyponatraemia and that the low sodium level reading from the laboratory was totally accurate. It also highlights that other blood tests should have been taken earlier.

Page 4 Para 5

*'The understanding of complex medical problems is always much easier with hindsight; in particular in this case there has been much recent publicity in both the lay and medical press which has led to a better appreciation of the dangers of hyponatraemia in children and helped to clarify the cause of this tragedy. Much of this information has only been available in the last five years'.*

The dangers of hyponatraemia and low sodium levels were clearly obvious to medical staff at the Belfast Royal Hospital following the death of Adam Strain in November 1995 and the subsequent inquest and investigation into hyponatraemia in June 1996. This case also refers to numerous medical reports on hyponatraemia such as the BMJ Arieff report dated 9 May 1992.

Dr Webb examined Claire on Tuesday 22 October 1996. He also examined Adam Strain on November 1995 defining acute cerebral oedema as a result of sudden fluid shift.

Page 4 Para 6

*'I feel that Claire's initial diagnosis and management was reasonable. A viral illness was a common and likely diagnosis and although her serum sodium was low it was not excessively so. Her fluid prescription was in line with the practice of the time and although current guidance would be to use fluid with high sodium content in this situation, this advice did not exist in 1996'.*

Comments as in paragraph 5 above plus statement made by Professor Young 'in Claire's case it was felt to be due to the syndrome of inappropriate antidiuretic hormone section (SIADH). The practice at that time would have been 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. (Reference Point 9 letter dated 12 January 2005 from Belfast Royal Hospital.)

Page 5 Para 1

*'I think there was also confusion about Claire's usual neurological status, which complicated her evaluation and led to an underestimate of the severity of the condition'.*

Page 5 Para 2

*'The initial and subsequent anti-convulsant treatment was logical, given the working diagnosis and it is unlikely it would have worsened the consequences of hyponatraemia although it may have masked the symptoms'.*

Was the working diagnosis correct considering that hyponatraemia was not thought at the time to be a major contributor to Claire's condition (letter 12 January 2005) although by 4:30 on Wednesday 23 October Dr Webb considered Claire to have SIADH leading to hyponatraemia and cerebral oedema.

If anti-convulsant treatment 'may have masked the symptoms of hyponatraemia' would it not then have an impact on the diagnosis?

Page 5 Para 3

*'The hyponatraemia was probably an associated feature of Claire's condition rather that the primary illness. It was most likely to have been a result of the combination of raised levels of anti-diuretic hormone together with intravenous infusion of fluid with low sodium content although the volumes infused do not fully account for the sodium becoming so low'.*

Hyponatraemia is defined as serum sodium less than 135 mmol/l. Acute onset may cause cerebral oedema and requires prompt diagnosis and correction.
Diagnosis involves careful history taking and a comprehensive clinical and physical examination by obtaining laboratory values of serum osmolality, urine osmolality and urine sodium.

I would agree that SIADH plus incorrect fluid type would result in hyponatraemia.

Page 5 Para 4

*'I think is most likely that hyponatraemia was the cause of the neurological deterioration.....'*

This defines that hyponatraemia was the cause of Claire's deterioration and highlights the mis-diagnosis that hyponatraemia was not thought at the time to be a major contributor to Claire's condition.
(Ref point 8 letter dated 12 January 2005 from Belfast Royal Hospital)

The possibility of the serum sodium result being an isolated artefact is highly unlikely given the accuracy of laboratory tests. This also highlights the lack of blood tests carried out considering doctors stated Claire was so unwell.

Page 5 Para 5

*.....'Assuming hyponatraemia was the cause, it is likely that identification of a low sodium level when the absence of a biochemical profile was noted, followed by the institution of a fluid restriction regime would have ameliorated its consequence. It is also possible that aggressive treatment at 21.00 when Claire's coma score reduced from 8 to 6 may have been affective. Although the measures taken at 23.30, when the sodium result was available, were of the correct type they were too little and too late'.*

I believe that this paragraph defines hyponatraemia as the cause of Claire's deterioration and also highlights the shortfall in her care management over a 27 hour period.

With a sodium level reading of less that 121mmol/l at 23.30 Claire's condition had deteriorated beyond the point of recovery and any other additional measures were too little too late. However as parents we were allowed to leave the hospital at 21.15 and were not informed of Claire's condition until 3.45 on Wednesday 23 October 1996.

I would like to make the following comments and highlight some points from Dr Maconochie report

Page 2 Para 11 and Para 12

*'Dr Webb suggested commencing more antiepileptic medication, hourly neurological recording and for her to have a CT the next day should she not 'wake up''.*

*'She was noted not to have responded to the antiepileptic medication and therefore additional medication was commenced'.*

Claire was not responding to several antiepileptic medications. Was this not an indication that other symptoms i.e. fluid build up, falling sodium levels and hyponatraemia were central to Claire's condition?

Between 17:00 and 21:30 how were the hourly neurological recordings carried out? My wife and I only recall a fairly general nursing care with the biggest alarm being Claire shaking off her finger pulse monitor.

Why was Dr Webb prepared to wait until the next day for a CT scan if Claire was considered as very unwell or in any danger?

Page 3 Para 2

*'Dr Webb prescribed antibiotic and anti viral medication to be started as a precaution albeit he thought the likelihood for either a bacterial or viral meningitis to be present was low; he asked for viral cultures to be taken to see if a viral infection could account for Claire's condition and that another anti-epileptic medication be started'.*

Dr Webb requested viral cultures at 17:00. Why was there a delay of over 4 hours before these samples were taken?
At 17:00 a viral infection was still being attributed to Claire's condition and yet another anti-epileptic medication was started. Why was hypouatraemia not considered at this stage?

Page 3 Para 3

*'The notes record that at 23.30, the results of the blood samples were available, showing hyponatraemia and her fluid management was altered. I will defer to Dr Bingham regarding the management of her fluid regime'.*

The blood test result at 23:30 show that Claire's sodium level had dropped to below 121mmol/l and that hyponatraemia was the cause of Claire's illness and deterioration. In Professor Young's opinion it was likely that Claire had deteriorated beyond the point of recovery by this time.
(Reference point 7c letter dated 12 January 2005 from Belfast Royal Hospital)

It is also Dr Bingham's view that any measures taken at 23:30 were too little too late.
Was this the first time that Doctors released Hyponatraemia was the major cause of Claire's illness?
Why was Claire not admitted to ICU at 23:30?
Why was Claire treated on Allen Ward for a further 3.5 hours leading to a respiratory arrest?
Why were we able to leave the hospital at 21:15 with no serious concerns for Claire's well being?
Why were we not informed by the hospital at 23:30 of this serious development?
Given that Claire had learning difficulties should more vigilance have been shown regarding her low sodium level on admission and the subsequent fall in sodium level?

All the above points raise serious questions about Claire's management plan, her management on Allen Ward and the management of her neurological presentation.

Page 3 Para 6

*'She was reviewed at 4:30 by Dr Webb, who considered her to have a syndrome of inappropriate anti-diuretic hormone production, leading to hyponatraemia and cerebral oedema.....'*
This diagnosis has changed from the initial diagnosis that hyponatraemia was not thought at the time to be a major contributor to Claire's condition.
(Reference point 8a lettered dated 12 January 2005)

Page 3 Para 10

*'Intensive care support was withdrawn from Claire at 18:45 and a death certificate for cerebral oedema secondary to status epilepticus was written'.*

Why was hyponatraemia not defined on the death certificate given that Dr Webb considered Claire to have SIADH leading to hyponatraemia and cerebral oedema?

