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## Ocular Manifestations of Child Abuse

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It is estimated that over two million children in the United States are victims of child abuse each year: four abused every minute.<sup>41,60</sup> Ocular injury is common in these children, and death is not rare. The omnipresence of child abuse in our society makes it likely that every ophthalmologist will at some time be called on to examine an abused child, either as part of the evaluation of an already identified case or as the initial care provider in a case where the history has not been established. The presenting sign of child abuse in fact involves the eye in 4% to 6% of cases.<sup>29,49</sup> An ophthalmologist with a sound understanding of the basic dynamics and types of child abuse, a working knowledge of its ocular manifestations, and an appropriate level of suspicion is in a good position to make this important and sometimes life-saving diagnosis.

In every state, all physicians, including ophthalmologists, are required by law to report to designated state or county agencies whenever there is a suspicion that abuse has occurred. Abuse need not be proven at the time of reporting. Investigation to determine the validity of a report of suspected abuse in fact often requires the efforts of many individuals, including social workers, law enforcement officers, and physicians specifically trained in this area. Failure to report at a threshold level of suspicion may lead to legal action against the physician, for which there is established precedent. But there is no physician liability for reporting, even if the suspi-

cion proves to be unfounded, as long as the report was generated without malicious intent.

This article will provide the reader with a conceptual framework for understanding child abuse, discuss the differentiation of nonaccidental injury from accidental injury and nontraumatic disorders, and review in some detail the most important ocular manifestations of abuse. We will focus on physical abuse, with particular attention to the shaken baby syndrome, but will briefly cover other forms, including medical neglect and sexual abuse.

### PHYSICAL ABUSE

Physical child abuse usually does not occur as a result of the premeditated intention of the perpetrator. It is rather a reflection of general loss of control, usually during a period of increased stress.<sup>60</sup> Poor education and lack of experience with child development and care can play a role, as can a history of prior victimization of the abusive adult. However, any person, regardless of race, religion, sex, profession, or socioeconomic status, may abuse a child. There is evidence of overreporting in cases with non-caucasian and socioeconomically disadvantaged perpetrators and under-reporting with perpetrators of higher socioeconomic status.<sup>37</sup> Although the abused child may be selected because of some "special" quality such as a physical handicap or illness,

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resemblance to a certain family member, or difficult behavior, in many cases there is no identifiable reason other than proximity for the choice of victim.

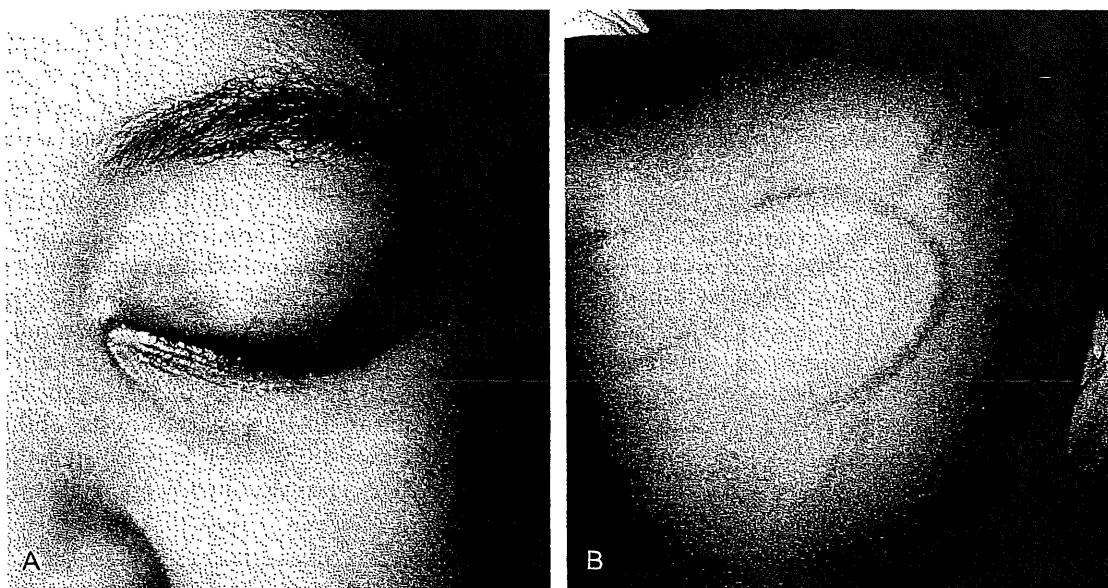
Physical abuse must be suspected when the history of injury is not consistent with physical findings or the developmental level of the child (e.g., a 6-month-old climbing out of a highchair and falling), or there is inconsistency between accounts given by different individuals at different times. A significant unexplainable delay in seeking medical attention should also arouse suspicion.

