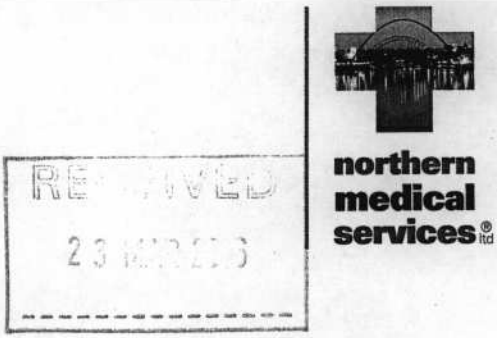
neurosurgery & spinal surgery
medico-legal services



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Ms P Fitzmaurice
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PO Box 50156
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Our ref: NVT/VT/SD/30585
Your ref:

Wednesday 8 March 2006

Dear Ms Fitzmaurice

Re: Hamill Inquiry

INTRODUCTION

I have tried to put a little more flesh on the bones of my Causation Report following a literature search.

REVIEW OF THE RELEVANT MEDICAL LITERATURE

Strich was the first person to describe neuropathological evidence of diffuse traumatic white matter injury[1]. Strich suggested that the mechanism was probably shearing of axons at the time of a traumatic head injury.

In the 1980's diffuse axonal damage was produced for the first time in experimental animals and the importance of rotatory injury become clear.

In 1989 Adams and others proposed a system by which the severity of diffuse axonal injury could be graded.

To some extent diffuse axonal injury is something of a misnomer in that the axons are not injured diffusely throughout the whole brain rather is the axonal injury predominant in discreet regions of the brain. Diffuse axonal injury were thought to require high velocity deceleration injuries and motor vehicle accidents and falls from a height are the commonest causes of severe diffuse axonal injury. It should also be accepted that there may be localised axonal injury in the brain stem as a consequence of injury at the cranio-cervical junction in the absence of axonal injury elsewhere. This localised axonal injury reflecting local injury does not have the same implication as diffuse axonal injury[4].

The use of markers for β – amyloid precursor protein (β APP) has been used to detect axonal damage in patients who have died within a few hours of a head injury. Positive β – APP axonal damage following trauma is evidence of traumatic axonal injury. The original description of diffuse axonal injury represents the most severe form of a diffuse traumatic axonal injury[5].

[redacted]

We also need to be aware that uncommonly hypoxia in the absence of trauma can be associated with axonal injury[6].

It remains the case that diffuse axonal damage is typically caused by accidents where a severe level of acceleration-deceleration occurs typically high velocity road traffic accidents and falls from a height.

In 1992 Graham et al[7] published 15 cases of fatal head injury caused by an assault where there was neuropathological evidence of diffuse axonal injury. In 10 of the 15 patients the diffuse axonal injury was severe (Grade 3). In some cases the assault was a punch or punches, in other cases there were further injuries to the head either an assault with a heavy object or kicking, in some cases the victim fell striking his head on the ground. Graham et al make the point that in most of their cases full details of the nature of the assault were not available.

Of further note is that diffuse axonal injury is a neuropathological diagnosis)it is a diagnosis made in patients who have died following their head injury) i.e. it is a diagnosis made following the most severe head injury. It is possible that mild axonal injury does occur in patients who survive traumatic head injury. Recently MR imaging in patient with mild traumatic brain injury has demonstrated changes which were thought to be an indicator of traumatic axonal injury[8].

There are a number of reports of axonal injury following assault. We have already considered the 15 patients reviewed by Graham et al. Grade 2 diffuse axonal injury has been identified in a man who died thirteen days after an assault. The paper contains the witness evidence that *"the victim was attacked while lying on the ground. The perpetrator stomped and kicked the victim and also struck the victim's head with a piece of cement"*. This suggests that severe injury is required to cause a grade 2 diffuse axonal injury following assault[9].

There is a report[10] of diffuse axonal injury which occurred in a 20 year old rugby player who *"was carrying the ball, was tackled and fell to grass covered ground, striking his head. The player immediately became unconscious and remain unconscious for fifteen hours at which time he was declared brain dead"*. The report notes generalised brain swelling, subdural and subarachnoid haemorrhages and cortical contusions with β APP positivity in the pons. It is difficult to be clear from this report whether the beta β APP positivity was a consequence of classical diffuse axonal injury, whether it merely represented traumatic axonal injury or whether it was a consequence of brain swelling which in turn may be been a direct consequence of the head injury or possibly a consequence of a head injury plus significant post-head injury hypoxia. In my view this patient is better considered to have a traumatic axonal injury rather than a classical diffuse axonal injury.

