# HER MAJESTY'S CORONER

DISTRICT OF GREATER BELFAST

John L Leckey LL.M.
H.M. Coroner
Coroner's Office
Courthouse
37 Church Road
Newtownabbey
Co. Antrim BT36 7LA
Northern Ireland

Telephone: [redacted]
Fax: [redacted]
E-mail: jleckey.rcj [redacted]

Ms Therese Brown
Risk Management Co-Ordinator
Altnagelvin Hospitals Health & Social
Services Trust
Altnagelvin Area Hospital
Glenshane Road
LONDONDERRY BT47 6SB

5th December 2001

Dear Ms Brown

**RACHEL FERGUSON, DECEASED**

I refer to my telephone conversation with you on 4th December and as arranged I am enclosing a copy of the post-mortem report of Dr Brian Herron and a copy of the supplemental report of Dr Clodagh Loughrey.

As I indicated to you I have decided to obtain an independent report from a Consultant Paediatric Anaesthetist. Several years ago I obtained a report in a not dissimilar case from Dr Edward Sumner, Consultant Paediatric Anaesthetist at Great Ormond Street Hospital for Children. I am seeking to ascertain if he would be willing to prepare a report in connection with the death of Rachel Ferguson and give evidence at the inquest. If he is agreeable I will let you have a copy of his report in advance of the inquest. In any event, I will write to you to confirm that he will be preparing a report for me and, if not, the identity of an alternative similar expert.

Please confirm that Dr Sumner may approach you direct for access to medical records. Also, I require statements as a matter of some urgency. I know Dr Sumner would require sight of these to enable him to prepare his report.

Yours sincerely

**J L LECKEY**
**HM CORONER FOR GREATER BELFAST**

**RF - ALTNAGELVIN**

**022-070-170**

# DEPARTMENT OF NEUROPATHOLOGY
# AUTOPSY REPORT

Autopsy No.: NPPM 61/2001
Name : FERGUSON, Rachel
Date of Birth:4-Feb-92 Hospital No.: CH 476554
Sex : F Hospital:RBHSC
Pathologist : Dr M Al-Husaini/
Dr B Herron Ward :PICU
Clinician :Dr P Crean
Date of Admission : 9-Jun-01
Date of Autopsy : 11/6/01 Date of Death : 10-Jun-01
Time of Autopsy : 10.00am Time of Death : 12.09 pm
Restrictions : Coroner's case
Organs Retained: Brain and spinal cord

ANATOMICAL SUMMARY SNOMED 3 CODES

History of appendicectomy 07/06/01 Altnagelvin, history of seizures 09/06/01 and brain stem death 09/06/01 at 12.09pm, acute cerebral oedema, aspiration pneumonia (see commentary).

P3-42000 TA0100 M36300

20/11/01

CLINICAL SUMMARY

She was admitted to Altnagelvin Hospital on 7/6/01 with abdominal pain and was diagnosed as having appendicitis. Appendicectomy was done on the same day and she was doing well after that. On 8/6/01 she was conscious and able to walk. However, she vomited 6 – 7 times but there was no fever or diarrhoea. On 9/6/01 at 3.00am she developed tonic seizures which lasted for 15 minutes. She received medication but did not improve. Soon after this she developed fixed dilated pupils with petechial haemorrhages on the anterior chest wall and possibly aspirated. An urgent CT scan showed possible subarachnoid haemorrhage with evidence of increased intracranial pressure. Electrolyte analysis showed sodium level of 118 mg./dl and potassium of 3 mg./dl. She was intubated and transferred to the RVH on 9/6/01. A second CT scan showed cerebral oedema and she was pronounced brain stem dead on 9/6/01 at 12.09 pm.

## EXTERNAL EXAMINATION

The body is that of a female child with features in keeping with that of the age of the deceased. She weighed 25 kg. Head circumference is 144 cm. Crow-heel is 120 cm. Average foot length is 19 cm. Petechial haemorrhages were noted on the anterior chest wall.

## INTERNAL EXAMINATION

BODY CAVITIES:

There is no pleural or pericardial effusion and there is no ascites.

HAEMATOPOIETIC SYSTEM:

Spleen This weighs 95.2 g. and appears unremarkable.

Histology shows this is congested.

Thymus This weighs 16 g.

This shows no abnormality.

