STATEMENT OF WITNESS

STATEMENT OF THOMAS FRANCIS FANNIN

AGE OF WITNESS[If over 21 enter 'over 21'] : OVER 21

I declare that this statement consisting of 2 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 11 day of July 1997

MICHAEL IRWIN *THOMAS FANNIN*
SIGNATURE OF MEMBER *SIGNATURE OF WITNESS*
by whom statement was recorded or received

Re: Robert Hamill (deceased), DOB: 12/09/71

I am a Consultant Neurosurgeon in the Regional Neurosurgical Unit at the Royal Victoria Hospital, Belfast (RVH). Mr Hamill was admitted under my care to the RVH on 27 April 1997. His history was of initial referral to the Craigavon Area Hospital, Surgical Unit, where he was admitted at approximately 2.45 am on 27 April 1997, having apparently been assaulted. We were given to understand that he had been hit with a bottle and put to the ground and immediately rendered unconscious. The referral notes indicated that his Glasgow Coma Scale on admission to Craigavon was 3 and that he had some decerebrate posturing of the left arm and a flicker of movement in the left leg. His pupils were equal and reacting. His blood alcohol was 221 mg%. He was intubated and ventilated in the Craigavon Area Hospital and taken to the RVH as the scanner in Craigavon was not working. A CT scan was performed. This showed soft tissue swelling in the scalp over the left temporo-parietal region and also over the right temporo-occipital region. No obvious intracranial abnormality was seen apart from a mild degree of frontal lobe atrophy. X-ray of the cervical spine and chest x-ray were satisfactory. He was treated by means of mechanical

ventilation and chemical paralysis. These paralysing drugs were stopped at approximately 10.00 am on 27 April 1997. He was extremely restless at that time and there appeared to be purposeful movements of the limbs. On occasions however his movements were spastic and decerebrate. He was sweating profusely. There were fluctuations in his pulse and blood pressure. It was considered that he was going through a stage of extreme irritation following closed head injury. His endotracheal tube was removed and it was believed that his agitation and abnormal limb movements should be treated by mild sedation using haloperidol. By 29 April 1997 he was less agitated, the sedation was stopped and he was transferred to the Neurosurgical Unit on that day. A nasal airway was in place. At that time his oxygen saturation was greater than 95%. Because of the recurrence of spasms haloperidol was reintroduced at a dose of 50 mg IV, twice daily. A further CT scan was performed on 30 April 1997 and this was unchanged being essentially normal. By 1 May 1997 it was apparent that Mr Hamill was not spontaneously opening his eyes. He was extremely agitated when disturbed and was therefore commenced on chlorpromazine. Some flexion movements of the right upper limb occurred at times but on other occasions he had quite marked extensor spasms in the left side with on occasions opisthotonos. His blood gases were satisfactory as were his oxygen saturations. I asked Dr J P McCann, Consultant Physician, in the Rehabilitation Service to see him, and his opinion was that Mr Hamill was showing signs of cerebral irritation. It was generally felt by most observers, myself included, that many of this man's problems were related to hypoxia

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presumably at the scene of the accident and perhaps compounded by a relatively high blood alcohol at the time of injury. Because of a pyrexia of up to 40 degrees Celsuis, blood cultures, urine analysis, sputum and a chest x-ray were carried out. Initially, it had been considered that this was a relatively minor brain injury but the possibility of secondary insult such as hypoxia and the effects of high blood alcohol had also been taken into account. There was a sudden, dramatic deterioration in his condition on the afternoon of 8 May 1997 and he ultimately died later that afternoon. Several possibilities were considered including the possibility of a pulmonary embolus from a deep venous thrombosis, a septicemic episode or possibly even a malignant neuroleptic syndrome which can occur occasionally and secondary to the use of neuroleptic drugs of various types. The death was reported to the Coroner.

SIGNATURE OF WITNESS THOMAS FANNIN

CHECKED AND CERTIFIED A TRUE COPY OF THE ORIGINAL SIGNED : Derek Faulkner QSO