Comment for the NIH 'Infant Neurotrauma' meeting, October 9 2002

The thing that should be concerning all of us here is the lack of proper evidence-base for much of what is generally believed.

I came to this field from adult head injury, with a particular interest in patterns of microscopic traumatic damage, and with a routine of taking large numbers of very large blocks from all head injuries.

The reason I started the NAI study that was published last year is that what I was finding in these brains was at variance with what was in the literature. It was claimed that infants with inflicted head injury suffered from diffuse traumatic axonal injury, but my experience was that they actually had very little in the way of traumatic brain damage.

So I looked at the evidence base for this belief. And here I have a slight advantage. The two papers which started this idea, and which have been so uncritically believed for all these years were those of Calder and Vowles, published in 1984 and 1987, from my department, by my predecessor, Carl Scholtz. The two papers use the same cases.

In their defence, a paper published in 1987 predates immunohistochemistry, and uses silver techniques, which are notoriously difficult to interpret. 1987 was also long before it was understood that trauma can produce a whole spectrum of white matter injury which falls short of DAI. I can give more detail of the inadequacies of the paper, but I will say that Carl Scholtz believed you could make a diagnosis of DAI on a single block of corpus callosum and parasagittal white matter, which would not be acceptable nowadays.

I have reviewed all but one of the cases from our files with Geoff Vowles, the first author of the 1987 paper, using immunohistochemistry, and I can report that only one case shows DAI – a young child with bilateral skull fractures.

There was no further similar study for 10 years after the Vowles paper, and as a result their findings became firmly entrenched in the literature.

In recent years immunohistochemistry for APP has been widely used in head injury. Confusion has arisen because forensic pathologists have found they had an easy way of detecting damaged axons, without really understanding what they were looking at. As a result the literature is full of neuropathologically inadequate case reports claiming to have found "DAI". And inevitably this has happened in paediatric head injury, as well as in adults. There is however no formal published neuropathological study of microscopic brain injury in infants apart from our own.

In summary, in my field there is no published evidencce for saying that the encephalopathy that these children suffer is the result of traumatic injury. Our work suggests that hypoxic brain injury is much more important than trauma, and we are supported in this by two recent studies looking at MR diffusion-weighted imaging. Secondary axonal damage occurs terminally in many of these cases, when the brain swells.

I give this as an example, from my field. I suspect that in very many areas of NAI poor studies are cited again and again in an uncritical way to finally become rock solid 'evidence', and that if one unravels this 'evidence' one finds that its not as robust as it should be, given what is at stake.

Jennian Geddes
London, 1 October 2002