MUSCULO-SKELETAL SYSTEM:

No fractures are seen. No muscle wasting is seen.

The muscles examined show no abnormality.

RESPIRATORY SYSTEM:

Lungs The right lung weighs 193 g. and left lung weighs 219 g. Both show haemorrhage in keeping with aspiration. There is no petechial haemorrhage on the plerual surface.

There is pulmonary oedema and haemorrhage with only occasional neurtrophils. These features suggest the possibility of aspiration.

CARDIOVASCULAR SYSTEM:

Heart This weighs 137 g. There is no atrial, valvular or ventricular lesion. The aorta is unremarkable. The coronary arteries appear normal.

There is patchy myocardial inflammation and pericardial inflammation in keeping with stress haemorrhage secondary to cerebral oedema. It does not appear ischaemic in distribution.

DIGESTIVE SYSTEM:

Oesophagus, stomach, small and large intestine There is no lesion.

Histology of the oesophagus shows no pathological abnormality. The small and large intestine are autolysed, but show no definite abnormality. There is no peritonitis.

The site of the appendectomy was clean and showed no inflammation.

Liver This weighs 783 g. and shows no focal lesion.

There is no abnormality.

Gallbladder This is present and the biliary system is patent.

Pancreas This shows no abnormality.

GENITO-URINARY SYSTEM:

Kidneys The right kidney weighs 770 g. The left kidney weighs 760 g. Both appear unremarkable. The collecting system is unremarkable.

There is no interstitial inflammation or acute pyelonephritis. There is no tubular necrosis and the glomeruli appear normal.

Bladder This is unremarkable.

ENDOCRINE SYSTEM:

Adrenals The combined adrenal weighs is 7.4 g.

These are unremarkable.

Thyroid This is unremarkable.

Parathyroids These are unremarkable.

Pituitary This is slightly congested, but shows no definite evidence of necrosis.

NERVOUS SYSTEM:

BRAIN DESCRIPTION

The brain weighs 1,475 g. On external examination the brain is quite pale. There is diffuse swelling with effacement of sulci and flattening of gyri. There is no infarct and there is no subarachnoid haemorrhage. On examination of the base of the brain the anatomy is normal. There is no abnormality to the blood vessels. There is bilateral uncal swelling and bilateral uncal necrosis.

On examination of the coronal sections the presence of diffuse cerebral oedema is confirmed with effacement of the ventricular system and slight caudal descent. There is no shift. There is evidence of diffuse hypoxic ischaemic necrosis due to perfusion failure with discoloration at the grey/white matter junction. However, there is no regional cortical lesion. The laminar pattern is normal. There are no heterotopias or suggestions of migration abnormality. The hypothalamus does not appear necrotic although the mamillary bodies are slightly elongated but this is more likely is an effect of cerebral oedema. There are no peticule haemorrhages. The brain stem and especially the pons appears normal. There is no intrinsic abnormality in the cerebellum. The cord appears normal.

9/7/01 DB

HISTOLOGY

Meninges

There is no acute meningitis. Very few inflammatory cells are present in the meninges which appear reactive in nature.

Cerebral Cortex

This has been examined in multiple areas and shows cerebral oedema and established diffuse hypoxic ischaemic necrosis as suggested by the macroscopic findings. There is no laminar necrosis and there is no abnormal inclusion seen. There is no encephalitis.

Deep Grey and White Matter

These show the changes of cerebral oedema, but no other abnormality.

Hippocampus

Oedema is present and very early acute changes of diffuse hypoxic ischaemic necrosis are seen.

Hypothalamus

Oedema is present, but there is no anatomical abnormality.

Brain Stem

This has been serially sectioned. It shows focal evidence of diffuse hypoxic ischaemic necrosis with neuronal necrosis. There is no evidence of central pontine myelinolysis.

Spinal Cord

There is no intrinsic abnormality, but a few mononuclear cells are present in the meninges suggestive of origin in the cerebellar tonsils.

