STATEMENT OF WITNESS

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

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

NOT SIGNED IN POLICE OFFICER'S PRESENCE

TO BE COMPLETED WHEN THE STATEMENT HAS BEEN WRITTEN

I declare that this statement consisting of 2 pages, each signed by me is true to tl best of my knowledge and belief and I make it knowing that, if it is tendered in evidence at preliminary enquiry or at the trial of any person, I shall be liable to prosecution if I have wilfu stated in it anything which I know to be false or do not believe to be true.

Dated this day of

SIGNATURE OF MEMBER by whom statement was recorded or received

Ian Young
SIGNATURE OF WITNESS

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

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

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

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

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

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

Form 38/36
(Plain)

SIGNATURE OF WITNESS..... Ian Young .....

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STATEMENT CONTINUATION PAGE

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

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

SIGNATURE OF STATEMENT MAKER: Ian Young

Form 38/36 [a]
(Plain)

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Dated this day of

Ian Young

SIGNATURE OF MEMBER by whom statement was recorded or received

SIGNATURE OF WITNESS

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

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

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

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

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

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

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

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

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

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

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(Plain)

SIGNATURE OF STATEMENT MAKER: Ian Young

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