Witness Statement Ref. No. 247/2

NAME OF CHILD: Claire Roberts

Name: Meenakshi Mirakhur

Title: Dr.

Present position and institution:

Retired December 2010.

Previous position and institution:
[As at the time of the child's death]
Consultant Neuropathologist, Royal Hospitals Trust (now Belfast Health and Social Care Trust).

Membership of Advisory Panels and Committees:
[Identify by date and title all of those between October 1996-August 2012]

Executive Committee Member, British Neuropathological Society: 2000-2010.
Treasurer, British Neuropathological Society: 2006-2010.

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

Witness statement reference No: 247/1.

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

Ref:	Date:	
WS-247/1	30th May 2012	Inquiry Witness Statement

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 such a number then please provide a copy of the document.

(1) **In October 1996 were you aware of:**

(a) **The Guidelines for Post-Mortem Reports, the Royal College of Pathologists, 1993 (Ref: 306-072-001)?** Yes.

(b) **The Report of the National Confidential Enquiry into Perioperative deaths:** Yes.

(2) **Do you agree with the Coroner's verdict and findings in the case of Claire Roberts (Ref: 091-002-002)?** Although I was not requested to give evidence by the Coroner at the Inquest, I am aware of his verdict and findings. As mentioned in my deposition (247/1) there was cerebral oedema and evidence of low grade inflammation in the brain.

(3) **In October 1996, what knowledge did you have of the case of Adam Strain?** I do not recall any knowledge about Adam Strain.

(4) **In October 1996 were you aware of the Arieff et al paper BMJ 1992 (Ref: 011-011-074)?** I cannot recall.

(5) **Did you see a copy of Dr. Alison Armour's Autopsy Report in the case of Adam Strain in 1996-1997 (Ref: 059-039-083)?** No.

(6) **Do you accept final responsibility for the Claire Roberts Autopsy Report in respect of:** It was a joint report as part of the team with Dr Herron in the context of the clinical information available at the time of Claire's death (this answer is for a, b, c and d below).

(a) **Accuracy;**

(b) **Content;**

(c) **Completeness;**

(d) **Presentation.**

(7) **Please identify any evidence of which you are aware in respect of Claire Roberts Autopsy, to confirm:**

(a) **The date and time the Autopsy Request Form was received?**

This information may be available from the mortuary.

(b) **The date and time the post mortem Consent Form was received?**

This information may be available from the mortuary.

(c) **The date the provisional Anatomical Summary (Ref: 090-005-007) was drafted?**

There is no record of this date.

(d) **The documents received, and whether any additional documents were sought?**

I cannot recall.

(e) **The verbal communications and additional information received?**

I cannot recall.

(f) **Whether a finalised copy of the report was signed and if so by whom?**

The final report is usually signed by the Pathologist but I cannot find a signed copy.

(g) **Whether a finalised and signed copy was sent out?**

It is normal practice to send out a signed copy to the clinicians but I cannot find a signed copy in our Departmental records.

(h) **Whether the Autopsy Report was presented to Audit meetings, Mortality meetings and/or Neuroscience Grand Round?**

Autopsy reports are usually presented to mortality and neurosciences meetings but there is no record in the Departmental files.

(8) **In respect of the draft version Autopsy Report dated 11th February 1997, and bearing your handwritten additions, please explain the meaning of the annotation at the top of page 1 *"DOCTOR S COPY COMPLETE + SENT 12/2/97"*?**

This is the draft report for the Pathologist to make corrections after it has been typed; only a final report with all the corrections made is sent. The secretaries may make a note (as on the top) for records.

(9) **Please indicate whether any assistance by way of second opinion or review was sought from any other pathologist apart from Dr. Herron?**

I cannot recall.

(10) **What procedures were adopted in 1996 if an inconsistency was detected between the information supplied in the Autopsy Request form and the information contained in the medical notes?**

I cannot recall what procedures were in place but the autopsy request form usually contains a summary of the medical notes.

(11) **Did you seek any clarification or further information from the requesting doctor?**

I cannot recall.

(12) **Did you seek a copy of the ICU Ward Discharge advices and diagnosis or were you supplied with a copy of the same?**

I cannot recall.

(13) **Please state whether you cross checked the medical notes with the Autopsy Request form to ensure completeness and accuracy of instruction, especially in relation to:**

(a) **Drug administration;**

(b) **Clinical history;**

(c) **Previous formulation of diagnosis?**

We do not normally do this.

