MEDICOLEGAL REPORT

ON

RACHEL FERGUSON

Deceased

dob: February 4th 1992

Died: June 10th 2001

Prepared for: John L Leckey LL.M
HM Coroner
Coroner's Office
Courthouse
37 Church Road
Newtownabbey
County Antrim BT36 7LA

By: Edward Sumner MA, BM, BCh, FRCA
Consultant Paediatric Anaesthetist
[redacted]

February 2002

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069A-065-242

My name is Edward Sumner and I am a consultant in Paediatric Anaesthesia with an interest in Intensive Care.
I was consultant at the Great Ormond Street Hospital for Children, London, from 1973 until June this year. I am the author of several textbooks on the subject and am the Editor-in-Chief of the Journal, *Paediatric Anaesthesia.*
Currently, I am the President of the Association of Paediatric Anaesthetists of Great Britain and Ireland.

In the preparation of this report I have carefully perused all the medical and nursing notes and statements presented to me, together with the reports of Dr Herron and Dr Loughrey.

I understand that my overriding duty is to the Court on matters which are within my expertise. I also believe that the facts I have stated in this report are true and that the opinions I have expressed are correct.

Rachel was born on 4th February 1992 and was a previously fit and well little girl with normal development.

On 7th June 2001 she was admitted to Altnagelvin Area Hospital via the Accident and Emergency Department complaining of sudden onset, acute abdominal pain with increasing severity at around 8 pm. She had eaten dinner at 5pm but after that had no appetite.

She was nauseated but was not vomiting. Her temperature was normal. The physical signs were of acute appendicitis with tenderness over Mcburney's point. Her weight was approximately 26kg.

Preoperative haematology and biochemistry was normal, notably the serum sodium was normal at137mmol.l^{-1}
The urine analysis showed proteinuria++
Consent for surgery and for rectal analgesia was taken from Mrs Ferguson in the theatre area. No premedication was administered and anaesthesia was induced at approximately 1130 pm. The anaesthetists were Drs Gund and Jamison and the surgeon Mr Makar.

The anaesthesia was routine and involved analgesia administered by the intravenous, rectal and local routes and a relaxant technique with intubation. She was also given an antiemetic. The anaesthetic form shows that she was given one litre of Hartmann's solution, but a witnessed, retrospective note states that only 200ml of this was actually infused.
Surgery finished after midnight on 8th June and postoperatively there seemed to be prolonged sedation from opioids, though she was awake in recovery by 0115. The IV infusion was to be recommenced in the ward.

The appendicectomy was routine. The peritoneum was clear and the appendix itself was mildly congested with an intramural faecolith. There was no Meckel's diverticulum.

Later on that day Rachel was noted to be apyrexial and free of pain, but she had vomited at 0800 and at 1015 she had a large vomit and again at 1300 and 1500. At 2115 the nurses noted "vomiting ++ (coffee grounds), colour flushed to pale, complaining of headache" and at 2300 there were three more small vomits. In spite of the vomiting Rachel had been able to walk during the day.

During this time she was receiving an intravenous infusion of solution 18 (0.18% saline with 4% dextrose) at a rate of 80ml per hour with a total of 540ml between leaving recovery and 0800 and a further1680ml between 8am and 4am the following morning (9th June) giving a total of 2220ml in 24hours. The fluid balance chart is confusing as the IV input is in the wrong column and I am not sure what is the significance of the AMT (150ml every hour). There is no note of any urine output or oral fluid intake, though it does say she was fasting during the night of surgery. There was no nasogastric tube at that stage.

On 9th June 2001 at 0315 Dr Johnson was called because Rachel had had a fit and had been incontinent. The seizure activity eventually responded to rectal and IV diazepam after 15 minutes. Oxygen was given. Although she was unresponsive, the other vital signs were normal and the blood sugar normal at 9.7mmol.l^{-1} An electrolyte disorder was suspected and this was urgently checked. The electrolyte results from 0330 were: sodium 119, potassium 3, chloride 90, CO_2 16 and magnesium 0.59 mmol.l^{-1} These were repeated at 0430 when the serum sodium was found to be 118, potassium 3 and CO_2 15mmol.l^{-1}

At 0630, the paediatric SHO noted that Rachel looked very unwell with pupils that were fixed and dilated. Her face was flushed with a rash and petechiae on the neck, probably from the vomiting. The chest was "rattly" and they wondered whether there had been aspiration into the lungs. The differential diagnosis at that stage was between the biochemical disorder and a cerebral lesion such as meningitis.

There is also an untimed note from the surgical registrar mentioning that Rachel was unresponsive with fixed, dilated pupils that she was intubated and that an emergency CT scan was organised.

