Witness Statement Ref. No. 224/4

**NAME OF CHILD: Claire Roberts**

**Name:** Brian Herron.

**Title: Dr.**

**Present position and institution:**

Consultant Neuropathologist

I lead the Regional Neuropathology Service for Northern Ireland.

I am the Lead Examiner for Northern Ireland for post mortem examinations for The Royal College of Pathologists.

I lead the Adult Post Mortem Service for the Belfast Health & Social Care Trust.

I am on the Coroner's list of doctors who can perform post mortems.

I am responsible for the vast majority of hospital Coroner's post mortems in Northern Ireland and interact with the Coroner's office on an almost daily basis.

I provide Expert Opinion Reports for the coroner.

I advise the coroner on post mortem certification on about 100 reports per year.

I provide a neuropathology referral service to the Northern Ireland Regional Forensic Medical Service, reporting approximately 100 referral cases per year.

I provide a neuropathology referral service for the Paediatric Pathology Regional Service. This Paediatric service constitutes about approximately 30% of my post mortem workload.

I have been involved in the reporting of adult and paediatric post mortems for over 20 years, the majority involving neurological conditions.

I am asked to provide Expert Reports locally, nationally and internationally.

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

Senior Registrar, Neuropathology, Royal Victoria Hospital, Royal Group Hospitals Trust.

**Membership of Advisory Panels and Committees:**
*[Identify by date and title all of those between October 1996-August 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: | |
|---|---|---|
| 096-006-032 | 25th April 2006 | Deposition to the Coroner. |
| WS-224/1 | 19th December 2011 | Witness Statement to the Inquiry. |
| WS-224/2 | 23rd December2011 | Witness Statement to the Inquiry. |
| WS-224/3 | 16th May 2012 | Witness 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 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 Confidential Enquiry into Perioperative deaths?***

*I am aware of this document but cannot say when I became aware of it.*

(2) ***Do you agree with the Coroner's verdict and findings in the case of Claire Roberts (Ref: 091-002-002)?***

*I copy my deposition to the Coroner at the time. It is beyond my expertise to comment on various aspects of the diagnosis in this case. As a Neuropathologist I must confine my answers to Neuropathological issues in context with the clinical findings.*

*The Deposition of Dr Brian Herron (096-006) 25th April 2006:-*

*"I found cerebral oedema which is the end stage of many diseases. I have been aware of the low sodium (hyponatraemia) but this may be caused by other diseases. There was mild inflammation of the brain. I did not find any virus to cause this though this does not exclude a virus. A pathologist cannot exclude epilepsy. I was not thinking of fluid management but SIADH. The main pathological finding was cerebral oedema with a little inflammation in the brain. In a typical case of encephalitis the degree of inflammation is more severe.*
*In response to Mr McCrea I weighed the brain. It was heavier than normal, but there had been abnormal development of the brain. 1300g would have been expected – Claire's was 1606g. This is higher than I would have expected.*
*I cannot recall what medical records I had available at the time. I knew of the low sodium level, the query about SIADH secretion and the respiratory arrest.*
*In addition there was some brain inflammation, possibly a viral infection. This could have resulted from a gastrointestinal infection.*
*I would have expected an infection to be the underlying cause. A metabolic cause cannot be excluded. It is difficult to say what part, if any her epilepsy played on her death."*

*As I am a neuropathologist I answered questions related to neuropathology. My deposition indicated that a number of factors could be considered relevant.*

*At no stage did I mention acute fulminant encephalitis as a diagnosis. In fact, I indicated that the*

*inflammation in the brain was "mild" and "a little" and that in a typical of a case of encephalitis the inflammation would be more severe. I thought an infection might be an underlying factor in her presentation. This seems reasonable given her presentation with gastrointestinal symptoms.*
*In a child with a predisposition to seizures, it may be that it only takes a much more subtle cerebral upset to cause neurological deterioration than in the case of a child with no previous history, a point Dr Squier also makes. In this case any amount of inflammation, no matter how little should be mentioned. (236-004-014) (68). Very rapidly progressive infectious encephalitis may cause death with little change in the brain. It is likely that Claire would have been predisposed to suffer from seizures early in any infectious or pyrexial illness due to her previous history and the hippocampal damage.*
*I did not exclude seizures being a factor in her illness.*
*I did not exclude a metabolic factor in her illness. (This would include issues relating to hyponatraemia).*