21/11/01 jl

COMMENTARY

She had her appendix removed on 07/06/01 and developed seizures on 09/06/01. At autopsy she had cerebral oedema and aspiration pneumonia from which she died. Specialist opinion was sought as to the likely cause of the cerebral oedema and a report is enclosed. The summary of this was that the oedema was caused by rapid fall in plasma sodium concentration as a result of net sodium loss, coupled with hypotonic fluid administration in a situation (ie. post operative state +/- vomiting) where a normal physiological response inhibited the effective excretion of the excess free water. The abnormality of sodium balance and thus the cerebral oedema which led to her death was thought to be caused by three main factors:- 1. Infusion of hypotonic fluids, 2. Profuse vomiting, 3. Anti-diuretic hormone (ADH) secretion.

Established changes related to sodium imbalance such as central pontine myelinolysis were not seen possibly due to short time period between her deterioration and death. The relative contribution of these factors are unknown and as a combination they led to the brain swelling which eventually led to her death.

Dr Brian Herron
Consultant Neuropathologist
Department of Pathology
Royal Group of Hospitals
Grosvenor Road
Belfast BT12 6BJ

24 October 2001

Dear Dr Herron

Re: Rachel Ferguson DOB 4/2/92
Ref: NPPM 61/2001

Thank you for asking me to look at the Altnagelvin records of this girl who had a major seizure approximately 28 hrs after an apparently uncomplicated appendicectomy. Significant hyponatraemia was noted after the seizure and she subsequently died, cerebral oedema being your major finding at autopsy. I have summarised relevant sections of the notes available to me, the page of origin identified by the title in italics:

This 9-year-old girl was admitted via A&E on the evening of 7/6/01 with abdominal pain (*"Accident & Emergency" sheet:* not legible due to photocopy quality).

Admission and pre-operative period
*"Clinical notes" Pg. 1*: patient was examined on the Children's ward (ward 6?) by the surgical SHO, who documented periumbilical pain, which had shifted to the right iliac fossa (McBurney's point); she had RIF tenderness, guarding and mild rebound tenderness. Absence of urinary symptoms was recorded. She was felt to have acute appendicitis and consent was obtained for appendicectomy. Intravenous fluids were prescribed.
*"Observation sheet" (nursing) Pg. 1(7/6/01)* documented abdominal pain and pain on urination.
*"TPR chart"*: on admission, patient was afebrile, BP was 103/61, weight was 25kg. An (undated, time 23:19) urinalysis printout indicated proteinuria++.
*"Parenteral nutrition fluids prescription sheet" (Pg.1):* Intravenous fluids ("No. 18 solution") were erected at 80mls/hr at 10.15pm.
(FBP/U&E checked: see table of biochemistry results below.)

Intra-operative / peri-operative period
*"Theatre nursing care plan"*: arrived at 11.20pm. Alert, not premedicated, IV infusion site right arm.
*"Surgeon's report"*: mildly congested appendix. Peritoneum clean. Flagyl prescribed.
*"Intra-operative nursing care":* received rectal Voltarol 12.5mg and paracetamol 500mg at 11.40pm.
*"Anaesthetic record" Pg1/2*: received ondansetron 2mg, fentanyl 50mg total, propofol 100mg, scoline 30mg, cyclimorph 5mg, mivacurium 2mg, metronidazole 250mg. Peri-operative event: "prolonged sedation due to opioids".
Hartmann's fluid 1L?: anaethetist's intention indicated, but administration not confirmed by fluid balance chart.

*"Parenteral nutrition fluids prescription sheet"(Pg.1)* (same document as for pre-op): Hartmann's fluid prescribed at 80mls/hr, signed by anaethetist, but deleted (unsigned).

Post-operative period prior to seizure

*"Fluid balance for IV fluids" (Pg. 1: 7/6/01):* Received total 540 mls No. 18 solution between 22.15 on 7/6/01 and 07.00 hrs on 8/6/01. No record of urine output.

*"Clinical notes" (Pg. 2:8/6/01)*: "Free of pain. Apyrexial. Continue observation."

*"Paediatric unit"sheet (7-8/6/01*) (Apparently a nursing record chart.) Temperature/respiratory rate/pulse/blood pressure recorded on return from theatre 01.55am, half-hourly until 4am, then 5am, 7am. BP range 78-96/41-57. Temp, resp rate and pulse only recorded 4 hourly from 9am until 21.15 on 8/6/01. Afebrile throughout. No problems documented until 21.15pm: "colour flushed→pale. Vomiting++. C/o headache."

