MR N V TODD MD FRCS
CONSULTANT NEUROSURGEON & SPINAL SURGEON

[redacted]

MEDICAL REPORT

Name	**Robert Hammil**
Date of Birth	**12 September 1971**
Type of Report	**Causation**
Our Ref	**NVT/SD/30585**
Your Ref	
Prepared at the request of	**Ms P Fitzmaurice** **Deputy Solicitor** **PO Box 50156** **London** **SW1E 6WX**
Prepared by	**Mr N V Todd MD FRCS** **Consultant Neurosurgeon and Spinal Surgeon** [redacted]
Date of Report	**21 November 2005**

SUMMARY

1 Mr Hamill died as a consequence of a head injury that followed an alleged assault on 27 April 1997.

2 The clinical picture was of a severe diffuse brain injury with coma from the time of injury and persistent coma until Mr Hamill's death.

3 CT scanning ante-mortem and examination of the brain post-mortem demonstrated no evidence of intracranial haematoma, no evidence of brain swelling or brain oedema nor any evidence that Mr Hamill died of a "pressure" death.

4 Histological examination of the brain showed no evidence of hypoxic ischaemic damage in neuronal structures that would be subject to such damage.

5 Histological examination of the brain demonstrated a diffuse axonal injury (a shearing injury to white matter tracts) widely distributed throughout the brain; in some areas that damage was thought by Professor Crane to be extensive.

6 Such a diffuse axonal injury, widespread and in parts extensive, would typically be associated with high velocity road traffic accidents where there are severe and abrupt acceleration/deceleration forces.

7 Such a pattern of injury does not usually follow a simple assault where a punch leads to the patient being knocked out briefly.

8 In my opinion Mr Hamill's head/brain was subjected to such significant forces that it caused the sort of severe axonal injury that is normally associated with high velocity road traffic accidents.

9 It is outwith my experience to comment upon whether such an injury could be caused by multiple blows to the head with fists, with striking the head one of more times with a weapon, with kicking the head one or more times as the unconscious victim lies on the ground, or any other potential mechanism of injury. Suffice to say that the levels of force applied must have been very much greater than those that occur in the average "Saturday night punch up" and more akin to the damage that is done in high speed motor vehicle accidents.

INTRODUCTION

10 This medical report was prepared for the Robert Hamill Public Enquiry at the request of Patricia Fitzmaurice (Deputy Solicitor).

11 In preparing this report I had available a bundle of medical records which included the following:

12 Copies of medical records from the Royal Hospital, Belfast.

13 A series of photographs taken post-mortem.

14 A post-mortem report.

15 Letters from Mr Fannin (Consultant Neurosurgeon) dated 14.05.97 and 07.07.97.

16 A letter from Mr Patel (Neurosurgical Registrar) dated 10.07.97.

17 A statement from Dr Lavery (Consultant in Intensive Care), undated.

18 The bundle of medical records has been paginated twice. I will refer to the first part of the bundle as I.page number and the second part as II.page number.

19 This report addresses issues in relation to causation.

GENERAL HISTORY

60 Mr Hamill was born on 12 September 1971. At the time of the alleged assault, 27.04.97, Mr Hamill was 25 years of age.

NARRATIVE HISTORY

61 Mr Hamill was taken to the Accident and Emergency Department at Craigavon Hospital on 27.04.97. Triage was carried out at 0205. The following note was made:

> *"? Involved in fracas in Portadown. ? hit on head by bottle. Unconscious. Breathing noisily. Wound to back of head.* (One word illegible) *anaesthetist called. 02. Nasal tube. Suction. 8.5 ET tube by an...".* **Attached to Letter 21.03.05**

62 The blood pressure was 160/103. The pulse was 100 beats per minute. The pupils were symmetrical 3mm in diameter and reactive to light. The Glasgow Coma Score was not recorded.