(3) ***In October 1996 were you aware of the Arieff et al paper (BMJ 1992, Ref: 011-011-074)?***

*I cannot recall if I was aware of this paper in 1996.*

(4) ***In October 1996, what knowledge did you have of the case of Adam Strain?***

*I did not know of the case of Adam Strain in 1996.*

(5) ***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?***

*I have seen no record of the timing for this.*

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

*I have seen no record of the timing for this.*

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

*I have seen no record of the date this was drafted.*

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

*As this case was in 1996 I have no specific recollection of which documents were received and whether additional documents were sought.*

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

*As this case was in 1996 I have no recollection of the verbal communications and additional information received.*

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

*I am aware that no signed copy of the final report has been found. I find this unusual as it has always been the practice that a report is not sent before it is signed. It is possible that a*

*signed covering letter was sent with the document and this has not been filed.*

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

*I am aware that no signed copy of the final report has been found. I find this unusual as it has always been the practice that a report is not sent before it is signed. It is possible that a signed covering letter was sent with the document and this has not been filed.*

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

*There is evidence to suggest that the case was prepared for a Neuroscience Grand Round (NSU) meeting, but there is no record of the meetings from 1997 in the department files. 35 mm slides were prepared by Dr Mirakhur and there is a record that material was required urgently for NSU. (**090-054-178**)*

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

*I assume that this means the post mortem copy was sent on the date stated.*

(7) ***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 answer for the procedures in 1996.*

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

*As this was in 1996 I cannot recall if any clarification or further information was sought from the requesting doctor.*

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

*As this was in 1996/1997 I do not know whether any other opinion was sought.*

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

*As this was in 1996/1997 I do not recall if I did seek or if I was supplied with a copy of this.*

(11) ***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?*

*As this was in 1996/1997 I cannot state whether I did or did not cross-check the medical notes, clinical history or previous formulation of the diagnosis.*

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

*Because this occurred in 1996 I have no recollection if I was provided with such an explanation.*

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

*Because this occurred in 1996 I have no recollection if I sought an explanation as to why this was an Autopsy restricted to "brain only".*

**(14)** ***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.***

*This is better answered by the recipients of the autopsy report.*

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

*A brain only autopsy is done to assist in coming to overall conclusions in a case. It is only with full integration of all the information including clinical, laboratory, radiological and pathological information that a cause of death is obtained. A Pathologist therefore is only one part of this process and it may is inappropriate for the Pathologist to indicate a cause of death on a brain only post mortem. I report approximately one hundred brain only post mortems a year and would rarely indicate a cause of death.*

*In many ways this can be compared to a radiological examination such as an MRI x-ray. In this case the neuroradiologist is provided with consent for an examination and a clinical history. The radiologist conducts the radiological examination along with his/her technical and scientific staff. The radiological report describes the relevant radiological findings e.g. cerebral oedema. This is used by the referring doctor to integrate with all of the other clinical and laboratory findings to come to a diagnosis.*

**(16)** ***"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;***

*In 1996 post mortems were done in a number of mortuaries throughout Northern Ireland. Over the years the services have integrated, initially in two locations, the Belfast City Hospital and Royal Victoria Hospital. There has also been a change in structure and*

*integration of different Trusts over the years. For the past 12 years all the post mortems from the Belfast area have been performed in the Royal Victoria Hospital Mortuary (apart from Forensic cases).*

*There has been further integration of services. Until the formation of the Belfast Trust, the Belfast City Hospital doctors performed post mortems on Belfast City Hospital, Musgrave Park and Lagan Valley Hospital patients, while the Royal Victoria Hospital covered the Mater, Ulster and Royal Victoria Hospitals along with specialist referral cases (Paediatric and Neuropathology).*

*For the last four years all of the adult post mortems were done or supervised by myself or Dr Mirakhur and for the last two years by myself alone.*

*I now run the service for all adult post mortems and for all Neuropathology post mortems in the Belfast Trust.*

*I personally write or edit all adult and neuropathology post mortem reports from the Trust. I am responsible for, and my name is present, on all reports.*

