STATEMENT OF WITNESS

STATEMENT OF: ELAINE HICKS, REGISTERED MEDICAL PRACTITIONER
Name *Rank*

AGE OF WITNESS (if over 21 enter "over 21"): OVER 21

NOT SIGNED IN POLICE OFFICER'S PRESENCE

TO BE COMPLETED WHEN THE STATEMENT HAS BEEN WRITTEN

I declare that this statement consisting of 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 13th day of February 2004

Elaine M. Hicks

SIGNATURE OF MEMBER by whom statement was recorded or received

SIGNATURE OF WITNESS

Re: Conor Mitchell (deceased) DOB: 12/10/87

This report has been prepared at the request of Her Majesty's Coroner.

I am a registered Medical Practitioner working as a Consultant Paediatric Neurologist, which post I have held since November 1983. I hold the following qualifications, MB BCh, Queen's University of Belfast, MA (Medical Ethics & Law) Queen's University of Belfast, Fellow of the Royal College of Physicians of London, Fellow of the Royal College of Paediatrics and Child Health, Diploma in Child Health (London).

This report is prepared with the assistance of the case-notes of the Royal Belfast Hospital for Sick Children, UNCH 334505.

I first met Conor Mitchell on 21 April 1988 when he was an inpatient under the care of Mr. VE Boston, Consultant Paediatric Surgeon. He had been referred by Dr. Paul Jackson, then working as a Consultant Paediatrician in Craigavon Area Hospital for investigation of a large head. Mr. Boston had asked me to see Conor because of concerns about neuro-developmental status. My note is dated 21 April 1988 and timed at 16.30 and I have noted that mother and grandmother were present. In this note, I detailed Conor's history including birth history and early life and the fact that his mother had been concerned about unsteadiness of his head control. At this stage, Conor was aged 6 months. It was thought that his head had always been large and had been measured by their local health visitor. Further questioning revealed that they felt he had adequate vision, but that he did not fix and follow well and there was concern that he could see better to the left than to the right. They gave a history that he would be interested and play with toys, his head control was a little unsteady, but improving, and that he could roll over and liked to stand rather than sit, although he needed held as his balance was poor. He was a healthy boy who had not had any significant medical illnesses.

I noted on examination that he was a robust boy, very active, who lay with head retraction and had an occipital frontal head circumference of 48cm. The anterior fontanelle was felt to be normal. On his growth chart, his head was noted to be above the 97th centile for age, and with previous head measurements it was possible to see that these had more or less followed the 97th centile line from birth, although there had

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been a slight divergence from the line between the ages of 4 and 6 months. There was a degree of scaphiocephaly and an excessive startle response. I thought vision was questionable, with possible menace, pupils poorly reactive to light, discs I felt normal, although I could not see his maculae well. He had conjugate eye movements, with no fix or follow, no nystagmus and would tend to have a gaze preference to the left. He had good facial expression, and hearing I thought was clinically normal. In the limbs, he had markedly increased tone at all levels, with fisted hands and a pronounced asymmetric tonic neck reflex to right and left side, with marked extensor thrust. Deep tendon reflexes were all very brisk to the level of being pathological, and plantar responses were extensor. I did not see him roll over and he made no definite reach for a toy. He had no balance or protective reflexes.

I have recorded my impression as that of spastic tetraparesis, with poor visual function and I wondered whether there was an element of hydrocephalus present and questioned the cause of this. The cerebral ultrasound report indicated large lateral and third ventricles. I have then made a note of my recommendations for further investigations, which included a CAT scan of brain, EEG, blood for TORCH titres (congenital infection screen), biochemical screening, chromosomes, urine for amino acids and I recommended ophthalmology consultation and a physiotherapy assessment. I have recorded that at that time I explained to mother and grandmother my concerns that this boy had a neuro-developmental problem. I have recorded that his mother immediately became upset and I have recorded a request for support and counselling input from social services and also contact with health visitor to obtain the serial head circumference measurements. Investigations revealed that biochemical screening was essentially normal for age, urinary amino acids normal, and full blood picture within normal limits for age. TORCH screening revealed herpes simplex, cytomegalovirus and rubella titres negative, there is no toxoplasma result in the chart that I can identify (this is unlikely to have any significance). Chromosomes are recorded as normal and EEG abnormal because of a poorly formed unresponsive background pattern with epileptiform abnormality in the left posterior region. The CAT scan was reported as showing appearances consistent with bilateral porencephalic cysts, more marked on the left. This was carried on 22 April 1988 and reported by Dr. McKinstry, Consultant Neuroradiologist. His conclusions would indicate that he was not concerned that there was evidence of ongoing raised intracranial pressure but that it was likely there had been an in-utero event.

Further notes have been made by myself, most particularly 22 April following the CAT scan indicating that I conveyed the results again to mother and grandmother. Again, I have recorded that his mother became extremely upset and a note was made that the general practitioner was contacted to inform him of the current situation. Conor was discharged home to be readmitted at a later date for investigations to be completed. In particular we investigated the cardiovascular system, with normal cardiology and echo, and Doppler studies of carotid arteries, which are recorded as normal. I indicated my willingness to review him at my outreach clinic in Craigavon Area Hospital.

Notes of Craigavon evaluations would be filed within the Craigavon Area Hospital notes, which are not available to me. There is, however, correspondence generated by myself on 5 May documenting my involvement to date and a letter from 16 May from

the outreach clinic in Craigavon Area Hospital at which time I discussed management of possible absence seizures with medication. A subsequent letter on 8 August 1988 indicates that he did not attend for review at the clinic and there is an additional letter of 23 January 1989 to Dr. Thompson, GP, reporting a telephone conversation with Conor's grandmother, at which time she explained involvement with alternate therapy programmes and our agreement that he need not come to my clinic for routine review as long as someone in Northern Ireland kept him under surveillance.