Page 3 Para 11

*'Claire Roberts was admitted with abnormal neurological symptoms and signs. The diagnosis of encephalitis/encephalopathy was made at an early stage of her admission.....'*

Was this diagnosis correct given that the symptoms for hyponatraemia are primarily related to the central nervous system and include signs of nausea, lethargy, disorientation, agitation, seizures, depressed reflexes aud focal neurological deficits?

Page 4 Para 2, 3 and 4

These paragraphs refer to Claire's care management. I refer to the points made on page 3 paragraph 3 and also to the timeline of Claire's treatment.

Summary

My wife and I now firmly believe that Claire's death is attributable to hyponatraemia, the delay in the recognition of this condition and the diagnosis and subsequent treatment Claire received at Belfast Royal Hospital.
We welcome the decision made by Mr Leckey H.M. Corner that an Inquest will be held into Claire's death and remain hopeful that Mr O'Hara QC will include Claire's case in the current inquiry into hyponatraemia related deaths

I would also like to refer to some extracts from articles published iu relation to hyponatraemia.

BMJ volume 304 9 May 1992. Arieff Report.

'In the prospective population the serum sodium concentration on admission was 138 (sd2) mmol/l. From three to 120 inpatient hours after hypotonic fluid administration patients developed progressive lethargy, headache, nausea and emesis with an explosive onset of respiratory arrest'.

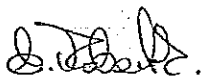
'This level of urine hypertonicity in the presence of hyponatraemia suggests that the plasma antidiuretic hormone concentration was raised. The onset of respiratory arrest was often explosive in nature and hyponatraemia was generally not considered as a possible cause'.

'Hyponatraemia in these children seems to have been caused by extensive extrarenal loss of electrolyte containing fluids and intravenous replacement with hypotonic fluid in the presence of antidiuretic hormone activity'.

'It is important to recognise that in children when there is substantial extrarenal loss of electrolytes a minimal positive balance of hypotonic fluid can lead to fatal hyponatraemia. Another major factor which may have contributed to the high morbidity among these children was the virtual absence of timely treatment in the presence of obvious symptoms.

'Recent studies show that recovery, even after the onset of seizures and apnoea may be possible if appropriate treatment is instituted in a timely manner'.

'When a paediatric patient receiving hypotonic fluids begins to have headache, emesis, nausea or lethargy the serum sodium concentration must be measured'.



Mr Alan Roberts
29 Sep. 05

re: **Claire Roberts (deceased)**

I have had sight of the medical notes for Claire Roberts, having been asked to comment on her attendance to the Royal Belfast Hospital for Sick Children on 21st October 1996. I understand that Dr Robert Bingham, consultant paediatric anaesthetist at the Hospital for Sick Children, Great Ormond Street, London has been asked to comment on the fluid management and the role that Claire Robert's hyponatraemia played in her subsequent demise.

I will outline the sequence of events from the medical notes and comment on her management.

The sequence of events in Claire Roberts' life from 21st October 1996

GENERAL PRACTITIONER
Claire Roberts had been referred to the Accident and Emergency Department by the General Practitioner with the following history:

9 year old girl with severe learning difficulties and a past medical history of epilepsy. He noted that she had been fit free for 3 years and had been weaned off her anti-epileptic medication eighteen months previously. She had not been speaking since coming home from school, where she had been noted as being very lethargic. She had vomited 3 times. Speech slurred earlier on.

The general practitioner noted on examination that she was pale, that her pupils were reacting but that she did not like light. There was no neck stiffness, no raised temperature but increased tone on the right side of her body, and the right plantar reflex was up going (*which is abnormal*). Her ear, nose and throat examination was normal. Her respiratory system was clear.

Correctly, the general practitioner was concerned about her neurological status and made a provisional diagnosis of further fits or underlying infection (*probably questioning the possibility of encephalitis, that is an inflammation of the brain*).

