

STATEMENT OF WITNESS

STATEMENT OF: DR MARY O'CONNOR

Name

Rank

AGE OF WITNESS (If over 18 enter "over 18"): OVER 18

*To be completed
when the statement
has been written*

I declare that this statement consisting of 4 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 at a preliminary enquiry or at the trial of any person, I shall be liable to prosecution if I have wilfully stated in it anything which I know to be false or do not believe to be true.

Dated this 12 day of APRIL 2006

William R Cross

Mary G O'Connor

*SIGNATURE OF MEMBER by whom
statement was recorded or received*

SIGNATURE OF WITNESS

WILLIAM R CROSS, D/SERGEANT

PRINT NAME IN CAPS

At the time of Adam's renal transplant on 27th November 1995 I was employed as a Consultant Paediatric Nephrologist in the Royal Belfast Hospital for Sick Children. I had taken up this post on 1st November 1995. Dr Savage and I had named responsibility for individual patients but provided cross cover for each other at nights, weekends and at times of holiday and emergency when the named Consultant was not available. Adam was a patient of Dr Savage's and preparation for his renal transplant had been made the previous evening on 26th November 1995. The renal transplant surgery was in progress when I arrived in the hospital on the morning of 27th November 2005. During a renal transplant the Consultant Nephrologist prescribes the immunosuppressive medication and is present to prescribe the immediate post-operative drugs and fluid regime. At some time during the morning of 27th November 1995 Dr Savage had commitments in the School of Medicine, Queen's University in his role as Senior Lecturer and hence I made myself available to attend to Adam's post-operative care. I was present in theatre

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towards the end of the operation (I do not recall precise timings). I was aware that at the end of surgery Dr Taylor discovered Adam to have fixed dilated pupils. My role in the Intensive Care Unit immediately following surgery was to make a clinical assessment of his condition, to request any necessary investigations and to prescribe any necessary fluids and drugs. I also contacted Dr Savage to inform him of the situation and he returned immediately from the university. My statement is based on my written notes as these events occurred almost 10 years ago. My notes confirm that I examined Adam at 12.05 pm on 27th November 1995. I have recorded that he did not breathe following surgery and that his pupils were observed to be fixed and dilated. There was no history obtained of any cardiovascular instability during the operation. Peri-operative blood pressure had been 118/78 mmHg and peri-operative CVP had risen to 30 mmHg. My note written at that time states that the CVP was known to be 17 mmHg at the start of procedure and in view of this high initial CVP the accuracy of recordings was uncertain. I had been informed that there had been difficulty inserting a central venous line at the start of the procedure and I made a presumption that this difficulty was due to previous multiple venous access. I assumed that he may have had one of his external jugular veins tied off, as this was common practice at the time of insertion of central lines in RBHSC in 1995. I had not read previous operation notes at this time to see if this was confirmed. However I felt it likely that the CVP measurement may have been unreliable. I summarised the fluid balance as recorded in the anaesthetic chart as I would do in all cases of renal transplantation. I have recorded that there was 911 ml of blood in the suction bottle during theatre presumed to be blood loss and that the total input during theatre consisted of 1 litre of HPPF, 500 mls of

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Hartmann's solution, 1500 mls of 0.18% saline 4% destrose solution and 250 mls of packed red cells. On examination I noted that Adam's pupils measured 7 mm and both were equal and fixed. I saw haemorrhages on both his right and left fundi and the disc margins were indistinct. I observed him to be puffy and recorded that the CVP measurement at the time of my examination was now 11 mmHg. I recorded that there had been 49 mls of output from his native kidneys from the time of transplantation and that there had been no recorded output to date from the transplanted kidney. It is possible to differentiate the outputs as there was a feeding tube inserted into the transplant ureter and an ordinary bladder catheter inserted separately into his bladder. My notes state that I queried two causes of his neurological abnormalities. I questioned in my notes whether he had "coned" due to cerebral oedema and noted that he had had high fluid intake and possible abnormal cerebral venous drainage. However it is normal for there to be a very positive fluid balance at the end of a renal transplantation as a high central venous pressure is required in order to perfuse the transplanted kidney adequately. I also queried whether a problem with the epidural may have led to cerebral problem. My plan of action was to give Mannitol in an effort to decrease any possible cerebral oedema and I agreed with Dr Taylor's management of hyperventilation. I restricted his fluid intake. An urgent urea and electrolyte profile, CT scan of brain and neurology opinion were sought. Routine anti-rejection therapy was also prescribed by myself.

My subsequent notes of approximately 1 hour 15 minutes later state that a chest x-ray had now been obtained showing that the central venous line was seen going through his neck vessels rather than downward toward the heart and I queried this may have caused some obstruction of venous return. I have also recorded that the

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post-operative serum sodium was 119 mmol/L and I queried that this was due to haemodilution. An emergency CT scan that afternoon confirmed generalised cerebral swelling. My analysis during the post-operative period was that the cerebral oedema was likely to be related to the drop in serum sodium from a pre-operative level of 139 mmol/L on 26th November 1995 to a post-operative level of 119 mmol/L at 1.00 pm on 27th November 1995. I assumed that his normal polyuric state complicated his fluid management and that his possible cerebral venous drainage may have made him more susceptible to cerebral oedema. My main role in the care of Adam was between 12.05 pm and approximately 1.00 pm on 27th November 1995 when Dr Savage took over his management. In response to specific questions from D/Sergeant Cross I can state: It is my normal practice during transplant surgery, if I am able to be present, to relay to parents information from theatre informing them as to the stage of the operation and generally how matters are progressing. I cannot recall a specific conversation with the mother of Adam Strain but it would have been my normal practice to have had such. After the operation I discussed the CVP figures with Dr Taylor and noted the initial reading of 17 mmHg was high and the later reading of 30 mmHg was very high but the conclusion was that these readings may have been unreliable. A subsequent chest x-ray confirmed the line to have been in the wrong position to measure CVP. I have recorded that the kidney was "bluish" at the end of theatre. This would have made me anxious to observe the urine output over the next days, however, in my experience kidneys which have appeared bluish at the end of theatre have later proved to be satisfactory after sometimes as much as 3 weeks, in that the recipient did not need to go back on dialysis.

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