63 There is a subsequent medical entry which reads as follows:

> *"assaulted ? left side head with bottle. Attacked by nine people.*
>
> *O/E pupil size 3-reacting. Pulse 100. BP 160/103. Unresponsive. Airway: breathing with upper airway obstruction will not open jaw to introduce Gudel airway. Breathing – 75% bagged.* (One word illegible)*: stable. Pupils equal and reacting. Plantars ↓↓. Left knee reflex decreased. No open injury. Abdomen soft. No limb function. GCS E: 1/4 V: 1/5 M: moved left foot to pain 3/6 – 5/15.*
>
> *Cervical collar. Anaesthetist → intubated. Brown venflon. Catheter".* **Attached to Letter 21.03.05**

64 I have seen a head injury chart from Craigavon Area Hospital. Prior to intubation Mr Hamill was said to have no eye opening, no vocal response, no motor response with 3mm reactive pupils. **I.129**

65 Mr Hamill was transferred to the Department of Neurosurgery at the Royal Victoria Hospital, Belfast (RVH). He was admitted under the care of Mr Fannin (Consultant Neurosurgeon). The clerking reads as follows:

> *"25 year old man referred by surgical unit in Craigavon Area Hospital. H/O alcohol intake +. Brought in by ambulance at 2.45am at CAH. Alleged H/O assault. ? hit with a bottle. Was unconscious GCS 4-5/15. Pupils small and reacting. Intubated and ventilated in CAH and transferred to RICU. Vital parametres stable. Pupils normal size reactive. Scalp haematoma. Blood alcohol 221.*
>
> *Clinically to rule out head injury. CT brain normal. Cervical spine – lateral view up to C6 normal, swimmers view C7-T1 junction normal – normal cervical spine".* **I.53**

66 The next note is at 10.40am on 27.04.97 and it reads as follows:

> *"Stopped sedation/relaxants. Beginning to open eyes. Scanty secretions. PH 7.43, PTO_2 3.2, PO_2 ...cardiovascular signs stable. Urinary output good. Await events".* **I.54**

67 I have seen nursing charts for 27 April. Until 10.00am Mr Hamill was recorded as being intubated. By 1400 there was no eye opening, incomprehensible sounds and localising (GCS 7/15). **I.87**

68 By 6.00pm on 27 April Mr Hamill was said to have:

> *"some purposeful movements. Very agitated. Not opening eyes. No speech. Moves all limbs and flexes to pain".* **I.54**

69 On 28.04.97 Mr Hamill initially had no eye opening to pain, incomprehensible sounds and localising. At about 10.00am the motor response dipped to extension and then recovered to localising. **I.87**

70 The medical entry for 28.04.97 reads:

> *"very restless moving all four limbs. Trying to get out of bed. Not conscious. Not obeying commands. Pupils equal and reacting. CT normal...".* **I.55**

71 On 29.04.97 the medical note reads:

> *"less agitated – off sedation. Gases ✓. Apyrexic ✓".* **I.55-56**

72 The nursing chart for 29 April records Mr Hamill to have no eye opening, no verbal response, localising with 3mm reactive pupils. **I.87**

73 On 29.04.97 Mr Hamill was transferred from the intensive care unit to the ward. He was said to be very agitated requiring sedation with midazolam. The Glasgow Coma Score was said to be 7 (E2 V2 M3). The sedative haloperidol was prescribed. **I.56-57**

74 On 30.04.97 a further CT scan was performed. I have seen a handwritten note which reads:

> *"no change from previous scan".* **I.57**

75 The nursing note for 30.04.97 notes that there was mild weakness in the right arm, severe weakness in the left arm and mild weakness in both legs. **I.19**

76 On 01.05.97 agitation was recorded. Midazolam and diazepam were given. **I.57-58**

77 On 1 May Mr Hamill was said to be:

> *"very distressed and agitated. GCS motor flexing on right extending left arching back. V1. E1. O_2 sats 97%".* **I.58**

78 I have reviewed the nursing charts from 3-5 May. There was no eye opening. The verbal response was generally none, occasionally incomprehensible sounds. The motor response was flexing. Pupils were consistently small and reactive to light. **I.75**