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

*See (a) above. The process of integration was in a number of stages.*

(17) ***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?***

*A brain only autopsy is done to assist in coming to overall conclusions in a case. It is only with full integration of all the information including clinical, laboratory, radiological and pathological information that a cause of death is obtained. A Pathologist therefore is only one part of this process and it may is inappropriate for the Pathologist to indicate a cause of death on a brain only post mortem. I report approximately one hundred brain only post mortems a year and would rarely indicate a cause of death.*

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(18) ***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?***

*As indicated above, as a Neuropathologist I can only comment on Neuropathological findings and there are other clinical and radiological issues that need to be considered in coming to a cause of death. Many of these are outside my expertise. As indicated above, a metabolic cause of death was not excluded as a factor (this includes the diagnosis of hyponatraemia).*

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

*I had often treated hyponatraemia as a junior doctor before 1996.*

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

*The Anatomical Summary is done to provide information either to the Pathologist or the referring clinicians as to the initial macroscopic findings during a post mortem.*

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

*Yes – since February 1996.*

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

*As indicated in previous depositions, given the history provided, there was no reason to refer the case to the Coroner.*

*Please also see paragraph 23 below.*

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

*Regarding reporting a case to the coroner.*

*In 1996 when I was involved in the post mortem of Claire Roberts I was a junior doctor in the Department of Neuropathology. Pathology and in particular Neuropathology was and is, a consultant led specialty. Trainees are closely supervised and all reports subject to scrutiny by the Consultant Neuropathologist. Post mortems would be discussed between the junior doctor and the Consultant at all stages, including before starting a post mortem.*

*There was no reason to refer this case to the Coroner based on the information provided at the time of the post mortem. However, it is unlikely, that if it was felt that a case should be referred to the Coroner, that a junior doctor would have done this. This would almost certainly have been done by the Consultant in charge of the case.*

*This is still the case. As a Consultant I often refer cases that come for non-coroner's consented post mortems, to the Coroner. I often advise requesting clinicians to refer cases to the coroner. I would not expect a doctor of Registrar grade to do this, as it is unlikely that they would have the experience to make these decisions.*

*Regarding reporting a death to the coroner it does indeed say in guidance that a death should be reported if 'the cause of death is not known'.*

*However this is general guidance and of more use to GPs who are unable to request a consented post Mortem or a limited post Mortem.*

*I have always understood that a death certificate should be written as accurately as possible but that the cause of death was recorded on the balance of probabilities. This seems to be the test in*

*Coroner's court anytime I have attended.*

*If a coroners post mortem was required, when the cause of death in a hospital patient was unknown, there would be no need for any consented post mortems.*

*It is frequently the case that the precise cause of death is not known in a hospital patient and there is either a full post mortem (if there is no clue as to where the problem lies), or a limited post mortem if there is some idea of where the problem is.*

*Examples include:*

*A post mortem limited to the abdomen in a man who comes in with abdominal pain and dies rapidly.*

*A post mortem limited to the chest in someone with acute chest pain.*

*A post mortem limited to brain only in someone with e.g. dementia or encephalitis.*

*This is normal and common practice in hospital medicine.*

*<u>Two factors have caused a great deal of difficulty and confusion for the Inquiry Team and myself in understanding the pathology processes in this case.</u>*

*1. The first of these is the naming of the Registrar and not the Consultant in the pathology report. This is especially the case with Claire Roberts where the Consultant Neuropathologist was the doctor responsible for the final report.*

*As a junior doctor in 1996 I would have had little or no part in the departmental policies followed but as Head of post mortem pathology for the Trust, I have since put procedures in place to make sure this not happen again (See paragraph 16).*

*2. The second factor was the lack of complete documentation when cases were retrieved before the Inquest. The usual procedure I follow when requested to attend an inquest, is to ask that all relevant documents relating to the case are made available to me. It is clear that in the case of Claire Roberts that not all documents were made available. It was not possible for me to ask for documents that I did not know existed.*

*Measures have been put in place to ensure that this does not happen in the future. A professional archivist has been appointed to the Trust Department of Pathology (since 2009) who manages the storage of autopsy reports. All documentation relating to any particular case is filed together and retrieving this documentation should ensure that all relevant material is present.*

***In relation to reports prepared by others I have made some comments.***

*With regard to authorship of the post mortem report, both Dr Mirakhur and I played a role.*

*I was responsible for the performance of the post mortem and the macroscopic brain description, both under consultant supervision.*

