Unnatural injuries

DAVID TAYLOR

1. Introduction

This article is based on four lectures that I was very proud to be invited to give recently: the 50th Francis I. Proctor lecture in San Francisco, the Angeline Parks Memorial Lecture in Washington, the Mackenzie Lecture in Glasgow, and the Mary Louisa Prentice Montgomery Lecture in Dublin. Each lecture was on a different aspect of this broad subject, with some overlap, but they all reflected personal clinical and medicolegal experience and drew on the collaborative efforts of the British child abuse working party¹ of which I had the honour of being chairman.

The term 'unnatural' is defined by the *Shorter Oxford English Dictionary* as including 'not in accordance with the usual course of nature', 'monstrous, abnormal', 'outraging natural feeling or moral standards, monstrously cruel or wicked'. In this paper I will use the first definition because it avoids the emotional undercurrents that inexorably drag us into conflicts with our own selves and interfere with our attempts to help those who we serve: the children. Natural injuries to children must include those such as road traffic accidents or the results of fisticuffs or horseplay as, although these can be intentional, they are often accidental or unintentional in their effect, or at least are a part of natural life.

Thus, ocular self-mutilation in severely developmentally delayed children might be considered natural injuries whilst similar injuries in persons with no history of physical illness may be considered unnatural. Any classification has weaknesses. In this case some self-inflicted injuries occur against the background of a disturbed psyche: these are, I feel, unnatural enough to be included.

2. Child abuse

Child abuse is a general term used to cover the various forms of damage inflicted on children, including neglect, sexual abuse, emotional abuse, induced illness and physical injury. Although this article concentrates on aspects of physical abuse, it is acknowledged that a child may have been subject to more than one kind of abuse. Non-accidental injury (NAI) is a term that, whilst it is most frequently used in the child abuse context, strictly encompasses other forms of injury including some sports, criminal injury or surgery!

Child abuse encompasses a number of different conditions, any of which may coexist. In the first part of this article I will concentrate on indirect injury and shaking.

Classification of child abuse

Physical abuse
 Indirect injury and shaking
 Direct trauma
 Smothering
 Poisoning
 Induced illness: Munchausen's syndrome
 by proxy
 Sexual abuse
 Neglect
 Emotional Abuse

History

Although child abuse is surely antediluvian, there is little to be found in the early literature; it mostly emerged during the medical enlightenments in the mid-twentieth century. An early record of injury by parents of their children is from France:² mostly the children reported were late in their first decade, very different from the situation today.

In 1946 John Caffey, a New York radiologist, published six cases in which there were unaccountable long bone fractures in infants whose principal problem was chronic subdural haematoma.³ He, and others referred to in his paper, had noted that the fractures were not necessarily caused by the same force that caused the subdural haemorrhage or by seizures: intentional ill treatment was suspected in one case. It was 25 years before Caffey clearly associated the phenomenon with a non-accidentally inflicted injury, coining the evocative term 'whiplash shaken infant syndrome'.^{4,5} No doubt he and many others were aware of the true nature of these injuries, as Kempe *et al.* in 1962⁶ had described the 'battered-child syndrome'.

Because eye injuries were soon recognised as a very important part of the total injury, ophthalmologists were involved from the very earliest years.⁷⁻¹²

The psychosocial background

Kempe *et al.*⁶ described two cases in which there was a history of psychiatric disturbance, and a history of abuse of the victim's parents during

David Taylor ✉
 Eye Department
 Great Ormond Street
 Hospital
 London WC1N 3JH, UK

Received:
 22 November 1999
 Accepted without revision:
 9 December 1999

childhood or low intelligence. However, they observed that there may be a range from 'people with a psychopathic personality or borderline socio-economic status ... to people with good education and stable financial and social background ... Psychiatric factors are probably of prime importance ... but our knowledge of these factors is limited.'

Later,¹³ Kempe confirmed these feelings and, noting that abuse was unusual in adopted children (adoption was then a middle and upper class phenomenon in the USA), they looked at the abused children of upper and middle class parents. He felt that 5% of involved parents were aggressive psychopaths, 5% had an 'encapsulated psychosis' against that child and 90% had 'serious problems in mothering'.

Although child abuse occurs in all socio-economic groups,⁵ it probably more often affects the deprived. The perpetrators differ according to the era of any study and where and how it was conducted. Mostly fathers or mother's male partners are responsible.¹⁴ Psychiatric and sociopathic abnormalities are frequent in parents and other antecedents.¹⁵

Epidemiology

Virtually never diagnosed before the 1960s, a series of papers^{6-9,16,17} led to the realisation that child abuse is a phenomenon of substantial proportions in nearly every society today.

Although it has always been very difficult to estimate the incidence accurately,⁵ it is suggested that, in the USA, 300 000 children are physically abused, 140 000 are sexually abused, and 700 000 are neglected or maltreated each year: this leads to about 1100 deaths each year.¹⁸ Accident and other referral centres report that non-accidental injuries are a major, or the most important, cause of serious head injuries,¹⁹⁻²³ especially in infants.^{19,24,25} The incidence of NAI in children in the USA in the 1970s may have been 6 per 1000.¹³ The USA National Study of the Incidence and Severity of Child Abuse suggested that hospitals recognised 77 000 cases in about a year at the end of the 1970s.²⁶ Indirect evidence comes from studies of the incidence of subdural haemorrhages, where the incidence has been found to be 12.8 per 100 000 children per year, or 21 per 100 000 infants under 1 year per year.²⁷

There is little doubt that child abuse is under-diagnosed,^{5,28} with consequences not only for the undiagnosed children but in lessening the actions taken to protect children and their siblings from further assault. Child abuse is also under-reported^{13,26} in some socio-economic groups and over-reported in others. Hospitals tend to under-report to child protection agencies.²⁶ Disproportionate numbers of unreported cases in one study²⁶ were from white, higher-income families and were subject to emotional abuse. Some types of injuries, such as burns, are probably under-diagnosed.²⁹

Risk factors for child abuse

When dealing with almost any child's eye problem it is worth thinking 'Does the child come from an at-risk group?' Premature, handicapped,³⁰ crying colicky babies and young infants are all at more risk. Abused children may be delayed in their intellectual, emotional, social and motor development, and mental retardation and motor hyperactivity may precede any abuse.³¹

Situations that may suggest child abuse

Are the carers themselves past victims of abuse?³²
Are the parents drug or alcohol abusers?²⁵
Have other children in the family been abused?
Does the family frequently attend casualty?
Is the family going to different casualty departments?
Does the general practitioner or social services have any more information?
How does each parent see and what do they expect of this child?¹³
How were the parents raised?¹³
Was there a family crisis at the time?¹³
Do the parents have help from family?¹³
It is important to communicate and coordinate. Where possible hand over to the professionals and you, the ophthalmologist, stick to the eyes. The important thing is to identify when to get other professionals involved.

Presentations that may suggest child abuse

The account of how the injuries occurred is inconsistent with their appearance
The apparent age of injuries is inconsistent with the account given, or a delay in presentation
Injuries of different ages, indicating a series of injuries
Multiple fractures particularly in different stages of healing
Unexplained injuries
An unusual lack of parental concern at the severity or extent of the injuries
Premature, handicapped and crying babies, siblings of abused children and children of previously abused parents are more at risk
Presentation at a time of family stress (moving house, unemployment, change of marital status, etc.)³³

Patterns of injuries that may suggest child abuse

Head injuries in infants or non-mobile children
Subdural or subarachnoid haemorrhages
Human bite marks, black eyes, or fingertip bruising
Retinal haemorrhages
Unusual fractures, e.g. acromion, metaphyseal and rib fractures
Cigarette burns, especially if multiple
Scalds, especially in the absence of splash marks
Bruising in inaccessible sites or unusual cuts or marks

Behavioural and other signs that may suggest child abuse

'Frozen watchfulness' (unnatural stillness and watchful observation) in the child
Aggressive, highly active playing or acting out
Unusual closeness or distance between parents
Excessive hostility or over-friendliness by the parents or child towards hospital staff
Previous child abuse in an infant^{6,27}
Previous child abuse in the family^{27,34}
Male sex of the child and the perpetrator^{5,27,35}

History taking in suspected child abuse

In paediatric ophthalmology, as much as in any other medical speciality, the taking of a detailed history and recording it in equal detail is always of paramount importance. This is especially important in cases of suspected child abuse, not only in the clinical management but also in any subsequent legal proceedings.

1. Take the history yourself – you will need to recount it if called to court; record it in the notes. Write what is said, i.e. 'went a bit blue' and not 'complained of apnoea'. The ophthalmologist who is involved at a later stage of the investigation of the child is liable to complicate the issue further with a fresh history, other than that specifically related to the eyes: there are usually experts in psychosocial issues and NAI involved at an early stage. Unnecessary history taking at a late stage by the ophthalmologist may damage the future relationship with the family if the child survives. Keep a professional detachment from the issues and stick to the eye examination and management itself and document this well, including photography if possible.
2. What is the past history of the child?
3. Is the story consistent with the injury?³⁶ Short falls do not cause serious injury.^{24,36,37}
4. Is the same story given consistently? Take care that discrepancy in histories is not due to a different question being asked.
5. Is the account of the cause of the injury and the timing of it consistent with its appearance?
6. Was there a delay in seeking medical advice?
7. Have there been any siblings who were admitted to hospital with an unclear diagnosis or who have died without a satisfactory explanation as to the cause?
8. Are the injuries of different ages?
9. Is there abnormal concern or lack of it shown by the parents? Is there unusual hostility or over-friendliness shown towards the hospital staff?
10. Does the child, or any sibling, show abnormal behavioural signs such as 'frozen watchfulness' (watchful observation with anxiety on being approached), aggressive play, or unusual distance or closeness with the parents?
11. Have there been any previous seizures, 'funny turns', 'blue attacks', etc.?

12. Does the child come from an 'at-risk' group?
13. Did the injury occur at an unusual time, i.e. at night (when the child would normally be asleep) or over a weekend? Abuse injuries tend to occur at odd hours and over long weekends.

Ophthalmic examination in a patient with suspected child abuse

1. Age-appropriate vision assessment.
2. Pupil responses, especially for a relative afferent defect.
3. External eye inspection, including lids, ears and face.
4. Refraction.
5. Examination with a slit lamp (portable?) if appropriate and possible, especially looking for iridodonesis, pupil sphincter rupture, cataract and subtle hyphaema.
6. Fundus examination with the pupil dilated, including indirect ophthalmoscopy, after discussion with the paediatrician about the safety of dilating the pupils and the effect that may have on the neuro-observations.
7. Clinical (including normal) findings, documented with annotated drawings.
8. Fundus photography and external photography, including normal features, correctly timed and dated. The use of wide-angle fundus cameras (i.e. Retcam) and image-grabbing video technology can enhance the documentation.
9. A non-ocular examination. This is essential unless the paediatrician or named doctor has become involved.
10. Collaboration with the paediatrician, especially to exclude a bleeding diathesis (full blood count, platelets, prothrombin time, partial thromboplastin time).
11. Follow-up arrangements for those with eye involvement who survive, as visual sequelae are common and often severe, requiring input from social and educational services.

Ocular signs of direct injuries

Direct injuries are those in which there is a direct blow to the eye or periocular tissues.

Periocular bruising

Lid (Fig. 1a) and subconjunctival bruising is common if there has been direct trauma.³³ Although, in the casualty department setting, usually due to accidental trauma, 'black eyes' should suggest an examination of the eyeball to search for ocular trauma³⁸ and if there are also retinal haemorrhages this may give rise to suspicions of NAI. Bilateral periocular bruising may be a sign of a fracture of the base of the skull.³⁹

Fig. 1. (a) Periocular bruising in a non-accidentally injured child. (b) This child presented with a severe chemical burn to the right eye on the day that the mother's partner had left the home. Although not proven in this case, such a presentation is highly suspicious of a non-accidentally inflicted injury. (c), (d) Non-accidentally inflicted repeated chemical injuries to a child.⁴²

Lid lacerations, orbital haemorrhage and inflammation

Punches or slaps (Fig. 8a) may result in severe periorbital or orbital haemorrhage;³³ lid laceration is less common, implying the use of an instrument.

A 1-year-old girl was in the care of her grandmother when she developed unilateral parotitis, followed by unilateral orbital cellulitis, thought to be due to the injection of the salt Alum.⁴⁰ She made a swift recovery after separation from the grandparents.

Unexplained lens dislocation or cataract

The finding of cataract or dislocated lenses³³ does not necessarily indicate direct eye trauma; it can occur with an indirect injury and there are of course many causes of these conditions that are accidental or pathological. In an infant it is rare to find significant cataract in a non-traumatic dislocated lens, so finding this combination should alert the clinician to the possibility of trauma, whether accidental or non-accidental. In non-accidentally dislocated lenses, the cause may be due to trauma to the lens itself or secondary to intraocular damage.¹⁷

Conjunctival and corneal injuries

Inflicted corneal injuries are rare: bilateral traumatically inflicted injuries were described by Auvert *et al.*⁴¹ and a unilateral case by Friendly.¹⁷ Two sibs presented⁴² with recurrent keratoconjunctivitis and one occasion of dermatitis, which led to the initial diagnosis of a self-

inflicted injury in one sibling (Fig. 1c, d). When the other sibling became involved and other aspects of the cases became apparent, the diagnosis of a non-accidentally inflicted injury was made, and subsequently confirmed. These cases, though rare, may be more frequent than is currently recognised (Figs. 1b, 2a-c).

A case⁴³ was described in which the 9-year-old patient had cyanoacrylate glue in one eye: the evidence for it being a non-accidental injury was circumstantial.