79 On 05.05.97 Mr Hamill was reviewed by Dr McCann from rehabilitation who made the following note:

> *"now nine days post-injury. Currently sedation → ? cerebral irritation secondary to hypoxia. GCS 6. No increased tone arms/legs. I will re-assess 1/52".* **I.60**

80 On 08.05.97 the note reads:

> *"cardiac arrest call 3.40pm. Ventilated. Cardiac output in femoral. Sinus tachycardia...cardiac arrest. Electro-mechanical disassociation. CPR commenced...".* **I.60**

81 Despite resuscitation there was no cardiac output and the pupils had been fixed and dilated for twenty minutes. Resuscitation was stopped after discussion with Mr Fannin and others. Mr Hamill's death was formally declared at 1703 on 8 May 1997. **I.61-62**

REVIEW OF WITNESS STATEMENT OF DR LAVERY (CONSULTANT IN INTENSIVE CARE) DATED 15.09.97

82 Dr Lavery's statement does not add a great deal to the history obtained from the medical records. His feeling is that:

> *"the patient's neurological status suggested significant hypoxic damage (probably acquired at the time of injury)".* **II.24**

REVIEW OF STATEMENT OF MR PATEL (NEUROSURGICAL REGISTRAR) UNDATED

83 Mr Patel has stated that he saw Mr Hamill:

> *"on 8 May 1997 at approximately 1530. When I saw the patient he was pale, there was no recordable blood pressure and his temperature was 42°C. There was marked perspiration noted. His pupils were equal at 2mm and reacting. The respiratory movements were very shallow and it appears that he had a respiratory arrest. The peripheral pulses were palpable...".*

84 Mr Patel then records the details of the post cardio-respiratory arrest. **II.26**

REVIEW OF STATEMENT OF MR FANNIN (CONSULTANT NEUROSURGEON) DATED 14.05.97

85 Mr Fannin records much of the history that has been noted above. He also noted that:

> *"there was evidence of bruising around the left temple. The following investigations were carried out: CT scans of brain which showed soft tissue swelling in the scalp of the left temporo-parietal*

region and also the right temporo-occipital region. No intracranial abnormality was detected. There was a suggestion of a mild degree of frontal lobe atrophy...

...in general his movements were spastic and decerebrate. His pupils remained equal and he was notably sweating profusely with fluctuations in his pulse and blood pressure. It was thought that he was going through a stage of extreme cerebral irritation following a closed head injury...

...his condition deteriorated dramatically on the afternoon of 08.05.97 when he became deeply cyanosed with peripheral shut-down and respiratory problems...

This was an extremely unexpected outcome and it was thought that he may have been suffering from a septicaemia or perhaps a pulmonary embolus...it is felt that this man sustained a relatively minor head injury that was in all likelihood hypoxic at the scene of the assault resulting in his extreme cerebral irritation and evidence of a decerebrate type brain stem injury". **Attached to Letter 21.03.05**

REVIEW OF POST-MORTEM PHOTOGRAPHS

86 There is a photograph of the left side of Mr Hamill's head. There is a left sided peri-orbital haematoma. On the left side of the forehead there is a linear partial thickness laceration of the skin with some skin contusion and perhaps a little swelling. The peri-orbital haematoma could have been caused by a direct blow to the left orbit, it could have been caused by an anterior fossa fracture. The linear laceration of the forehead is most likely to have been caused by an object of about the size of the laceration, perhaps a bottle, perhaps a hammer or other similar

object but probably not a fist (speaking as an expert neurosurgeon not an expert forensic pathologist).

87 There is a more frontal photograph which probably suggests that there are bilateral peri-orbital haematomas.

88 There are a number of other photographs showing bruising to various parts of the body.

89 There is a photograph of what I take to be the left side of the brain with the dura intact. There is a small left sided extradural haematoma.

90 There is a view of the skull base with brain removed and the dura reflected. There is a left sided extradural haematoma lying on the floor of the anterior cranial fossa. I suspect this haematoma was in continuity with the convexity extradural haematoma. This haematoma probably lies under the left frontal skin contusion noted above.

