# HER MAJESTY'S CORONER

DISTRICT OF GREATER BELFAST

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Our Ref: JLL/gr/29200

3 October 2005

Dear Peter,

**CLAIRE ROBERTS – DECEASED**

I am enclosing a copy of a further statement I have received from Mr Alan Roberts. I have sent a copy of the statement to Dr Bingham and Dr Maconochie, and I have asked each to provide me with a written response as soon as possible before I make arrangements for the Inquest hearing.

I would ask you to circulate this statement amongst the staff involved at the Royal Belfast Hospital for Sick Children and again, written responses would be useful at this stage.

Yours sincerely

**J L LECKEY**
**HM CORONER FOR GREATER BELFAST**

THE ROYAL HOSPITALS
04 OCT 2005
LITIGATION MANAGEMENT

4/10/05

4/10/05
GRW

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 around 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 bounce 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 hyponatraemia 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 and 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 in 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

Monday 21st Oct.
Tuesday 22nd Oct.
Wednesday 23rd Oct.
1pm
8pm
Sodium level
132 mmol/L
Incorrect fluid / care management
Sodium level fall over 24 hour period without monitoring (132 to 121)
No urine tests
No blood tests
Fluid levels rising / SIADH
1pm
4pm
5pm
DR Webb medication input
9pm
Blood cultures to check "viral infection"
Sodium level
121 mmol/L
11:30pm
Still not admitted to ICU
Patients not informed
3am
Claire still on Allen ward
Claire has respiratory arrest
Moved to I.C.U.