Intraocular haemorrhages and retinal detachment

Although retinal detachment and vitreous haemorrhage may be due to direct injury,³³ it is much more frequently associated with indirect trauma. Vitreous base avulsion suggests a contusion injury with profound distortion of the eye causing different movements of the sclera and vitreous base, due to their different elasticities. This may suggest abuse,⁴⁴ but by itself does not prove it. Vitreoretinal adhesions at the vitreous base are stronger in the child than in the adult and avulsion probably requires more distortive forces than are possible with shaking injuries alone.⁴⁴

Pupil abnormalities

Abnormalities of the pupil reactions may be due to intracranial problems, but some, predominantly unilateral cases are due to direct injury to the iris; these are usually accompanied by other intraocular injuries and haemorrhage.

Fig. 2. (a) This 5-month-old baby presented with a 3 month history of watering and sticky eyes. (b) Old sub-ungual haemorrhages were noted. (c) Both eyes had deep corneal scarring in the lower half, with perforation, anterior synechia and cataract formation in one eye. (d) A subdural haemorrhage was found on CT scanning. Within 18 months the child's younger infant sibling was admitted with a subdural haemorrhage. (e) NAI. Extensive, multi-layered haemorrhages virtually obscure the retina and have broken into the vitreous. The optic disc is visible in the centre of the picture. (f) NAI. Chronic papilloedema from raised intracranial pressure associated with a chronic subdural haemorrhage. The haemorrhages are grouped around the optic disc, which may suggest that they are associated with the papilloedema itself rather than the event that caused the subdural haemorrhage.

Ocular signs of indirect injuries

Subconjunctival haemorrhages

Subconjunctival haemorrhages are fairly common in the normal neonate,⁴⁵ and may be seen in infants and children who have had an assault in which the central venous pressure has been raised.

Haemorrhages in and around the eye

The detection of retinal haemorrhages is a vital part of the investigation of an infant suspected of suffering child abuse: retinal damage is a very important cause of long-term morbidity and may be important in subsequent medicolegal proceedings.¹ It is vital that the pupils are

dilated for examination by indirect ophthalmoscopy, preferably with indentation, provided that the doctor in charge is satisfied this will not interfere with the monitoring of the child. In those cases where pupil dilatation is not advised, an initial examination with a direct ophthalmoscope or an 'all-pupil' indirect ophthalmoscope should be undertaken in the first instance. Documentation of the retinal and other changes is vital, preferably by expert photography.

Scoring systems

Few clinical or pathological studies have used an objective scoring system to assess the severity of eye or brain injuries in child abuse.^{25,46-48} There are no studies published which use a scoring system to compare the severity of non-accidental injuries with accidental injury or other conditions with overlapping signs, symptoms or autopsy findings, in either fatal or non-fatal cases. Wilkinson *et al.*⁴⁷ used a scoring system to demonstrate statistically that 'retinal hemorrhage predicts neurologic injury in the shaken baby syndrome.'

An autopsy study,^{49,50} reviewed by Snodgrass,⁵¹ used a trauma level scoring system to estimate relative trauma in the eyes and brains of 23 infants who had died as a result of unequivocal NAI. Of these children, 16 died from central nervous system (CNS) injuries due to shaking, without external signs of head trauma, and 7 died from non-cerebral injuries. Eye injuries were found in 81% of those dying of their head injuries and in 17% of those dying of non-cerebral injuries. Ten of the 16 patients with subdural haematomas had retinal detachments and a greater number of other eye injuries than the 6 who did not have retinal detachments at autopsy. Using this system, separate 'haemorrhage' and 'total eye' scores were derived from the presence of haemorrhage or focal retinal detachments in the eye, and 'the intracranial (CNS) score' from specific lesions in the CNS.

Scoring systems have a considerable potential as an objective means of establishing the severity of trauma inflicted to the eyes and CNS in infants; however, existing systems used by individual groups require further documentation and validation before they can be implemented fully in practice.

The application of scoring systems should only be used in addition to other social and clinical information. Natural disease such as ruptured vascular malformation⁵² or haemorrhagic disease of the newborn⁵³ could produce high eye and CNS trauma scores, which might together suggest NAI.

Age of abused children

Retinal haemorrhages are more frequently described in NAI below 1 year of age^{54,55} and they become less frequent and less severe in older children.

Location and type of retinal haemorrhages

Clinically, haemorrhages are seen most easily and frequently at the posterior pole (Fig. 3a-d),⁵⁶ however the retinal periphery is often difficult to examine clinically. An autopsy study⁴⁹ demonstrated that subretinal and subhyaloid haemorrhages were more common in the peripheral retina than at the macula or around the optic disc, and least common at the equator, but this pattern has not been assessed in survivors of traumatic episodes. Typically all layers of the retina (Figs. 2e, f; 3a-d; 4d-f) are involved,^{10,11,49,54,57-59} but haemorrhages confined to one layer are also found.^{54,59} The haemorrhages may have white centres that consist of fibrin.⁶⁰

Purtscher's retinopathy,⁶¹ in which there are retinal exudates as well as haemorrhages, is also found in child abuse;⁶² since it occurs mostly after chest compression in various situations,^{45,63-5} it probably suggests an increased role of raised central venous pressure. Air emboli may play a role⁶⁶ and in NAI it is likely that there is a combined effect, with acceleration-deceleration being very important.⁶⁷

Accidental and non-accidental trauma

A number of studies have established that retinal haemorrhages are rare after accidental trauma in infants and young children.^{19,58,68-72} Retinal haemorrhages may be seen after very severe accidental head injury^{72,73} causing skull fractures and diffuse or focal intracranial damage;⁴⁶ most of these accidents can be independently corroborated.²⁴ In the studies by Buys *et al.*³⁷ and Duhaime *et al.*²⁴ a total of 148 children under 3 years old with accidental injury were specifically examined for retinal haemorrhage. This was found in only one case: a high-speed car accident. The two studies included 58 skull fractures, 40 falls from higher than 4 feet (1.2 m), many onto hard surfaces, 8 falls down stairs and 11 car accidents; after excluding the one child involved in a high-speed car accident, none developed retinal haemorrhages. In contrast, retinal haemorrhages were found in 12 of 27 suspected abuse cases. When children receive fatal injuries after reportedly short falls, they are more likely to be non-accidental.³⁶

Retinal haemorrhages were not associated with witnessed accidental trauma unless it had been very severe: 100 consecutive cases of head injury in children under 2 years were examined for retinal haemorrhages. In 73 of the children, the injury was due to a fall and 24 were considered due to NAI. Household falls were neurologically benign, even with skull fractures. Only 10 patients had retinal haemorrhages, of whom 9 were in the 24 child abuse cases and 1 was involved in a high-speed road traffic accident.²⁴

Seventy-nine children under 3 years old had fundus examinations after head injury. Seventy-five of the children had accidental injuries, 1 child had injuries of undetermined cause: they all had normal fundi. All 3 abused children had retinal haemorrhages.³⁷

Fig. 3. (a) NAI. Multiple, small, flame-shaped haemorrhages. (b) NAI. Multiple 'domed' (subhyaloid) haemorrhages. The white spots at the apex of many of the haemorrhages are light reflexes. (c) NAI. Multiple, multi-layered retinal haemorrhages, including subhyaloid and intraretinal haemorrhages and haemorrhages on the optic disc. (d) NAI. A widespread deep retinal haemorrhage has extended to become subhyaloid. (e), (f) Flame-shaped and blot haemorrhages in a child with a bleeding disorder. Haemorrhages in patients with bleeding disorders are rare, usually only occur if the bleeding disorder is very severe and are usually small and well defined.

Tangential acceleration is associated with much more brain deformation and shear than an equivalent linear acceleration. This may explain the propensity for retinal haemorrhage to occur with shaking injury in infants.²⁴

Betz *et al.*⁴⁶ suggested that retinal haemorrhages 'exceeding 20–30% of the total retinal area could not be explained by a single traumatic event and must be regarded as a very strong indicator for violent shaking'. Their study, however, did not include controls and lacked some details in the groups studied.⁷⁴ Although it is much more likely that massive retinal haemorrhages are caused by a NAI, it cannot be said that less extensive haemorrhages are less likely to be associated with NAI.

The biomechanics of retinal haemorrhages: Glossary

A number of terms are used in the literature to describe various forms of acceleration:

Positive acceleration ('acceleration') results in an increase in speed, *negative acceleration* ('deceleration') results in a decrease in speed

Linear acceleration (positive or negative) is acceleration in a straight line and is measured in distance/s/s

Angular or rotational acceleration is acceleration in a plane about an axis: it is measured in units of angle (degrees or radians)/s/s. It is, in the child abuse context, usually a compound of acceleration in three planes. The sagittal plane is about an axis going roughly between the ears ('pitch'), the axial or horizontal plane is about an axis between the jaw and the top of the head ('yaw'), and the coronal plane is about an axis from the nose to the occiput ('tilt')

Tangential acceleration is the linear acceleration measured in distance/s/s, perpendicular to the radius of a circle; it results in shear forces that depend on the radius of the circle, the weight of the object and the rate of acceleration

Jerk is the term used for the rate of change of acceleration and is measured in distance/s/s/s.

The biomechanics of retinal haemorrhages

There is reliable evidence to show that retinal damage can be produced by severe angular positive or negative acceleration forces alone, without impact and without direct ocular injury: it has been suggested that this may result from direct vitreous traction on the retina.⁴⁹ The relative additional contribution of an associated sudden rise of intracranial or central venous pressure is certain.

Direct evidence that angular acceleration without impact can produce retinal haemorrhages, as suggested by Caffey,⁵ includes the finding of retinal haemorrhages in adults after emergency aircraft ejection without impact and in experiments in which subjects experienced deceleration above 40G for fractions of a second.⁷⁵

Retinal haemorrhages are also described after road traffic accidents in which there has been no direct trauma to the

eye,⁷⁶ and in 'bungee-jumpers'.^{77–79} Supportive evidence comes from retinal haemorrhages seen after confessed shaking injuries.^{4,24,76}

Much of the indirect evidence linking brain and eye injury is based on the good correlation between the severity of ocular injury and the severity of the intracerebral injury. This enables information gathered on the forces required for cerebral injury to be extrapolated to eye injury.^{47,49,57} Subdural haematomas and other cerebral haemorrhages and tears were produced by whiplash injury without impact in monkeys. This occurred only in subjects that were concussed. The acceleration threshold for injury in humans was predicted to be 6000 radians/s/s or 342 000 deg/s/s.¹⁶

The acceleration threshold for cerebral injury calculated from animal experiments was easily exceeded by shake and impact in life-sized and head-weight-equivalent infant models.²⁰ This threshold could not be reached by violent shaking alone but this may have reflected deficiencies in the model construction.⁷⁶ Not all infants with retinal haemorrhages associated with abuse have signs of impact injury.^{21,49,76,80}

No absolute value can be given for the angular acceleration forces and jerk required to produce retinal bleeding or other injury, but there is good evidence that they must be considerable. Retinal haemorrhages are not reported in quite severe household falls, even when associated with skull fractures, but occur after very severe motor accidents.^{24,71,76}

Retinal haemorrhages are associated with types of cerebral injury that have been shown experimentally to require severe concussive forces. Subdural haemorrhages (which are commonly associated with retinal haemorrhages in NAI) are formed when there is surface vascular damage during short-duration angular acceleration loading at high rates of acceleration, as in deceleration-impact injuries in falls. Diffuse axonal injury (DAI), mostly seen after motor accidents, is usually associated with coronal plane angular acceleration of longer duration and at a lower rate.⁸¹

By making comparisons with intracerebral lesions, the finding of retinal haemorrhages with additional features, such as perimacular folds, subretinal haemorrhages, choroidal haemorrhage and vitreous haemorrhage, indicates a greater severity of applied force.^{47,49}

Blunt head trauma

Blunt head trauma, in addition to angular acceleration, is probably necessary to produce the most serious eye problems in child abuse, as the deceleration produced in impact injuries is greater than that produced when the head is flexed or extended to its limits without impact.^{20,24} Elner *et al.*⁸³ found evidence of head injury at autopsy of all 10 of their cases of suspected child abuse, although the signs were sometimes occult. However, evidence of associated head injury is not always present in severe cerebral injury with coexisting eye injuries.^{21,49,55,80} Gilliland and Folberg⁷⁶ found that, of 80

infants dying of NAI, over half had impact injuries without evidence of shaking, over a third had evidence of combined shaking and impact, and the remaining minority had exclusively shaking-induced injuries.

Hypoxia and ischaemia

Hypoxia and ischaemia, sometimes ongoing, play a significant role in determining the severity of cerebral injuries, whether it is due to the nature of the trauma itself⁸⁴ or to non-specific trauma-induced apnoea.⁸⁵ It is likely that similar mechanisms apply to the severity of eye damage (Fig. 4f).

Increasing retinal haemorrhages

It is 'common sense' that retinal haemorrhages will increase in extent and severity rapidly while the trauma and its immediate effects are in progress. However, many of the intracranial events that are associated with retinal haemorrhages do progress gradually: subdural haemorrhages, for instance, may progressively enlarge⁸⁶ and further haemorrhages occur in chronic subdural haemorrhages, at least in adults.⁸⁷ Therefore, the finding of increasingly extensive intraocular haemorrhages could reflect observer error, further trauma, further haemorrhages as a result of continuation of the effects of the original trauma, or a spread of the haemorrhage from one layer or area of the retina to another.

Birth-related haemorrhages

Retinal haemorrhages are common at birth, occurring in between 2.6% and 59% of newborn infants.⁸⁸⁻⁹¹ The incidence is increased in prolonged labour, assisted delivery, toxemia, in the children of older primiparae, and possibly with parenteral vitamin E and dinoprostone induction of labour. The incidence appears to be decreased after caesarean section and breech delivery.

The finding of retinal haemorrhages in a large proportion of normal neonates⁸⁸ has caused a dilemma in some cases of suspected child abuse: are the haemorrhages due to abuse or natural causes?