REVIEW OF POST-MORTEM REPORT

91 I have reviewed a post-mortem report issued by Professor Crane (Professor of Forensic Medicine and Consultant Pathologist).

92 The date of death was confirmed as 8 May 1997. The post-mortem examination was carried out the following day, 9 May 1997.

93 Professor Crane identified the following injuries to the outside of the head:

> *"1. An area of red abrasion, 15mm diameter on the left side of the forehead above the temple and close to the hairline. Within it was a fine linear pinkish-red scar, 2cm long.*

2. *A resolving reddish-purple bruise, 25mm long and up to 9mm broad across the upper eyelid of the left eye.*

3. *A spot of abrasion, 2mm diameter on the naso-labial fold close to the left nostril".*

94 Internal examination of the head was recorded as follows:

"Scalp: When reflected there was an area of bruising 6x3cm in the left temporalis muscle and another area of bruising 3x1cm over the right parietal region on its undersurface.

Skull: Of normal thickness and density, varying between 3 and 10mm. In the relatively thin frontal bone on the left side there was a fissured fracture, 7cm long which extended downwards and medially into the roof of the left orbit overlying which was a little extradural haemorrhage.

Brain: 1,548gm. It seemed rather bulky with flattening of the convolutions and grooving of the cerebellar tonsils. The meningies appeared congested but translucent although the cerebrospinal fluid appeared a little cloudy".

95 The gross examination of the brain following fixation showed:

"on external examination there was no subdural or subarachnoid haemorrhage and there was no meningitis. No surface contusions were seen. There was no asymmetrical swelling and no evidence of uncal or tonsillar herniation or necrosis. The vessels of the Circle of Willis showed no evidence of atheromatous degeneration or aneurysm formation.

On coronal sectioning there was no cortical atrophy. Focal haemorrhages were present within the deep white matter of both frontal lobes in a parasagittal location. They were also present in the left thalamus and internal capsule. In additional there was diffuse vascular congestion throughout the white matter. No focal haemorrhage was present in the corpus callosum although it appeared congested. There was no evidence of midline shift or internal herniation but there was a mild degree of ventricular compression. There was no caudal descent of the mamillary bodies.

On sectioning the brainstem there was diffuse punctuate haemorrhage. The cerebellum showed no macroscopic abnormality".

96 Microscopic examination of the fixed brain showed:

"Brain: Sections were taken from the right and left frontal lobes, the corpus callosum, both hippocampi, the hypothalamus, cerebellum, midbrain and pons.

Frontal lobes: Examination of the sections from the right and left frontal lobes showed the presence of white matter contusions. This was associated with tissue necrosis and a macrophage reaction. Surrounding this was extensive diffuse damage. There was no evidence of hypoxic/ischaemic necrosis of the cortex. There was no meningo-encephalitis.

Corpus Callosum: Examination showed diffuse axonal damage.

Left Internal Capsule: Examination showed extensive diffuse axonal damage.

Hypothalamus: Examination showed no evidence of haemorrhagic necrosis. There was however external damage in the hypothalamic region.

Midbrain: Examination showed diffuse axonal damage. There was no secondary brainstem haemorrhage. The substantia nigra showed no evidence of degenerative change.

Pons: Examination showed the features of severe diffuse axonal damage.

Cerebellum: Examination showed no evidence of cortical necrosis. The Purkinje cells were preserved. A small gliotic scar was present in the white matter.

Immunohistochemistry for neurofilament protein and for the myelin stain LFB/H & E confirmed the distribution and severity of diffuse axonal damage".

96 Professor Crane came to the following conclusions:

"Death was a result of the head injuries which he sustained. Externally his injuries appeared trivial; there was a small area of abrasion on the left side of the forehead, a bruise on the upper eyelid of the left eye and a small sport of abrasion close to the left nostril. Even internally the injuries did not seen particularly severe with only two areas of bruising on each side of the undersurface of the scalp and the small almost hairline fracture in the front part of the skull running into the roof of the left eye socket. Detailed examination of the brain however and in particular its microscopic examination revealed widespread damage within its substance of a type as diffuse axonal injury.