A thirty year old man died three days after bring interrogated by a security service[11]. A CT scan demonstrated malignant brain oedema without intracranial haemorrhage. At post-mortem there was a right sided subdural haematoma causing right parietal lobe compression. The brain was markedly swollen, there was *"massive"* right sided uncal

herniation and *"severe"* herniation on the left. There was subarachnoid haemorrhage. There were no brain contusions. Histological examination was reported only as demonstrating *"diffuse axonal injury particularly in the corpus collosum and retinal haemorrhages"*. It was subsequently found that interrogation by the security services had involved violent shaking which was thought to be the cause of the fatal head injury. Thus it appears that, rarely, fatal brain injury can be cause by repetitive shaking only. In my view this case does not equate to classical diffuse axonal injury. This is a traumatic axonal injury associated with severe brain swelling, a compressive right parietal acute subdural haematoma and transtentorial herniation.

Geddes et al[5] make the point succinctly as follows:

> *"with our current state of knowledge we would say it is difficult to be confident in any given situation about the relative contribution of different types of assault to the cerebral pathology found at post-mortem"*.

FURTHER OPINION

Mr Hamill died as a consequence of a grade 3 (severe) diffuse axonal injury. That is to say there was a severe diffuse axonal injury in the absence of any other lesion specifically no contusion, no extra-axial of haematoma, no transtentorial herniation, no brain swelling.

In my opinion assessing causation of death in this case comes down in part to the burden of proof that the tribunal will apply. What I think we can say with some confidence is the following:

(i) Isolated grade 3 traumatic diffuse axonal injury is most commonly a consequence of severe acceleration/deceleration forces that occur for example following high velocity road traffic accidents or falls from a significant height.

(ii) The paper by Geddes et al[5] clearly records that grade 3 diffuse axonal injury can occur in patients who do not have an intracranial haematoma, who do not have diffuse swelling and who do not have evidence of raised intracranial pressure i.e. an assault can be the cause of injuries similar to Mr Hamill's injury.

(iii) The nature of the injury that caused Mr Hamill's severe diffuse axonal injury is speculative. Graham's paper makes the point that the nature of those assaults was largely unknown. The Raisenen paper and the Pounder paper suggest that death following a rugby tackle or death following severe shaking are caused by mechanisms other than isolated diffuse axonal injury.

(iv) The Imajo paper is a single case of a patient who had a severe assault *"the victim was attacked while lying on the ground. The perpetrator stopped and kicked the victim and also struck the victim's head with a cement piece"*. This prolonged

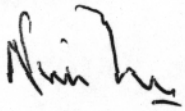
and severe assault led to a grade 2 diffuse axonal injury. If we rely on this single case report (which would be unwise) we have an implication that Mr Hamill's assault was at that level of severity or worse.

I have quoted Geddes et al's comments about the difficulty in coming to any conclusion about the nature of an assault placed upon the neuropathological findings seen at postmortem. However what I think we can say is that on a balance of probabilities Mr Hamill suffered a prolonged and violent assault rather than a single blow to the head. I say this on the basis that the vast majority of grade 3 diffuse axonal injuries are caused by severe acceleration/deceleration forces such as occur following high velocity road traffic accidents and/or falls from a significant height. However we are left with a small number of fatal head injuries who have pure grade 3 diffuse axonal injury following an assault (where the mechanism is unknown, Geddes et al[5]) or a single person with a grade 2 diffuse axonal injury who suffered a prolonged and violent assault. On that basis, and in my opinion, it is not beyond reasonable doubt that Mr Hamill did not suffer a relatively minor assault, perhaps being punched once, falling to the ground and striking his head, for example on a curb stone which led to such sufficient deceleration forces that a shearing injury as severe as a grade 3 diffuse axonal injury occurred.

I enclose a number of scientific papers for the assistance of the inquiry.

With kindest regards.

Yours sincerely



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