There are four usual morphological types of neonatal haemorrhages: superficial retinal (splinter or flame shaped), intraretinal (dot/blot/domed), preretinal/subhyaloid (Fig. 4a) and subretinal. Birth-related haemorrhages are thought to occur during labour⁸⁸ by leakage from retinal venules and capillaries and they are similar in appearance to haemorrhages occurring after central retinal vein occlusion or raised intracranial pressure. It is suggested⁸⁸ that hypercapnia and hypoxia causes vessel dilatation and compression during labour leading to raised intracranial pressure and impaired venous return, leading to vessel rupture and haemorrhage. There seems to be no association between neonatal retinal haemorrhages and platelet count, plasma fibrinogen level or the Thrombotest result.⁹¹

Haemorrhages present after birth disappear rapidly, with very few exceptions; this has been summarised in table form by Von Barsewisch.⁸⁸ The finest nerve fibre layer (NFL) haemorrhages can disappear in 24 hours, whilst even extensive NFL haemorrhages are usually gone within a few days. Half of all haemorrhages disappear within 48 hours⁹⁰ and Sezen⁸⁹ found that 2.6% persisted at 72-120 hours after birth. In one case, the haemorrhage persisted for 6 weeks. Larger subhyaloid and larger intraretinal haemorrhages seem to persist for the longest time.⁹¹ Most neonatal retinal haemorrhages have no visual sequelae,⁹² although there are occasional cases of long-term visual impairment reported.⁹³

Many of the studies mentioned above were not truly longitudinal and further studies would be helpful.

Unilateral retinal haemorrhages

The mechanisms whereby unilateral or asymmetrical haemorrhages could occur is interesting; there could be anatomical differences in the optic nerve such as those that purportedly give rise to asymmetries in papilloedema.¹⁰¹ It is perhaps more likely that there is an asymmetry in the injury, with one eye receiving a greater insult than the other, due to the way in which the acceleration forces are distributed. This may be similar to a proposed association between unilateral ear bruising and ipsilateral cerebral oedema.¹⁰² For head injuries, shear forces are greater the more peripheral to the centre of rotation;¹⁰³ thus one eye could receive more accelerative trauma than the other. It is unlikely that there is a large enough difference in the hydrostatic forces (intracranial and intravascular pressure) to affect one eye more than the other. There may be additional local injury, enough to make one eye worse affected than the other.

Unilateral optic atrophy has been noted in a child presumed to have had an optic canal fracture.¹⁷

Unilateral retinal haemorrhages in child abuse		
Authors	Findings	Reference
Ludwig and Warman, 1984	4 of 20 cases of shaken infants with unilateral retinal haemorrhages,	22
Roussey <i>et al.</i> , 1987	2 of 10 cases of retinal haemorrhages in NAI	33
Wilkinson <i>et al.</i> , 1989	3 of 14 consecutive cases of presumed shaken baby had unilateral retinal haemorrhages	47
Budenz <i>et al.</i> , 1994	13 autopsy cases, 3 of 9 with direct head trauma, had unilateral retinal haemorrhages	57
Betz <i>et al.</i> , 1996	1 of 7 babies with 'physical child abuse' had unilateral haemorrhages on histopathology	46
Tyagi <i>et al.</i> , 1997	3 consecutive cases	100

Fig. 4. (a) Birth-related haemorrhages. There are multiple, multi-layered haemorrhages, with a few prominent subhyaloid haemorrhages in the centre of the picture. Photograph by courtesy of Dr Andrew Q. McCormick, Vancouver, BC, Canada. (b) Leukaemic macular haemorrhage. There is a whitish area which represents an aggregation of white blood cells. The white spot at the top of the haemorrhage is an artefact. (c) Subhyaloid haemorrhage in a child with profound anaemia. Generally, retinal haemorrhages in bleeding disorders are rare, small, few in number and well defined. (d) NAI. The greenish background colour of the retina that has multiple haemorrhages visible in this picture suggests longevity. The bright red vitreous haemorrhages lying in front of the retina are rounded and smooth, which is a change that evolves during organisation and resolution. The bright red colour may suggest recent haemorrhage, but the colour is dependent on so many factors that it alone is not a good indicator of the age of an intraocular haemorrhage. (e) NAI. Extensive vitreous haemorrhage with areas of yellow organisation together with a greenish background given by altered blood products suggests that the haemorrhages occurred at least several days before the photograph was taken. (f) NAI. Widespread retinal haemorrhages and a greenish background hue suggest a long-standing injury, while the pale optic disc and thinned arterioles indicate that ischaemia may have played a role.

The intracranial associations of retinal haemorrhages

The significance of the retinal haemorrhages goes far beyond the eye: there is a close correlation between the severity of retinal haemorrhages and intracranial injuries,^{47,49,104} although it is sometimes difficult to estimate the severity of or even to diagnose intracranial injury on the initial CT scan.¹⁰⁵ It is usual for NAI patients with subdural haemorrhages and other intracranial injuries to have retinal haemorrhages, but the association is not invariable;⁴⁹ between 11%¹⁹ and 39%⁵⁴ of abused children do not have retinal haemorrhages. Also, most subdural haemorrhages in children, especially in infants, are associated with child abuse and with eye abnormalities.²⁷

The differential diagnosis of retinal haemorrhages in childhood

What is the differential diagnosis of retinal haemorrhages associated with child abuse? There are numerous conditions that give rise to retinal haemorrhages in infants and children, but they all have their own special diagnostic features.

1. Leukaemia (Fig. 4b). Ten per cent of children with acute lymphatic leukaemia have intraretinal haemorrhages and 3% have white-centred haemorrhages. The leukocyte count is substantially abnormal in all cases and most are thrombocytopenic;¹⁰⁶ not all leukaemic children with retinal haemorrhages, however, have other abnormal blood parameters.¹⁰⁷ A 4-week-old child with myelogenous leukaemia and neurofibromatosis presented with a high fever that was initially suspected to be caused by child abuse.¹⁰⁸
2. Haemorrhagic disease of the newborn. There appears to be no association between the Thrombotest results and birth-related intraocular haemorrhages;⁹¹ this had been suggested in 1941.¹⁰⁹
3. Retinopathy of prematurity (ROP). ROP can be differentiated by the associated findings.
4. Sick cell retinopathy.¹¹⁰ Haematological studies and the presence of new vessels can differentiate sick cell retinopathy.
5. Neonatal arterial hypertension.¹¹¹
6. ECMO treatment. Although some patients having extra-corporeal membrane oxygenation (ECMO) have intraretinal haemorrhages and other changes,^{97,98,112,113} they are not necessarily related to the ECMO itself and are insufficiently serious or frequent to necessitate screening of ECMO patients.^{114,115}
7. Metabolic disease. Vitreous haemorrhages have been described in children with severe galactosaemia.¹¹⁶ Children presenting with the effects of glutaric aciduria type 1 frequently have subdural effusions and may have subdural haemorrhages due to rupture of bridging vessels due to cerebral atrophy.^{117,118} Retinal haemorrhages have been noted

in association with subdural haemorrhage or effusion, but a satisfactory explanation is not yet forthcoming.¹¹⁹

8. Other bleeding disorders (Figs. 3e–f, 4c). In Henoch-Schönlein purpura, transient punctate retinal haemorrhages were described in 4 of 22 children aged 8 months to 14 years.¹²⁰ Resolving haemorrhages may appear ophthalmoscopically like vitelliform lesions.¹²¹ Gayle *et al.*¹²² have said that it is necessary to exclude a bleeding diathesis in children with suspected child abuse. However, retinal haemorrhages are probably rare even in children with abnormal bleeding studies, although there is a need for studies to confirm this. Intracranial haemorrhage is infrequent after accidental head trauma in children with a congenital coagulopathy.¹²³ Extensive retinal haemorrhages have been described in two teenagers with von Willebrand's disease,¹²⁴ a common and variable disorder. Vitreous and subarachnoid haemorrhages are described in severe protein C deficiency with disseminated intravascular coagulation.^{125,126} Evidence of a bleeding disorder is not uncommon in child abuse and the two conditions are not mutually exclusive,^{127,128} this may be due to the consumption of coagulation factors in the formation of intracranial haemorrhages, whatever the cause. Advice from a haematologist or other specialist may be necessary in this situation.
9. Scurvy causes vascular fragility and bleeding. It may occur through low dietary vitamin C intake or iron overload as seen in thalassaemic patients who have repeated blood transfusions.¹²⁹ Eye manifestations are rare: subconjunctival haemorrhages and superior orbital subperiosteal haemorrhages are the most common findings.^{129,130} From the literature, it seems that ocular lesions are mostly seen in severe scurvy, although conjunctival lesions were seen early in the course of 5 of 9 adult volunteers who were vitamin C deprived.
10. Maternal ingestion of cocaine. Birth-trauma-like haemorrhages which last longer than normal have been described in neonates born to cocaine addicts.¹³¹ In addition, these children are at high risk of NAI.
11. Meningitis. Retinal haemorrhages have been associated with meningococcal meningitis.⁹⁴
12. Intracranial vascular malformation. Spontaneous subhyaloid and retinal haemorrhages occurred in an infant with an intracranial arterial aneurysm,¹³² and an optic sheath haemorrhage was described in a child with a ruptured sub-arachnoid malformation.⁵² In both cases NAI had been suspected.
13. Vitreous haemorrhage has been recorded with optic disc drusen, in tuberous sclerosis¹³³ and in X-linked retinoschisis.¹³⁴

14. Chronic, severe papilloedema associated with diagnosable intracranial pathology may cause peripapillary haemorrhages and acute, severely raised intracranial pressure may cause widespread haemorrhage.
15. Multiple retinal haemorrhages commonly occur following intraocular surgery in infants.^{135,136}
16. Retinal haemorrhages are often present in older children with severe hypertension.

Most conditions of childhood that cause retinal haemorrhages have distinguishing clinical features and further investigations are only indicated when they are clinically appropriate. A 'battery' of tests for all cases is not appropriate.

Seizures and retinal haemorrhages

In adults with seizures, retinal haemorrhages were not noted in any of 560 visits to an emergency department that resulted in 7 deaths and 553 injuries.¹³⁷ A case of extensive retinal haemorrhages was described in a 32-year-old man after status epilepticus 3 months after surgery for Arnold Chiari malformation.¹³⁸

A study in which 33 children, not including any neonates, were examined ophthalmoscopically within 48 hours of hospital admission for an episode of seizures of various types revealed no retinal haemorrhages. This was despite some of the children having vomited or having had cardiopulmonary resuscitation;⁹⁵ after this study was completed the authors reported a case of a premature infant who had seizures from 10 days and was found to have unilateral retinal haemorrhages at 23 days. A study from the same hospital¹³⁹ suggested that the upper limit of the 95% confidence interval of retinal haemorrhages following convulsions in children under 14 years old, is less than 5/100. Therefore, convulsions in young children only very rarely cause retinal haemorrhages.¹

Cardiopulmonary resuscitation, Valsalva's manoeuvre and retinal haemorrhages

Closed chest cardiac massage, even by trained personnel, causes a marked rise in intracranial pressure.¹⁴⁰ Violent chest compression in child abuse may cause retinal haemorrhages;^{62,141,142} mostly it was felt that the retinal haemorrhages predated the cardiopulmonary resuscitation (CPR). Attempted CPR by untrained individuals has been advanced as an innocent cause of retinal haemorrhages in a single case report,¹⁴³ although NAI was not convincingly excluded.

There have been other reports of retinal haemorrhages occurring after CPR without other explanation for the haemorrhages.^{99,144} One neonate with meconium aspiration who had CPR during the first day of life before being put onto ECMO suffered seizures and vitreous haemorrhages were noted on the 11th day.⁹⁸ Gilliland and Luckenbach¹⁴⁵ found no case with retinal haemorrhages in their autopsy study of 169 cases, 131 of which had had prolonged resuscitation.

Kanter,¹⁴¹ a paediatrician, found retinal haemorrhages on non-mydriatic ophthalmoscopy in only 1 of 45 children who had not had prior trauma and were successfully or unsuccessfully resuscitated: that child had severe arterial hypertension and seizures. Of 9 trauma victims, 5 had retinal haemorrhages; 4 of these were correctly suspected of being due to child abuse and the other had head and chest injuries in an automobile accident. He could not examine a further 5 children who had corneal clouding or miosis. He concluded 'When retinal haemorrhage is detected in the pediatric patient after CPR, prior trauma should be assumed. Retinal haemorrhage should not be attributed to the mechanical effects of CPR'. This study¹⁴¹ may have contained a number of methodological weaknesses.¹⁴⁶

Experimental evidence from studies on piglets showed no retinal haemorrhages despite high, monitored intracranial and intrathoracic pressures,¹⁴⁷ although there are a number of differences between pig and human retinas.

CPR alone is therefore very unlikely to cause retinal haemorrhages, even if carried out by unskilled individuals.¹

Valsalva's manoeuvre may be associated with retinal haemorrhage in adults,^{148,149} but with the possible exception of pertussis, no cases have been described in children.¹

The clearing of retinal haemorrhages

The rate of clearing of retinal haemorrhages is important for determining prognosis, in the understanding of the disease process and sometimes in medicolegal matters; it is not as clearly understood as changes with time in skin bruises, which may show changes that allow approximate dating of the injury.¹⁵⁰ In the eye, however, the colour changes that occur in skin bruising are different, the changes are so gradual and the retinal background contrast so variable that accurate dating of retinal haemorrhages to within days, based on their ophthalmoscopic appearance, is not possible (Fig. 4d-f). The colour of a retinal haemorrhage is influenced at least as much by its size as by its age. Brown-yellow ochre membranes are consistent with injuries at least 1 month old, and the presence of bright red fresh haemorrhage together with ochre membranes strongly suggests haemorrhages of different ages.¹

Mild retinal haemorrhages in NAI probably clear rapidly, 'flame-shaped' birth-related haemorrhages may resolve within 24 hours^{89,91} and moderately severe retinal haemorrhages clear within a few weeks.¹⁵¹ Severe, widespread, retinal and vitreous haemorrhages may take many months to clear, and usually are associated with substantial residual damage.^{152,153} If subretinal and intraretinal haemorrhages are found in the absence of flame-shaped haemorrhages this may suggest that the injury occurred at least 24 hours previously because of different clearing rates, but there are no studies to corroborate this. Dating and timing can be very difficult on morphological grounds alone.¹⁵⁴

Fig. 5. (a), (b) Photomontage of bilateral perimacular folds in NAI. In both eyes a roughly circular ridge can be seen extending from the optic disc to surround the macula. The apex of the ridge is pale and in some areas the elevation of the folds is revealed by a shadow cast from the photographic flashlight. (c) Perimacular folds. When there are extensive retinal or vitreous haemorrhages perimacular folds are either obscured or difficult to see. The presence of a perimacular fold can be assumed from the curvilinear pale line seen below the optic disc. (d) NAI. A vitreous haemorrhage obscures widespread retinal damage. In child abuse, if there is a vitreous haemorrhage there are usually widespread retinal haemorrhages and damage which, though not visible ophthalmoscopically, may be detected by electrophysiology. (e) Perimacular fold. The elevation of the fold can be seen because of the shadow at its border.

