Form 20

CORONERS ACT (NORTHERN IRELAND) 1959

Deposition of Witness taken on WEDNESDAY the 5th day of FEBRUARY 2003 at inquest touching the death of RAYCHEL FERGUSON, before me J L LECKEY Coroner for the District of GREATER BELFAST as follows to wit:-

The Deposition of DR JOHN GORDON JENKINS
of SCHOOL OF MEDICINE, QUEENS UNIVERSITY, BELFAST who being sworn upon his oath, saith

My name is John Gordon Jenkins and I am a Senior Lecturer in Child Health at Queen's University, Belfast. I have 20 years experience as a Consultant Paediatrician initially at the Waveney Hospital, Ballymena and subsequently at Antrim Hospital. I qualified in Medicine from Queens University, Belfast in 1974 and subsequently obtained my Doctorate with Honours in 1980. I became a member of the Royal College of Physicians of the United Kingdom by examination in 1977 and was elected to Fellowship of the Royal College of Physicians of Edinburgh in 1989. I became a founder fellow of the Royal College of Paediatrics and Child Health in 1997. This report has been prepared following review of photocopied material from the case notes relating to the admission of this girl to Altnagelvin Hospital in June 2001, together with other material.

Rachel was admitted with abdominal pain suggestive of acute appendicitis on 7.6.01 and subsequently underwent emergency appendicectomy. She was healthy and well with approximate weight 26 kgs and her preoperative blood investigations were normal (serum sodium 137 mmol/l). Post-operatively she was initially felt to be making good progress but had vomiting and headache. At approximately 03.00 on 9.6.01 she began to have severe seizure activity with further subsequent deterioration despite resuscitation and intensive care. She subsequently died and evidence on

CT scan and at post-mortem was consistent with the diagnosis of cerebral oedema related to hyponatraemia. Her sodium was found to be 119 at 03.30 on 9.6.01 with a repeat specimen at 4.30 giving a result of 118, associated with low levels of potassium and magnesium.

Solution 18 (0.18% saline with 4% dextrose) has been routinely used in Paediatric medical practice for a very long time and is rarely associated with any acute electrolyte disturbances such as were seen in this tragic case. However, this is largely related to the range of conditions commonly seen by Paediatricians and cared for within the medical (as opposed to surgical) environment. By and large these are not associated with the syndrome of inappropriate secretion of antiduretic hormone. It has become increasingly recognised in recent years that a regime utilising solution 18 may not provide the right balance of sodium and free water for children with some clinical conditions, and particularly where there is an increased likelihood of failure to excrete water. This would include situations of stress, pain and nausea, and may be particularly common in the post-operative period. It is the combination of excessive loss of sodium (for example in vomitus) with water retention (as a result of excessive secretion of antidiuretic hormone) which leads to a fall in the concentration of sodium in body fluids and increased risk of brain swelling (cerebral oedema).

This was well described in an editorial in the Journal "Paediatric Anaesthesia" in 1998 by Dr Arieff, but did not receive widespread publicity in journals likely to be read by most Paediatricians or Surgeons caring for children at that time. The potential dangers were highlighted to a wider clinical community in an article published in the British Medical Journal of 31.03.01 by Halberthal et al. However, this topic is not well covered in a number of standard paediatric texts. Most Paediatric Units were still using their traditional regimes based on solution 18 until further concerns were raised within Northern Ireland in September 2001 as a result of two deaths. Steps were taken to convene a Working Group who have subsequently prepared and distributed guidance on the prevention of hyponatraemia in children under cover of a letter from the Chief Medical

Officer dated 23rd March 2002. This highlights the danger of this condition and gives guidance as to how this can be minimised in clinical practice.
It seems that some individuals can develop this condition in circumstances which are clinically no more severe than those experienced by many patients in the post-operative period, but the reasons for this variation in susceptibility are currently not well understood. It has been suggested that females and children may be particularly at risk. It is for this reason that guidance has now been prepared and issued to increase awareness of this poorly recognised condition and to ensure that Units providing care for children take steps locally to introduce care pathways and / or fluid management regimes which take account of this possibility and minimise the risks of occurrence.

The deterioration in Raychel's condition occurred rapidly. The possibility of an electrolyte disturbance being the cause of the fit was considered by Dr Johnson and efforts made to obtain electrolyte results from the laboratory urgently. However, even by the time these became available her condition had further deteriorated and her pupils were found to be dilated and not reacting to light. (evidence that increased intracranial pressure due to cerebral oedema has already caused pressure damage within the brain.) Despite prompt resuscitation and further investigation and management this damage proved irreversible and led to her death.

