MEDICAL REPORT

Raychel FERGUSON

Dob: 4th February 1992

Prepared by Dr Fenella Kirkham MB BChir MD FRCPCH

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&

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Prepared under the instruction of the Hyponatraemia Enquiry

My name is Dr Fenella Jane Kirkham and I qualified MBChir in 1978. I trained in paediatrics from 1979 and in paediatric neurology from 1982. I obtained the MRCP in 1982 and was awarded FRCP in 1994. I was a founding member of the Royal College of Paediatrics and Child Health in 1997. I have been a Consultant Paediatric Neurologist since 1990. From 1990 to 2001, I was Senior Lecturer in Paediatric Neurology at the Institute of Child Health, London, with an honorary contract at Great Ormond Street Hospital. I am currently working part-time as a Consultant Paediatric Neurologist at Southampton General Hospital (6 PAs) and part-time as a clinical academic (6PAs). I was promoted to Reader at the Institute of Child Health in 2002 and to Professor in 2006. I have a research interest in coma, intracranial hypertension and ischaemic brain damage in childhood and have published widely on these subjects. I obtained my MD on 'Cerebral haemodynamics in children in coma' from the University of Cambridge in 2009. I was senior editor for the book 'Cerebrovascular Disease and Stroke in childhood' published by MacKeith Press in 2011.

I have been asked to provide a report on Raychel Ferguson (Date of birth: 4th February 1992). I have had access to the photocopied notes from Royal Belfast Children's Hospital but not to her general practice notes. I cannot find her neonatal notes in the bundle I have been sent although they are listed on the front page.

1. Raychel Fergson was born on the 4th February 1992.
2. On the 7th June 2001 at 20:01, Raychel presented to Casualty at Altnagelvin hospital complaining of sudden onset of abdominal pain at around 16:30 which had increased in severity and was accompanied by nausea and pain on urination. She was tender over McBurney's point and there was rebound and guarding but bowel sounds were normal. Blood pressure was 126/76.
3. Haemoglobin of 11.7 g/dl with a haematocrit of 0.335, a mean cell volume of 84.6, RDW of 12.4 and white cell count of 9.06. On admission sodium was recorded as 137 mmol/L with a chloride of 107.
4. Appendicectomy was performed from 23:45 on 7th June to 00:40 on 8th June. Raychel received 200ml of Hartmann's solution during the procedure.
5. Raychel was prescribed 80ml/hour of 0.18% sodium chloride 4% dextrose post-operatively and during the day on 8th June.
6. Raychel started to vomit at 08:00 on 8th June and continued to vomit during the day (at 10:30, 13:00, 15:00, 21:00, 22:00 and 23:00)
7. Raychel became gradually more listless during the day.
8. Raychel was restless at 00:35 but settled back to sleep and she was apparently asleep but rousable at 02:00.
9. Raychel had an episode of hypertonicity which may have been a convulsion at 0300 on 9th June. Her pupils reacted to light at this stage and she pushed an oxygen mask away so her Glasgow coma

score was probably around 9/15 (Motor 5 for localising; Verbal 2 for restlessness; Eye opening 2 for opening to pain).

10. The intermittent tonic episodes continued and Raychel was described as being more unwell. At 04:00 the pupils were sluggishly reacting to light.

11. Between 04:00 and 04:45 Raychel's limbs became flaccid and she appears to have deteriorated to Glasgow coma score 3 (Motor 1 for flaccid; Verbal 1 for no response; Eye opening 1 for no opening). Her pupils became fixed and dilated.

12. Raychel's oxygenation deteriorated and she was intubated around 04:45. There were copious dirty secretions consistent with aspiration of stomach contents.

13. Low sodiums of 119 and 118 mmol/L, a low chloride of 90 mmol/L, a low magnesium of 0.59 mmol/L and a low bicarbonate of 15 mmol/L were recorded.

14. Urinary sodium was 90 mmol/L

15. I cannot see a serum ammonia to exclude a Reye-like illness. The AST was a little above the normal range at 59 umol/L (upper limit of normal 42).