Pathological studies suggest that the finding of haemosiderin in a haemorrhage suggests it is more than about 3 days old,¹⁵⁵ although in current forensic practice it is generally agreed that haemosiderin begins to appear around 48 hours after injury in bruises in skin and subcutaneous tissue. If there are some haemorrhages with

and some without haemosiderin, this suggests that the haemorrhages are of different ages,^{83,156} but it is possible that there are minor differences in the biochemical ageing of haemorrhages at different sites or even within a single organ. There is no detailed research giving the time of appearance of haemosiderin in retinal tissues.

The persistence of haemosiderin following birth-related haemorrhage at any site is also poorly documented; current opinion favours the disappearance of haemosiderin from soft tissues within 6–8 weeks after birth. Xanthochromic staining of the relatively avascular dura may persist for much longer. If haemosiderin is found in the relatively vascular eye tissues in infants of more than 2–3 months old it is likely that this is from a haemorrhage occurring after birth.

Intrascleral haemorrhages

Haemorrhages in the sclera around the optic disc have been noted histopathologically.^{60,67,83} This finding is strongly associated with trauma,⁷³ particularly child abuse^{83,154} in an appropriate setting. A proposed mechanism is that acceleration and deceleration motion tears the intrascleral ciliary vessels that form the circle of Zinn and Haller;⁶⁰ the bilaterally seen in many cases suggests that the mechanism is head motion rather than eye or localised trauma.

Optic nerve sheath haemorrhages

Haemorrhages in and around the optic nerve sheath are common in fatally or otherwise seriously injured children.^{49,56–58,67,73,83} When found together with retinal haemorrhages they are very suggestive of child abuse,¹⁵⁴ but, like any other injury, cannot be considered pathognomonic.

When at the optic nerve–scleral junction, the pathogenesis may be similar to that of intrascleral haemorrhage,⁶⁰ the haemorrhage occurring in the sclera and the adjacent optic nerve sheath. Elsewhere in the optic nerve, a sudden rise in intracranial pressure,^{157,158} and cycles of acceleration and deceleration,^{56,67} are the most likely causes, giving rise to haemorrhages in and around the optic nerve subarachnoid space.

Perimacular retinal folds

There are no fundus abnormalities that can be described as pathognomonic of child abuse. Severe retinal haemorrhage and perimacular folds are frequently seen in NAI,^{56,83,154,159,160} but they cannot be said to be pathognomonic¹⁶¹ as they occur in severe accidental injury and Terson's syndrome.⁹⁶ However, they are much more likely to be seen in child abuse. The presence of perimacular folds (Fig. 5a–e) may suggest that there have been cycles of acceleration and deceleration,^{56,160} perhaps also with impact.⁸³ Perimacular folds may be unilateral.¹⁵⁴

Defects of pupil reactions

With very severe intraocular or intrasheath haemorrhages, amaurotic pupils may be found. If there is an interocular difference in the extent of the damage, a relative afferent defect may be found.

Third nerve and other cranial nerve palsies are due to intracranial events. The observation of pupil reactions is a vital part of the monitoring of many of the children admitted to hospital with head injuries. It may be so important that the paediatrician or intensivist in charge of the case may not wish pupil-dilating drops to be instilled: this precludes a full examination of the fundus, but direct ophthalmoscopy, or the use of a small pupil indirect ophthalmoscope can help. Short-acting mydriatics, such as tropicamide 0.5–1%, may be useful.

Retinal detachment

Retinal detachments are seen after indirect injuries in child abuse¹⁶² and because they suggest greater trauma,⁴⁹ not necessarily direct (Fig. 7d), has taken place, they are often associated with severe intracranial injuries.⁴⁹

Retinal pigmentation

Peripheral retinal pigmentation or chorioretinal lesions have been noted, presumably due to the various effects of trauma.¹⁶² The frequency of peripheral subhyaloid haemorrhages in less severe trauma⁴⁹ may account for the peripheral location.

Ocular sequelae

Because follow-up is frequently erratic in NAI, an accurate prognosis is impossible. It is certain, however, that those with eye signs at the time of admission have a high incidence of residual eye defects¹⁶³ and a poor visual prognosis³³ due to both eye (Fig. 6a–f) and brain damage, especially if the eye problem is severe.¹⁵²

Haemorrhagic retinal cysts, retinoschisis^{83,154,164,165} and retinal detachments (Fig. 7d) are probably much more frequent in child abuse than in accidental trauma. Optic disc neovascularisation has been described following severe retinosclerosis in a severely shaken infant.¹⁶⁶

Systemic signs of child abuse

Cerebral trauma and subdural haemorrhage

Cerebral oedema may be ipsilateral to unilateral ear bruising due to the shared rotational acceleration affecting one side of the head more than the other.¹⁰² As in the eye, timing of injury is difficult; the CT changes are not really time-specific.¹⁶⁷ From the ophthalmologist's perspective children with non-accidentally inflicted head injuries may present in a number of ways, including cranial nerve palsies (Fig. 7a–c), headaches in older infants and children, or suspected poor vision.

Bruising, burns and skin marks

Examination of the child for bruising with a Woods (ultraviolet) lamp may help in dark-skinned children. It is important to note any tenderness: bruises can, and should, be photographed but tenderness cannot be, so this information should be recorded. Tenderness may be

Fig. 6. (a), (b) NAI. Bilateral profound optic atrophy with some prepapillary fibrosis resulting from severe retinal and vitreous haemorrhage. (c), (d) NAI. Optic atrophy and marked macular pigment disturbance. (e), (f) Severe optic atrophy and preretinal fibrosis resulting from widespread retinal and vitreous haemorrhage.

present especially if there are underlying fractures. Bruises cannot be easily aged and may change in colour at different rates in different sites or if they are of different volume even if of the same age (Figs. 1a, 7e, 8a).

It is important to know the prevalence (12%) and distribution of bruises in normal children.¹⁶⁸ They are on the front of the body, mostly single or only a few, on the face, head and shins, in mobile children.

Bruises cannot reliably be dated even by an experienced clinician, but bruises with some yellowing are likely to be more than 18 hours old. The question of whether a bruise is older or younger than another can only be answered on a 'balance of probabilities basis'.¹⁵⁰

The clinician should pay particular concern to bruising around the head, especially behind the ear (Fig. 7f), subcapsular bruises, and if there are finger

marks at shaking sites or nail gouges (Fig. 7e). Careful photography can identify a particular bite (note that bites can be self-inflicted). If the child has died, consider calling in the police forensic photographer if there is not a photographer with appropriate experience available.

Burned children may have clues to the non-accidental nature of the injury from the clinical history, the social background or the examination,^{29,169} and if there is any suspicion they should be appropriately investigated. They tend to be younger children; if the burns involve the buttocks or perineum they are more likely to be non-accidental.¹⁷⁰

Cigarette burns can be accidental, unless multiple or linear, instead of round (Fig. 8b), especially if they are at different stages of healing or associated with other burns or scalds.¹⁷¹ Accidental scalds usually have splash marks.

Fig. 7. (a), (b) NAI. A 2-year-old child who presented with a sudden onset squint after a few days of irritability. In (a) there is failure of abduction of the right eye and in (b) there is failure of abduction of the left eye due to a bilateral sixth nerve palsy. (c) Same patient as in (a) and (b). There is bilateral mild papilloedema. There was a subdural haematoma on CT scanning. The childminder was prosecuted for child abuse. (d) Retinal detachment in a child with brain damage admitted to be due to child abuse 5 years after the injury. (e) NAI. Nail gouge on a child's arm. (f) NAI. Post-auricular bruising from ear pulling. A tear of the frenulum is another common injury in child abuse.

Fractures

Radiographs are important even if the child has died. This is the province of the paediatrician, radiologist or pathologist, not the ophthalmologist. Some fractures, such as rib and metaphyseal fractures, may not be apparent from clinical examination alone.¹⁷²

Ribs

Rib fractures in young children are unusual and therefore suspicious. Most rib fractures in infants are caused by NAI. Rib fractures in children are rarely the result of compression from CPR.¹⁷³ Costovertebral fractures are hard to see, especially when acute on

Fig. 8. (a) Finger marks on a baby's face after a slapping injury. Direct ocular injury should be suspected in such injuries. (b) Purported cigarette burn. Linear or multiple cigarette burns are likely to be due to child abuse. (c) Subdural haematoma in NAI. The upper MRI scan shows an obvious bilateral haemorrhage with a fluid level on the right side of the picture; the lower MRI scan shows a subtle subdural collection. (d) MRI scan of a bilateral subacute subdural haemorrhage with membrane formation. (MRI scan courtesy of Dr Kling Chong, Great Ormond Street Hospital.)

Procedures for the ophthalmologist who suspects child abuse: The Royal College of Ophthalmologists Statement

All NHS Trust hospitals that deal with children should have a *Named Doctor (ND)* and *Named Nurse (NN)* with particular expertise in child protection. They have responsibility for providing appropriate training and dissemination of local child protection guidelines. The ND is usually a consultant paediatrician, but in an Eye Hospital the ND may be an Ophthalmologist who will need to have links with the Named Doctor (Paediatrician) in a neighbouring trust.

In addition, every Health Authority or Board appoints a *Designated Doctor* and *Designated Nurse* in child protection, who are available for advice.

Local guidelines should be readily available to all staff working with children: they identify key personnel together with relevant telephone numbers including those of the local Social Services and the Police Child Protection offices.

Suspecting abuse or neglect

Many forms of child abuse may involve the eye and they may coexist. The Ophthalmologist mainly encounters physical abuse (indirect trauma, shaking, smothering and direct eye trauma), and occasionally induced illness (Munchausen syndrome by proxy), sexual abuse, neglect, and emotional abuse.

What to do if you suspect child abuse or neglect

Professionals should not intervene on their own and all suspicions should be discussed with the hospital social worker and ND. When child abuse is felt to be occurring there is a responsibility to inform the Social Services office.

1. If a trainee *suspects* abuse or neglect, there should be immediate *consultation* with a senior colleague, the senior nurse of the ward or department, and the consultant Ophthalmologist in charge of the case to confirm suspicions of abuse.
2. There should be early *consultation* with the Named Doctor and Named Nurse, who will frequently be responsible for the further investigation and general medical management. In Trusts where there is no Named Doctor, the consultant Ophthalmologist or the Paediatrician in charge of the case should decide the lines of responsibility and discuss the case with the Designated Doctor.
3. *Admission* may be necessary if the named doctor is not readily able to see the child or if there are grave injuries or serious suspicions about the immediate risks to the child. A full history must be taken and an examination of the patient made, including non-ocular areas of the body if the Paediatrician has not yet become involved. There must be full *documentation* of the history, including what is said by all parties, and

the physical findings must be noted, with annotated drawings and photography where possible. Early involvement of a paediatrician is advisable.

4. If, after consultation, abuse or neglect is still considered a possibility, a *referral* will be made by the Named Doctor to the Social Services Department, via the hospital's social worker, if there is one, or directly if there isn't. The responsibility for investigating suspected child abuse lies with the Local Authority Social Services department and the Police Child Protection team.

Presentations, injuries or behavioural states which should alert the clinician

Children at risk

Premature, handicapped, and crying babies.
Siblings of abused children.
Children of previously abused parents.

Worrying factors in the presentation

The account of how the injuries occurred is inconsistent with their appearance.
The apparent age of injuries is inconsistent with the account given, or a delay in presentation.
Unexplained injuries.
Injuries blamed on siblings.
Multiple attendances at A&E departments.
An unusual lack of parental concern at the severity or extent of the injuries.

Eye signs suggestive of abuse

Retinal haemorrhages.
Periocular bruising, lid lacerations.
Unexplained lens dislocation or cataract.
Unexplained conjunctival or corneal injuries, especially in the lower half of the eye.

Other signs of abuse

Head or face injuries in infants or non-mobile children.
Subdural or subarachnoid haemorrhages.
Bite marks, scalds or fingertip bruising.
Cigarette burns, especially if multiple.
Unusual injuries in inaccessible sites, e.g. neck, armpits, groin.

Neglect

When a child presents dirty and unkempt or where there is worrying (e.g. aggressive, hyperactive) behaviour, this should be discussed with the hospital social worker and consideration given to discussing this further with the GP or Health Visitor. Similar procedures should be observed when parents behave aggressively towards their children, or show unusual behaviour towards hospital staff. This particularly applies if drug or alcohol abuse is suspected.

Principles

Informed Consent to medical examination should be obtained from an adult with parental responsibility for the child, and from the child, in a manner appropriate for age and level of understanding. Medical examination can be carried out with only the child's consent when, in the opinion of the doctor, the child has sufficient understanding.

Refusal to give consent. If the carer or the child refuses to give consent or to co-operate with admission or treatment, the doctor should inform the Consultant in charge or the Named Doctor immediately: it may be necessary to consider emergency legal action, initiated by the Social Services Department or the Police.

Children's rights. Children have a right to know what is going on. They should not be made promises that cannot be kept, and their views and wishes should be taken into consideration. They should be given the opportunity to explain what has happened to them, but probing and confrontational 'disclosure' interviews should not be carried out. Physical examinations should be few, and carried out in a suitable environment by appropriately trained staff and in the presence of a trusted adult.