Conclusion

Having carefully studied the statements provided by the doctors and nurses involved in Raychel's care my impression is that they acted in accordance with established custom and practice in the Unit at that time. Raychel's untimely death highlights the current situation whereby one sector of one sector of the medical profession can become aware of risks associated with particular disease processes or procedures through their own specialist communication channels, but where this is not more widely disseminated to colleagues in other specialities who may provide care for patients at risk from the relevant condition. In the circumstances relating to this incident,

it was only the tragic deaths of two children in Northern Ireland which alerted the wider clinical community to these concerns. These have subsequently been assessed and relevant guidance prepared and disseminated as outlined above.

TAKEN before me this 5TH DAY OF FEBRUARY 2003

Coroner for the District of Greater Belfast

STRICTLY PRIVATE & CONFIDENTIAL

Rachel Ferguson (Deceased) - Inquest at Belfast Coroner's Court, February 2003

Date of birth: 04.02.92 **Date of death: 10.06.01**

My name is John Gordon Jenkins and I am a Senior Lecturer in Child Health at Queen's University, Belfast. I have 20 years experience as a Consultant Paediatrician initially at the Waveney Hospital, Ballymena and subsequently at Antrim Hospital. I qualified in Medicine from Queens University, Belfast in 1974 and subsequently obtained my Doctorate with Honours in 1980. I became a member of the Royal College of Physicians of the United Kingdom by examination in 1977 and was elected to Fellowship of the Royal College of Physicians of Edinburgh in 1989. I became a founder fellow of the Royal College of Paediatrics and Child Health in 1997. This report has been prepared following review of photocopied material from the casenotes relating to the admission of this girl to Altnagelvin Hospital in June 2001, together with other material.

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Solution 18 (0.18% saline with 4% dextrose) has been routinely used in Paediatric medical practice for a very long time and is rarely associated with any acute electrolyte disturbances such as were seen in this tragic case. However, this is largely related to the range of conditions commonly seen by Paediatricians and cared for within the medical (as opposed to surgical) environment. By and large these are not associated with the syndrome of inappropriate secretion of antidiuretic hormone. It has become increasingly recognised in recent years that a regime utilising solution 18 may not provide the right balance of sodium and free water for children with some clinical conditions, and particularly where there is an increased likelihood of failure to excrete water. This would include situations of stress, pain and nausea, and may be particularly common in the post-operative period. It is the combination of excessive loss of sodium (for example in vomitus) with water retention (as a result of excessive secretion of antidiuretic hormone) which leads to a fall in the concentration of sodium in body fluids and increased risk of brain swelling (cerebral oedema).

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Rachel Ferguson (Deceased) - Inquest at Belfast Coroner's Court, February 2003

18 until further concerns were raised within Northern Ireland in September 2001 as a result of two deaths. Steps were taken to convene a Working Group who have subsequently prepared and distributed guidance on the prevention of hyponatraemia in children under cover of a letter from the Chief Medical Officer dated 25.03.02. This highlights the danger of this condition and gives guidance as to how this can be minimised in clinical practice.

It seems that some individuals can develop this condition in circumstances which are clinically no more severe than those experienced by many patients in the post-operative period, but the reasons for this variation in susceptibility are currently not well understood. It has been suggested that females and children may be particularly at risk. It is for this reason that guidance has now been prepared and issued to increase awareness of this previously poorly recognised condition, and to ensure that Units providing care for children take steps locally to introduce care pathways and/or fluid management regimes which take account of this possibility and minimise the risks of occurrence.

The deterioration in Rachel's condition occurred rapidly. The possibility of an electrolyte disturbance being the cause of the fit was considered by Dr Johnston and efforts made to obtain electrolyte results from the laboratory urgently. However, even by the time these became available her condition had further deteriorated and her pupils were found to be dilated and not reacting to light (evidence that increased intracranial pressure due to cerebral oedema had already caused pressure damage within the brain). Despite prompt resuscitation and further investigation and management this damage proved irreversible and led to her death.

Conclusion

Having carefully studied the statements provided by the doctors and nurses involved in Rachel's care my impression is that they acted in accordance with established custom and practice in the Unit at that time. Rachel's untimely death highlights the current situation whereby one sector of the medical profession can become aware of risks associated with particular disease processes or procedures through their own specialist communication channels, but where this is not more widely disseminated to colleagues in other specialties who may provide care for patients at risk from the relevant condition. In the circumstances relating to this incident, it was only the tragic deaths of two children in Northern Ireland which alerted the wider clinical community to these concerns. These have subsequently been assessed and relevant guidance prepared and disseminated as outlined above.

Dr J G Jenkins MD FRCP FRCPCH
Senior Lecturer in Child Health & Consultant Paediatrician