Editorial

Paediatric head injury

Minor trauma to the head is common in childhood and does not require any medical or surgical treatment. Nevertheless, head injury in infancy and childhood is the single most common cause of death (Luerssen et al., 1988) and permanent disability. Measurable deficits occur even after mild to moderate head injury but are markedly greater after severe injury. They include impaired cognition, motor impairments, disruption of attention and information processing, and psychiatric disturbances (Adelson and Kochanek, 1998).

Despite the frequency of the sequelae of head injury in childhood, there is relatively little information about the structural basis of the clinical deficits. The classical literature suggests that the immature brain and its coverings, at a time when it is rapidly acquiring new information, respond differently from the adult brain when subjected to an equivalent amount of mechanical force, whether mediated by contact or inertial loading. However, any discussion about the mechanisms of injury must consider uncertainties about non-accidental injuries sustained in the context of child abuse.

For obvious reasons it has been difficult to determine the true epidemiology of non-accidental injury, although child abuse is considered to be the most common cause of head injury in infants younger than 2 years of age and is second only to road traffic accidents as a cause of death in childhood. Recently, Duhaime and colleagues have reported that non-accidental injuries account for nearly 25% of all head-injured children under the age of 2 years admitted to hospital (Duhaime et al., 1992). One specific mechanism of injury involved the child’s head being struck forcefully against a hard surface, the resulting injuries closely mimicking those resulting from a fall from a height, the pathology being a consequence of both impact and large inertial forces.

The first child abuse syndrome to be widely recognized was that of the ‘battered child’. The term ‘shaken baby syndrome’ was applied to infants with acute subdural haematoma and subarachnoid haemorrhage, retinal haemorrhages and periostial new bone formation at epiphiseal regions of long bones; it was attributed to the to and fro shaking of a child’s body producing a whiplash motion of the child’s head on the neck (Caffey, 1974). The term ‘shaken baby syndrome’ has been questioned, evidence suggesting that most of these injured infants have evidence of blunt trauma to the head and that the inertial forces generated by shaking alone were insignificant compared with those caused by impact (Duhaime et al., 1987, 1998). These and related studies gave origin to the term the ‘shaking impact syndrome’ in which it was recognized that, although many of the infants had been shaken as part of their injury, much—if not most—of the brain damage involved the head striking against a surface with sufficient inertial force to cause severe injury and subdural haematoma. Current debate revolves around the question as to whether shaking alone is sufficient to cause injuries observed in infants with the ‘shaking impact syndrome’. The current belief is that head-injured infants are likely to have undergone shaking followed by sudden inertial injury from impact.

In contrast to the neuropathological studies of traumatic brain injury in adults, the equivalent literature in paediatric head injury is much more limited. However, studies have described particular features that include contusional tears and injury to axons (Vowles et al., 1987; Shannon et al., 1998; Gleckman et al., 1999). There were similar findings in the Glasgow cohort of fatal head-injured children (Graham et al., 1989), in which detailed studies on 87 children aged between 2 and 15 years identified a range of pathologies remarkably similar to those seen in adults, the only difference being the increased prevalence of diffuse brain swelling. Therefore, given this literature, why is it that there are continuing uncertainties about the nature, the distribution and the pathologies in accidental and non-accidental injury in infants and children? Is there more to be learned from the careful study of cohorts of patients using standardized techniques? The answer is undoubtedly yes, and many of these particular issues are addressed by Geddes and colleagues in this issue (Geddes et al., 2001a, b). These authors have undertaken a meticulous clinicopathological correlation in 53 cases of non-accidental paediatric head injury.

Thirty-seven of the 53 cases were infants aged 20 days to 9 months with head injury, and 16 were children aged between 13 months and 2 years 6 months. When the data were analysed by median age at head injury, statistically significant patterns of age-related damage emerged. In particular, axonal damage with a distribution characteristic of diffuse axonal injury was found to be unusual in infants, any axonal damage being restricted to the craniocervical junction. But when axonal damage was present in children
older than 12 months, the pattern was similar to that of diffuse axonal injury in adults. The most important finding was that the predominant neurohistological abnormality in the cases of non-accidental injury in infants was due to hypoxia and not diffuse axonal injury. Although not commented upon it is not clear if, when interpreting this type of case material, there may be a need to consider the vascular complications of hypoxia and/or raised intracranial pressure.

Identification of different patterns of injury in different age groups has resonance in clinical practice and now provides a reference point for future clinical and neuropathological studies. This work not only provides the basis for the future management of patients, but also serves to remind us of the continuing value of the autopsy and the proper examination of retained organs using modern standardized techniques.

David I. Graham
Department of Neuropathology,
Institute of Neurological Science,
Southern General Hospital,
Glasgow, UK

References