This condition, most frequently encountered in acceleration/deceleration injury as a result of road traffic accidents is also well recognised as occurring as the result of repeated blows to the head such as by punching or kicking and this would seem the most likely mechanism of injury in this case. It was ultimately the effects of the brain injury which were eventually responsible for his death in hospital...".

"On his initial admission to hospital...an analysis carried out at that time revealed an alcohol concentration of 221mg per 100ml. Such a level would leave no doubt that he was moderately intoxicated at the time of the incident. Also it is well recognised that alcohol intoxication exacerbates the effects of head injuries and may have played a part in the fatal outcome in this case".

REVIEW OF RADIOLOGICAL IMAGING

97 I have not seen the radiological imaging carried out at the RVH nor have I seen reports of that imaging.

98 Notes within the medical records suggest that there was no obvious abnormality of any of the CT scans of the head that were performed.

OPINION

99 Mr Hamill died as a consequence of head injuries suffered on 27 April 1997 the consequence, as far as I can tell, of an alleged assault. At the risk of simplifying a complex subject might I ask the enquiry to consider a simple classification of the types of damage that can be caused following head injury?

100 **A haematoma or contusion** - A haematoma is a blood clot. The blood clot can lie outside the dura (extradural), just beneath the dura but outside the brain (a

subdural haematoma) or within the brain itself (an intracerebral haematoma or contusion).

101 A blood clot causes problems by virtue of local or generalised mass effect. The brain sits within the skull which is a bony, rigid and largely inflexible container. The brain has a limited capacity to cope with intracranial mass lesions (before there are increases in intracranial pressure (ICP) (the Munro-Kellie doctrine). Significant intracranial haematomas will cause increased intracranial pressure (the generalised effect of the increased mass) and/or brain shifts (the consequence of localised mass effect).

102 Mr Hamill did not suffer a significant intracranial haematoma. His death was not a consequence of raised intracranial pressure.

103 **Brain swelling** – The brain can be swollen either because of an increase in the cerebral blood volume or because of an increase in the extracellular or intracellular fluid volume. Increased cerebral blood volume is a cause of brain swelling when there is post-traumatic failure of the auto-regulatory mechanisms that control cerebral blood flow and cerebral blood volume. It typically occurs in young patients of children following high velocity accidents such as road traffic accidents. It causes a generalised rise in intracranial pressure.

104 An increase in the extracellular fluid volume (vasogenic oedema)commonly occurs around a brain contusion.

105 Sharp increases in the cerebral blood volume following auto-regulatory failure or significant brain oedema would lead to raised intracranial pressure and death would be associated with signs of localised raised intracranial pressure (typically brain shift) and/or generally raised intracranial pressure (tentorial or tonsillar herniation). There is no evidence in Mr Hamill's case to suggest that either of those two consequences of head injury were a relevant matter.

106 **Diffuse axonal injury** – In the diffuse axonal injury pattern of head injury axons (which are the connections between different nerve cells) become torn. We consider this to be a "shearing" injury with bundles of axons in nerve fibre tracts becoming disrupted. A useful analogy might be to consider that the brain is composed of millions of electrical cables (like telephone cables) which are all interconnected and where there are large bundles of cables connecting different parts of the brain much as there might be a large bundle of telephone cables connecting the UK with the USA. Diffuse axonal injury typically occurs where there are abrupt acceleration/deceleration forces applied to the head. One cause would be where a vehicle travelling at speed, say 60 or 70 miles per hour, abruptly stops for example after hitting a brick wall. The head at one moment is moving through space at 70 miles per hour and then milliseconds later the body stops moving. These forces are transmitted to the brain and this results in axonal tearing or shearing. An alternative mechanism for axonal tearing or shearing would be repeated blows to the head that also lead to similar sorts of acceleration/deceleration forces occurring. There was some evidence that rotational injury is particularly damaging.

107 The diffuse axonal injury pattern of injury was a major contribution and possibly the only contribution to Mr Hamill's death.

