- 19 Krakow K, Polizzi G, Riordansva P, *et al.* Recovery of visual field constriction following discontinuation of vigabatrin. *Seizure* 2000;**9**:287–90.
- 20 Schmitz B, Jokiel B, Schmitz T, *et al.* Visual field defects under treatment with vigabatrin, carbamazepine and valproate. A prospective study. *Epilepsia* 1999;**40**(suppl 2):257.
- 21 Wilson EA, Brodie MJ. Severe persistent visual field constriction associated with vigabatrin. Chronic refractory epilepsy may have a role in causing these unusual lesions. *BMJ* 1997;**314**:1693.
- 22 Harding GFA, Wild JM, Robertson KA, *et al.* Separating the retinal electrophysiologic effects of vigabatrin: treatment versus field loss. *Neurology* 2000;**55**:347–52.
- 23 Koul RL, Aithala GR, Joshi RM, *et al.* Lamotrigene and vigabatrin in intractable seizures in children. *Neurology India* 1998;**46**:130–3.
- 24 The Royal College of Ophthalmologists (prepared by by Nick Asbury). *The ocular side-effects of vigabatrin (Sabril)—information and guidelines for screening.* Royal College of Ophthalmologists, 17 Cornwall Terrace, Regents Park, London NW1 4QW, April 2000.

Pathology of nonaccidental brain injury

It is commonly believed that severely shaken babies have diffuse axonal injury to the brain and that the forces needed to produce such injury are greater than can be explained by shaking alone and imply that the head has also struck a solid surface. Now a detailed neuropathological study (Jennian F Geddes and colleagues. *Brain* 2001;**124**:1290–8 and 1299–306; see also editorial, *ibid* 1261–2) has suggested that diffuse axonal injury is, in fact, uncommon in these infants and that their diffuse brain damage may be secondary to cervical injury and consequent hypoxia and ischaemia.

They examined the brains of 53 children who had died of presumed nonaccidental brain injury, 37 aged under 1 year (“infants”) and 16 aged 13 months to 8 years (“children”). The infants were less likely than the older children to have extracranial injuries but they were more likely to have evidence of previous trauma. Subdural haemorrhage was documented in 43 cases and was recent in 38. Of these 38, however, only four were large enough to cause a volume effect, the remaining 34 being described as trivial (“thin film”). No child under the age of 8 months had a large subdural haemorrhage.

Axonal damage was assessed using immunocytochemistry for β -amyloid precursor protein, a more sensitive method than previous silver stains. Diffuse brain swelling with raised intracranial pressure was considered to be the cause of death in 29 of the 37 infants and 13 of the 16 older children. Microscopic evidence of hypoxia-ischaemia was found in 32 infants and 11 older children, vascular axonal damage in 13 infants and 8 children, and diffuse axonal injury in only two infants and one child.

Eight infants had no signs of head impact and were thought to have been shaken. Seven of these had severe brain swelling and the eighth had severe hypoxic-ischaemic changes after surviving for 5 months. Two of these infants had cervical spinal cord or nerve root damage. The neuropathological findings were similar in these eight infants and the 29 who had evidence of head impact. In all, 11 cases, all infants, had evidence of localised axonal injury at the craniocervical junction or cervical cord. Twenty eight infants and five children had apnoea or cardiorespiratory collapse prior to presentation.

Diffuse axonal injury is uncommon in children who die of nonaccidental brain injury; most have diffuse brain swelling and hypoxic-ischaemic damage.

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