Since Kempe's landmark paper in 1962 defined the *battered child syndrome*,<sup>53</sup> the reported manifestations of physical abuse have grown enormously in number and variety. The head is the most common target in physical abuse, and head injuries are the most frequent cause of death secondary to child abuse in general.<sup>74,85</sup> Facial involvement has been reported in 11% to 45% of cases, with ocular signs in 5 to 61 percent.<sup>15,24,29,49,50</sup> Virtually any form of trauma to the eye may be a result of physical abuse. Ophthalmological findings in physically abused children can be divided into three categories: direct ocular injury, indirect ocular injury, and visual abnormality secondary to nonocular injury. Direct ocular trauma usually is caused by blunt impact. Ex-

amples of indirect injury are optic nerve compression and retinal hemorrhage secondary to intracranial bleeding. Cortical trauma may cause blindness though the eyes themselves are left unharmed.

### Periorbital and Conjunctival Injury

Ecchymosis, lacerations, abrasions, and burns involving the eyelids and periorbital tissues have frequently been reported to occur in cases of physical child abuse.<sup>1,12,29,39,40,49,50,77,90</sup> It is always important for the ophthalmologist to ascertain whether or not such physical findings are consistent with an alleged history of accidental injury. Patterns of ecchymosis such as the loop mark left by a cord or belt, or a bruise with detached radial linear elements indicative of a hand slap can reveal the nonaccidental nature of an injury. Nonspecific ecchymosis in the periorbital area may be associated with a more characteristic lesion of abuse elsewhere (Fig. 1). Ludwig and Warman<sup>61</sup> suggested that any bruising in an infant under the age of 6 months be considered suspicious for child abuse. Children under the age of 4 years usually have nonspecific types



**Figure 1.** *A*, Nonspecific ecchymotic swelling of left upper eyelid. *B*, Examination of the back of the same child reveals loop mark ecchymosis secondary to a belt beating.

of soft tissue injuries. Johnson et al<sup>50</sup> observed that implements are commonly used on children 4 to 12 years old, while fists and feet inflict most injuries to children beyond the age of 12 years.

Although bilateral injury should always lead to suspicion of nonaccidental etiology, blows to the forehead (accidental or nonaccidental) may cause marked bilateral periorbital ecchymosis, which characteristically becomes increasingly infraorbital over time. A blunt injury to the temporal region can result in unilateral periorbital ecchymosis.<sup>84</sup> When bruising is the only manifestation of injury, one must exclude the presence of a bleeding diathesis by obtaining a complete blood count, platelet count, and clotting studies. It must be remembered, however, that the presence of a bleeding diathesis does not rule out the coincident occurrence of child abuse.

The dating of ecchymosis, although somewhat imprecise,<sup>84,97</sup> may be helpful both in confirming or refuting the history and in identifying potential perpetrators based on who had contact with the child at the likely time of injury. Table 1 provides a general guide to the progressive color changes in nonperiorbital ecchymosis over time. In children with darkly pigmented skin, subtle changes, particularly early erythema, can be difficult to detect. Periorbital ecchymoses tend to look up to several days older than they truly are and may persist in a green-yellow stage longer than bruises at other sites. This is due to more abundant collection of blood in the relatively loose periorbital tissues than under other areas of skin.<sup>84,97</sup>

Nonaccidental burns involve the face in less

than 10% of cases.<sup>43</sup> In comparison with accidental burns, nonaccidental thermal injuries tend to be more focal, have deeper tissue penetration, and are more often associated with evidence of mechanical skin trauma.<sup>42,43,52,66,86</sup> Accidental burns from hot liquid are usually asymmetric, show lines tracing flow of the hot liquid away from the primary site, and may have satellite splatter burns. Some authors have suggested that facial burns in the absence of submental and axillary burns are inconsistent with a history of the child having pulled hot liquid down off of a stove or table and are indicative of an inflicted burn.<sup>52</sup> In my own experience, this rule has not always held true. Suspicion of abuse inevitably arises when cigarette burns of the skin are encountered. Accidental burning of the eyelids or cornea by a cigarette held in the hand of a sitting or standing adult is, however, a not uncommon occurrence in toddlers.