(14) ***"The policy at that time was to enter the name but not the grade of the junior doctor on the Autopsy Report and Provisional Anatomical Summary. The consultants name was not reported on these Reports. This policy has changed and the consultants name is now recorded on all Autopsy Reports"*** **(WS-224/3 p.4 at Q2(b)). In respect of this statement please:**

(a) **Identify policy referred to;**

I cannot recall.

(b) **State date of change to said policy.**

I cannot recall.

(15) **Please advise as to why you did not advance an opinion as to the most likely cause of death on the basis of all known information?**

It is inappropriate to give a cause of death after a brain only autopsy; that is what was consented for in this case.

(16) **Were you provided with an explanation as to why this was an autopsy restricted to 'brain only'?**

I cannot find this in the autopsy request form. The nature of a consented autopsy is usually determined by the consent provided.

(17) **Did you seek an explanation as to why this was an Autopsy restricted to 'brain only'?**

Pathologists are not usually involved in obtaining the consent. The autopsy was conducted according to the consent that had been provided.

(18) **Would you have expected the Autopsy Report to have been:**

(a) **Inserted into the Medical Notes of Claire Roberts;**

(b) **Sent to her General Practitioner.**

(for both a and b above). The pathologists send the report to the clinician.

(19) **Why does the Autopsy Report not contain a *"cause of death"* section?**

Please see answer to 15 above.

(20) **Given the information provided to you of respiratory arrest, cerebral oedema, SIADH and serum sodium of 121 mmol, why did you not implicate hyponatraemia as a possible cause of**

death?

Hyponatraemia is a clinical diagnosis. However, it is recorded in the autopsy report that a metabolic cause for cerebral oedema cannot be excluded.

(21) **In 1996, did you consider hyponatraemia to be treatable condition?**

This is outside my area of expertise.

(22) **Did you consider reporting the death of Claire Roberts to H.M. Coroner? If not why not?**

There was no indication for the pathologist to do so at that time.

As regards the reporting of a death to the Coroner, the guidance is that the death should be reported "if the cause of the death is not known". This is a general guidance which may be of use to GPs also who may not be able to request an autopsy. A Coroner's post-mortem is requested if the cause of death is not known in a hospital patient; there will be no need for a consented post-mortem under such circumstances.

(23) **Was the provision neuropathological services in the RBHSC in 1996 subject to accreditation? If so please provide details of the same.**

Yes since February 1996.

(24) **What was the purpose of the Provisional Anatomical Summary?**

The purpose is to record the naked eye examination findings immediately after carrying out an autopsy.

(25) **Please provide any further comments you think may be relevant, together with any documents or materials.**

PLEASE SEE NEXT PAGES FOR FURTHER COMMENTS

I wish to make the following further comments in relation to the reports of expert witnesses Dr Squier and Dr Harding.

It is important to clarify that the expert Neuropathologists' reports of Dr Squier and Dr Harding were prepared following the establishment of the Hyponatraemia enquiry after 2005 whereas our autopsy report was prepared in 1997 in the original clinical context of epilepsy, seizures, probability of viral encephalitis, cerebral oedema and learning disabilities. I would like to re-state that we raised the possibility of a metabolic cause for cerebral oedema in our original autopsy report. I would also like to emphasize that there are no specific structural lesions in the brain specific to hyponatraemia.

I would like to highlight the following three additional points:

1. Viral encephalitis. While Dr Squier and Dr Harding state that they could not confirm the presence of viral encephalitis, Dr Squier has described cellular reactions in perivascular space and brain parenchyma. It is also important to note that the quality of the slides may have deteriorated between when these were made and when these experts saw them. I am also not sure if the experts made new slides from the blocks but if they did, these new slides may not be from the same areas of the brain as are shown in the slides prepared in 1997.

In relation to Dr Harding's statement (document 235-002), Dr Harding had been asked whether in his experience an acute fulminant encephalitis causing cerebral oedema, coning and death in the space of three days could occur in the absence of clear neuropathological changes possibly as a result of the rapidity of such an infection? Dr Hardings's response was to the effect that his experience did not support this contention. He stated: "Given the degree of marked brain swelling noted clinically (including papilloedema on CT scan) and confirmed at post-mortem, I consider it extremely unlikely that microscopic evidence and encephalitis would not be evident by three days. I have seen it occurring within 36 hours."

My response to this is that neither Dr Herron nor myself suggested that there was acute fulminant encephalitis (Please see the autopsy report).

Dr Squier agrees that the diagnosis of meningo-encephalitis is possible. See (236-004-014) (68). Very rapidly progressive infectious encephalitis may cause death with little change in the brain.

