modelling from data on infants' skulls, brains, and neck structures, rather than living animals. Any studies on immature animal models, if performed, will need to be validated against the known mechanical properties of the human infant. Pending completion of such studies, the reviews by Lantz and Donohoe are a valuable contribution and provide a salutary check for anyone wishing to cite the literature in support of an opinion. Their criticisms of lack of case definition or proper controls can be levelled at the whole literature on child abuse. If the issues are much less certain than we have been taught to believe, then to admit uncertainty sometimes would be appropriate for experts. Doing so may make prosecution more difficult, but a natural desire to protect children should not lead anyone to proffer opinions unsupported by good quality science. We need to reconsider the diagnostic criteria, if not the existence, of shaken baby syndrome.

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Shaken baby syndrome

Pathological diagnosis rests on the combined triad, not on individual injuries

S haken baby syndrome is a form of physical non-accidental injury to infants, characterised by acute encephalopathy with subdural and retinal haemorrhages, occurring in a context of inappropriate or inconsistent history and commonly accompanied by other apparently inflicted injuries.^{1 2} Injuries to the neck and spinal cord may also be present. Controversy surrounds the precise causation of the brain injury, the retinal and subdural haemorrhages, as well as the degree of force required and whether impact in addition to whiplash forces is needed.^{1 3 4} Although most discussion has concerned fatal injuries of this nature, not all are lethal, but they may be associated with subsequent neurological disability of varying severity.

Expert medical evidence about inflicted injury must have scientific validity, but applying the evidence based criteria appropriate to clinical practice entails some difficulties.⁵ In clinical practice medical management of defined clinical problems can be compared and best practice distinguished by clinical outcomes. Conversely, in inflicted paediatric injuries, one is presented with the outcome, investigation follows rather than precedes that outcome, and the history may be incomplete or deliberately misleading. A need exists for an impartial and intelligent assessment, but how may this be achieved in practice?

Because of the serious implications of diagnosing inflicted injury such as shaken baby syndrome, every case must be evaluated in detail, taking account of all the circumstances surrounding the injury and considering the pathological features in full, rather than attempting to evaluate the significance of each component.

In shaken baby syndrome, it is the combined triad of subdural and retinal haemorrhage with brain damage, as well as the characteristics of each of these components that allow a reconstruction of the mechanism of injury, and assessment of the degree of force employed. The application of rotational acceleration and deceleration forces to the infant's head causes the brain to rotate in the skull. Abrupt deceleration allows continuing brain rotation until bridging veins are stretched and ruptured, causing a thin layer of subdural haemorrhage on the surface of the brain. This is not a space occupying lesion; its importance is in indicating the mechanism of injury. The retinal haemorrhages, which are characteristically extensive, occupy much of the circumference of the globe and extend through all the layers of the retina and similarly result from rotational acceleration and deceleration forces.

The mechanism of brain damage is problematic. Traditional wisdom has suggested shearing forces operating within the brain substance with consequent axonal damage.⁶ Geddes et al, in a careful neuropathological study of head injuries in children using β amyloid precursor protein immunostaining, observed that the predominant changes in infants with evidence of shaking were hypoxic-ischaemic rather than the diffuse axonal injury seen in older children and adults with fatal head trauma.^{7 8} These authors thought that acceleration and deceleration forces might damage the neuraxis to cause apnoea, with consequent ischaemic insult causing diffuse cerebral oedema.

Unfortunately, this logical idea was followed in a second paper by the statement, "Although mechanisms of Editorial p 719 Clinical review p 754 Letters p 766 Personal view p 775

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shaking must vary and nobody really knows how babies are injured, it may not be necessary to shake an infant very violently to produce stretch injury to its neuroaxis," a conclusion that is not supported by data in the paper and that has lead to considerable controversy among expert witnesses in court.8 It ignores the evidence for the force required to produce the triad of injuries, in fatal instances of shaken baby syndrome, obtained from evaluating the other components. Clearly, if "gentle" shaking were capable of causing fatal injury, such events would be an everyday occurrence. There is abundant evidence that minor head trauma, so common in the domestic context, is only very rarely associated with severe intracranial injury.9-11

Further confusion has been sown by a more recent contribution by Geddes et al.12 This describes the neuropathological findings in the brains of infants dying of non-traumatic cerebral hypoxia. Random examination of sections of dura showed intradural haemorrhage evident only at the microscopic level. On this basis they thought that all the components normally indicative of shaken baby syndrome might result from hypoxic damage alone, dural and retinal haemorrhage being due to brain swelling consequent on cerebral hypoxia. However, subdural haemorrhage in shaken baby syndrome is a macroscopic, not a microscopic, finding, and the comment on retinal haemorrhage has even less foundation in that no examination of the eyes was made.

As shown by Lantz et al in this issue, even when a particular detail has been claimed to be pathognomonic of shaken baby syndrome, the diagnosis should not rest on this feature alone¹³ (p 754). This careful case study reinforces the need for meticulous identification of the complexity of the injury and evaluating the findings against the validity of the explanation offered. It is also true that retinal haemorrhages can have causes other than shaking and that space occupying subdural haemorrhages causing death can occur in witnessed accidental injuries in children.¹⁴ However, of the patients Plunkett described, the youngest was 12 months old, which is outside the age group in which most cases of shaken baby syndrome occur.

The pathological diagnosis of shaken baby syndrome requires careful evaluation of the character and extent of all components of the injury and should

not rest merely on the presence or absence of one or more of the constituent lesions. The basic triad should have all the necessary features for confident diagnosis and the conclusion that undue force has been applied. Damage to the neck or spinal cord is further useful confirmation, and the presence of gripping injuries, while often absent, can provide further weight. Other inflicted extracranial injuries provide evidence of abuse even if they are not contemporaneous with the head injury.

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Risk assessment for spinal injury after trauma

The guidelines are simple and evidence based

bout 600-700 people sustain acute traumatic injuries to the spinal cord in the United Kingdom each year. Previously published data indicate that the injury to the spinal cord remains unrecognised in 4-9% of individuals.¹² Inadequate management of patients with injury to the spinal cord has the potential to lead to neurological deterioration, additional functional handicaps, and possibly medical litigation. Thousands of patients, however, routinely present to primary care centres every day with injuries to the neck and back. The immediate care and appropriate assessment of patients with spinal injury is a

skill that is expected of all doctors. General practitioners and hospital doctors with little or no training and experience of caring for patients with trauma might have to help the victims of a recent accident. They will certainly have to advise patients who complain of spinal pain after injury. This article is written to guide clinicians in these situations.

The evidence base for this subject has improved recently with some large scale studies from North America.3 4 Several consensus guidelines have been published by the National Institute for Clinical Excellence and the British Trauma Society.5 6 Most of