INITIAL ASSESSMENT AT ROYAL BELFAST HOSPITAL FOR SICK CHILDREN

She attended the Children's Hospital with her mother at 18.57 hours and was seen at 19.15 hours.
Her vital signs were: respiratory rate 24 breaths per minute, heart rate 96 beats per minute, which are both elevated for her age. She did not have a raised temperature.

The history and examination were largely unchanged from examination made by the GP. Her speech was noted to be very slurred and that she was hardly speaking. She was drowsy and the neurological examination was abnormal in ~~that~~ the right side of her body and there was an increased tone noted. There were bilateral down going plantars i.e. now both right and left plantar reflexes were abnormal. There was clonus in both ankles, which is indicative of abnormal neurological functioning.

The decision to admit her was made with a provisional diagnosis of ? encephalitis, signed by Dr O'Hare.

INITIAL ADMISSION NOTES

Claire was admitted to Allan ward.

8pm The first medical notes of her admission were made at 8 pm.

She was noted to be sitting up and staring vacantly, only responding intermittently to voice, but responded to pain. A full examination could not be conducted owing to her inability to co-operate. She ascribed a preliminary diagnosis of viral infection. I note that encephalitis had been scoured out of the notes.

Appropriate investigations were instigated and she was managed by being commenced on intravenous fluids and in the notes intravenous diazepam prescribed if she were to show seizure activity. She was to be reassessed after the intravenous fluids had been given. This is signed by Dr O'Hare.

At midnight she was seen by the SHO who recorded that she was slightly more responsive and that she did not have the clinical features of meningitis.

The sodium is 132 mmol/l which is on the low side of normal. She had a rasied white cell count which may be indicative of infection.

The next day, on the ward round was lead by Dr Sands. Claire had not had any seizure activity and showed little response as compared with her normal status.

On examination her pupils were sluggish in response to light stimulus and she showed abnormal long tract signs (that is, abnormal neurological responses).

From these findings, Dr Sands made the differential diagnoses of non-fitting status, encephalitis or encephalopathy and it was noted that she should have diazepam which is part of the treatment for the control of seizures. Dr Webb, consultant neurologist was contacted. Dr Gaston who was the consultant in charge of her long term management previously was to be contacted.

DR WEBB

Dr Webb subsequently wrote in the notes at 4 pm on 22nd October 1996 that he found that she did not have any features of meningitis, that she was rousable to voice, that she withdrew from ~~power~~ painful stimuli, that she had clonus at both ankles, had increased tone and that she was siting up staring vacantly and not obeying commands.

'I don't have a clear picture of the prodrome and yesterdays episodes. Her motor findings today are probably long standing but this needs to be checked with notes. The picture is of acute encephalopathy most probably postictal in nature.'

Dr Webb suggested commencing more antiepileptic medication, hourly neurological recording and for her to have a CT the next day should she not 'wake up'.

She was noted not to have responded to the antiepileptic medication and therefore additional medication was commenced.

Dr Webb reviewed Claire at 17:00, noting that she had had the loading dose of the anti-epileptic medication, phenytoin (*initially this drug is given over a period of time, as a large dose so that satisfactory blood levels are achieved*) and a dose of midazolam which is also used in the treatment of seizures.

He noted that she continued to appear to be 'largely unresponsive', that she responded to deep pain by flexing her left arm and had a facial grimace. There was no vocalisation noted.

He obtained further history of the stiffening on the right hand side of the body being present on Monday with focal signs. There was contact with a cousin who had had gastrointestinal upset the preceding day.

Dr Webb prescribed antibiotic and anti viral medication to be started as a precaution albeit he thought the likelihood for either a bacterial or viral meningitis to be present was low; he asked for viral cultures to be taken to see if a viral infection could account for Claire's condition and that another anti-epileptic medication be started.

The notes record that at 23.30, the results of the blood samples were available, showing hyponatraemia and her fluid management was altered. I will defer to Dr Bingham regarding the management of her fluid regime.