2. Cerebral oedema. With regard to Dr Squier's point about the methods of assessing cerebral oedema (Deposition 236-004-021):

Dr Squier was asked (Question 5B): Please comment on whether brain weight is useful to record, but is not per se an accurate indicator of pathological processes or oedema. In reply Dr Squier stated that Haussmann concluded that fresh brain weight compared with standard tables for expected weight for age is the most reliable criterion for grading brain swelling. It is also important for assessing brain atrophy.

I would like to say that while fresh brain weight may be a reliable criterion of brain swelling, it

may not always be feasible to weigh fresh brain. It is the responsibility of the pathologist 'on the spot' to make a decision in each individual case as to the optimum method of assessing the brain when confronted with the extreme softening of the brain in order to avoid handling artifacts.

Dr Squier was asked (Question C): Please comment on whether cerebral oedema is better diagnosed by examining a carefully fixed brain weeks after the initial autopsy, by looking for swelling of the surface of the brain and by microscopic examination of the brain. Dr Squier stated that examination of the fixed brain is also helpful in determining the degree of swelling. She stated that she believed that microscopy was less helpful and that histological assessment of oedema does not correlate with other markers of swelling or the clinical estimation of brain water (Hausmann 2006).

With regard to the paper by Hausmann, 'Values for Morphological Parameters for Grading of Brain Swelling', I would like to state that his paper involved 42 individuals all over the age of 15 and none with a previous neurological disorder. He examined only one histological parameter in coming to his conclusion and we find this paper of little relevance to our conclusion.

Dr Harding seems to agree that brain weight per se is not a strong indicator of cerebral oedema (096-027 of 22nd August 2007).

3. Neuronal migration disorder: Dr Harding and Dr Squier in their statements say that there is either no convincing evidence (Harding) or that there is no evidence of this (Squier).

The experts appear to interpret the findings differently from ourselves. They appear to regard the presence of subependymal neurons as part of normal anatomical structure whereas we have interpreted their origin to be from arrested migration the clinical manifestations of which may include mental handicap or learning disability.

Dr Harding does not specify the nature of these cells (subependymal neurons) whereas Dr Squier is quite specific about them to be of hypothalamic origin. Also Dr Squier states that the subependymal cells are likely to be residual germinal matrix. This is somewhat unusual as germinal matrix usually disappears by 36-40 weeks of gestation. Dr Squier's footnote comment re germinal matrix states young people but does not specify age.

I also wish to highlight the some differing opinions of the two experts employed by the enquiry:

Statement of Dr Harding (096-027) of 22 August 2007:

The basal ganglia and thalamus as well as the hippocampus are unremarkable. Sections from the brain stem and cervical spinal cord are similarly unremarkable. Dr Squier however describes at length the pathological changes in hippocampus and the cervical spinal cord.

The only relevant observation according to Dr Harding is of brain swelling (naked eye) as judged by increased brain weight after fixation (1606 gm), the normal for girls of this age being 1200 gm, effacement of gyri and the uncal prominence. However these are rather weak indicators not supported by major downward shift of the brain and cerebellum which is common in severely swollen brains and by the microscopy (lack of vacuolation of white matter). This is in contrast to Dr Squier's interpretation of white matter oedema.

This seems to disagree with Hausmann who suggests the brain weight to be the best indicator of cerebral oedema.

Dr Harding: The child was said to suffer from seizures, none were witnessed prior to hospital admission and certainly not status epilepticus. Moreover the neuropathological sequalae of status were not present nor was there damage to the hippocampus which may be seen in children with chronic epilepsy. This is in complete contrast to Dr Sequier's statement who describes the changes in hippocampus at length.

Statement of Dr Squier (236-003-001)

Paragraph 19: Hippocampal pathology. The Hippocampus shows gliosis predominantly in hilum and CA1. Dr Harding describes no damage to hippocampus

Paragraph 25: there is diffuse gliosis in the white matter and the superficial cortex. It is difficult to time this accurately; so the effects of hypoxia or infection before or at the time of birth cannot be readily distinguished from the effects of later seizures or even the terminal brain oedema. Dr Harding does not describe these changes.

Spinal cord: this is normally formed. The tissue is oedematous and the nerve cells appear shrunken and pyknotic. The tissue is not distorted or fragmented. There is a mass of cerebellar tissue outside the cord in subarachnoid compartment. Dr Harding describes no major downward shift of brain or cerebellum. He also states that the sections from spinal cord are unremarkable.

Many of the points raised in consolidated report have been dealt with in my statement above. I would like to add that the Expert Clinicians have pointed out that Claire's clinical presentation was suggestive of viral infection/encephalitis.

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

Signed: Meenakshi Hirachan **Dated:** 18/9/12