*There is no evidence that I had any involvement in Claire's case between the brain cut (28/11/1996) and December 2004. There is no evidence that I had any involvement in the interpretation of the histology, the drafting of the post mortem report or in the conclusions made within the report in 1997. (This should not be taken to indicate any disagreement with these processes).*

*In my opinion therefore, documentation that has been supplied to Dr Squier is in areas incorrect. The brain description is mine but there is no evidence that any of the rest of the report is mine. It has been presented to Dr Squier as written by Dr Herron. This point has been made many times to the Inquiry.*

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

*It is important to clarify that the Inquiry Neuropathologists' reports were issued following the establishment of, and in response to the Hyponatraemia Inquiry after 2005 whereas the post mortem and its report were made in 1996/1997 in the original clinical context of epilepsy, seizures, viral encephalitis, cerebral oedema and learning disabilities. It is also important to re-state that the possibility of a metabolic cause for the cerebral oedema was considered possible in the original post mortem report in 1997 and at the Inquest in 2006.*

*I would further like to highlight the following points:*

*Viral encephalitis- The material that Dr Harding and Dr Squier viewed was at least 10 years old when examined. The stains on these glass slides deteriorate significantly in even a short period of time. In fact it seems that Dr Squier made her own slides and therefore did not necessarily see the same material reported in Belfast. Neuropathological changes, especially when mild may be focal in the brain.*

*The two neuropathologists engaged by the Inquiry state that they could not confirm the presence of meningoencephalitis, although Dr Squier described cellular reactions in perivascular spaces and brain parenchyma. She also accepts the presence of inflammatory cells in the 35mm images taken of the brain histology.*

*In relation to Dr Harding's statement (document 235-002): Dr Harding had been asked – whether in your 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 the result of the rapidity of such an infection?*

*Dr Hardings's response was- my experience does not support this contention. 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 of encephalitis would not be evident by three days. I have seen it occurring within 36 hours.*

*An acute fulminant encephalitis was never suggested by Dr Herron or Dr Mirakhur as a diagnosis. (Please see the post mortem report and the Inquest documentation).*

*Dr Squier agrees that the diagnosis of meningo-encephalitis is still possible: (236-004-014) (68). Very rapidly progressive infectious encephalitis may cause death with little change in the brain. It is likely that Claire would have been predisposed to suffer from seizures early in any infectious or pyrexial illness due to her previous history and the hippocampal damage.*

*(2360004-014)(74) Q. Is it possible that if Claire's illness and death was precipitated by meningo-encephalitis, there might be no evidence thereof in Claire's brain?*
*This is possible, particularly if the disease is rapidly progressive.*

*<u>Cerebral oedema</u>:*

*With regard to Dr Squier's point about the methods of assessing cerebral oedema (deposition 236-00421).*

*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- 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.*

*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 brain and by microscopic examination of the brain. Dr Squier stated- Examination of the fixed brain is also helpful in determining the degree of swelling. I believe microscopy is less helpful. 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 R Hausmann, 'Values for Morphological Parameters for Grading of Brain Swelling.' His paper involved 42 individuals all over the age of 15 and none with a previous neurological disorder. He examined only one histology parameter in coming to a conclusion and I find this paper of little relevance to my conclusion.*

*Dr Squier at times describes the degree of cerebral oedema as severe 236-003-006(22), but in paragraph (b) page 8 where she sees actual photographs of the brain she describes mild narrowing of the lateral ventricles and no tentorial notching (which would be features of severe oedema).*

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

*Neuronal migration disorder:*

*Dr Harding and Dr Squier in their statements say that there is either no convincing evidence (Dr Harding) or that there is no evidence (Dr Squier) of this.*

*The neuropathologists who have originally made the blocks are in a better position to assess this as they know the anatomical sites in the brain from where the tissue blocks were taken.*

*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. Her footnote comment regarding germinal matrix states 'young people' but does not specify age.*

*I would also like to highlight the following widely differing opinions of the two experts employed by the inquiry: These are just some examples and there are others.*

***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 the 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 (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. It also contradicts her impression of severe oedema and cerebellar herniation.*

*This also 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 Squier's statement who describes the changes in the hippocampus at length.*

