Vitreoretinal traction is a major factor in causing the haemorrhagic retinopathy of abusive head injury? – No

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For the past 20 years, vitreoretinal traction has been held to be a major mechanism for the generation of retinal haemorrhage in non-accidental injury in infancy. The presence of circinate macular folds, and a lesion termed ‘traumatic retinoschisis’ have been proposed as indicating severe vitreoretinal traction due to shaking, and by implication, only to result from the application of extreme violence. Some recent evidence, and clinical experience of the behaviour of partially detached vitreous, casts doubt on this hypothesis and this has implications for the degree of certainty with which the presence of retinal haemorrhage and circinate macular folds can be used as a marker for extreme violence done to an infant.

Child abuse is likely to be as old as humanity, but only recently has the combination of subdural and retinal haemorrhages with encephalopathy in infancy been recognised as being due, in some, if not all, cases, to inflicted trauma. The mechanism by which the trauma gives rise to the clinical findings remains the subject of hypothesis and conjecture.

Subdural haemorrhage (SDH) in abused children was first described by Tardieu.¹ Over 80 years later, Caffey described unexplained fractures of the long bones and SDH in 6 children,² but did not overtly cite inflicted trauma as the cause. It was a further 16 years before Kempe coined the term ‘Battered Child Syndrome’ to explain such findings.³

The first description of retinal haemorrhage (RH) in abused children was by Gilkes and Mann.⁴ They suggested that RH arose as a result

of a rise in the intracranial and the intraocular venous pressure, which could arise because of chest compression while the child was being shaken. Further descriptions of RH in abused children were given by Harcourt and Hopkins, who also described the visual impairment which could result not only because of ocular but also cerebral injury.^{5,6}

Guthkelch, a British neurosurgeon, first postulated that the cause of SDH in Battered Child Syndrome was a shaking injury, causing rotational forces within the cranium which disrupted vessels bridging the subdural space.⁷ He commented that, at the time, a ‘good shaking’ was considered by many British parents socially more acceptable and less dangerous than a blow to the head.

In 1974, Caffey coined the term ‘Whiplash Shaken Infant Syndrome’⁸ and postulated that many battered babies were really shaken babies. In commenting on the pathogenic significance of ocular lesions in these children, Caffey agreed with other authors of the time that ‘some of the affected infants are the victims of over vigorous *manipulations* (sic), not battering.’ He went on to comment that: ‘The pathogenesis of retinal hemorrhages in the manual WLS (whiplash shaking) of infants and children cannot be evaluated satisfactorily without a consideration of the incidence, nature and persistence of idiopathic retinal hemorrhages of the newborn.’ before going on to cite an increase in blood viscosity and polycythaemia as the major causal factors.

The concept that retinal haemorrhage arose in shaking injuries because of vitreous traction on the retina was first proposed by Greenwald *et al* in 1986.⁹ They coined the term ‘traumatic retinoschisis’ to refer to the appearances described in their series, which consisted of five

children with features compatible with inflicted trauma (although criminal prosecution occurred in only one case). Cystic retinal lesions, partially or completely filled with blood, were described at the posterior pole in four cases, in two of which cysts developed (in one case after clearance of delayed vitreous haemorrhage) after an initial evaluation had shown retinal haemorrhage only. All five cases had reduced or electronegative ERGs in at least one eye, indicating damage to the inner layers of the retina. They proposed that back and forth movement of the lens during a shaking episode transferred tractional forces through the vitreous to the posterior pole of the eye, causing splitting of retinal layers. Further descriptions of circinate perimacular folds, considered to result from vitreoretinal traction attributable to shaking, followed.¹⁰

Pathological support for the vitreous traction theory came from papers by Massicote *et al*¹¹ and Green *et al*.¹² Massicote *et al* noted partial detachment of the vitreous except at the apices of retinal folds—confirming, in their view, the role of vitreous traction in the formation of folds.

Green *et al* found subhyaloid haemorrhage and retinal detachment to be most frequent at the retinal periphery and around the optic nerve—the sites of the strongest vitreoretinal adhesion. They did not, however, describe retinoschisis.

Massicote *et al* also noted massive retinal haemorrhage at the vitreous base in one of their cases, and described a haemorrhagic cavity beneath the internal limiting membrane in one of their patients, which they described as schitic. In fact, despite the continued use of the term ‘traumatic retinoschisis’, true retinoschisis, as opposed to separation of the internal limiting membrane, has never been described pathologically due to inflicted head trauma in children.

In contrast, Emerson *et al*,¹³ found retinal haemorrhage to be more common in the mid periphery of the retina rather than at the vitreous base. Furthermore, Emerson *et al* did not find vitreous detachment peripheral to macular folds and cast doubt on vitreomacular traction as the aetiology of circumferential macular fold formation. They proposed that venous leakage led to the formation of a haemorrhagic schisis cavity, which expanded, pulling surrounding retina centripetally into a circumferential fold.

In other respects, it seems unlikely that shaking of an infant would result in significant vitreoretinal traction, or that this would lead to retinal haemorrhage.

Clinical experience of the behaviour of partially detached vitreous, and of vitrectomy surgery, where attached vitreous may have to be peeled away from the retinal surface, suggests that vitreous traction on the retina causes retinal tears rather than haemorrhage.

Furthermore, the eye is ‘designed’ to rotate, for example during saccadic eye movements, during which angular

accelerations of up to 700° per second may be achieved, and the vestibulo-ocular reflex is likely to mitigate the effects of rotation of the head on the eye.¹⁴ Retinal haemorrhages are not observed after saccadic eye movements, nor in cases of nystagmus, or opsoclonus. Rotational forces are intentionally applied to the eye by some surgeons during strabismus surgery—the ‘spring back balance test’ of Jampolsky,¹⁵ without causing haemorrhage.

Neither does vitreoretinal traction explain the frequent finding of RH (and when looked for, SDH¹⁶) in normal neonates, nor why the frequency of RH is significantly increased (reaching up to 75%) after Ventouse delivery,^{17,18} indicating a role for venous congestion by suctional forces transmitted through the fontanelle.

Does the precise mechanism whereby retinal haemorrhage occurs, in cases of inflicted trauma, matter? It is clear that inflicted trauma can give rise to subdural haemorrhage, encephalopathy, retinal haemorrhage, subhyaloid and sub internal limiting membrane haemorrhage, and circinate macular folds; and it is very likely that these findings can arise from shaking an infant without any impact or injury. However without a clearer understanding of the processes involved in the pathogenesis of these findings, it remains impossible, despite the assertions of some authors,¹⁹ to be certain that all infants demonstrating them have been the victims of attempted, or actual, murder.

Conflict of interest

The author has been a paid expert witness in court cases related to child abuse.

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