



Mr N V Todd MD FRCS
Consultant Neurosurgeon & Spinal Surgeon

Ms P Fitzmaurice
Deputy Solicitor
PO Box 50156
London
SW1E 6WX

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 injury¹. 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 discrete 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 injury⁴.

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 injury⁵.