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MEDICOLEGAL REPORT

ON

RACHEL FERGUSON

Deceased

dob: February 4th 1992

Died: June 10th 2001

Prepared for: Northern Ireland Police service
Fermanagh District Command Unit
48 Queen Street
Enniskillen BT74 7JR

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

September 2005

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. In particular the letter of Dr Loughrey, Consultant Chemical Pathologist (page 168) to Dr Herron, consultant neuropathologist is extremely helpful, provides an excellent chronology and a very lucid explanation of the cause of the cerebral oedema from which Raychel died.
I am in total agreement with her point of view.

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.

I must stress that the comments I make and the answers to questions posed are only my opinion.

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. Dr Jamison did not stay after the induction of anaesthesia.

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, but in her statement Dr Jamison suggests the volume was 300 ml.
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.

Postoperatively she was nursed on the children's ward, but had been admitted under the care of the consultant surgeon, Mr Gilliland. The trainee surgeon, Mr Makar who performed the surgery had written Raychel for Hartmann's solution intravenously while she was in the A and E Department but this was changed to Dextrose/Saline at the request of the nursing staff on the children's ward (Staff Nurse Noble) as this was the regime in use there at that time.
After the operation, on the morning of the 8th June she was seen by the trainee surgeon Mr Zafar who found her to be well, free from pain and apyrexial and that observations should continue. He did not see her again until the time of the resuscitation. She was also seen by Mr Makar on that morning. There is no note that the fluid therapy was reviewed at this time.

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She 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.
According to the statement of Staff Nurse Rice, she had asked one of the paediatric SHOs to write up another bag of dextrose saline and then later the surgical JHO wrote Raychel for an anti-emetic around 6 pm at the request of the nurses. The signature for this is not clear.
It is not clear from the notes whether she was seen again by any member of the medical staff until 0315 when she suffered the seizure.

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.
Dr Johnston the junior paediatrician wrote a good report (page158) and acted appropriately. He discussed the clinical situation with his next superior, Dr Trainor who informed the duty consultant paediatrician, Dr McCord. The surgeons were also informed and were present at the time of resuscitation, as were the anaesthetists.

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 0430 the anaesthetist, Dr Aparna Date 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.

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.

The verdict at the Inquest was cause of death: a) Cerebral oedema b) Hyponatraemia

I would like to make the following comments:

1. Rachel was a previously fit and healthy little girl suffering from mild appendicitis.
2. She was being nursed on the children's ward where the paediatricians would be in charge, but had been officially admitted under the care of the surgeons. However, I imagine the regimes operating in that ward at that time would prevail, as seen by the request of the nursing staff for the surgeon to change

the type of intravenous fluid to that in use on that ward. Probably the role of the surgeon would be confined to looking after the surgical aspects of the postoperative management. It is possible that the medical care could fall between two stools.

3. 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. The vomiting was certainly recognised by the nurses and had been duly noted and the doctors were informed. However, there is no indication that the severity of the vomiting was ever recognised and there was no attempt to quantify the sort of volumes that were being lost.
4. Because Raychel had only had an appendicectomy and she would have been expected to make a full and rapid recovery, then it is possible that there was no thought that anything could go wrong, so severe vomiting was underestimated and correct fluid therapy not instituted.
5. 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 <u>it is essential</u> 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.
6. 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.
7. 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 might 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.
8. 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 which seems to be the pattern in patients who suffer cerebral oedema from this condition.

9. 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 and during this period symptoms are not necessarily severe – headache, vomiting etc. When this mechanism cannot cope any longer, then the pressure in the head 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.
10. Dr Jenkins conclusion in his report for the Coroner, dated 30th January 2003 (page11) reads "Raychel'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" I note he mentions the paper from Halberthal et al from the BMJ in 2001, rather than the BMJ paper from nine years earlier. Dr Fulton, Medical Director of the Altnagelvin Trust at the time of Raychel's death set up a Critical Incident enquiry. Dr Nesbitt, Clinical Director of Anaesthesia suggested that dextrose/saline should not be used in paediatric surgical patients and from this stemmed the publication of Guidelines on Hyponatraemia which are now used in Northern Ireland (and elsewhere in the UK) His statement is on pages 26/7 and dated April 2002.

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.
In my opinion Raychel's death was caused by a systems failure, rather than by individuals at fault.

References:

Arieff AI, et al. Hyponatraemia and death or permanent brain damage in healthy children.
BMJ. 1992 May 9;304(6836):1218-22.

