

STATEMENT OF WITNESS

(CJ.Act, 1967, s.9 MC Act 1980, ss.5A(3a) and 5B MC Rules 1981, r.70)

Statement of: Professor R A Risdon**Age of Witness:** (if over 18 enter 'over 18'): Over 18**Occupation of Witness:** Emeritus Professor, Consultant Paediatric Pathologist**Address:** Department of Histopathology
Great Ormond Street Hospital for Children NHS Trust
London WC1N 3JH**Telephone:** [REDACTED]**Fax:** [REDACTED]

This statement, (consisting of 3 pages each signed by me), is true to the best of my knowledge and belief and I make it knowing that if it is tendered in evidence, I shall be liable to prosecution if I have wilfully stated anything which I know to be false or do not believe to be true.

Dated: 2 June, 2006**Signed:** *R A Risdon***Signature witnessed by:** *K. Wray***Adam STRAIN (DOD 28.11.1995)****Introduction**

I am Professor R A RISDON and my qualifications are MD (London), FRCPath, DMJ. I retired from NHS practice as a Consultant Paediatric Pathologist and Head of Department at Great Ormond Street Children's Hospital, in March 2004. Since that time I have practiced as a Paediatric Forensic Pathologist as a Partner in Forensic Pathology Services. I continue to be up to date in CPD and have had an annual appraisal at GOS where I have an Honorary Consultant Contract. I have had considerable experience in both adult and paediatric renal pathology providing a specialist service for Addenbrooke's Hospital Cambridge, The Royal London Hospital and Great Ormond Street during my various appointments to those hospitals over the last 30 years. I have contributed extensively to the literature in renal pathology, in peer reviewed journals and have contributed chapters on paediatric renal subjects in the two major American textbooks on renal pathology.

Over the past 30 years, providing opinions on biopsy specimens from renal transplants has been part of my responsibility.

Clinical History

In November 1995, Adam was aged 4 years. He had congenital obstructive uropathy and had developed chronic renal failure.

AS - PSNI**Signed:** *R A Risdon***Signature witnessed by:** *K. Wray***093-031-081**

On the 26th November 1995 he was admitted for renal transplantation. The insertion of the graft was performed on the 27th November 1995, surgery commencing at approximately 07:00 hours and lasting 4 hours. Adam did not recover from the anaesthetic and, following brain stem tests he was pronounced dead at 09:15 hours the following day (28th November 1995).

A post-mortem examination was conducted the following day on the 29th November 1995 by Dr ARMOUR.

At an Inquest on the 18th June 1996, the medical cause of death was given as 1a Cerebral oedema, due to 1b Dilutional hyponatraemia and impaired cerebral perfusion during a renal transplant operation for chronic renal failure (Congenital obstructive uropathy).

Material Provided

1. A number of tissue samples from the post-mortem examination mounted in paraffin wax. These included sections of the 'native' kidneys as well as a section of the transplant.
2. A letter from the Director of Renal Transplantation at Greater Glasgow NHS.
3. A letter from UK Transplant.
4. The post-mortem report by Dr ARMOUR.
5. A medico-legal report from Professor P J BERRY dated the 23rd March 1996.

Instruction.

The letter of instruction dated the 16th May 2006 from Detective Sergeant William CROSS contained the specific request that I examine the tissue samples provided to indicate to the Police whether it was possible to state with any certainty the time of the infarction of the donor kidney.

Histology

Multiple sections have been cut from the blocks of tissue from the two 'native' kidneys and the transplant kidney. A variety of special stains have been applied to these tissues.

Native Kidneys

Sections of both kidneys show end stage renal disease with extensive tubular atrophy and loss, interstitial fibrosis and chronic inflammation, glomerulosclerosis and a number of cystic structures. There is disproportionate loss of cortical compared with medullary structures. A focus of metaplastic hyaline cartilage is seen and there are so-called 'primitive ducts' in the medullary areas.

The appearances are of end stage renal dysplasia and are entirely consistent with the clinical evolution into chronic renal failure requiring renal transplantation.

Transplant Kidney

Sections show complete coagulative necrosis of the graft. The basic renal architecture is recognisable in 'ghost' form, but the proximal tubular cells are

completely necrotic and lack nuclei. In the glomeruli, some distal tubules and some blood vessels pyknotic nuclear material is still visible.

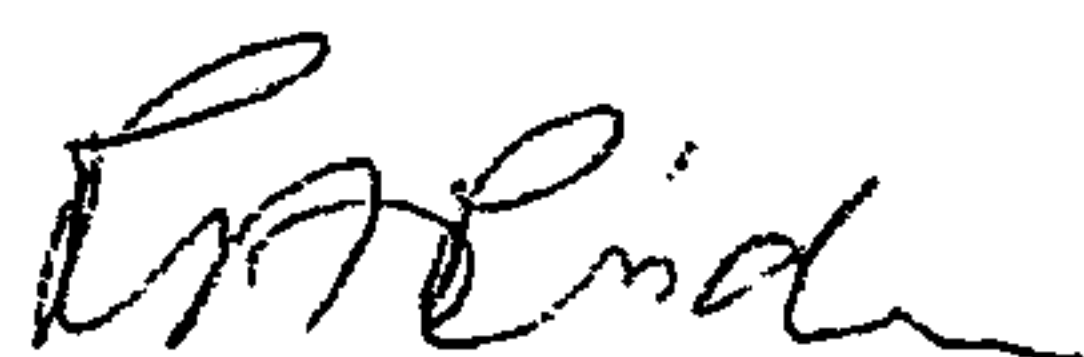
Comments.

1. This child survived only 24 hours after the transplant operation and the post-mortem was performed the day after death.
2. In my opinion the changes seen in the transplant kidney are more advanced than would be expected after only 24 hours of non-perfusion.
3. In my opinion the transplanted kidney must have suffered significant ischaemic damage prior to its insertion for this degree of ischaemic damage to be apparent at post-mortem.
4. This opinion is supported by the fact that the other kidney from the same donor failed to function when transplanted to a different patient in Glasgow. This would suggest that both the kidneys from this donor had suffered significant ischaemic damage before transplantation.

AS - PSNI

093-031-083

Signed:



Signature witnessed by:

