

Professor of Medicine I.S. Young BSc MD FRCP FRCPI FRCPath

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Telephone: Direct line:

Our Ref:

Statement

5<sup>th</sup> May 2005

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 hyponagremia 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. 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

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 21<sup>st</sup> 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 22<sup>nd</sup> October 1996, states: "I note (N, biochemistry profile".)

Claire received intravenous fluid replacement following admission and throughout the day of the 22<sup>nd</sup> (or 1 Note that the day of the 22<sup>nd</sup>) 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 22<sup>nd</sup> October. A note timed 23.30 on the 22<sup>nd</sup> 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 2/3<sup>rds</sup> of previous values. A note was taken to send urine for osmolality although there is no record of a result.

At approximately 3am on 23<sup>rd</sup> October Claire suffered a respiratory arrest and was noted to have fixed dilated pupils. She was transferred to the Pediatric 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 23<sup>rd</sup> October form Dr David Webb indicated the likely diagnosis of syndrome of inappropriate ADH production with hyponatremia, hypo-osmolality and cerebral oedema following prolonged epileptic seizures. Claire subsequently died on the 23<sup>rd</sup> 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 hyponatremia 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

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access to statements from all of the staff involved in Claire's care.

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