Witness Statement Ref. No. 111/3

NAME OF CHILD: Adam Strain

Name: C S McKinstry

Title: Dr

Present position and institution: Consultant Neuroradiologist, Royal Group of Hospitals

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

Membership of Advisory Panels and Committees:
[Identify by date and title all of those between January 1995-December 2004]

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

111/1 - 5/4/2011 Inquiry Witness Statement
111/2 - 3/1/2012 Inquiry Witness Statement

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

Ref:	Date:	

Other points you wish to make including additions to any previous Statements, Depositions and o Reports

[Please attach additional sheets if more space is required]

I have been Consultant Neuroradiologist at the Royal Group of Hospitals since 1987, having trained in Radiology in Belfast, and in Neuroradiology in Newcastle upon Tyne. I was honorary research fellow in MRI at the Hammersmith Hospital, London, 1985-86 and in 1996 I was awarded a travelling scholarship in Paediatric Neuroradiology by the Hospital for Sick Children, Toronto.

I supervised and interpreted the CT scan performed on Adam Strain in the Department of Neuroradiology at the Royal Victoria Hospital, on 27th November 1995 (please see my previous Witness Statements to the Inquiry), and also the previous CT scan of brain performed on 7th July 1995. For the purposes of this report, I have reviewed these scans again in the context of the Expert Report provided by Professor Fenella Kirkham and the response to that report by Dr Waney Squier.

The request form for the CT scan of brain states "Renal transplant. At end of procedure, pupils fixed and dilated. ?Bleed. ?Brain oedema." The CT scan shows generalised brain swelling with compression of the ventricular system and effacement of the cortical sulci and basal cisterns, consistent with generalised brain oedema, ie swelling of the brain.

Prof Kirkham has suggested that the cause of Adam's previous neurological symptoms may have been cerebral venous sinus thrombosis (CVST), and that the fatal brain swelling which occurred following his renal transplant was due to acute on chronic venous sinus thrombosis, in combination with PRES (posterior reversible encephalopathy syndrome).

Regarding the possibility of CVST, the CT scan with intravenous contrast performed on 7th July 1995 shows no abnormality to suggest this diagnosis. The venous sinuses are well demonstrated and appear normal. However, the scans have not continued to the skull base and therefore the jugular bulbs have not been shown. Signs of CVST on CT scanning include filling defects in the sinus following iv contrast, cortical haemorrhagic infarction and brain swelling, none of which are present on this scan. However the absence of these signs would not exclude the diagnosis, in the correct clinical context.

I have again reviewed the scan performed on 27th November 1995. This shows generalised brain swelling, without specific involvement of one part of the brain. This appearance alone is non-specific and could be ascribed to a number of toxic, metabolic and vascular disorders. Acute CVST could cause brain swelling and would not be excluded by this scan. Regarding PRES, the typical appearance is of cerebral oedema causing low density in the white matter of the parietal and occipital lobes, with associated brain swelling. However, generalised oedema and swelling may occur and a diagnosis of PRES could not be excluded. MRI is a much more sensitive method of delineating the changes associated with PRES, particularly in the acute phase (1).

In PRES, the parts of the brain typically affected are the occipital and parietal lobes, ie the "posterior" parts of the cerebral hemispheres. Incomplete or asymmetrical involvement of the parietal and occipital lobes, and of other parts of the brain including the posterior fossa structures, is not uncommon. However, some involvement of the parietal and occipital lobes is seen in 98% of cases (1,2). Changes of PRES confined to the brainstem have been rarely reported (2,3).

In support of this diagnosis, Prof Kirkham quotes Dr Anslow's report on the CT scan performed on 27th November 1995, which describes "changes particularly severe in the posterior fossa" (para 27 of h

report). The posterior fossa structures of the brain are the brainstem and cerebellum, which are less frequently involved in PRES, and usually in combination with changes in the occipital and parietal lobes.

In paragraph 50, Prof Kirkham states that PRES led to the "rapid development of mainly posterior cerebral oedema". It is not clear whether this refers to the posterior fossa structures to which reference had already been made in Dr Anslow's report, or the typical "posterior" distribution in the cerebral hemispheres referred to above. In paragraph 55, she states that "the rapid development of posterior cerebral oedema will have pushed the cerebellum down to the foramen magnum", and again the exact meaning of "posterior" is unclear.

In her response to the query "can she exclude PRES?" Dr Squier states that "the hindbrain has been relatively well sampled" and that this did not show evidence of oedema. The hindbrain refers to the brainstem and cerebellum, but it is not clear whether the other parts of the brain more typically involved in PRES have also been examined.

In conclusion, therefore, the CT scan performed on 7/7/95 shows no evidence of cerebral venous sinus thrombosis, although the jugular bulbs have not been included in the scan and this diagnosis cannot be completely excluded.

The CT scan on 27/11/95 shows non-specific generalised brain swelling and there are no particular changes to suggest a diagnosis of PRES, although this diagnosis cannot be excluded. The interpretation that this scan shows brain swelling mainly affecting the posterior fossa structures, and that this therefore supports a diagnosis of PRES, appears out of keeping with the typical distribution of the cerebral changes seen in this condition. It is not clear whether post mortem examination of the brain to exclude PRES has included the cerebral hemispheres as well as the posterior fossa structures.

References:

1. Bartynski WS & Boardman JF. Distinct imaging patterns and lesion distribution in posterior reversible leucoencephalopathy syndrome. American Journal of Neuroradiology 2007:28:1320-7.

2. McKinney AM et al. Posterior reversible encephalopathy syndrome: incidence of atypical regions of involvement and imaging findings. American Journal of Roentgenology 2007:189(4):904-912.

3. McCarron MO & McKinstry CS. Vanishing brainstem oedema. Journal of Stroke and Cerebrovascular disease 2008:17(3):156-157.

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

Signed:  **Dated:** 15/3/2012