[NB: No *"Observation sheet"* for 8/6/01 is present in copy of notes I received (7/6/01 and 9/6/01 both present: see above and below).]

*"Parenteral nutrition fluids prescription sheet"(Pg.2):* 1L No.18 solution prescribed at 80mls/hr and erected at 12.15pm. [A second litre of 0.9% NaCl was apparently prescribed early on 9/6/01: from subsequent nursing notes.]

*"Fluid balance for IV fluids" (Pg. 2: 8/6/01):* Received 1520mls No.18 solution between 08.00 on 8/6/01 and 04.00 on 9/6/01. No record of urine output. No record of oral intake, if any. Seven episodes of vomiting documented between 08.00 on 8/6/01and 01.00 am on 9/6/01, with "coffee-grounds" mentioned latterly, but no measure of volume ("large vomit", "vomit++").

*"Observation sheet"* (nursing) 9/6/01: 03.05am: major seizure. Bloods taken at 3.30am for electrolytes: see table.

Likely pathogenesis of cerebral oedema

I have little doubt that the cerebral oedema which you noted at autopsy was caused by an intracellular fluid shift as a result of rapid fall in tonicity of the extracellular fluid (ECF). As you can see from the table of biochemistry results, I have estimated a fall of 37mOsm/L (293 to 256 mOsm/L) over approximately 30 hrs. Sodium is the predominant extracellular cation and as such is the major determinant of extracellular tonicity. The cell membrane is relatively impermeable to sodium due to an active sodium pump mechanism, and rapid changes in the concentration of sodium in the ECF (in either direction) result in significant fluid shifts to maintain osmotic equilibrium between the intracellular and extracellular compartments. The brain is particularly susceptible to the effects of such fluid shifts and profound neurological damage such as occurred in this case has been well-described in association with rapid increases and decreases in plasma sodium concentrations. Cerebral oedema with its attendant acute neurological features is characteristic of rapidly-developing hyponatraemia.

I believe that in this case the fall in plasma sodium concentration and thus ECF tonicity was caused by a combination of 3 main factors:

1. infusion of hypotonic parenteral fluids (No. 18 solution contains 31mmol Na in 1 litre 4% glucose solution, one-fifth the concentration of plasma);

2. profuse vomiting in the post-operative period. Although vomitus contains 70-100mmol of sodium /L, which is relatively less than plasma (at 140mmol/L), if the ECF volume is replaced as in this case with fluids containing very little sodium, the net effect is a significant salt loss, with little or no water deficit;
3. Anti-diuretic hormone (ADH) secretion, known to be associated with stress (e.g. surgery), vomiting and pain, is likely to have been a major contributor to the overall picture by inhibiting excretion of excess free water.

The relative contributions of these factors will remain unknown. Normally administration of generous volumes of hypotonic fluids will result in a brisk diuresis, and certainly this will be noted by most healthy people who can tolerate drinking large amounts of dilute fluids without consequence. However in this case excess ADH secretion for the reasons mentioned above might have resulted in a net positive fluid balance and an inappropriately concentrated urine. Urine osmolarity was indeed inappropriately high in the sample taken after the seizure (measured last week on the sample obtained by you from Altnagelvin laboratory), and the low urea notable in the post-seizure serum samples, relative to that on admission, might indicate relative water excess as a consequence of ADH action. However whether this was a cause or effect of the cerebral oedema cannot be judged and no plasma or urine samples are available from the post-operative but pre-seizure period. Unfortunately no record of fluid balance was apparent. A low urinary output might have given an early sign of evolving problems.

(I also measured cortisol in the post-seizure blood sample and this was appropriately elevated, excluding adrenal insufficiency as a cause of the hyponatraemia.)

In summary, I believe that the cerebral oedema which you noted at autopsy was caused by a rapid fall in plasma sodium concentration as a result of a net sodium loss coupled with hypotonic fluid administration in a situation (i.e. post-operative state ± vomiting) where a normal physiological response inhibited the effective excretion of the excess free water

I hope this has been of some help. Please do not hesitate to contact me if further clarification is required.

Best wishes.