Parents' or carers' rights. Carers are entitled to know what is going on and to be helped to understand the steps being taken, but the child's welfare is paramount. If the child is under a Child Protection Order or accommodated by the Local Authority, arrangements for contact with the family should be clarified with Social Services.

Evidence. Therapeutic needs take precedence over evidential requirements. Accurate and unbiased records are essential for case conferences and legal proceedings which it may be the Ophthalmologist's duty to take part in.

References

1. Working together under the Children Act 1989. London: HMSO, 1991.
(Child Protection: Medical Responsibilities, Child Protection: Arrangements between the NHS and other agencies, are addenda to the above)
2. Child abuse and the eye. Report of the British Ophthalmology Child Abuse Working Party. Eye 1999;13:3-10.
3. Duhaime A-C, *et al.* Non-accidental injury in infants. N Engl J Med 1998;338:1822-9.
4. Handle with Care. NSPCC document for parents. National Centre, Curtain Road, London EC2 3NH.

This document was prepared by the Ophthalmology Child Abuse Working Party (ref. 2), March 2000.

standard anteroposterior radiographs, as they overlie the region of the transverse process and may just appear as slight widening of the neck of the rib. Similar forces to adjacent ribs lead to a line of fractures or 'string of pearls'. Anterolateral thoracic compression during shaking is the probable mechanism of the injury of multiple posterior rib fractures near the costo-transverse articulation, caused by the abuser shaking in an anteroposterior direction with the palms laterally and thumbs on the chest. Single fractures and costochondral junction fractures¹⁷⁴ may be due to local impact and may involve injuries to the viscera.

Scapula

Scapular fractures are difficult to identify but rare except in massive direct trauma or child abuse. The acromion is the most common site of fracture, e.g. avulsion where the deltoid muscle attaches or where the latissimus dorsi and serratus anterior attachments are stretched by dragging by the arm. They are unusual fractures and any unusual fracture is considered suggestive of child abuse.

Limbs

Certain fracture patterns are suggestive of NAI.^{3,175} avulsion of a metaphysis, chipped edges of a metaphysis or an epiphysis with isolated small fragments. Metaphyseal injury is easily missed unless good-quality radiographs are taken.¹⁷⁶ Metaphyseal lucency and a 'bucket-handle' appearance are the same lesion. Periosteal new bone formation suggests that the injury is 7-10 days old, which may be significant if there are fractures of different stages of healing. Spiral diaphyseal fractures of long bones are highly suspicious (but not pathognomonic) of NAI if found in children too young to be very mobile. A rotational force applied to the extremity during vigorous handling and shaking causes spiral and oblique fractures.

Investigation of suspected child abuse

The minimum investigations suggested by Jayawant *et al.* in addition to the paediatric assessment and management²⁷ for subdural haemorrhages were: Full multidisciplinary social assessment. Ophthalmoscopy by an ophthalmologist. Skeletal survey, supplemented by a bone scan or a repeat survey at around 10 days. Coagulation screen. CT or MRI (Fig. 8c, d).

The doctor's attitude

Physicians, because of their own feelings and their difficulty in playing a role that they find hard to assume, may have great reluctance in believing that parents were guilty of abuse. They may also find it difficult to initiate proper investigation so as to ensure adequate management of the case. 'Above all, the physician's duty

and responsibility to the child requires a full evaluation of the problem and a guarantee that the expected repetition of trauma will not be permitted to occur.¹⁶

Whilst being compassionate for the child and the family in the situation that they find themselves in, the doctors involved must be dispassionate in that they need to remember who the patient is, and what the consequences are of any management decisions made. Above all they need to be non-judgemental.

Giving evidence in court

The ophthalmologist is often called upon to give vital evidence in court, frequently in a murder or manslaughter case. The consequences of the verdict, whether of guilty or not guilty, to the accused, to the child, to the family and to friends are so colossal that the medical evidence must be rigorously accurate. An incorrect guilty verdict can have consequences as severe as a correct one.

The evidence given needs to be based on the following principles:

1. Factual accuracy. It is vital that the factual evidence is as accurate as possible. This depends on detailed note-taking during the child's admission (often many months or years before the court case), good preparation and clear presentation. It was not long ago that certain physical signs alone were given credence as being pathognomonic of child abuse. One of these was perimacular folds, which, although highly suggestive of NAI, are found occasionally in natural injuries.
2. Impartiality. Advocacy is not the medical expert's role, but from time to time an expert gives evidence that is biased by his or her personal views and preconceptions. Fortunately, judges and juries usually detect the more obvious examples, but evidence that is or appears weighted in one or other direction can obscure the truth or obstruct the path to justice.
3. Clarity. All written statements and presentations to the court must be as clear and unambiguous as possible. As an expert witness, it is essential to choose words carefully and deliberately so that what is said is meant and what is meant is said.

Prevention

Caffey⁵ supported the need for 'a massive nation-wide educational campaign to alert everyone responsible'. There may be further need for campaigns that draw parents' and other carers' attention to the fact that the effects of shaking may be more severe than expected.¹⁷⁷⁻¹⁷⁹ The ambitious 'FULL STOP' campaign of the National Society for the Prevention of Cruelty to Children is under way in the United Kingdom. Education of personnel involved with childcare is also important.¹⁸⁰

Conclusion

It is evident that the role of the ophthalmologist in child abuse is an important one, and some of the ophthalmologist's duty to the patient and to the court is based on rather shaky foundations. Further studies and evidence-based research are needed.¹

3. Self-inflicted injury

In young adults, self-inflicted injuries often occur as part of Munchausen's syndrome¹⁸¹ in which patients inflict a variety of injuries on themselves to deceive their doctor into believing that they have a natural disease. No medical speciality is free from such patients, and their self-injurious behaviour, which is usually part of a more widespread psychiatric disorder, demands as much or more skill and care as any other condition, as the prognosis can be very poor.¹⁸² Munchausen's syndrome is a rather flamboyant term but it is useful in that the characteristic that separates it from other self-inflicted injuries is the deception, resulting often in hospital admission, and the mimicry of a natural disease process. The eye and adnexae can be affected in many ways in adults: by self-inflicted orbital emphysema,¹⁸³ voluntary nystagmus, periorbital cellulitis, corneal ulcers or alkali burns.¹⁸² In children, the underlying disorder is different, and the most common disorders are dermatitis, keratoconjunctivitis, ocular self-mutilation and trichotillomania. Ocular Munchausen's syndrome, however, is occasionally recognised in older children: a case was described in an 11-year-old Italian girl who induced a keratoconjunctivitis with chalk grains.¹⁸⁴

Dermatitis and keratoconjunctivitis

Self-inflicted injury to the surface of the eye and the skin around the eye is rarely recognised in childhood, but it is important in that it may be a warning of much more serious problems affecting the child such as physical or sexual abuse. In adults there are often a number of associated complex psychological and emotional problems;¹⁸⁵ sometimes there may be more serious underlying psychopathology.¹⁸⁶ When dermatitis or keratoconjunctivitis artefacta affects adults with a long history of psychiatric illness¹⁸⁷ recovery only occurs with a change in the patient's circumstances.¹⁸⁷ Some adult patients are malingerers, classically trying to escape military service.

It is important to distinguish between benign neurotic excoriations, in which the patient admits to the self-destructive nature of their problem, and factitious disease.¹⁸⁸ In the latter the patients deny the cause of their injury, are deceptive and their motivation is to play the sick role. They are much more difficult to treat, requiring skilled psychiatric intervention.

The diagnosis is suggested by the combination of sharply delineated lesions in the inferior and nasal quadrants of the cornea and bulbar conjunctiva, an unconcerned attitude ('belle indifférence') and other psychological features.¹⁸⁹ The dermatitis is very variable in its character (Fig. 9a).

Fig. 9. Self-inflicted dermatitis around the eye and cheek. (b) Trichotillomania of the eyelashes. This boy was the subject of a lot of stress-related conflict with his ambitious father. (c) Autoenucleation. Photograph by courtesy of Dr John Crompton and Dr George Pietris, Adelaide. A young adult 'born-again' Christian male suffered from religious preoccupations for 3 days, then had an experience where he 'could look into the rest of his life and saw he was a child-molester'. He plucked out both eyes using no instruments: he had no pain and little bleeding, one optic nerve had part of the chiasm attached.

It is important to rule out congenital anaesthesia,¹⁹⁰ which most frequently occurs in young children. In children, self-inflicted injuries mostly occur in mentally retarded children, especially those with Lesch-Nyhan and Smith-Magenis syndromes.

Trichotillomania

Trichotillomania is a common condition, with a prevalence of between 1% and 5%,¹⁹¹ in which the affected person pulls out hair, sometimes including the

eyelashes and eyebrows (Fig. 9b). Loss of eyelashes occurs with skin diseases such as alopecia, ichthyosis, ectodermal dysplasia and others, following radiation or chemotherapy, after lid infections or endocrine disease. For dermatitis to induce loss of lashes it must be at least moderately severe.¹⁹² A full differential diagnosis is given by Mawn and Jordan.¹⁹³ The diagnosis is often one of exclusion.¹⁸⁵

The onset is often in youth;¹⁹⁴ boys, although affected about half as frequently as girls, have an earlier age at onset (8 years)¹⁹¹ but are less likely to pull out their

eyebrows and eyelashes.¹⁹² Pulling scalp hair from one or more sites is the most usual manifestation in both sexes. Sometimes the affected person has a sensation of irritation or itching which can be intense.¹⁹⁵

Trichotillomanics frequently conceal their habit. The hair pulling can become ritualised, with close inspection of the pulled hair, and the hair may be swallowed. Examination of the lids or brows shows healthy skin with multiple broken hair shafts.

Trichotillomania in childhood is not usually thought to be a serious condition,^{196–198} as the hair loss is reversible on cessation of the hair pulling, but sometimes it may become a severe chronic illness.¹⁹⁴ Some patients are depressed, or may have an obsessive-compulsive disorder. Stress is implicated as a precipitating factor in many cases: imminent examinations, parental divorce or separation,¹⁹¹ teasing or bullying.

Management is best carried out in association with a dermatologist, to reassure that the skin is healthy, a psychiatrist or general practitioner. Most cases respond to reassurance and explanation; some require behaviour therapy and drug treatment may sometimes help.¹⁹⁹

Ocular self-mutilation: direct eye trauma

Ocular self-mutilation typically occurs in young men with a history of severe psychiatric illness, especially schizophrenia, serious criminality or drug abuse.²⁰⁰ Many have committed other acts of self-mutilation.²⁰¹ The injury can be performed in many ways: knife, razor blade or scissor²⁰² lacerations, sharpened domestic articles,²⁰³ injection of substances²⁰¹ or direct blows.

In childhood, self-inflicted injuries occur in the severely mentally retarded. The injury may be by laceration,²⁰⁴ but is more frequently a result of severe direct blows²⁰⁵ or headbanging.²⁰⁶ It is important to be sure that the injuries are a result of self-inflicted injury and that they are not inflicted by carers, as these children are at risk for child abuse. Self-inflicted injuries occur more frequently in certain syndromes, mostly associated with severe mental retardation; the London Dysmorphology Database²⁰⁷ lists 15 syndromes, of which Lesch-Nyhan and Smith Magennis are the most important to ophthalmologists.

Autoenucleation

Autoenucleation, or Oedipism, is rooted in history. The most well known reference is in the Bible, Matthew 5:29, wherein Jesus says 'and if thy right eye offend thee, pluck it out, and cast it from thee'; there is a similar passage in Matthew 18:9. Whether these were meant literally is still open to debate.

In Sophocles' play *Oedipus Rex*, Oedipus cleansed himself of the sin of marrying his own mother, Queen Jocasta, and killing his father, the Theban king, by gouging out his own eyes using a piece of his mother's jewellery. Saint Lucy of Syracuse,²⁰⁸ now the patron saint of ophthalmology, was a pious Sicilian girl who looked

on a man lustfully and, in proper remorse, plucked out the eyes that had offended to give them to God. He, in His permissive wisdom, gave her two more, so that 'she then had two good ones as well as the two squashy ones'.²⁰⁹ Saint Triduana of Scotland,²¹⁰ and Saint Medana of Ireland, both carried out autoenucleation to impress their suitors, but, in Trevor-Roper's²⁰⁹ words, 'both missed out on the replacements.'

There have been a number of case reports, but the act is probably very rare: it is the stunning effect that it has on all persons involved in each case that prompts publication of so many cases. There have been some useful reviews.^{201,208,211–213} Because sophisticated instruments are not necessary it has probably been performed for aeons: the first published case in recent years was in the German literature in 1846.^{214,215}

Mostly, hallucinating young adult schizophrenics of either sex perform autoenucleation and coexisting drug and/or alcohol abuse is frequent.²⁰¹ It has been suggested that autoenucleation is unique to Western Christian culture,²¹⁶ because of literal interpretation of the Christian biblical text by religiously preoccupied persons psychotic at the time of the act. Rarely, mentally retarded children²¹⁷ perform it. They usually manage to remove more optic nerve than an ophthalmologist does using fancy instruments,^{209,218} although the removal of parts of the chiasm²¹⁹ is an unwelcome extension of this skill (Fig. 9c). Frequently, Matthew 5:29 is cited by the delusion victim as the justification for the act. They often have cognitive distortions, sexual deviancy²²⁰ and other self-injurious behaviour.^{201,220,221} The removal of one or both^{222,223} eyes is performed with or without the aid of instruments such as scissors, teaspoon handles, chisels or knives,²²⁴ and most autoenucleators report little pain.

The diagnosis is usually self-evident but the self-inflicted nature of the injury is sometimes concealed by a history of accidental trauma or spontaneous proptosis that is compatible with orbital malignancy.²²⁵

If the eye has not been irretrievably damaged visual recovery can occur: systemic steroids may help.²²¹

The surgical management consists, most importantly, of preservation of as much conjunctiva as possible, creation of fornices, removal of any eye remnants, leading ultimately to the fitting of an artificial eye. Antibiotic cover is essential.

All patients need to be under the care of a psychiatrist as they are all suffering or have recently suffered from one or another form of acute psychosis and further self-injury or suicide²²⁶ may occur.

4. Induced illness: Munchausen's syndrome by proxy

Munchausen's syndrome was a term used to describe adults who presented themselves with false illness stories.¹⁸¹ The term was adapted by Dr Roy Meadow²²⁷ who used the term 'Munchausen's syndrome by proxy' to apply to children who were presented with a false illness story by someone else (a proxy). The illness needs to meet the following criteria:²²⁸

1. Intentional production of feigning of physical or psychological signs or symptoms in another person who is under the individual's care.
2. The motivation of the perpetrator's behaviour is to assume the sick role by proxy.
3. External incentives for the behaviour (such as economic gain) are absent.
4. The behaviour is not accounted for by another mental disorder.

These criteria have the advantage that they limit the use of the term to those cases in which the perpetrator's behaviour is motivated by the need to assume the sick role by proxy²²⁹ or another attention-seeking behaviour. This thus excludes a number of other forms of child abuse, overanxious parents, 'doctor shopping' and others. Caution needs to be exercised to avoid including parents who exaggerate genuine symptoms or signs.²³⁰

The perpetrator is most frequently a woman but men are also implicated without the active collusion of the child's mother.²³¹ The perpetrator usually has a history of psychiatric problems, including Munchausen's syndrome^{231,232} or other factitious illnesses. Other events in the home, such as fire or death of pets, or a history of fabricating vainglorious or bizarre stories around their lives, are other features of perpetrators.²³¹

Although the clinical manifestations are protean, ocular involvement in Munchausen syndrome by proxy is rare; orbital cellulitis has been caused by the probable injection of Alum by the victim's grandmother who also suffered from Munchausen syndrome⁴⁰ and pupil dilatation was a feature in the child of a potential trainee emergency room technician.²³³ The dividing line between Munchausen's by proxy and child abuse can be quite fine; for instance Taylor and Bentovim's cases⁴² have been described as Munchausen syndrome by proxy in various publications, but the perpetrator's behaviour was abusing, not meeting the criteria listed above.

Diagnosis can only be made by due vigilance to the possibility of an induced illness; patients in whom the signs and symptoms do not fit with recognised disease patterns need to be carefully analysed because a wrong diagnosis can have disastrous consequences. For inpatients or institutionalised children, video surveillance may have a role, sometimes with dramatic consequences.²³⁴

The therapeutic approach varies with each case, child protection procedures need to be considered in every case, psychiatric and social worker involvement is usual, and the initial and long-term placement of victims is paramount. The prognosis, however, is poor,^{235,236} for the victim and for siblings, and long-term follow-up by a paediatrician is essential.²³⁶

Finally, the words of Meadow²²⁷ deserve to be repeated 'This paper is dedicated to the many caring and conscientious doctors who tried to help these families, and who, although deceived, will rightly continue to believe what most patients say about their children, most of the time.'

I would like to thank my colleagues in the Child Abuse Working Party (reference 1) for their help and particularly Isabelle Russell-Eggitt, who helped with the sections dealing with systemic signs. Ken Nischal and Lorraine Cassidy read and commented on the manuscript. James Badenoch QC made helpful comments on the section on giving evidence in court. Dr Christine Hall has been a helpful colleague for many years and has generously contributed to the section on fractures. Dr John Crompton and Dr George Pietris gave Figure 9c. The Iris Fund for the Prevention of Blindness and the Ulverscroft Foundation have generously supported our work at Great Ormond Street for many years.

References

1. The Ophthalmology Child Abuse Working Party. Child abuse and the eye. *Eye* 1999;13:3-10.
2. Tardieu A. Étude médico-légale sur les sévices et mauvais traitements exercés sur des enfants. *Annales d'Hygiène Publique et de Médecine Légale* 1860;13:361-98.
3. Caffey J. Multiple fractures in the long bones of infants suffering from chronic subdural haematoma. *AJR* 1946;56:163-73.
4. Caffey J. The whiplash shaken infant syndrome: manual shaking by the extremities with whiplash-induced intracranial and intraocular bleedings, linked with residual permanent brain damage and mental retardation. *Pediatrics* 1974;54:396-403.
5. Caffey J. On the theory and practice of shaking infants: its potential residual effects of permanent brain damage in mental retardation. *Am J Dis Child* 1972;124:161-9.
6. Kempe CH, Silverman FN, Steele BF, Droegenmueller W, Silver HK. The battered child syndrome. *JAMA* 1962;181:17-24.
7. Kiffney GT. The eye of the 'battered child'. *Arch Ophthalmol* 1964;72:231-3.
8. Gilkes MJ, Mann TP. The fundi of battered babies. *Lancet* 1967;II:468.
9. Harcourt B, Hopkins D. Ophthalmic manifestations of the battered baby syndrome. *BMJ* 1971;III:398.
10. Jensen AD, Smith RE, Olson MI. Ocular clues to child abuse. *J Pediatr Ophthalmol* 1971;8:270-2.
11. Mushin A, Morgan G. Ocular injury in the battered baby. *Br J Ophthalmol* 1971;55:343-7.
12. Mushin AS. Ocular damage in battered baby syndrome. *BMJ* 1971;III:402-4.
13. Kempe CH. Paediatric implications of the battered baby syndrome. *Arch Dis Child* 1971;46:28-37.
14. Lazoritz S, Baldwin S, Kini N. The whiplash shaken infant syndrome: has Caffey's syndrome changed or have we changed the syndrome? *Child Abuse Neglect* 1997;21:1009-14.
15. Oliver JE. Successive generations of child abuse. *Br J Psychiatry* 1985;147:484-90.
16. Ommaya AK, Faas F, Yarnell P. Whiplash injury and brain damage: an experimental study. *JAMA* 1968;204:285-9.
17. Friendly DS. Ocular manifestations of physical child abuse. *Trans Am Acad Ophthalmol Otolaryngol* 1971;75:318-32.
18. Sedlak A. Study of national incidence and prevalence of child abuse. Bethesda, MD: Westat, 1987.
19. Billmire ME, Myers PA. Serious head injury in infants: accident or abuse? *Pediatrics* 1985;75:340-2.
20. Duhaime AC, Gennarelli TA, Thibault LE, Bruce DA, Margulies SS, Wiser R. The shaken baby syndrome: a clinical, pathological and biomechanical study. *J Neurosurg* 1987;66:409-15.
21. Hadley MN, Murphy A, Rekate HL, Sonntag V. The infant whiplash-shake injury syndrome: a clinical and pathological study. *Neurosurgery* 1989;24:536-40.

22. Ludwig S, Warman M. Shaken baby syndrome: a review of 20 cases. *Ann Emerg Med* 1984;13:104-7.
23. Duhaime A-C, Christian CW, Rorke LB, Zimmerman RA. Nonaccidental head injury in infants: the 'shaken-baby syndrome'. *N Engl J Med* 1998;338:1822-9.
24. Duhaime AC, Alario AJ, Lewander WJ, Schut L, Sutton LN, Seidl TS, *et al.* Head injury in very young children: mechanisms, injury types and ophthalmologic findings in 100 hospitalised patients younger than two years of age. *Pediatrics* 1992;90:179-85.
25. Golstein B, Kelly MM, Bruton D, Cox C. Inflicted versus accidental head injury in critically injured children. *Crit Care Med* 1993;21:1328-32.
26. Hampton RL, Newberger EH. Child abuse incidence and reporting by hospitals: significance of severity, class and race. *Am J Public Health* 1985;75:56.
27. Jayawant S, Rawlinson A, Gibbon F, Price J, Schulte J, Sharples P, *et al.* Subdural haemorrhages in infants: population based study. *BMJ* 1998;317:1558-61.
28. Lloyd B. Subdural haemorrhages in infants [editorial]. *BMJ* 1998;317:1538-9.
29. Hobbs CJ. When are burns not accidental? *Arch Dis Child* 1986;61:357-61.
30. Glaser D, Bentovim A. Abuse and risk to handicapped and chronically ill children. *Child Abuse Neglect* 1979;3:565-75.
31. Morse CW, Shaler OJZ, Friedman SB, Rochester NY. A three year follow-up study of abused and neglected children. *Am J Dis Child* 1970;120:439-46.
32. Spinetta JJ, Rigler D. The child abusing parent: a psychological review. *Psychol Bull* 1972;7:296-304.
33. Roussey M, Betremieux P, Journeil H, Lefrançois MC, Urvoy M. L'ophtalmologiste et les enfants victimes de sévices. *J Fr Ophtalmol* 1987;10:201-5.
34. Meadow R. Unnatural sudden infant death. *Arch Dis Child* 1999;80:7-14.
35. Brown JK, Minns RA. Non-accidental head injury, with particular reference to whiplash shaking injury and medico-legal aspects. *Dev Med Child Neurol* 1993;35:849-69.
36. Chadwick DL, Chin S, Salerno C, Landsverk J, Kitchen L. Deaths from falls in children: how far is fatal? *J Trauma* 1991;31:1353.
37. Buys YM, Levin AV, Enzenauer RW, Elder JE, Letourneau MA, Humphreys RP, *et al.* Retinal findings after head trauma in infants and young children. *Ophthalmology* 1992;99:1718-23.
38. Waterhouse W, Enzenauer RW, Parmley VC. Inflammatory orbital tumour as an ocular sign of a battered child. *Am J Ophthalmol* 1992;114:510-2.
39. Reece RM, Grodin MA. Recognition of non-accidental injuries. *Pediatr Clin North Am* 1985;32:41-59.
40. Feenstra J, Merth IT, Treffers PDA. Een geval van het syndroom van Münchhausen bij proxy. *Tijdschr Kindergeneesk* 1988;56:148-51.
41. Auvert B, Degos R, Theron H-P. Syndrome des enfants battus (syndrome de Silverman): un cas avec atteinte cornéenne. *Bull Soc Ophtalmol Fr* 1971;71:1092-6.
42. Taylor D, Bentovim A. Recurrent nonaccidentally inflicted chemical eye injuries to siblings. *J Pediatr Ophthalmol* 1976;13:238-42.
43. Blinder KJ, Scott W, Lange MP. Abuse of cyanoacrylate in child abuse. *Arch Ophthalmol* 1987;105:1632-3.
44. Gonzales CA, Scott IU, Chaudry NA, Oster AS, Hess DJ, Murray TG. Bilateral rhegmatogenous retinal detachments with unilateral vitreous base avulsion as the presenting signs of child abuse. *Am J Ophthalmol* 1999;127:475-7.
45. Jain IS, Singh YP, Grupta SL, Gupta A. Ocular hazards during birth. *J Pediatr Ophthalmol Strabismus* 1980;17:14-6.
46. Betz P, Eisenmenger W, Lignitz E, Miltner E, Puschel K. Morphometrical analysis of retinal hemorrhages in the shaken baby syndrome. *Forensic Sci Int* 1996;78:71-80.
47. Wilkinson WS, Han DP, Rappley MD, Owings CL. Retinal hemorrhage predicts neurologic injury in the shaken baby syndrome. *Arch Ophthalmol* 1989;107:1472-4.
48. Holloway M, Bye AME, Moran K. Non-accidental head injury in children. *Med J Aust* 1994;160:786-9.
49. Green MA, Lieberman G, Milroy CM, Parsons MA. Ocular and cerebral trauma in non-accidental injury in infancy: underlying mechanisms and implications for paediatric practice. *Br J Ophthalmol* 1996;80:282-7.
50. Parsons MA, Liebermann G, Milroy CM, Green MA. Relative trauma scoring in non-accidental injury to infants: a forensic study. *Proceedings of the XIth Congress of the European Society of Ophthalmology* 1997:1177-82.
51. Snodgrass GJ. The eye and the brain in non-accidental injury involving young children [editorial]. *Comment. Br J Ophthalmol* 1996;80:275.
52. Weissgold DJ, Budenz DL, Hood I, Rorke LB. Ruptured vascular malformation masquerading as battered/shaken baby syndrome: a nearly tragic mistake. *Surv Ophthalmol* 1995;39:509-12.
53. Shemie S, Cutz E. Late haemorrhagic disease of the newborn: a fatal presentation of hepatobiliary atresia masquerading as shaken baby syndrome. *J Intensive Care Med* 1995;10:315-8.
54. Riffenburgh RS, Sathyavagiswaran L. The eyes of child abuse victims: autopsy findings. *J Forensic Sci* 1991;36:293-9.
55. Frank Y, Zimmerman R, Leeds NMD. Neurological manifestations in abused children who have been shaken. *Dev Med Child Neurol* 1985;27:312-5.
56. Munger CE, Peiffer RL, Bouldin TW, Kylstra JA, Thompson RL. Ocular and associated neuropathologic observations in suspected whiplash baby syndrome: a retrospective study of 12 cases. *Am J Forensic Med Pathol* 1994;101:559-65.
57. Budenz DL, Farger MG, Mirchandani HG, Park H, Rorke LB. Ocular and optic nerve hemorrhages in abused infants with intracranial injuries. *Ophthalmology* 1994;101:559-65.
58. Rao N, Smith RE, Choi JH, Xiaohu X, Kornblum RN. Autopsy findings in the eyes of fourteen fatally abused children. *Forensic Sci Int* 1988;36:293-9.
59. Riffenburgh RS, Sathyavagiswaran L. Ocular findings at autopsy of child abuse victims. *Ophthalmology* 1991;98:1519-24.
60. Lin KC, Glasgow BJ. Bilateral periophtic intrascleral hemorrhages associated with traumatic child abuse. *Am J Ophthalmol* 1999;127:473-5.
61. Purtscher O. Noch unbekannte Befunde nach Schädeltrauma. *Ber Zusammenkunft Dtsch Ophthalmologie Ges* 1910;36:294-301.
62. Tomasi LG, Rosman NP. Purtscher retinopathy in the battered child syndrome. *Am J Dis Child* 1975;129:1335-7.
63. Kelley JS. Purtscher's retinopathy related to chest compression by safety belts. *Am J Ophthalmol* 1972;74:278-83.
64. Harada K, Hayashi T, Anegawa S, Torigoe R, Araki T, Murakoa Y, *et al.* Vitreous hemorrhage after head injury with chest compression: case report. *No To Shinkei* 1994;46:1095-9.
65. Morgan OG. A case of crush injury to the chest associated with ocular complications. *Trans Ophthalmol Soc UK* 1945;65:366-8.
66. Burton TC. Unilateral Purtscher's retinopathy. *Ophthalmology* 1980;87:1096-105.
67. Ober RR. Hemorrhagic retinopathy in infancy: a clinicopathologic report. *J Pediatr Ophthalmol Strabismus* 1980;17:17-20.
68. Elder JE, Taylor RG, Klug GL. Retinal haemorrhage in accidental head trauma in childhood. *J Paediatr Child Health* 1991;27:286.