Data are available regarding the time of exposure at various temperature levels required to inflict a burn of a given thickness.<sup>26,67,68</sup> At 113°F (45°C), a first degree burn takes 2 hours to develop, and a full-thickness burn 3 hours. At 140°F (60°C) such burns develop in only 3 seconds and 5–6 seconds, respectively. In one survey, 80% of homes had an achievable bathtub water temperature of greater than 130°F (54°C), at which as little as 1 minute of exposure is required to produce a full-thickness burn.<sup>26</sup>

Subconjunctival hemorrhage (SCH) occurs in up to 4% of child abuse victims, either in isolation or in association with other facial injury, and may be due either to direct trauma or to hemodynamic forces induced by chest compression.<sup>29,49,79</sup> Transient SCH is present in 0.5% to 13% of newborns as a result of the normal birth process, typically with limited size at the nasal and temporal limbus.<sup>7,48</sup> Extensive SCH evident beyond the first 1 to 2 weeks of life should be considered suspicious of nonaccidental injury.

Forced chemical instillation into the eye may lead to conjunctivitis with disproportionate involvement of the lower palpebral and bulbar conjunctiva, presumably due to a Bell's response as the lids are forcibly separated.<sup>10,87</sup>

**Table 1. Progression of Color Change in Ecchymosis**

AGE OF ECCHYMOSIS	COLOR OF ECCHYMOSIS
0–24 hours	red, dark red or reddish blue
24–48 hours	blue or early purple
2–5 days	dark blue or dark purple
5–7 days	green or yellowish green
10–14 days	yellow or brown
2–4 weeks	ecchymosis resolved

*Adapted from Ludwig S: Child abuse. In Fleisher G, Ludwig S (eds): Textbook of Pediatric Emergency Medicine. Baltimore, Williams & Wilkins, 1983, p 1027; and Wilson EF: Estimation of the age of cutaneous contusions in child abuse. Pediatrics 60: 750, 1977; with permission.*

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Table 2. Anterior Segment Injuries Reported in Association with Child Abuse\*

Cornea
Microcornea/phthisis <sup>29,70</sup>
Enlarged cornea <sup>29,90</sup>
Epithelial defect <sup>10,73</sup>
Chemical keratitis <sup>87</sup>
Ulceration <sup>73</sup>
Stromal scarring <sup>29,40,90</sup>
Blood staining <sup>29</sup>
Descemet's breaks <sup>29</sup>
Edema/nonspecific haze <sup>29,70,90</sup>
Anterior chamber
Hyphema <sup>29,39,49,90</sup>
Post-inflammatory membrane <sup>29</sup>
Iridocorneal angle
Glaucoma (open- or closed-angle) <sup>29,70,90</sup>
Iris/pupil
Complete or sector traumatic mydriasis <sup>40,49</sup>
Sphincter tears <sup>94</sup>
Iris atrophy <sup>70</sup>
Iridodialysis <sup>90</sup>
Ectropion uveae <sup>70</sup>
Anisocoria <sup>29,39,55,61,70</sup>
Abnormal reactivity to light <sup>30,31,33,39,55,73</sup>
Afferent pupillary defect <sup>33,54</sup>

\* Not listed in order of frequency

### Anterior Segment and Lens Injury

Anterior segment injuries are relatively uncommon in physically abused children, but when present, they tend to be associated with severe trauma and a poor prognosis. Table 2 lists acute and chronic anterior segment findings that have been reported to result from child abuse. These are usually caused by direct blunt trauma, but manifestations such as microphthalmos, neovascular glaucoma, and phthisis may be consequences of indirect ocular damage in the shaken baby syndrome (see below). Pupillary abnormalities may reflect either iris trauma or injury to the afferent or efferent nerves that control the light reaction.

I have seen several cases of hyphema secondary to belt beatings. These tend to occur when the child who is being beaten at another site unexpectedly turns, exposing the eye to a blow. Although the eye injury itself may have been unintentional, this type of uncontrolled discipline leading to serious trauma clearly must be regarded as physical abuse and reported.