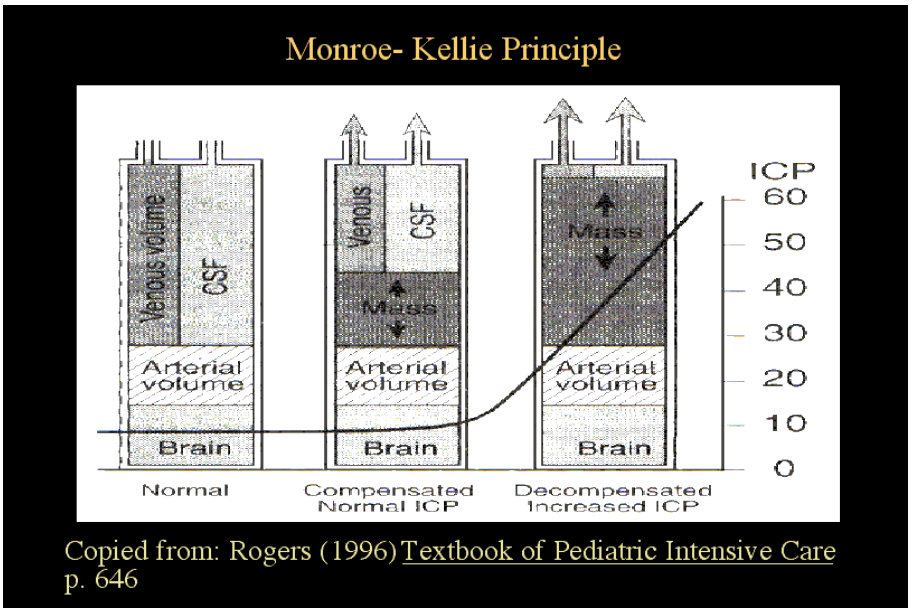
16. I cannot see an Amylase to exclude pancreatitis.

17. Raychel had a CT scan which was initially reported as showing subarachnoid haemorrhage. A neurosurgical opinion was obtained and a further CT scan with contrast was obtained which showed no additional findings. Dr McKinstry, the consultant neuroradiologist,

felt that there was no evidence of subarachnoid haemorrhage but that the appearances were those of cerebral oedema.

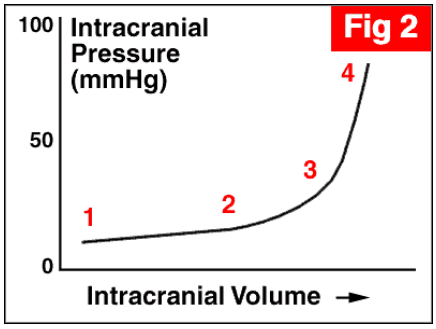
18. A post mortem was carried out on the 11th June 2001 by Dr Herron who reported the cause of Raychel's death as cerebral oedema secondary to hyponatraemia.

19. The intracranial contents consist of three components; brain, blood (arterial and venous) and cerebrospinal fluid (Figure 1).



If there is a mass lesion or the volume of one of these components increases e.g. there is cerebral oedema leading to increased volume of the brain, there is some reserve capacity related to (i) reduction of venous blood volume by compression and/or drainage into the jugular veins and (ii) reduction of CSF volume by increased absorption in the subarachnoid space over the brain and around the spinal cord. This is difficult to predict on an individual basis, but may be less in children as there is little cerebral atrophy meaning that the skull is almost completely full of brain. When the volume of the contents of the skull exceeds the reserve capacity, the intracranial

pressure can go up extremely quickly because of the shape of the pressure-volume curve. The relationship between increasing volumes and intracranial pressure are shown in the enclosed diagram.



20. Acute reduction in conscious level associated with cerebral oedema on neuroimaging has been reported in water intoxication/dilutional hyponatraemia in childhood (Arieff et al 1992, Boetzkes et al 2010, Radojevic et al 2012) as well as menstruating women (Ayus et al. A few children involved in apparently minor trauma or after undergoing tonsillectomy, appendicectomy or orchidopexy have died with no other cause identified, although the series reporting most of these childhood deaths was from more than 20 years ago. The possibility of co-existing metabolic disease predisposing to cerebral complications was not considered. Neuroimaging was less sophisticated in the 1990s so that cerebral co-morbidities, e.g. vascular pathologies such as venous sinus thrombosis, would not have been excluded. Even now neuroimaging is not necessarily comprehensive: for example the child with fatal hyponatraemia after renal transplant described by Cansick et al (2009) had had meningitis, which may be associated with compromise of the venous sinuses (Sebire et al 2005).

21. Raychel's death associated with cerebral oedema occurred in 2001, i.e. in a similar era to Arieff's series, with similar concerns that cerebral and metabolic co-morbidities may not have been excluded.

22. Raychel was vomiting profusely and may have become sufficiently dehydrated to be at risk of venous thromboembolism which could have involved the cerebral venous sinuses. Her haematocrit was a little low on admission, consistent with iron deficiency, a risk factor for cerebral venous sinus thrombosis (Sebire et al 2005). Venous sinus thrombosis is difficult to exclude even in 2012 but may be associated with subarachnoid haemorrhage, acute cerebral swelling and brain death as well as seizures (Sebire et al 2005).