***Statement of Dr Squier (236-003-001)***

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

*Paragraph 25 (Dr Squier): 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 (Dr Squier): 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 the subarachnoid compartment. Dr Harding describes no major downward shift of the brain or cerebellum. He also states that the sections from the spinal cord are unremarkable.*

*With regard to the consolidated report (310-002-002) I would like to comment on some issues.* ***Reporting of Neuropathological Findings*** *(Dr Mirakhur, Dr Herron).*

*It must be noted that four Consultant Neuropathologists have given opinions in this case.*

*Dr Herron was a Senior Registrar in 1996 and a Consultant of some experience in 2006. He had spent two of the three years before the death of Claire Roberts as a Research Fellow investigating human and animal models of encephalitis. He had been to local, national and international conferences on these topics and presented and published many research papers.*

*Dr Mirakhur was an established Consultant in a Department that was world famous for its work on neurovirology and encephalitis research.*

*With regard to the opinions of Dr Herron and Dr Mirakhur, it must be noted that the post mortem report did not at any stage indicate there was an acute encephalitis. It certainly did not diagnose acute fulminant encephalitis. The post mortem report considered various pathological processes including mild subacute encephalitis and considered a metabolic cause (this would include inappropriate ADH secretion and other causes of hyponatraemia) could not be excluded.*

*In addition, at the Inquest Dr Herron indicated that there was only a little inflammation of the brain and the inflammation in encephalitis was typically more severe. He also considered other processes possible, including metabolic diseases and he could not exclude seizures being a factor in Claire's death.*

*As mentioned in previous depositions, the role of the neuropathologist is to describe pathological processes and not specific diagnoses. There are issues that are outside the expertise of the pathologist. These include clinical, radiological and other laboratory*

*investigations. It is only when all of these are integrated that a conclusion can be made in an individual case.*

*With regard to the conclusions of others:*

*As mentioned previously Dr Squier and Dr Harding are fundamentally opposed with each other in many critical areas of neuropathological interpretation that their opinions are in conflict with the expert clinicians interpretation of the clinical picture and the laboratory findings.*

*CSF was taken at the time of post mortem which supports an inflammatory condition of the meninges and this is specifically addressed by Dr Dewi Evans.*

*Dr Harding offers no explanation for the child's encephalopathic presentation. It is unlikely to be due to the sodium level at the time of admission:*

*3.1 "This was reported at midnight as 132, just below the lower limit of the normal range (135). Dr Scott-Jupp points out that such a level is common in paediatric practice and does not, in itself, require a management change."*

*Many of the experts conclude that an acute viral infection/encephalitis is a reasonable clinical diagnosis in this case:*

*(15.2) The clinical presentation is consistent with an acute viral infection. The fact that no laboratory confirmation or undisputed neuropathological evidence is available in no way rules out this possible precipitating cause. Professor Neville regards it as a reasonable suggestion.*

*(15.4) Encephalitis/Meningoencephalitis – All experts agree it was reasonable to assume this as a provisional diagnosis on admission.*

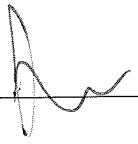
*(15.4.1) Professor Cartwright considers the clinical presentation, illness progression and blood and CSF consistent with an acute and fulminating encephalitis. Some support for this view came from an interpretation of post mortem CSF findings by Dr Dewi Evans, an expert for the PSNI. Dr Mirakhur/and/or Dr Herron stated that they had seen histological evidence of a subacute low grade encephalitis, but this is gainsaid on review of the brain tissues by Professor Harding and Dr Squier. Furthermore in response to a question from Professor Cartwright, Professor Harding states that an acute and fulminating encephalitis causing cerebral oedema, coning and death in the space of three days cannot occur in the absence of Neuropathological changes. (Acute fulminating encephalitis was Professor Cartwright's term and was never used by Dr Mirakhur or Dr Herron).*

*In previous reports Dr Harding has never mentioned coning and in fact this appears to contradict his interpretation of the findings.*

*(15.4.2) Professor Cartwright notes that he cannot reconcile the opinions of Professor Harding and Dr Dewi Evans, but in view particularly of the CSF findings he maintains his opinion that encephalitis was likely.*

*(15.4.3) Dr Scott-Jupp considers it plausible that the presenting illness was viral encephalitis.*

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

**Signed:** 

**Dated:** 14/9/12