Arieff AI. Postoperative hyponatraemic encephalopathy following elective surgery in children. Paediatric Anaesthesia 1998; 8: 1-4
Huskisson L Fluid balance: all aspects. In: Paediatric Anaesthesia. Eds: Sumner E, Hatch DJ. London Arnold 1999

Halberthal M et al. Lesson of the week: Acute hyponatraemia in children admitted to hospital: retrospective analysis of factors contributing to its development and resolution.
BMJ. 2001 Mar 31;322(7289):780-2.

RAYCHEL FERGUSON

In the preparation of this report I have carefully perused the documentation presented to me by the Police Service of Northern Ireland.

I must stress that the comments I make and the answers to questions posed are only my opinion.

My opinions have not changed since my report for the Coroner dated February 2002.

The verdict at the Inquest was cause of death: a) Cerebral oedema b) Hyponatraemia.

The letter of Dr Loughrey, Consultant Chemical Pathologist (page 168) to Dr Herron, consultant neuropathologist is extremely helpful, provides an excellent chronology and a very lucid explanation of the cause of the cerebral oedema from which Raychel died. I am in total agreement with her point of view.

Raychel was a previously fit little girl who underwent an appendicectomy late on the 7th June 2001. Postoperatively she was nursed on the children's ward and was therefore likely to have been under the care of the paediatricians as far as fluid management was concerned. The trainee surgeon who performed the surgery had written Raychel for Hartmann's solution while she was in the A and E Depar tment but this was changed to Dextrose/Saline at the request of the nursing staff on the children's ward (Staff Nurse Noble) as this was the regime in use there at that time.

She was given 200ml of Hartmann's solution in theatre by the anaesthetists.
Postoperatively she repeatedly vomited, though this was never quantified, nor was the volume of urine passed.
She had been given a total of 2220ml of the dextrose/saline solution over the first postoperative 24 hours.
On the 9th of June at 0315 Raychel had a fit and at that time the sodium was found to be 119, potassium 3 and magnesium 0.59 mmol/l, a picture of severe dilution. By 0630 her pupils were fixed and dilated.

After the operation she was seen by the trainee surgeon Mr Zatar the following day who found her well early in the morning, and did not see her again until the time of the resuscitation. She was also seen by Mr Makar in the morning.
According to the statement of Staff Nurse Rice, she had asked one of the paediatric SHOs to write up another bag of dextrose saline and then later the surgical JHO wrote Raychel for an anti-emetic around 6 pm, the signature for this is not clear

It is not clear from the notes whether she was seen again by the medical staff until 0315 when she suffered the seizure. At that time she was seen by Dr Johnston a junior paediatrician who wrote a good report (page158) and acted appropriately. He discussed the clinical situation with his next superior, Dr Trainor who informed the duty consultant paediatrician, Dr McCord. The surgeons were also informed and were present at the time of resuscitation, as were the anaesthetists.

In my opinion Raychel's death was caused by a systems failure, rather than by individuals at fault.
She was being nursed on the children's ward where the paediatricians would be in charge. The regimes operating in that ward at that time would prevail, as seen by the request of the nursing staff for the surgeon to change the type of intravenous fluid to that in used on that ward. I imagine that the role of the surgeon would be confined to looking after the surgical aspects of the postoperative management.

There was a failure on the part of the nursing staff to take the postoperative vomiting seriously and not to measure or at least estimate the sort of volumes being lost in this way. There was a great deal of discussion on this in the Coroner's Court. However, it might well be that a child having had "only" an appendicectomy would not be put on a strict intake/output regime.

There was a collective ignorance of the need to replace losses from vomiting with saline or Hartmann's solution, rather than dextrose/saline. This latter solution is only appropriate for use as a maintenance agent.
There was also a collective ignorance of the need to initially restrict fluids for the first 24 hours postoperatively because of the phenomenon of inappropriate ADH secretion and water retention.

I am enclosing a list of references relating to the phenomenon of hyponatraemia, the most important of which is the Arieff paper in the British Medical Journal from 1992. It might have been expected that earlier papers such as that in the New England Journal (the most prestigious medical journal in the world in my opinion) or that from Acta Paed Scand could have gone into the collective consciousness.

Dr Jenkins conclusion in his report for the Coroner, dated 30th January 2003 (page11) reads "Raychel'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" I note he mentions the paper from Halberthal et al from the BMJ in 2001, rather than the BMJ paper from nine years earlier.

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