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