Yours sincerely,

Clodagh Loughrey MD MRCP MRCPath
Consultant Chemical Pathologist
Belfast City Hospital

RF - ALTNAGELVIN

022-070-179

Table of relevant laboratory results: Rachel Ferguson (DOB 4/2/92)
(*tests performed *post-mortem*)

| **Serum** | | | | | |
|---|---|---|---|---|---|
| | **Pre-operatively** | **Post-seizure** | | | |
| **Date** | 7/6/01 | 9/6/01 | 9/6/01 | 9/6/01 | 9/6/01 |
| **Time received in lab** | 9pm approx. | 4.06am | 4.40am | 9.22 am | 3pm |
| Lab. No. | 01633 | 01742 | 01747 | 5380 | (RBHSC) |
| | | | | | |
| Na (mmol/L) | 137 | 119 | 118 | 119 | 130 |
| K (mmol/L) | 3.6 | 3.0 | 3.0 | 3.4 | |
| Cl (mmol/L) | 107 | 90 | 90 | 90 | |
| CO2 (mmol/L) | 22 | 16 | 15 | 22 | |
| Urea (mmol/L) | 4.8 | 2.3 | 2.1 | 2.5 | |
| Creat (mmol/L) | 47 | 44 | 43 | 22 | |
| Glucose(mmol/L) | 7.2 | 9.9 | 11 | 7.1 | |
| T prot (mmol/L) | 69 | 71 | 72 | 68 | |
| Osmol (mOsm/L) (calc) | 293* | 256* | | 255* | |
| | | | | | |
| **Urine** | | | | | |
| **Date** | | | | 9/6/01 | |
| **Time** | | | | 9am | |
| Lab. No. | | | | 5425 | |
| Na (mmol/L) | | | | 90 | 13 |
| Osmol (mOsm/L) | | | | 382* | 73 |

Cor.1

DEPARTMENT OF NEUROPATHOLOGY

INSTITUTE OF PATHOLOGY, GROSVENOR ROAD, BELFAST

Name: FERGUSON Rachael Age: 9 yrsSex: F P.M.No.: NPPM 61/2001

Date of Admission:09/06/01 Ward:PICU, RBHSC Date of Death:09/06/01

Date of Autopsy:11/06/01

Place of Autopsy: Mortuary, RVH

On the instruction of H.M. Coroner, Mr. J.L. Leckey, LL.M. – Dr. Department of Neuropathology, Institute of Pathology, Grosvenor Road, Belfast, Northern Ireland, made a post mortem examination on the body of:-

Rachael Ferguson
Aged: 9 yrs

Identified to Dr B Herron By Constable MB Adams
Grosvenor Rd

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CLINICAL SUMMARY

She was admitted to Altnagelvin Hospital on 7/6/01 with abdominal pain and was diagnosed as having appendicitis. Appendicectomy was done on the same day and she was doing well after that. On 8/6/01 she was conscious and able to walk. However, she vomited 6 – 7 times but there was no fever or diarrhoea. On 9/6/01 at 3.00am she developed tonic seizures which lasted for 15 minutes. She received medication but did not improve. Soon after this she developed fixed dilated pupils with petechial haemorrhages on the anterior chest wall and possibly aspirated. An urgent CT scan showed possible subarachnoid haemorrhage with evidence of increased intracranial pressure. Electrolyte analysis showed sodium level of 118 mg./dl and potassium of 3 mg./dl. She was intubated and transferred to the RVH on 9/6/01. A second CT scan showed cerebral oedema and she was pronounced brain stem dead on 9/6/01 at 12.09 pm.

My findings are consistent with death having taken place on

| | | |
|---|---|---|
| (I) | Disease or condition directly Leading to death: | Ia. Cerebral oedema due to |
| | Antecedent causes. Morbid conditions, if any, giving rise to the above cause, stating the underlying conditon last. | b. Hyponatraemia |
| (II) | Other significant conditions, contributing to the death but not related to the disease or condition causing it. | II. |

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COMMENTARY

She was admitted on 07/06/01 for an appendicectomy because of abdominal pain. On 09/06/01 she developed seizures and was noticed to have a low sodium level in her blood. Expert opinion was sought on the cause of this and an additional report is enclosed. It suggested the low sodium was due to a combination of three causes including infusion of fluids post operatively which contained a low sodium concentration. Other factors were the vomiting in the post operative period and the stress of surgery causing secretion of other chemicals. The expert stated that the relative contributions of these factors are unknown and as a combination they led to this brain swelling which eventually led to her death.

AM 4/2/01