At about 3 am on the 23rd October 1996, she had a respiratory arrest and developed fixed dilated pupils. She was intubated and transferred to the paediatric intensive care unit.

At 4am her examination revealed fixed dilated pupils, with bilateral papilloedema (*suggesting raised intracranial pressure, i.e. the pressure within her brain was elevated*), and that she was no longer responsive to painful stimuli.

She was reviewed at 4.30 am by Dr Webb, who considered her to have the syndrome of inappropriate anti-diuretic hormone production, leading to hyponatraemia and cerebral oedema. He also thought that she had coned (*that is her brain due to swelling had become forced down the main outlet within the skull, causing irreparable damage to the brain*), following prolonged seizure activity. She had had mannitol, medication which is used to decrease brain swelling by drawing out fluid by virtual of its osmotic effects, but that this had had no effect on the pupillary size, and that there were no eye moments. This composite picture is alarming and suggested that brain death had occurred.
Dr Webb requested an urgent CT of Claire's head which confirmed the clinical findings of diffuse swelling of the brain, without any focal abnormality being seen (recorded in the notes at 5 am on 23rd October 1996).

At 6 am the brain stem death criteria were applied by Dr Webb.

The laboratory sample at the time of brain stem testing recorded the sodium level as 129 mmol/l.

These brain stem test criteria were repeated and confirmed at 18:25.

Intensive care support was withdrawn from Claire at 18:45 and a death certificate for cerebral oedema secondary to status epilepticus was written.

CONCLUSIONS

Claire Roberts was admitted with abnormal neurological symptoms and signs. The diagnosis of encephalitis/ encephalopathy was made at an early stage of her admission and measures taken to treat the likely diagnosis of non-convulsive epilepsy. There was a background of seizure activity in her past medical history and hence the probability of this diagnosis was high, given her presentation.

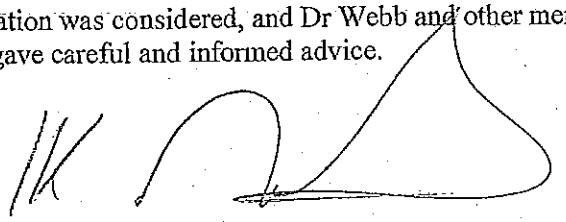
Dr Webb played a key part in the management of Claire and his initial assessment was considered and defined a clear management plan that incorporated treatment of meningitis (although as he rightly said, this was an unlikely diagnosis, however, it was essential to instigate treatment for meningitis. Failure to do so would lead to permanent disability and a possible death if meningitis had been present).

The underlying diagnosis was that of non-convulsive status epilepticus; a condition where the electrical activity of the brain is disrupted but there are not the somatic manifestations of seizure activity apparent such as is seen with other forms of fitting such as jerking convulsions. If untreated non-convulsive status epilepticus can lead to worsen any disability and be a cause of death. The term status meaning that the fitting is established (e.g. one definition for status epilepsy is that it has lasted for more than 20 minutes).

The management plan to treat the possibility of non-convulsive status epilepticus was correct at the time of practice.

Claire Robert's subsequent management was correct and her course of management on the ward and PICU was appropriate. The parents were invited to return for further discussions after the withdrawal of treatment.

I have not commented upon the hyponatraemia which has been addressed by Dr Bingham. Claire Robert's treatment from the point of view of management of her neurological presentation was considered, and Dr Webb and other members of the team looking after Claire gave careful and informed advice.



Dr Ian Maconochie FRCPCH FFAEM
Consultant in Paediatric A&E Medicine

CH 7

Medicolegal Report on Claire Roberts
D.O.B: 10th January 1987

By

Dr R M Bingham FRCA

This report has been compiled using the following documents:

- Photocopies of the clinical notes from Claire's admission to the Royal Hospital for Sick Children, Belfast on 21st October 1996.
- Statement compiled by Mr Alan Roberts following a meeting with Dr N Rooney, Dr H Stein, Dr A Sands and Professor Young
- Statement from Dr Heather Steen compiled on the 16th March 2005

BACKGROUND TO CASE

Claire Roberts was admitted to the A & E department of the Royal Hospital for Sick Children at 19.15hrs on the 21st October 1996 with a history of malaise for 1 day and hourly vomiting and drowsiness since the same afternoon.