69. Alario A, Duhaime A, Lewander W, Tsirias W, Wallach M, O'Shea J. Do retinal haemorrhages occur with accidental head trauma in young children? [abstract] *Am J Dis Child* 1990;144:445.
70. Eisenbrey AB. Retinal haemorrhage in the battered child. *Childs Brain* 1979;5:40-4.
71. Reiber GD. Fatal falls in childhood. How far must children fall to sustain fatal head injury? Report of cases and review of the literature. *Am J Forensic Med Pathol* 1993;14:201-7.
72. Johnson DL, Braun D, Friendly D. Accidental head trauma and retinal haemorrhage. *Neurosurgery* 1993;33:231-5.
73. Gilliland MGF, Luckenbach MW, Chenier TC. Systemic and ocular findings in 169 prospectively studied child deaths: retinal hemorrhages usually mean child abuse. *Forensic Sci* 1994;68:117-32.
74. James DS, Leadbetter S. Letter to the editor. *Forensic Sci Int* 1996;82:255.
75. Lyle DJ, Stapp JP, Button RR. Ophthalmologic hydrostatic pressure syndrome. *Am J Ophthalmol* 1957;44:652-7.
76. Gilliland MG, Folberg R. Shaken babies: some have no impact injuries. *J Forensic Sci* 1996;41:114-6.
77. David DB, Mears T, Quinlan MP. Ocular complications of bungee jumping. *Br J Ophthalmol* 1994;78:234-5.
78. Jain BK, Talbot EM. Bungee jumping and intraocular haemorrhage. *Br J Ophthalmol* 1997;78:236-7.
79. Chan J. Ophthalmic complications of bungee jumping. *Br J Ophthalmol* 1994;78:239.
80. Guthkelch AN. Infantile subdural haematoma and its relationship to whiplash injuries. *BMJ* 1971;11:430-1.
81. Gennarelli TA. Head injury in man and experimental animals: clinical aspects. *Acta Neurochir Suppl* 1983;32:1-13.
82. David TJ. Shaken baby (shaken impact) syndrome: non-accidental head injury in infancy. *J R Soc Med* 1999;92:556-61.
83. Elner SG, Elner VM, Arnall M, Albert DM. Ocular and associated systemic findings in suspected child abuse. *Arch Ophthalmol* 1990;108:1094-101.
84. Shaver EG, Duhaime A-C, Curtis M, Gennarelli LM, Barrett R. Experimental acute subdural haematoma in piglets. *Pediatr Neurosurg* 1996;25:123-9.
85. Johnson DL, Boal D, Baule R. Role of apnea in nonaccidental head injury. *Pediatr Neurosurg* 1995;23:305-10.
86. Till K. Subdural haematoma and effusion in infancy. *BMJ* 1968;III:400-2.
87. Hashimoto N, Sakakibara T, Yamamoto K. Two fluid-blood density levels in chronic subdural hematoma. *J Neurosurg* 1992;77:310-11.
88. Von Barsewisch B. Perinatal retinal haemorrhages: morphology, aetiology and significance. Berlin: Springer-Verlag, 1979.
89. Sezen F. Retinal haemorrhages in newborn infants. *Br J Ophthalmol* 1970;55:248-53.
90. Giles CL. Retinal haemorrhages in the newborn. *Am J Ophthalmol* 1960;49:1005-10.
91. Baum JD, Bulpitt CJ. Retinal and conjunctival haemorrhage in the newborn. *Arch Dis Child* 1970;45:344-9.
92. Bonamour G. Le pronostic éloigné des hémorragies rétiniennes du nouveau-né. *Bull Soc Fr Ophtalmol* 1949;62:227-36.
93. Zwaan J, Cardenas R, O'Connor PS. Long-term outcome of neonatal macular haemorrhage. *J Pediatr Ophthalmol Strabismus* 1997;34:286-8.
94. Fraser SC. Retinal haemorrhage in meningitis [letter]. *Eye* 1995;9:659-60.
95. Sandramouli S, Robinson R, Tsaloumas M, Willshaw HE. Retinal haemorrhages and convulsions. *Arch Dis Child* 1997;76:449-51.
96. Keithahn MAZ, Bennett SR, Cameron D, Mieler WF. Retinal folds in Terson syndrome. *Ophthalmology* 1993;100:1187-90.
97. Sethi SK. Retinal haemorrhages after extracorporeal membranous oxygenation. *North Carolina Med J* 1990;51:246.
98. Carney MD, Wortham E, Al-Mateen KB. Vitreous hemorrhage and extracorporeal membrane oxygenation. *Am J Ophthalmol* 1993;115:391-3.
99. Goetting MG, Sowa B. Retinal haemorrhage after cardiopulmonary resuscitation in children: an etiologic reevaluation. *Pediatrics* 1990;85:585-8.
100. Tyagi AK, Willshaw HE, Ainsworth JR. Unilateral retinal haemorrhages in non-accidental injury. *Lancet* 1997;349:1224.
101. Miller NR. Walsh and Hoyt's clinical neuro-ophthalmology. Vol. 1 Baltimore: Williams and Wilkins, 1982.
102. Hanigan W, Peterson R, Njus G. Tin ear syndrome: rotational acceleration in pediatric head injuries. *Pediatrics* 1987;80:618-22.
103. Ommaya AK, Gennarelli TA. Cerebral concussion and traumatic unconsciousness. *Brain* 1974;97:633-54.
104. Zimmerman RA, Bilianuk LT, Bruce D, Schut L, Uzzell B, Goldberg H. Interhemispheric acute subdural hematoma: a computed tomographic manifestation of child abuse. *Neuroradiology* 1978;16:39-40.
105. Giangiacomo J, Khan JA, Levine C, Thompson VM. Sequential cranial computed tomography in infants with retinal hemorrhages. *Ophthalmology* 1988;95:295-9.
106. Guyer DR, Schachat AP, Vitale S, Markowitz JA, Braine H, Burke PJ, et al. Relationship between fundus lesions and hematologic parameters at diagnosis. *Ophthalmology* 1989;96:860-4.
107. Robb RM, Ervin LD, Sallan SE. A pathological study of eye involvement in acute leukemia of childhood. *Trans Am Acad Ophthalmol Otolaryngol* 1978;76:90-101.
108. Shaw NJ, Eden OB. Juvenile chronic myelogenous leukemia and neurofibromatosis in infancy presenting as ocular hemorrhage. *Pediatr Hematol Oncol* 1989;6:23-6.
109. Maumenee AE, Hellman LM, Shettles LB. Factors influencing plasma prothrombin in the newborn infant: the effect of antenatal administration of vitamin K on the incidence of retinal hemorrhage in the newborn. *Bull Johns Hopkins Hosp* 1941;68:158-61.
110. Luxenberg MN. The evolution of salmon-patch hemorrhages in sickle cell retinopathy. *Arch Ophthalmol* 1989;107:1814-5.
111. Skalina MEL, Annable WL, Kliegman RM, Fanaroff AA. Hypertensive retinopathy in the newborn infant. *J Pediatr* 1983;103:781-6.
112. Young TL, Quinn GE, Baumgart S, Schaffer DB. Extracorporeal membrane oxygenation causing unilateral retinal vasculopathy in neonates. *Ophthalmology* 1992;99(Suppl):149.
113. Patrias MC, Rabinowicz IM, Klein MD. Ocular findings in infants treated with extracorporeal membrane oxygenator support. *Pediatrics* 1988;82:560-4.
114. Varn MM, Donahue ML, Saunders RA, Baker JD, Smith CM, Wilson ME. Retinal examinations in infants after extracorporeal membrane oxygenation. *J Pediatr Ophthalmol Strabismus* 1997;34:182-5.
115. Pollack JS, Tycksen L. Prevalence of retinal hemorrhages in infants after extracorporeal membrane oxygenation. *Am J Ophthalmol* 1996;121:297-303.
116. Levy HL, Brown AE, Williams SE, de Juan E. Vitreous hemorrhage as an ophthalmic complication of galactosemia. *J Pediatr* 1996;129:922-5.
117. Morris AAM, Hoffmann GF, Naughten ER, Monavari AA, Collins JE, Leonard JV. Glutaric aciduria and suspected child abuse. *Arch Dis Child* 1999;80:404-5.

118. Hoffmann GF, Athanassopoulos S, Burlina AB, Duran M, DeKlerk JBC, Lehnert W, *et al.* Clinical course, early diagnosis, and prevention of disease in glutaryl-CoA dehydrogenase deficiency. *Neuropediatrics* 1996;27:115-23.
119. Hoffmann GF, Naughten ER. Abuse or metabolic disorder? [letter] *Arch Dis Child* 1998;78:399.
120. Petersen S, Taaning E, Söderström T, Pilgaard B, Traenebjærg L, Christensen T, *et al.* Immunoglobulin and complement studies in children with Schönlein-Henoch syndrome and other vasculitic diseases. *Acta Paediatr Scand* 1991;80:1037-43.
121. Raja SC, Fekrat S, Connor TB. Bilateral macular vitelliform lesions in a thrombocytopenic patient [case report]. *Arch Ophthalmol* 1995;113:411-3.
122. Gayle MO, Hered RW, Harwood-Nuss A, Kisson N. Retinal hemorrhage in the young child: a review of etiology, predisposed conditions, and clinical implications. *J Emerg Med* 1995;13:233-9.
123. Dietrich AM, James CD, King DR. Head trauma in children with congenital coagulation disorders. *J Pediatr Surg* 1994;29:28-32.
124. Shiono T, Abe S, Watabe T, Noro M, Tamai M, Akutso Y, *et al.* Vitreous, retinal and subretinal hemorrhages associated with von Willebrand's syndrome. *Graefes Arch Clin Exp Ophthalmol* 1992;230:496-7.
125. Pulido JS, Lingua RW, Cristol BS, Byrne SF. Protein C deficiency associated with vitreous hemorrhage in a neonate. *Am J Ophthalmol* 1987;104:546-7.
126. Cassels-Brown A, Minford AMB, Chatfield SL, Bradbury JA. Ophthalmic manifestations of neonatal protein C deficiency. *Br J Ophthalmol* 1994;78:486-7.
127. O'Hare AE, Eden OB. Bleeding disorders and non-accidental injury. *Arch Dis Child* 1984;59:860-4.
128. Hymel KP, Abshire TC, Luckey DW, Jenny C. Coagulopathy in pediatric abusive head trauma. *Pediatrics* 1997;99:371-5.
129. Sloan B, Kulwin DR, Kersten RC. Scurvy causing bilateral orbital hemorrhage. *Arch Ophthalmol* 1999;117:842-3.
130. Hood J, Hodges RE. Ocular lesions in scurvy. *J Clin Nutr* 1969;22:559-67.
131. Silva-Araujo J. Maternal ingestion of cocaine. *Retina* 1996;16:411-8.
132. McLellan NJ, Prasad R, Punt J. Spontaneous subhyaloid and retinal hemorrhages in an infant. *Arch Dis Child* 1986;61:1130-2.
133. Atkinson A, Sanders MS, Wong V. Vitreous hemorrhage in tuberous sclerosis. *Br J Ophthalmol* 1973;57:773-9.
134. George NDL, Yates JRW, Bradshaw K, Moore AT. Infantile presentation of X-linked retinoschisis. *Br J Ophthalmol* 1995;79:653-7.
135. Christiansen S, Munoz M, Capo H. Retinal hemorrhage following lensectomy and vitrectomy in children. *J Pediatr Ophthalmol Strabismus* 1993;30:24-7.
136. Mets M, Del Monte M. Hemorrhagic retinopathy following childhood cataract extraction [letter]. *Arch Ophthalmol* 1986;104:975-9.
137. Kirby S, Sadler RM. Injury and death as a result of seizures. *Epilepsia* 1995;36:25-8.
138. Feyi-Waboso AC, Beck L. Minerva column. *BMJ* 1997;314:688.
139. Tyagi AK, Scotcher S, Kozeis N, Willshaw H. Can convulsions alone cause retinal hemorrhages in infants? *Br J Ophthalmol* 1998;82:659-60.
140. Rogers MC, Nugent SK, Stidham GL. Effects of closed chest cardiac massage on intracranial pressure. *Crit Care Med* 1979;7:454-6.
141. Kanter RK. Retinal hemorrhages after cardiopulmonary resuscitation or child abuse. *J Pediatr* 1986;108:430-2.
142. Carter J, McCormick AQ. Whiplash shaking syndrome: retinal hemorrhages and computerised axial tomography of the brain. *Child Abuse Neglect* 1983;7:279-86.
143. Bacon CJ, Sayer GC, Howe J. Extensive retinal haemorrhages in infancy: an innocent cause. *BMJ* 1978;1:281.
144. Weedn VVW, Mansour AM, Nichols MM. Retinal hemorrhage in an infant after cardiopulmonary resuscitation. *Am J Forensic Pathol* 1990;11:79-82.
145. Gilliland MGF, Luckenbach MW. Are retinal hemorrhages found after resuscitation attempts? *Am J Forensic Med Pathol* 1993;14:187-92.
146. Levin A. Retinal haemorrhages after cardiopulmonary resuscitation or child abuse [comment on]. *Pediatr Emergency Care* 1986;2:269-70.
147. Fackler JC, Berkovitz ID, Green WR. Retinal haemorrhages in newborn piglets following cardiopulmonary resuscitation. *Am J Dis Child* 1992;146:1294-6.
148. Duane TD. Valsalva hemorrhagic retinopathy. *Trans Am Ophthalmol Soc* 1972;70:298-313.
149. Georgiou T, Pearce IA, Taylor RH. Valsalva retinopathy associated with blowing balloons [letter]. *Eye* 1999;13:686-7.
150. Stephenson T. Ageing of bruising in children. *J R Soc Med* 1997;90:312-4.
151. Giangiacomo J, Barkett KJ. Ophthalmoscopic findings in occult child abuse. *J Pediatr Ophthalmol Strabismus* 1985;22:234-7.
152. Matthews GP, Das A. Dense vitreous hemorrhages predict poor visual and neurological prognosis in infants with shaken baby syndrome. *J Pediatr Ophthalmol Strabismus* 1996;33:260-5.
153. Ferrone PJ, de Juan E. Vitreous hemorrhage in infants. *Arch Ophthalmol* 1994;112:1185-9.
154. Rohrbach JM, Benz D, Friedrichs W, Thiel H-J, Wehner H-D. Okuläre Pathologie der Kindesmisshandlung. *Klin Monatsbl Augenheilkd* 1997;210:133-8.
155. Gilliland MGF, Luckenbach MW, Massicotte SJ, Folberg R. The medicolegal implications of detecting hemosiderin in the eyes of children who are suspected of being abused [letter]. *Arch Ophthalmol* 1991;109:321-2.
156. Elner SG, Elner VM, Albert DM, Arnall M. The medicolegal implications of detecting hemosiderin in the eyes of children who are suspected of being abused [reply to letter from Gilliland *et al.*]. *Arch Ophthalmol* 1991;109:322.
157. Lambert S, Johnson TE, Hoyt CS. Optic nerve sheath and retinal hemorrhages associated with the shaken baby syndrome. *Arch Ophthalmol* 1986;104:1509-12.
158. Muller PJ, Deck JHN. Intraocular and optic nerve sheath hemorrhage in cases of sudden intracranial hypertension. *J Neurosurg* 1974;41:160-6.
159. Gaynon MW, Koh K, Marmor MF, Frankel LR. Retinal folds in the shaken baby syndrome. *Am J Ophthalmol* 1988;106:423-5.
160. Massicotte SJ, Folberg R, Torczynski E, Gilliland MGF, Luckenbach MV. Vitreoretinal traction and perimacular retinal folds in the eyes of deliberately traumatized children. *Ophthalmology* 1991;98:1124-7.
161. Tongue AC. The ophthalmologist's role in diagnosing child abuse. *Ophthalmology* 1991;98:1009-11.
162. Aron JJ, Marx P, Blanck MF, Duval R, Luce R. Signes oculaires observés dans le syndrome de Silverman. *Ann Oculist (Paris)* 1970;203:533-46.
163. Hollenhorst RW, Stein HA. Ocular signs and prognosis in subdural and subarachnoid bleeding in young children. *Arch Ophthalmol* 1958;60:187-92.
164. Greenwald MJ, Weiss A, Oesterle CS, Friendly DS. Traumatic retinoschisis in battered babies. *Ophthalmology* 1986;93:618-25.
165. Morris R, Kuhn F, Witherspoon CD. Hemorrhagic macular cyst [letter]. *Ophthalmology* 1994;101:1.