There are of course many nontraumatic entities that can cause anterior segment findings similar to those reported in child abuse. It is essential that the ophthalmologist look for

other signs of trauma, as nearly all reported anterior segment injuries in abused children have been accompanied by additional evidence of ocular or nonocular trauma. The rare occurrence of spontaneous hyphema in a child with juvenile xanthogranulomatosis should be associated with characteristic skin or iris lesions. The etiology of corneal injuries and hyphema resulting from forceps-assisted vaginal delivery is usually obvious.<sup>48</sup>

Various forms of cataract have been reported as consequences of physical abuse in childhood.<sup>29,54,70,77</sup> To my knowledge, there has been no reported case of a cataract secondary to nonaccidental trauma without other evidence of ocular or nonocular trauma. Trauma is the most common cause of lens subluxation or dislocation in childhood, and there have been a number of reported cases involving abuse.<sup>29,70,90</sup> Nontraumatic disorders that cause spontaneous lens displacement (e.g., Marfan syndrome, homocystinuria, Weill-Marchesani syndrome, syphilis)<sup>72</sup> must obviously be ruled out in such cases. Compared with traumatic dislocation, spontaneous ectopia lentis is more likely to be bilateral, although marked asymmetry may make detection in the less involved eye difficult. In reported cases of lens subluxation secondary to child abuse, the lens has been displaced in all directions except upward (which is the most common direction in Marfan syndrome, the most common cause of spontaneous displacement).

### Vitreoretinal Injury

Since Kiffney first described ocular injury as a result of child abuse in a case of bilateral total rhegmatogenous retinal detachment,<sup>54</sup> there have been many reports confirming that vitreoretinal damage can be a devastating consequence of nonaccidental trauma. This is the third most common ocular manifestation of child abuse (after retinal hemorrhage and periorbital ecchymosis).<sup>40</sup> All reported patients with vitreoretinal damage other than retinal hemorrhage due to child abuse have had other evidence of ocular or nonocular trauma as well.

Unilateral and bilateral retinal detachment due to abuse is most often rhegmatogenous.<sup>54,70</sup> Giant tears and large (usually

temporal) dialyses are common.<sup>29,94</sup> A single case of peripapillary retinal avulsion has been described.<sup>73</sup> One case of exudative ("serosanguinous") detachment has been reported, presumed to be secondary to dissection of intra-retinal hemorrhage into the subretinal space.<sup>55</sup> Tractional detachment may result from organization of vitreous hemorrhage.

Although there are other causes of retinal detachment in childhood, the presence of this finding (especially when bilateral) in the absence of a clear underlying diagnosis or risk factor should always lead to suspicion of non-accidental injury. Retinal detachment associated with retinoblastoma, Coats' exudative retinopathy, X-linked juvenile retinoschisis, or toxocara infestation is in general easily distinguished from post-traumatic detachment. Retinopathy of prematurity can of course lead to retinal detachment in small premature infants, but it must also be kept in mind that premature babies are at increased risk to become victims of child abuse. Congenital retinal dialysis is a rare occurrence that is usually associated with nasal lens coloboma.<sup>46</sup> Peripheral retinal degeneration with hole formation and detachment is not uncommon in adolescents with high myopia, but is quite unusual in an infant or preschool child. Once again, the ophthalmologist must be alert for historical evidence of abuse such as delay in seeking treatment, risk factors such as a stressful social situation, and other physical findings suggestive of injury or neglect.

Damage to the macula can result from traumatic retinoschisis in the shaken baby syndrome (see below) or from blunt contrecoup injury.<sup>98</sup> Macular edema (Berlin's edema, commotio retinae)<sup>28,30,33,73,88</sup> and exudate<sup>32,85,88</sup> may be seen acutely in battered children. Holes, cysts, gliosis, and other forms of macular scarring have been reported as chronic manifestations of abuse.<sup>31,33,39,70</sup> In a large series of physically abused children, Aron et al<sup>5</sup> found numerous peripheral chorioretinal scars, either atrophic or hyperpigmented, which were thought to have been caused by blunt trauma.

Retinal and vitreous hemorrhage, the most common ocular manifestations of child abuse, are discussed below in reference to the shaken baby syndrome. Central retinal artery occlusion has been reported to occur in

abused children; the responsible mechanism is unclear.<sup>29,33,49</sup> Curiously, avulsion of the vitreous base, a finding pathognomonic for trauma (especially when it occurs in the superonasal quadrant),<sup>19</sup> has not been reported in association with child abuse.

