

CORONERS ACT (Northern Ireland), 1959

Deposition of ~~Witness~~ taken on _____ the _____ day
of _____ 20 _____, at inquest touching the death of
_____, before me

Coroner for the District of _____

as follows to wit: -

The Deposition of ^{PROFESSOR} ~~DR~~ IAN YOUNG

of _____

(Address)

who being sworn upon his oath, saith

Mr. McCrea:

My review is as set out in this deposition. I gave a verbal report to Dr Michael McBride the Medical Director. I agree that hyponatraemia may have contributed significantly to Claire's death. I agree with Dr. Bingham's comments relating to fluid management. How her fluid management would be different. She had been given the standard fluid management used at that time. The drop in sodium level occurred over a 25 hour period. The symptoms of this case as described by Drs Bingham and Macconachie. Claire's symptoms were in keeping with any of the three causes I have given at 1(b) in the formulation of the cause of death. In Claire's case the focus was on meningoencephalitis & status epilepticus. The symptoms were indicative also of hyponatraemia. Claire had the potential for electrolyte imbalance. I was not involved with the Adam Strain case. I am unaware of any circular issued subsequent to that inquest. A ~~more~~ blood sample every 24 hours would be good clinical practice. It is impossible to say if the drop in sodium

level would have been linear. I examined
Claire's fluid regime, I agree that the amount
of fluid given between 8pm + ~~7pm~~^{2am} was
greater than planned. I agree with Dr. Benjamin
that this did not make any substantial
difference to Claire. We do not have an
particular accurate picture of Claire's fluid
balance or fluid losses were not recorded.
A blood test would have shown a drop. An
interpretation of a urine sample would have
been complex - I think it would have shown
that Claire was adequately hydrated. A CT
scan would have shown cerebral oedema.

Antony:

Tai Long

CR - CORONER

TAKEN before me this 4th day of May 2006

091-010-061

hml Co. No. Coroner for the District of ~~Greater~~ Northern

CORONERS ACT (NORTHERN IRELAND) 1959

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

The Deposition of ^{Professor} ~~Dr~~ Ian Young

Who is being sworn upon his oath, saith

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

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

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

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

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

At approximately 3am on 23rd October Claire suffered a respiratory arrest and was noted to have fixed dilated pupils. She was transferred to the Paediatric Intensive Care Unit. At 4am it is noted that pupils were fixed and dilated and there was bilateral papilloedema. A note at 4.4.am on the 23rd October from Dr David Webb indicated the likely diagnosis of syndrome of inappropriate ADH production with hyponatraemia, hypo-osmolality and cerebral oedema following prolonged epileptic seizures. Claire subsequently died on the 23rd October at 18.45 hours. A death certificate was issued indicating cerebral oedema secondary to status epilepticus.

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

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

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

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

notes prior to Dr Webb's written note, and this seems inconsistent with Dr Bingham's interpretation of the comment.

2. On page 4, paragraph 1, Dr Bingham indicates that it is unlikely that the serum sodium on admission (132mmol/l) was the cause of Claire's presenting symptoms. I think that this is an important point, with which I agree. While Claire's sodium was low on admission, the degree of hyponatraemia was relatively minor and was unlikely to be making a significant contribution to her presentation.
3. As indicated by Dr Bingham, urine output from Claire was not measured. Dr Bingham believes that there is sufficient recorded information relating to wet nappies to conclude that urine output was reasonably high. I do not think that it is possible to reach any conclusion as to whether urine output was high or low.
4. Dr Bingham indicates that the intravenous fluid volume recorded in Claire's notes would not be sufficient to account for the fall in her serum sodium. In contrast, I do not think that it is possible to reach any firm conclusion on this matter in the absence of any record of urine volume or urinary sodium concentration. I believe that the changes in Claire's serum sodium are entirely consistent with the recorded intravenous fluid intake when possible urinary losses of water and sodium are taken into account.
5. In his report, Dr Bingham raises the possibility that the serum sodium measurement of 121mmol/l was wrong. The laboratory measurement of sodium is extremely accurate. Assuming that an appropriate sample was taken (and there is nothing in the notes to suggest that sample collection was difficult), I believe that the possibility of an inaccurate laboratory result is negligibly small.

In addition to the above comments, I would like to make one comment in response to the letter from Mr Alan Roberts dated 29th September 2005. Mr Roberts refers to my earlier statement that: 'The practice at that time would be firstly, to restrict fluid intake and secondly, to consider administration of fluid with a high content of sodium, if symptoms attributable to hyponatraemia were present'. This statement was made in response to a question about the action taken when Claire's serum sodium was noted to be 121 mmol/l. In my opinion, when Claire was initially admitted her serum sodium of 132mmol/l was unlikely to have made a significant contribution to her

presenting symptoms, although serum sodium was slightly below the lower reference limit and therefore in the hyponatraemic range. ~~I produce a further statement c6,~~ I produce my formulation of the cause of death c6.

4th MAY
TAKEN before me this day of ~~April~~ 2006

H. L. Keating

Senior Coroner for Northern Ireland

CG

THIS CERTIFICATE MUST BE DELIVERED WITH THE DECEASED'S MEDICAL CARD WITHIN FIVE DAYS TO THE REGISTRAR FOR THE DISTRICT IN WHICH THE PERSON (a) DIED OR (b) WAS ORDINARILY RESIDENT (WITHIN NORTHERN IRELAND) IMMEDIATELY BEFORE DEATH FOR INSTRUCTIONS TO INFORMANTS SEE OVERLEAF

MEDICAL CERTIFICATE OF CAUSE OF DEATH

Births and Deaths Registration (Northern Ireland) Order 1976, Article 25(3)

To be signed by a Registered Medical Practitioner WHO HAS BEEN IN ATTENDANCE during the last illness of the deceased person and given to some person required by Statute to give information of the death to the Registrar. (SEE OVERLEAF)

FOR USE OF REGISTRAR
Entry No
District

Name of Deceased
Usual Residence
Place of Death
Date of Death
Date on which last seen alive and treated by me for the undermentioned conditions
Whether seen after death by me

Whether seen after death by another medical practitioner

These particulars not to be entered in Death Register

CAUSE OF DEATH
I Disease or condition directly leading to death*
Antecedent causes
Morbid conditions, if any, giving rise to the above cause, stating the underlying condition last.
II Other significant conditions contributing to the death, but not related to the disease or condition causing it.
(a) cerebral oedema
(b) meningococcal meningitis status epilepticus
(c) hypoparathyroidism with excess ADH production

*This does not mean the mode of dying eg heart failure, asphyxia, etc. It means the disease, injury or complication which caused death.

I hereby certify that the above-named person has died as a result of the natural illness or disease for which he has been treated by me within twenty-eight days prior to the date of death, and that the particulars and cause of death above written are true to the best of my knowledge and belief.

Signature
Residence
Date
Qualifications as registered by General Medical Council

The Health Service Number of the deceased should be entered here by the certifying doctor.

091-010-000

CR - CORONER