Claire had a history of developmental delay and moderate learning difficulty and she attended Torbank Special School[1]. She also had hospital admissions for seizure activity between the ages of 6 months and 4 years. She was treated for these with anti-epileptic medication but this was discontinued when she had no further fits.

From the admissions notes it would appear that when well, Claire could construct meaningful sentences, walk, run and climb stairs unaided. She fed herself with supervision but needed help with dressing. It was noted that she favoured her left side.

On examination following her admission her temperature and pulse rate were normal but it was noted that her speech was slurred and that she was hardly speaking. Her pupils and fundii were normal and there was no neck stiffness[2]. There is a note that her muscle tone was raised and the left sided tendon reflexes were brisker than those on the right[3]. Later, on the ward this was reversed and it was also noted that she had "cogwheel rigidity" of the right arm and ankle clonus[4].

On admission to the ward there is a statement that she was "not responding to parents voice" but she was "responding to deep pain". There is no mention of Claire's state of hydration, which I assume means that there were no concerns. Certainly her heart rate was normal and increased heart rate is one of the earliest sign of dehydration.

A presumptive diagnosis of "viral illness" was made, blood samples[5] were taken and intravenous fluids were started at a rate of 64ml/hr.

On reassessment at midnight Claire was "slightly more responsive" and there were no signs of meningism so it was decided to observe her overnight and re-assess in the morning. The results of the blood tests were also available and were normal except for a raised white cell count[6] and a low serum sodium (132mmol/l).

The following day there was a ward round with Dr Sands where Claire's continued lack of responsiveness was noted with the comment "vagueness/vacant (apparent to parents)" and it was agreed to contact Dr Gaston; Claire's Community Paediatrician.

A diagnosis of "non-fitting status"[7] was made and a dose of diazepam[8] was prescribed.

Later the same day (16.00) Claire was reviewed by a neurologist who noticed that she appeared to have improved following the diazepam. It is noted that Claire withdrew from a painful stimulus

[1] I think this is the correct name; the writing is difficult to read from the photocopy.
[2] Abnormal fundii (the optic nerves at the back of the eye) are associated with brain swelling. Neck stiffness is a symptom of meningitis.
[3] There is a reference here to a GP letter, which I do not have.
[4] All these are signs of abnormalities in the control the central nervous system (CNS) exerts over the peripheral nervous system. These could be longstanding, related to Claire's developmental delay or signs of CNS irritability related to the current illness.
[5] Full blood count, serum electrolytes, blood group and antibody screen and viral titres.
[6] This is consistent with an infective process.
[7] A condition in which there is epileptic electrical activity in the brain but no muscle twitching.
[8] A tranquillising drug which is an effective anti-epileptic.

and opened her eyes to voice prompts but did not talk. On examination there were "reduced movements right side?" and her reflexes were symmetrically brisk. This was interpreted as "probably long standing but this needs to be checked with notes". Her fundii were again normal. The summary states "The picture is of acute encephalopathy most probably posticfal in nature[9]. I note no biochemistry profile". A loading dose of the anti-epileptic drugs phenytoin and midazolam were prescribed.

Claire was re-assessed by the neurologist at 17.00 following the drugs. Her level of consciousness had deteriorated so that she only responded to pain. It was decided to start anti-biotic and anti-viral drugs in case Claire had meningo-encephalitis[10] although this was not felt to be a likely diagnosis. Another anti-convulsant (sodium valproate) was also prescribed.

The nursing notes and observations at this time also note that she was only responding to painful stimuli. At 21.00 an episode of screaming and drawing up of arms was noted on the "Record of Attacks Observed" and at this time a reduction in the GCS[11] from 8 to 6 occurred.