108 **Metabolic injury** – The brain is metabolically active. This can be seen when one considers that the weight of the brain (1.5kg in a 75kg adult) is 2% of the body's mass yet the blood flow to the brain is 20% of the blood flow to the body generally. The brain has no store of energy metabolites and it relies on a continuous delivery of oxygen and glucose from the blood in order to maintain cerebral metabolism.

109 If there is a failure of the delivery of energy metabolites to the brain there can be rapid and indeed catastrophic impairment of neuronal function. For example following a cardiac arrest there is no delivery of energy metabolites to the brain. Following profound hypoglycaemia there may be substantial impairment of

glucose to the brain. Following hypoxic events there may be impaired oxygen delivery to the brain. Any or all of these mechanisms can cause primary brain injury.

110 It has long been known that a primary traumatic brain injury can be exacerbated by the secondary effects of metabolic impairment. Patients with a given level of a traumatic head injury will have more profound neurological impairment and a poorer long-term outcome if they had for example suffered hypoxia or hypotension at the time of the head injury and/or subsequently.

111 Let us consider Mr Hamill's injuries. From the CT scan there appeared to be scalp swelling over the left tempero-parietal region and over the right tempero-occipital region. From the post-mortem examination there was an area of abrasion with a skin scar on the left side of the forehead above the temple with underlying bruising in the left temporalis muscle. There was also another area of bruising over the right parietal region.

112 The CT scan and the external examination of the head confirm soft tissue/scalp injuries over the left frontal region and the right temporo-parietal occipital region (for convenience let us say above the right ear). The left frontal injury was associated with a linear scar fracture which extended into the roof of the orbit and that in turn was associated with a small extradural haematoma. We can conclude that Mr Hamill, on a balance of probabilities, suffered as a minimum two significant blows to the skull; one to the left frontal region and one to the right side of the head (approximately above the right ear). It is possible that one of the soft tissue scalp lesions occurred as a consequence of Mr Hamill's head striking the ground.

113 Mr Hamill also had an abrasion and bruising of the left eye which could either have been caused by a blow to the eye or it could have been caused by blood tracking from the left anterior fossa fracture (probably the former in that blood

tracking from an anterior fossa fracture typically causes a widespread periorbital haematoma (a panda eye) which was not reported by Professor Crane.

114 There is also the possibility that Mr Hamill suffered further blows to the head no one of which was sufficiently severe to cause a scalp contusion but which might have contributed to the totality of his brain injuries.

115 We know that from the moment that Mr Hamill was first assessed at Craigavon Area Hospital he had suffered a severe brain injury, he was unconscious, at times being recorded as having no eye opening, no verbal response and no motor response (GCS 3/15) or flexing to pain with no eye opening and no verbal response (GCS 5/15). [We must remember that the Glasgow Coma Scale has as its lowest score 1 in each of the three domains i.e. 3/15 is the lowest possible score on the Glasgow Coma Scale]. Thus there can be no doubt that Mr Hamill had a severe brain injury at the time that he was first assessed. There are many patients who initially do not appear to have suffered a major primary injury for example they may be alert, orientated, talking or perhaps confused who subsequently deteriorate because of the effects of raised intracranial pressure. Such a pattern would be very typical following an extradural haematoma although any haematoma, contusion or swelling could cause such a pattern of injury i.e. the subsequent neurological impairment is a consequence of the subsequent development of mass effect and raised intracranial pressure and not the primary injury.

116 Mr Hamill was probably rendered unconscious in the earliest moments following the alleged assault. He would then have been lying on the ground. If an unconscious patient is lying flat on his or her back they can *"swallow their tongue"*: loss of consciousness can lead to impairment of reflex protection of the airway and essentially the tongue obstructs the pharynx leading to hypoventilation and hypoxia. It is for this reason that unconscious patients should be placed into the recovery position where gravity helps the tongue to fall forwards clearing the

airway. In hospital we would put in a Gudel airway to achieve the same effect and/or intubate the patient if that was necessary.