23. It is possible that Raychel had a metabolic problem exposed by the operation or a co-existing infection, e.g. of the urinary tract. The most likely in my opinion would have been that she was heterozygous for ornithine carbamyl transferase deficiency, a condition which can present with profuse vomiting and cerebral oedema secondary to hyperammonaemia (diagnosed as Reye's syndrome before the enzyme was identified and aspirin was discontinued in children). A patient undergoing appendicectomy but with pancreatitis and hyperammonaemia has been reported (Pedal et al 1984).

24. I have not been able to locate Raychel's height so far but her blood pressure may have been intermittently higher than the appropriate centile, a risk factor for posterior reversible encephalopathy syndrome.

25. If Raychel developed any primary cerebral problem during 7th-9th June, she would have been at risk of hyponatraemia secondary to cerebral salt-wasting as well as inappropriate antidiuretic hormone secretion and any effect of the hypotonic fluids. The urinary sodium measurement should be reviewed with the expert in fluid balance in the light of these possibilities.

26. In answer to paragraph 132 under Requirements: I think that Raychel's brain damage became irreversible between 04:00 and 04:45 when her pupils became fixed and dilated and her respiration required mechanical support with bagging and then intubation.

27. My report will require modification when I have been able to view Raychel's earlier history and her family history from the general practice notes and have access to the reports from the expert neuroradiologist, neuropathologist and expert in fluid balance. In particular, the question of whether there was a subarachnoid haemorrhage or whether that pathology can be definitely excluded is of considerable importance. On the balance of probabilities, she was vomiting because of a developing intracerebral problem which may have been exacerbated by, but not necessarily caused by, the administration of hypotonic fluids.

Declaration

I Fenella Kirkham DECLARE THAT:

1. I understand that my duty in providing written reports and giving evidence is to help the Court, and that this duty overrides any

obligation to the party by whom I am engaged or the person who has paid or is liable to pay me. I confirm that I have complied and will continue to comply with my duty.

2. I confirm that I have not entered into any arrangement where the amount or payment of my fees is in any way dependent on the outcome of the case.

3. I know of no conflict of interest of any kind, other than any which I have disclosed in my report.

4. I do not consider that any interest which I have disclosed affects my suitability as an expert witness on any issues on which I have given evidence.

5. I will advise the party by whom I am instructed if, between the date of my report and the trial, there is any change in circumstances which affect my answers to points 3 and 4 above.

6. I have shown the sources of all information I have used.

7. I have exercised reasonable care and skill in order to be accurate and complete in preparing this report.

8. I have endeavoured to include in my report those matters, of which I have knowledge or of which I have been made aware, that might adversely affect the validity of my opinion. I have clearly stated any qualifications to my opinion.

9. I have not, without forming an independent view, included or excluded anything which has been suggested to me by others, including my instructing lawyers.

10. I will notify those instructing me immediately and confirm in writing if, for any reason, my existing report requires any correction or qualification.

11. I understand that;

    1. my report will form the evidence to be given under oath or affirmation;

    2. questions may be put to me in writing for the purposes of clarifying my report and that my answers shall be treated as part of my report and covered by my statement of truth;

    3. the court may at any stage direct a discussion to take place between experts for the purpose of identifying and discussing the expert issues in the proceedings, where possible reaching

an agreed opinion on those issues and identifying what action, if any, may be taken to resolve any of the outstanding issues between the parties;

4. the court may direct that following a discussion between the experts that a statement should be prepared showing those issues which are agreed, and those issues which are not agreed, together with a summary of the reasons for disagreeing;

5. I may be required to attend court to be cross-examined on my report by a cross-examiner assisted by an expert;

6. I am likely to be the subject of public adverse criticism by the judge if the Court concludes that I have not taken reasonable care in trying to meet the standards set out above.

12. I have read Part 35 of the Civil Procedure Rules and the accompanying practice direction including the “Protocol for Instruction of Experts to give Evidence in Civil Claims” and I have complied with their requirements.

13. I am aware of the practice direction on pre-action conduct. I have acted in accordance with the Code of Practice for Experts.

Statement of Truth

I confirm that I have made clear which facts and matters referred to in this report are within my own knowledge and which are not. Those that are within my own knowledge I confirm to be true. The opinions I have expressed represent my true and complete professional opinions on the matters to which they refer.

Fenella Kirkham FRCPCH

Professor in Paediatric Neurology

Consultant Paediatric Neurologist

8th Feb 2012

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