At 23.30, the results from a previous blood test were available. The serum sodium level was extremely low (121mmol/l). The SHO contacted his Registrar who suggested reducing the rate of fluid administration to 2/3 of its current value. Urine was sent for osmolality testing[12].

At 03.00 Claire had a sudden respiratory arrest and developed fixed and dilated pupils[13]. She was resuscitated with mask ventilation with oxygen followed by tracheal intubation[14]. She was transferred to the ICU where she was put on a mechanical ventilator given mannitol[15] and an infusion of dopamine[16]. Examination revealed fixed and dilated pupils, bilateral papilloedema[17] and no response to painful stimuli. A CT scan showed severe swelling of the brain and no focal abnormalities.

Brain stem death tests at 06.00 showed that the criteria for brain stem death were fulfilled. Serum sodium levels at the time of the brain stem testing were 133mmol/l on the intensive care unit blood gas analyser and 129mmol/l on the laboratory sample.

A second set of tests performed at 18.25 confirmed brain stem death and ventilation was discontinued.

## OPINION ON CAUSATION

On admission, Claire's initial diagnosis was of an unspecified "viral illness" but the slight improvement in her level of consciousness led the team to a plan of observation and expectant management. It appears to me that there was difficulty in deciding how much of Claire's condition was related to her underlying developmental delay and how much was due to the acute illness; this is highlighted by the comment "vagueness/vacant (apparent to parents)" noted following the ward round on the morning after her admission. This difficulty may have contributed to the delay in recognition of the serious nature of her condition.

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[9] Brain injury (probably temporary) as a result of the status epilepticus.
[10] CNS infection.
[11] Glasgow Coma Score; an internationally accepted measure of depth of coma – the lower the number the deeper the coma.
[12] A measure of its concentration as a cause of low sodium is the production of urine of inappropriately high concentration.
[13] A sign of severe swelling of the brain with an increase in the pressure within the skull.
[14] A tube passed into her lungs to aid mechanical ventilation.
[15] A drug which reduces brain swelling
[16] A drug which increases blood pressure in an effort to improve the blood flow to the swollen brain
[17] Swelling of the fundii or optic nerves, which had previously been normal.

The admission diagnosis is reasonable however and acute encephalopathy (viral or ictal) is a likely cause of the presenting illness. Although the serum sodium was initially low and officially defined as hyponatraemia[18] I think it is unlikely that this was the cause of her presenting symptoms. Serum sodium levels in this range are reasonably common in children admitted to hospital in acute illness[19] and symptomatic hyponatraemia is usually associated with much lower sodium levels than this. In addition hyponatraemia is known to accompany both acute encephalopathy and nausea and vomiting.

The decision to start Claire on intravenous fluids was reasonable, as she did not appear well enough to hydrate herself. The choice of fluid was 0.18% saline with 5% glucose, which has been the standard fluid, used for maintenance fluid therapy in children for 50 years. The volume prescribed was in line with widely used recommendations for full maintenance fluid therapy. There were, however reasons why Claire may have required fluid restriction. These include a low level of metabolism as she had reduced consciousness and a possibly reduced urine output due to the secretion of anti-diuretic hormone[20], which often accompanies both encephalopathy and nausea and vomiting. A record of poor urine output could confirm this but in fact there are several notes of her passing urine recorded on the fluid charts and on one occasion it was noted that there was a large volume.

Despite the fact that Claire may have been receiving more fluid than she required, it is not clear why her serum sodium fell to such a low level. The intravenous fluid volume charted in the notes would not be sufficient to account for this, even in the absence of any water excretion by the kidneys. It is possible she was receiving water by other means such as drinking but there is little record of this in the ward notes and her reduced conscious level would make it unlikely. Another possibility is that she was passing urine with very high sodium content but although there is a record of a urine osmolality sample[21] being sent at 23.30 on the 22nd October I have not been able to find the results. Finally, it is possible that the result was inaccurate as the sodium levels in the ICU at 06.00 on the 23rd October were much higher (133mmol/l (blood gas analyser) or 129mmol/l (laboratory)).