117 It is possible that Mr Hamill had an obstructed airway following a coma-producing head injury and the subsequent neurological impairment was a combination of a primary traumatic head injury plus a secondary metabolic injury to the brain.

118 Both the CT scans and the neuropathology confirm that there was no intracranial haematoma and there was no brain swelling. Internal examination of the unfixed brain showed some modest tonsillar grooving which was probably a consequence of the immediately ante-mortem perfusion failure. However Professor Crane specifically noted that there was *"no asymmetrical swelling and no evidence of uncal or tonsillar herniation or necrosis...no evidence of midline shift of internal herniation...no caudal descent of the mamillary bodies"*. We can conclude with a high degree of certainty that Mr Hamill's death was not a "pressure" death.

119 In my opinion we are left with three possibilities which are that Mr Hamill's death was (i) largely due to diffuse axonal injury, (ii) was largely due to a post-concussional hypoxic injury or (iii) it was a mixture of both.

120 Professor Crane's microscopic examination of the fixed brain demonstrated diffuse axonal damage and white matter contusions (both a hallmark of the diffuse axonal injury pattern of head injury) very extensively in the brain including the frontal lobes, corpus callosum, internal capsules, hypothalamus, mid-brain and pons. By contrast Professor Crane was unable to identify evidence of hypoxic/ischaemic necrosis in the cortex. If there was a significant metabolic (in this case hypoxic) injury to the brain it would affect those parts of the brain that are metabolically most active. The grey matter contains a high proportion of nerve cells bodies which are metabolically very active. White matter tracts by contrast are by contrast are less metabolically active. Grey matter has a blood flow which is approximately four times greater than white matter which reflects

the different metabolic requirements of grey and white matter. If there had been a significant hypoxic injury to the brain one would expect to see histological evidence of damage to cortical neurons in excess of any damage seen in white matter tracts. The cerebellar Purkinje cells are metabolically very active and one would expect to see evidence of damage to the Purkinje cells. In Mr Hamill's case there was no evidence of damage to these metabolically active structures. By contrast damage was exclusively to white matter tracts. On a balance of probabilities Mr Hamill's death was almost exclusively a death consequent upon diffuse axonal injury with little or no contribution from any other cause.

121 It might be helpful to wonder why Mr Fannin (a very experienced consultant neurosurgeon) and Dr Lavery (an experienced intensive care consultant) should both have thought that Mr Hamill was probably suffering from a hypoxic injury? In my opinion they probably thought that the mechanism of injury was hypoxic because they were aware of somebody who had a severe brain injury with no evidence of a haematoma or contusion or brain swelling i.e. a diffuse and severe brain injury where the mechanism of injury had apparently been an assault. Simple assaults do not normally cause diffuse axonal injury which is more commonly a consequence of high speed road traffic accidents (which are required to cause the abrupt acceleration/deceleration forces that are needed to damage axons in this way). It is obviously the case that the Accident and Emergency wards of most hospitals contain large numbers of patients who have been subject to relatively simple assaults where a punch has left the victim knocked out for a few minutes and he recovers in hospital, going home the following day with no more than headache for a few days. Even in the boxing ring where boxers are trained to deliver severe punches of enormous power and they are trained to deliver them to the head to try and cause loss of consciousness, diffuse axonal injury is very rare (obviously boxers can die because they develop blood clots).

122 My conclusions are as follows:

(i) Mr Hamill's head/brain was subjected to such acceleration/deceleration forces that there was a shearing injury to white matter tracts throughout the brain and the degree of axonal injury was considered by Professor Crane to be *"extensive"*.

(ii) That diffuse axonal injury was probably the sole cause of Mr Hamill's death.

(iii) Such a wide-spread and extensive diffuse axonal injury to the brain would normally be associated with high velocity road traffic accidents which is perhaps one reason why Mr Fannin and Dr Lavery felt that there was a hypoxic basis for the injury. They did not expect such an injury to be a consequence of a simple assault.

(iv) A single "knock out punch" would almost never be associated with such an injury.

(v) On a balance of probabilities Mr Hamill suffered a blow or blows of great force to cause this pattern of brain injury.