166. Brown SM, Shami M. Optic disc neovascularisation following severe retinoschisis due to shaken baby syndrome [letter]. *Arch Ophthalmol* 1999;117:838-9.
167. Willman KY, Bank DE, Senac M, Chadwick DL. Restricting the time of injury in fatal inflicted head injuries. *Child Abuse Neglect* 1997;21:929-40.
168. Carpenter RF. The prevalence and distribution of bruising in babies. *Arch Dis Child* 1999;80:363-6.
169. Hight DW, Bakalar HR, Lloyd JR. Inflicted burns in children. *JAMA* 1979;242:517-20.
170. Montrey JS, Barcia PJ. Nonaccidental burns in child abuse. *Southern Med J* 1985;78:1324-6.
171. Keen JH, Lendrum J, Wolman B. Inflicted burns and scalds in children. *BMJ* 1975;IV:268-9.
172. Hall CM, Shaw DG. Non-accidental injury. *Current Imaging* 1991;3:88-93.
173. Feldman KW, Brewer DK. Child abuse, cardiopulmonary resuscitation, and rib fractures. *Pediatrics* 1984;73:339-42.
174. Ng CS, Hall CM. Costochondral junction fractures and intra-abdominal trauma in non-accidental injury (child abuse). *Pediatr Radiol* 1998;28:671-6.
175. Kleinmann PK. Skeletal trauma: general considerations. In: *Diagnostic imaging of child abuse*. Baltimore: Williams & Wilkins, 1987:5-28.
176. Rogers LF, Poznanski AK. Imaging of epiphyseal injuries. *Rev Radiol* 1994;191:297-308.
177. Carty H, Ratcliffe J. The shaken baby syndrome: parents and other carers need to know of its dangers. *BMJ* 1995;310:344-5.
178. Showers J. 'Don't shake the baby': the effectiveness of a prevention programme. *Child Abuse Neglect* 1992;16:11-8.
179. Haviland J, Ross Russell RI. Outcome after severe non-accidental head injury. *Arch Dis Child* 1997;77:504-7.
180. Bruce DA, Zimmerman RA. Shaken impact syndrome. *Pediatr Ann* 1989;18:482-4.
181. Asher R. Munchausen's syndrome. *Lancet* 1951;I:343-5.
182. Rosenberg PN, Krohel GB, Webb RM, Hepler RS. Ocular Munchausen's syndrome. *Ophthalmology* 1986;93:1120-3.
183. Winans JM, House LR, Robinson HE. Self-induced orbital emphysema as a presenting sign of Munchausen's syndrome. *Laryngoscope* 1983;93:1209-11.
184. Cruciani F, Santino G, Trudu R, Balacco Gabrieli C. Ocular Munchausen syndrome characterised by self-introduction of chalk concretions into the conjunctival fornix. *Eye* 1999;13:598-9.
185. Ugurlu S, Bartley GB, Otley CC, Baratz KH. Factitious disease of periocular and facial skin. *Am J Ophthalmol* 1999;127:196-201.
186. Feldman MC. Factitious disease of periocular and facial skin [letter]. *Am J Ophthalmol* 1999;128:392-3.
187. Sneddon I, Sneddon J. Self-inflicted injury: a follow-up study of 43 patients. *BMJ* 1975;III:527-30.
188. Stark A III, Sherman MD. Factitious disease of periocular and facial skin. *Am J Ophthalmol* 1999;128:392.
189. Jay JL, Grant S, Murray SB. Keratoconjunctivitis artefacta. *Br J Ophthalmol* 1982;66:781-5.
190. Trope G, Jay J, Dudgeon J, Woodruff G. Self-inflicted corneal injuries in children with congenital corneal anaesthesia. *Br J Ophthalmol* 1985;69:551-4.
191. Graber J, Arndt W. Trichotillomania. *Compr Psychiatry* 1993;34:340-6.
192. Orentreich D, Orentreich N. Patchy eyelash loss [letter]. *JAMA* 1984;252:684.
193. Mawn LA, Jordan DR. Trichotillomania. *Ophthalmology* 1997;104:2175-8.
194. Cohen LJ, Stein DJ, Simeon D, Spadaccini E, Rosen J, Aronowitz B, et al. Clinical profile, comorbidity and treatment history in 123 hair pullers: a survey study. *J Clin Psychiatry* 1995;56:319-26.
195. Hallopeau M. Alopecie par grattage (trichomanie ou trichotillomanie). *Ann Dermatol Syphilol* 1889;10:440-1.
196. Hamdan-Allen G. Trichotillomania in childhood. *Acta Psychiatr Scand* 1991;83:241-3.
197. Smith JR. Trichotillomania: ophthalmic presentation. *Aust NZ J Ophthalmol* 1995;23:59-61.
198. Huismans H. Ein Fall von Trichotillomanie. *Klin Monatsbl Augenheilkd* 1976;168:850-1.
199. Swedo S, Lenane M, Leonard H. Long term treatment of trichotillomania (hair pulling). *N Engl J Med* 1993;329:141-2.
200. Noel L-P, Clarke WN. Self-inflicted ocular injuries in children. *Am J Ophthalmol* 1982;94:630-3.
201. Kennedy BL, Feldmann TB. Self-inflicted eye injuries: case presentation and a literature review. *Hosp Community Psychiatry* 1994;45:470-4.
202. Chapman FM, Dickinson AJ, Figueredo FC. Self-induced cicatricial conjunctivitis [letter]. *Eye* 1999;13:674-6.
203. Witherspoon DC, Feist FW, Morris RE, Feist FM. Ocular self-mutilation. *Ann Ophthalmol* 1989;21:674-6.
204. Noel L-P, Clarke WC. Self-inflicted ocular injuries in children. *Am J Ophthalmol* 1982;94:630-3.
205. Ashkenazi I, Shahar E, Brand N, Bartov E, Blumenthal M. Self-inflicted ocular mutilation in the pediatric age group. *Acta Paediatr Scand* 1992;81:649-51.
206. Spalter HF, Bemporad JR, Sours JA. Cataracts following chronic headbanging. *Arch Ophthalmol* 1970;83:182-4.
207. Winter R, Baraitser M. London dysmorphology database. Oxford: Oxford University Press, 1998.
208. Aung T, Yap EY, Fam HB, Law NM. Oedipism. *Aust NZ J Ophthalmol* 1996;24:153-7.
209. Trevor-Roper PD. The psychopathic eye. *Br J Hosp Med* 1980;23:138-41.
210. Foster J. The legend and shrine of Saint Triduana. *Br J Ophthalmol* 1953;37:763-5.
211. Brown R, Al-Bachari MAH, Kambhampati KK. Self-inflicted eye injuries. *Br J Ophthalmol* 1990;75:496-8.
212. Krauss HR, Yee RD, Foos RY. Autoenucleation. *Surv Ophthalmol* 1984;29:179-87.
213. Gerhard JP. A propos des automutilations oculaires. *Bull Soc Fr Ophthalmol* 1968;68:622-6.
214. Bergmann GH. Ein Fall von religiöser Monomanie, die eine unerharte Selbstvestummelung bei Geistkranken. *Allg Z Psychiat* 1846;3:365-80.
215. Bergmann GH. Ein Fall von religiöser Monomanie, die unerhöret Selbstverletzung veranlasste. *Z Augenheilkd* 1846;1:365-80.
216. Shiwach RS. Autoenucleation - a culture specific phenomenon: a case. *Compr Psychiatry* 1998;39:318-22.
217. Kinnas SJ, Brady HR. Oedipism and Down's syndrome: case report and review of the literature. *Ann Ophthalmol* 1985;17:431-2.
218. Pillai S, Mahmood MA, Limaye SR. Complete avulsion of the globe and optic nerve. *Br J Ophthalmol* 1987;71:69-72.
219. Krauss HR, Yee RD, Foos RY. Autoenucleation. *Surv Ophthalmol* 1984;29:179-87.
220. Clarke RA. Self-mutilation accompanying religious delusions: a case report and review. *J Clin Psychiatry* 1981;42:243-5.
221. Wolff RS, Wright MM, Walsh AW. Attempted autoenucleation. *Am J Ophthalmol* 1996;121:726-8.
222. Stannard K, Leonard T, Holden G, Shilling J. Oedipism reviewed: a case of bilateral ocular self-mutilation. *Br J Ophthalmol* 1984;68:276-80.
223. Byrnes VA, Shier JM. A case of self-enucleation involving both eyes. *Am J Ophthalmol* 1949;32:268-9.
224. Jones NP. Self-enucleation and psychosis. *Br J Ophthalmol* 1990;74:571-3.
225. Tamai M, Mizuno K, Okuyama S. Self-inflicted proptosis misdiagnosed as orbital malignancy. *Am J Ophthalmol* 1983;96:812-4.

226. Khan JA, Buescher L, Ide CH, Pettigrove B. Medical management of self-enucleation. *Arch Ophthalmol* 1985;103:386-9.
227. Meadow R. Munchausen syndrome by proxy: the hinterland of child abuse. *Lancet* 1977;II:343-5.
228. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. (DSM IV). 4th ed. Washington: American Psychiatric Association, 1994.
229. Meadow R. What is, and what is not. 'Munchausen's syndrome by proxy?' *Arch Dis Child* 1995;72:534-8.
230. Morley CJ. Practical concerns about the diagnosis of Munchausen's syndrome by proxy. *Arch Dis Child* 1995;72:528-30.
231. Meadow R. Munchausen syndrome by proxy abuse perpetrated by men. *Arch Dis Child* 1998;78:210-6.
232. Verity CM, Winckworth D, Burman D, Stevens D, White RJ. Polle syndrome: children of Munchausen. *BMJ* 1979;II:422-3.
233. Wood PR, Fowlkes J, Holden P, Casto D. Fever of unknown origin for six years: Munchausen syndrome by proxy. *J Fam Pract* 1989;28:391-5.
234. Southall DP, Plunkett MCB, Banks MW, Falkov AF, Samuels MP. Covert video recordings of life-threatening child abuse: lessons for child protection. *Pediatrics* 1997;100:735-60.
235. Bools CN, Neale BA, Meadow SR. Follow up of victims of fabricated illness (Munchausen syndrome by proxy). *Arch Dis Child* 1993;69:625-30.
236. Davis P, McClure RJ, Rolfe K, Chessman N, Pearson S, Sibert JR, *et al.* Procedures, placement, and risks of further abuse after Munchausen syndrome by proxy, non-accidental poisoning and non-accidental suffocation. *Arch Dis Child* 1998;78:217-21.