Witness Statement Ref. No.



NAME OF CHILD: RAYCHEL FERGUSON (LUCY CRAWFORD)

Name: CAROLINE GANNON

Title: Doctor

Present position and institution: Consultant paediatric pathologist Northern Ireland Regional Paediatric Pathology Service Royal Victoria Hospital, Belfast

Previous position and institution: [*As at the time of the child's death*]

April 2000: Specialist registrar in paediatric pathology, Hammersmith and Queen Charlotte's Hospital, London

Membership of Advisory Panels and Committees: [Identify by date and title all of those between January 2000 – August 2012]

Group B Streptococcus working group, 2011-2012

Previous Statements, Depositions and Reports: [Identify by date and title all those made in relation to the child's death]

OFFICIAL USE: List of previous statements, depositions and reports attached:

Ref:	Date:	
WS-281/1	11-10-2012	Statement to the Inquiry

IMPORTANT INSTRUCTIONS FOR ANSWERING:

Please attach additional sheets if more space is required. Please identify clearly any document to which you refer or rely upon for your answer. If the document has an Inquiry reference number, e.g. Ref: 049-001-001 which is 'Chart No.1 Old Notes', then please provide that number.

If the document does not have an Inquiry reference number, then please provide a copy of the document attached

- (1) Arising out of your answers to question 2 of WS-281/1, please address the following additional matters:
 - (a) You are directed to Dr. O'Hara's reports which comprise a provisional report dated 17 April 2000 and a final report dated 12 June 2000, as well as a report dated 6 November 2003 which was written on the instruction of HM Coroner: see pages Ref: 013-017-054 through to Ref: 013-017-065. Please clarify which of these reports you had access to at the time you were asked to review the pathological findings of Dr. O'Hara.

I had access to Dr O'Hara's provisional and final reports dated 17 April 2000 and 12 June 2000, and the report dated 6 November 2003.

(b) In answer to question 2(h) you have stated that you "would have reached the same conclusion" as Dr. O'Hara had about the cause of death. For the avoidance of doubt please state the conclusion that you would have reached, and explain how you would have reached such a conclusion.

I reached the conclusion that the pathological diagnoses present at the time of death were bronchopneumonia in the lungs, and cerebral oedema of the brain. Both of these disease processes are readily identifiable on inspection of the histological sections of the lung and brain tissue made at the time of the autopsy. From the written report, Dr O'Hara appears to have made an extensive search for the presence of gastro-enteritis, both in submitting samples for bacteriological culture and electron microscopy, and processing tissue for histological examination looking for cellular damage as a result of infection. He reached the conclusion that he could not see any structural injury to the bowel lining, but that this wasn't unexpected. I agree with his conclusion: some of the organisms that cause diarrheal type illnesses do not produce any structural alteration in the bowel lining.

Furthermore, I agreed with Dr O'Hara's written comment that cerebral oedema is the terminal event in several different disease processes. Bronchopneumonia is a cause of hypoxia (low oxygen levels) which can cause cerebral oedema. It is not possible histologically to determine what proportion of the cerebral oedema was caused by bronchopneumonia, if any, and what proportion caused by another disease process. Had this been my case, I may possibly have placed more emphasis on the presence of bronchopneumonia (depending on discussion with clinical staff), and may have placed bronchopneumonia in part II of the formulation of the cause of death-as a disease process present at the time of death-as this appears to be a significant disease process.

(c) When you considered Dr. O'Hara's reports on behalf of the Coroner, did you give any consideration to the contribution played by dilutional hyponatraemia in causing the

cerebral oedema suffered by Lucy Crawford? If so, please indicate what consideration you gave to that issue and explain the conclusions which you reached.

Dilutional hyponatraemia is a diagnosis that cannot be made by a histopathologist alone. The pathological diagnosis provided by Dr O'Hara, made on examination of the brain both grossly (naked eye inspection) and histologically, was 'cerebral oedema'. Cerebral oedema is a descriptive term used to describe the appearance of excessive water accumulating within the brain tissue. Cerebral oedema can occur in response to trauma, tumours, inflammation, ischaemia, drugs and toxins, amongst other causes. Furthermore, this child also had established bronchopneumonia: this is a severe chest infection that can cause hypoxia, and hypoxia is a cause of cerebral oedema such as tumour or infection, but dilutional hyponatraemia i.e. a fluid-electrolyte disorder, cannot be definitively diagnosed microscopically. This is a diagnosis reached by clinicopathological correlation.

Clinicopathological correlation is an objective summary and correlation of the clinical findings in a particular case with the gross and microscopic findings and with the results of other studies performed at autopsy, ultimately to describe the cause of death and elucidate the sequence of events leading to death. The pathologist may not necessarily have sufficient clinical background or expertise, or access to all of the clinical information required to perform this alone to reach a formal diagnosis of the cause of death. Clinicopathological correlation is best carried out in a setting such as a mortality meeting with the clinicians who had cared for the patient present, to discuss all relevant findings and reach a consensus as to the causation of death. In normal circumstances, paediatric autopsy cases are discussed at hospital mortality meetings attended by the pathologist who performed the autopsy and the clinicians who cared for the patient: I assume that this case was discussed by Dr O'Hara with the patient's clinician, but I was not privy to any discussion.

My interpretation was that cerebral oedema was present on microscopic examination of the brain, but that the determination of the cause of the cerebral oedema and the potential role of dilutional hyponatraemia would require more detailed clinical interpretation of the fluidelectrolyte status of the patient, an interpretation that is outwith my area of expertise. However, I would also question the role of bronchopneumonia in the causation of cerebral oedema, or the worsening of pre-existing cerebral oedema.

THIS STATEMENT IS TRUE TO THE BEST OF MY KNOWLEDGE AND BELIEF

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Signed:

Caroline Gannon

Dated: 28th December 2012