123 It is beyond my expertise to say whether such damage could be caused by a single blow of great violence as might be caused for example by kicking the head with a steel toe capped boot or whether it would require the head to be kicked repeatedly "like a football" or whether it could be caused by one or more blows with a weapon such as a hammer or whether it could be caused by multiple blows with fists.

SUMMARY

124 Mr Hamill died as a consequence of a head injury that followed an alleged assault on 27 April 1997.

125 The clinical picture was of a severe diffuse brain injury with coma from the time of injury and persistent coma until Mr Hamill's death.

126 CT scanning ante-mortem and examination of the brain post-mortem demonstrated no evidence of intracranial haematoma, no evidence of brain swelling or brain oedema nor any evidence that Mr Hamill died of a "pressure" death.

127 Histological examination of the brain showed no evidence of hypoxic ischaemic damage in neuronal structures that would be subject to such damage.

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129 Such a diffuse axonal injury, widespread and in parts extensive, would typically be associated with high velocity road traffic accidents where there are severe and abrupt acceleration/deceleration forces.

130 Such a pattern of injury does not usually follow a simple assault where a punch leads to the patient being knocked out briefly.

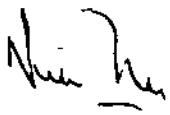
131 In my opinion Mr Hamill's head/brain was subjected to such significant forces that it caused the sort of severe axonal injury that is normally associated with high velocity road traffic accidents.

132 It is outwith my experience to comment upon whether such an injury could be caused by multiple blows to the head with fists, with striking the head one of more times with a weapon, with kicking the head one or more times as the unconscious victim lies on the ground, or any other potential mechanism of injury. Suffice to say that the levels of force applied must have been very much

greater than those that occur in the average "Saturday night punch up" and more akin to the damage that is done in high speed motor vehicle accidents.

This report has been prepared in my role as advisor to instructing solicitors. It should not be disclosed without written authority.

Yours sincerely



Mr N V Todd MD FRCS
Consultant Neurosurgeon and Spinal Surgeon

CURRICULUM VITAE (CONDENSED)

NAME	Nicholas Vyner Todd
QUALIFICATIONS	MBBS ~ University of London, 1978 FRCS ~ RCS England, 1982 MD ~ University of London, 1987 Diploma in Law (CPE) ~ University of Northumbria, 2001
DISTINCTIONS	Hunterian Professor Royal College of Surgeons, London 1985
CURRENT APPOINTMENTS	Consultant Neurosurgeon Newcastle Upon Tyne NHS Trusts
	Honorary Lecturer University of Newcastle Upon Tyne
	Honorary Consultant Neurosurgeon Royal Victoria Hospital Belfast
	Honorary Consultant Neurosurgeon James Cook University Hospital Middlesbrough
	Neurosurgery/Spinal Surgery Adviser National Institute for Clinical Excellence

CLINICAL INTERESTS

I work as a neurosurgeon taking patients with the widest range of neurological and neurosurgical conditions. I would consider myself expert in the management of head and spinal injury, ischaemic and haemorrhagic cerebro-vascular disease, paediatric and adult brain tumours and central nervous system infection. I provide specialist services in paediatrics particularly to children with brain tumours. I also provide specialist services in the field of spinal surgery. I am the sole surgeon providing a Regional service for the assessment and surgical treatment of rheumatoid disorders of the cervical spine.

ACADEMIC

In 1983 – 1984 I was a Medical Research Council training fellow in Neurosurgery undertaking experimental research in the area of ischaemic cerebro-vascular disease. I have subsequently undertaken clinical research in various areas including haemorrhagic cerebro-vascular disorders, head injury, spinal disorders, brain tumours, trauma and image-guided surgery. I have published a higher degree and have over 100 neurosurgical publications or abstracts.

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MEDICO-LEGAL WORK

I have provided many hundreds of reports, for both personal injury litigation and medical negligence litigation. I have appeared in the County Court and High Court on many occasions. I would estimate that I am currently instructed approximately 60% by plaintiffs, 40% by defendants.