At 0830 the anaesthetist was urgently summoned as Rachel had stopped breathing. He found her to be cyanosed and still vomiting. She was intubated without the need for any drugs, given antibiotics, intravenous 0.9% saline with magnesium and catheterized. Suctioning down the tracheal tube produced copious dirty secretions.

Later, the CT scan showed evidence of subarachnoid haemorrhage with raised intracranial pressure and at the request of the neurosurgeons a second, enhanced scan showed no evidence of a subdural collection of pus.

She was transferred to the intensive care unit and then to Belfast at 1110 at a time when she was hypothermic and with a negative fluid balance of one litre.

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Rachel eventually died the following day at 1209.

The postmortem examination was carried out on 11th June by Drs Al-Husaini and Herron. They found diffuse swelling of the brain with flattening of the gyri and effacement of the sulci. There was bilateral uncal swelling and uncal necrosis, plus evidence of diffuse hypoxic ischaemic necrosis due to perfusion failure. Their conclusion was that Rachel died from cerebral oedema due to hyponatraemia.

I would like to make the following comments:

1. Rachel was a previously fit and healthy little girl suffering from mild appendicitis.
2. Postoperative vomiting is very common indeed and has a variety of causes notably as a reaction to anaesthetic agents particularly the opioids such as fentanyl and morphine, but also after interference with the peritoneum. Vomiting is also a sign of rising intracranial pressure. Rachel was given antiemetic drugs, but suffered very severe and prolonged vomiting. We know this because of the presence of "coffee grounds" which is a sign of gastric bleeding and also the petechiae seen on her neck from straining.
3. It has been known for many years that after surgery there an accumulation of fluid in the extravascular space and that some degree of fluid restriction is necessary postoperatively for 24 to 48 hours. This known to be caused by the inappropriate secretion of Antidiuretic Hormone (ADH). The commonest regime to cope with this and prevent the deleterious effect of the excess water is to give 2ml per kilo body weight per hour for the first 24 hours of a solution such as 0.18% saline with 4 or 5% dextrose and then a little more the following day. During this time it is essential to replace gastrointestinal losses with an equal volume of 0.9% saline (normal saline) together with a potassium supplement until the patient is back to a normal feeding regime. Rachel was given approx 4ml per kilo per hour of the no 18 solution and no saline replacement for the vomiting losses.
4. Vomiting causes a severe loss of both water and electrolytes. Sodium and acid are lost from the stomach in the vomiting and as a compensatory mechanism the kidneys in trying to conserve sodium allow a net loss of potassium. If these dual electrolyte losses are not replaced with normal saline, but only a fluid containing 30mmol.l^{-1} then a state of hyponatraemia will develop acutely. The extent of the severe electrolyte losses seen in this case is reflected in the very low level of serum magnesium.
5. There is no doubt that Rachel suffered severe and prolonged vomiting. In my opinion there should have been fluid supplements administered, probably as early as 1030 on 8th June after the large vomit. It would also have been very prudent to check the electrolytes in the evening of that day, as the vomiting had not settled down by that stage. It is very uncomfortable, but with prolonged and severe vomiting after an abdominal operation, a nasogastric tube to drain the stomach and allow the gastric losses to be accurately quantified should have been passed. There is no evidence of any attempt to measure the gastrointestinal losses or the urine output – both essential for correct fluid therapy.

6. By the late evening of the 8th June, Rachel had become extremely hyponatraemic, hypokalaemic and hypomagnesaemic. Hyponatraemia is usually defined as a serum sodium of less than 128mmol.l^{-1} so the levels found in Rachel were very low indeed and the changes from the normal values found preoperatively had occurred very quickly.
7. The brain is very sensitive indeed to acute changes in serum sodium levels and cerebral oedema from hyponatraemia with catastrophic consequences is very well documented in the medical literature. Although the skull is a rigid structure,as the brain swells, the intracranial pressure does not rise at once because CSF and blood are displaced from the cranium, but when this mechanism cannot cope, then the pressure rises rapidly and the brain is forced down into the foramen magnum – a situation known as "coning". At this stage there would be seizures and vomiting with the rise in intracranial pressure followed by changes to the pupils and loss of consciousness. Brain death follows if steps to reduce the cerebral swelling are not taken immediately as the intracranial pressure exceeds that of the blood supply. Rachel's clinical course vividly illustrates this.

To conclude and summarize, I believe that Rachel died from acute cerebral oedema leading to coning as a result of hyponatraemia. I believe that the state of hyponatraemia was caused by a combination of inadequate electrolyte replacement in the face of severe postoperative vomiting and the water retention always seen postoperatively from inappropriate secretion of ADH.

References:

Huskisson L Fluid balance: all aspects. In: Paediatric Anaesthesia. Eds: Sumner E, Hatch DJ. London Arnold 1999

Arieff AI. Postoperative hyponatraemic encephalopathy following elective surgery in children. Paediatric Anaesthesia 1998; 8: 1-4