My next contact occurred on 10 May 2003. The chart indicates that the note was made at 17.50. On this occasion, I was asked to consult on Conor who had been admitted to the Paediatric Intensive Care Unit by transfer from Craigavon Area Hospital. Conor had had a relatively brief illness, but had markedly deteriorated, hence the transfer and his status at this time. My note indicates that I reviewed the course of his current illness with Conor's mother, from documentation that we had received from Craigavon Area Hospital, from the notes made by staff in R.B.H.S.C after arrival and by talking to the staff. I later talked to his grandmother. The salient points I noted were as follows.

- Conor had been his usual self until approximately 10 days previously.
- He then became unwell, with a reduced appetite, malaise, apparently a pink throat and was seen by his GP who prescribed symptomatic treatment.
- His family thought he improved to some extent until about a week previously when his throat apparently got worse, he began to vomit and again was seen by the GP who felt he had an infection in throat and ears and prescribed Penicillin.
- On this treatment he improved for a day or two then became worse with more marked vomiting and his antibiotic was changed to Amoxicillin.
- However, he did not return to baseline, although the eating improved a little bit, but he continued to vomit.
- He had some "jiggly" movements of his legs, but no overt seizures at this stage. He was felt to be lethargic and his urine was cloudy.
- Three days previously, he had become much worse and was taken to Craigavon Area Hospital where they apparently had considered him dehydrated, recommended intravenous fluids and he was admitted. At that time, I gather that electrolytes showed urea a little raised at 7.8, we had no note of the potassium level, sodium was 138, haemoglobin 13.6, white cell count 19, CRP <5.
- The family expressed concern that the IV site was unsatisfactory as he was having episodes where he would stiffen, in that his head would go back, his arms were flexed, his legs out straight and it was felt that he possible had reduced level of consciousness, and these episodes occurred repeatedly. This led to the IV being removed and later re-sited. It was thought that the site might be painful, thus causing these symptoms.
- A more major event then occurred in which he stopped breathing, his head went back, he became blue and stiff all over, and following this, he was moved to Intensive Care in Craigavon Area Hospital. (My note indicates PICU, but this I feel must be an error of notation as there is no PICU in Craigavon). He was intensively managed here with ventilation and apparently had not breathed since that time. Among other medications he had required adrenalin and he had been given dilute IV fluids because of a raised sodium at 149, although the total volume of fluids was

unclear from our notes and records. In addition, he was treated with IV Acyclovir and Ciproxin.

- A CAT scan had been considered to show sub-arachnoid haemorrhage, plus the long standing changes. He was given IV Phenytoin, but no sedatives. There had been concern that he was in fact brain dead, but subsequent to this, he was transferred to PICU.

In reviewing the past medical history, I have noted that diagnosis of cerebral palsy at 6 months as per my previous account above. He had always had a large head and was always a large baby overall. Although he had suffered from epileptic seizures, his family considered these not to have been a significant problem until the very recent past and had generally manifested as occasional staring attacks only. He was on treatment with Sodium Valproate an anti-epileptic drug. Conor had had intensive input from his family along the lines of alternative therapies. His neuro-development had remained very compromised, although they had felt he had improved to the state that could creep around the house, use his hands better and that he knew the family and could interact. He lived with his mother and her partner in an extension on the grandparents' house.

Examination noted that he appeared tall, with a head circumference of 59cm and scaphiocephaly. I have noted that he appeared thin. He made no spontaneous respiratory effort and no movement at rest. I elicited no response to voice, light touch on face, arms or hands, trunk and thigh. Any touch on his feet caused flexion at hip and knee, and dorsiflexion at the ankle. There was no response to deep pain in the cranial nerve distribution or over the sternum. His pupils were 8mm irregular and unreactive, doll's head movements were negative, fundi showed small, possibly slightly pale discs, vessels were normal, there was no movement of his face, there was no corneal reflex or eyelash reflex, there was no gag and tracheal suction revealed no response. Deep tendon reflexes were possibly present in the right upper limb and at both ankles.

My impression was that of a boy with long standing neurological dysfunction in the form of cerebral palsy and epilepsy with learning difficulties, in whom early cerebral imaging by CAT scan had revealed changes considered to be the result of an antenatal insult, and who had recently suffered severe cerebral and brain stem decompensation. I indicated that it was possible that this represented irreversible damage to the nervous system.

I addition, I have noted that I thought the course of the recent illness was not totally clear, possibly a viral infection, but that I felt he seemed to have had, by history, multiple tonic epileptiform seizures, and then a cardio respiratory arrest, which seems to have been due to acute brain stem compression, possibly cerebral swelling, possibly with an element of hypoxia-ischaemia. I have indicated earlier in my notes that I was uncertain about fluid balance. I have noted that I talked to his grandmother, who felt that Conor had got "a lot of fluid" in Craigavon and that medical input was inadequate. In addition she was adamant that he had had a significant number, probably at least 12 major stiffening episodes, which sounded to me like generalised

tonic seizures. During this time, she felt he had become increasingly puffy and more lethargic prior to the acute and severe deterioration.

In my discussions with the family at that time, I indicated to them that I felt it unlikely he would recover from this illness.

Unfortunately, this is how things turned out and I documented clinical visits on 11 May at 13.30 and 12 May at 12.00. On the latter occasion, my notes indicated that there were some atypical movements of abdominal muscles, which I was uncertain whether they were of cerebral or reflex origin. That time, I felt unable to confirm absolute brain and brain stem death. Subsequent to this, the movements became more clearly those of a reflex nature and were not of respiratory effort. As he gradually deteriorated and with extensive communication between staff and the family, Conor was allowed to die.