Assuming the result was accurate and irrespective of the mechanism for the fall in serum sodium, it is likely that this was the cause of the deterioration in Claire's condition on the evening of the 22nd October; a sodium level of 121mmol/l is known to cause brain swelling and convulsions which can progress to respiratory arrest and death. It is not however possible to completely exclude the chance that the sodium level was inaccurate and that acute encephalopathy was involved in - or even central to - the deterioration.

**IN CONCLUSION:**

The understanding of complex medical problems is always much easier with hindsight; in particular in this case there has been much recent publicity in both the lay and medical press which has led to a better appreciation of the dangers of hyponatraemia in children and helped to clarify the cause of this tragedy. Much of this information has only been available in the last five years.

I feel that Claire's initial diagnosis and management was reasonable. A viral illness was a common and likely diagnosis and although her serum sodium was low it was not excessively so. Her fluid prescription was in line with the practice of the time and although current guidance would be to use fluid with higher sodium content in this situation, this advice did not exist in 1996.

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[18] A serum sodium level of less than 136mmol/l

[19] Pediatrics 2004: 113;1279-1284

[20] A hormone which reduces the amount of water the kidneys excrete.

[21] A measurement of the electrolyte content of the urine

I think there was also confusion about Claire's usual neurological status, which complicated her evaluation and led to an underestimate of the severity of the condition.

The initial and subsequent anti-convulsant treatment was logical, given the working diagnosis and it is unlikely it would have worsened the consequences of hyponatraemia although it may have masked the symptoms.

The hyponatraemia was probably an associated feature of Claire's condition rather than the primary illness. It was most likely to have been a result of the combination of raised levels of anti-diuretic hormone together with the intravenous infusion of fluid with low sodium content although the volumes infused do not fully account for the sodium becoming so low.

I think it is most likely that hyponatraemia was the cause of the neurological deterioration on the evening of the 22nd October culminating in the respiratory arrest at 03.00 on the 23rd. It is not, however possible to completely exclude the possibility that the serum sodium result was an isolated artefact and the deterioration was due to acute encephalopathy.

It is difficult to be certain whether this tragedy could have been prevented but assuming hyponatraemia was the cause, it is likely that identification of a low sodium level when the absence of a biochemical profile was noted, followed by the institution of a fluid restriction regime, would have ameliorated its consequences. It is also possible that aggressive treatment at 21.00, when Claire's coma score reduced from 8 to 6, may have been effective. Although the measures taken at 23.30, when the sodium result was available, were of the correct type they were too little and too late.

**Statement**

I understand that it is my duty to help the Court with those matters, which are within my expertise, and in relation to which my advice has been sought, and I have complied with that duty.
I believe that the facts I have stated in this report are true and that the opinions I have expressed are correct.

Dr Robert Bingham
Consultant Paediatric Anaesthetist

14 April 2005

# 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

*TO BE COMPLETED WHEN THE STATEMENT HAS BEEN WRITTEN*

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

D W Webb

*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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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.'*

**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.***

**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.

**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.

**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.*

*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.'*

## 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.

| | |
|---|---|
| Best Eye opening | Spontaneously (4)<br>To speech (3)<br>To pain (2)<br>No eye opening (1) |
| Best Verbal response | Orientated (5)<br>Confused, conversant (4)<br>Inappropriate words (3)<br>Vocalisation (2)<br>None (1) |
| Best Motor response | Obeys commands (6)<br>Localises pain (5)<br>Flexion withdrawal (4)<br>Abnormal flexion (3)<br>Abnormal extension (2)<br>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.

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.

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).



SIGNATURE OF STATEMENT MAKER: D Webb

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).

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