Forensic investigation may also be aided by the dating of vitreoretinal injury. Fresh hemorrhage and retinal edema suggest recent trauma. Gliosis, pigmented demarcation lines around retinal detachments or holes, intra-retinal cysts in detached retina, fixed retinal folds in a rhegmatogenous detachment, and vitreous organization are all signs of chronicity.

#### Traumatic Disorders of Eye Movement

A number of abnormalities of ocular movement have been reported in abused children. All have appeared to be secondary to disruption of efferent or afferent nerve supply rather than direct orbital or muscular trauma. In fact, in only two published cases has retrobulbar injury (with proptosis) been suspected in association with child abuse.<sup>12,29</sup>

As early as 1928, a disorder of eye movement (right gaze preference), was described in the setting of neurologic decompensation of an abused child.<sup>1</sup> Sixth nerve palsy,<sup>49</sup> nystagmus,<sup>33,39,54,61</sup> ophthalmoplegia,<sup>12,28,33</sup> and internuclear ophthalmoplegia<sup>33</sup> have since been reported. Strabismus (esotropia or exotropia) is found in up to one third of physically abused children, especially following shaking injury.<sup>12,29,33,39,40,70,79</sup>

#### SHAKEN BABY SYNDROME

Although the association of fractures, subdural hemorrhage, and retinal hemorrhage in infants had been noted earlier,<sup>12</sup> it was not until the 1970s that Caffey<sup>13,14</sup> recognized the significance of this constellation of findings (which he termed the *whiplash shaken infant syndrome*), and proposed the currently accepted explanation for it.<sup>14</sup> In what is now generally known as the shaken baby syndrome (SBS), an infant (nearly always under 2 years old), is violently shaken so that its head undergoes repetitive extreme anterior-posterior, side-to-side, and/or rotatory movement with repeated abrupt deceleration.<sup>13,14,23,61</sup>

Some authors believe impact deceleration also plays a critical role.<sup>22</sup> Unfortunately, Caffey<sup>14</sup> failed to appreciate the violent nature of this form of nonaccidental trauma when he proposed that the syndrome could be caused by playful tossing of a baby in the air, vigorous burping, use of jumper seats, head banging, and various playground activities in children up to 4 to 8 years old. We have since come to understand that these benign activities cannot cause the injuries of SBS.

The cardinal triad of features in SBS consists of central nervous system, skeletal, and ocular injury. Brain damage is aggravated by the disproportionately large size of the infant's head, weak cervical musculature, the large capacity of the unfused cranial vault (which allows for movement of the cerebral hemispheres), and the vulnerability of the incompletely myelinated infant brain. Shaken infants may sustain subarachnoid, subdural, or parenchymal hemorrhages as a result of tearing of bridging veins and direct contusion. Cerebral edema may be so dramatic that it results in obstruction of the major intracranial arteries. Typically, these changes are demonstrated by computed tomography (CT), although in some cases magnetic resonance imaging (MRI) is required for detection.<sup>9,58,81</sup> The cervical spinal cord may also be involved.

The perpetrator of shaking injury sometimes compresses the infant's chest so firmly as to cause posterior or posterolateral rib fractures, which are typically multiple, bilateral, and vertically aligned (Fig. 2). If the baby is shaken while being held by an arm or leg, forces on the long bones may result in stripping of the loosely attached periosteum, subperiosteal hemorrhage, and corner or "bucket handle" fractures, which represent avulsions of the metaphyseal bone where the periosteum is attached. Such rib and long bone findings are virtually pathognomonic of nonaccidental injury, especially when seen in the company of central nervous system and ocular damage. In many cases no fractures are found, however.

Characteristically, shaken infants do not usually show external evidence of trauma as do battered babies. Initial clinical manifestations are often seemingly benign (gastrointestinal distress, poor appetite, irritability),<sup>61</sup> but rapid progression to seizures, coma, and death may ensue. Early recognition is critical to avert spontaneous deterioration from injuries already sustained and to prevent further trauma from repeated shaking. Because of the central position of eye findings in SBS, the ophthalmologist plays a key role in its diagnosis.

Many forms of ocular injury, both acute and

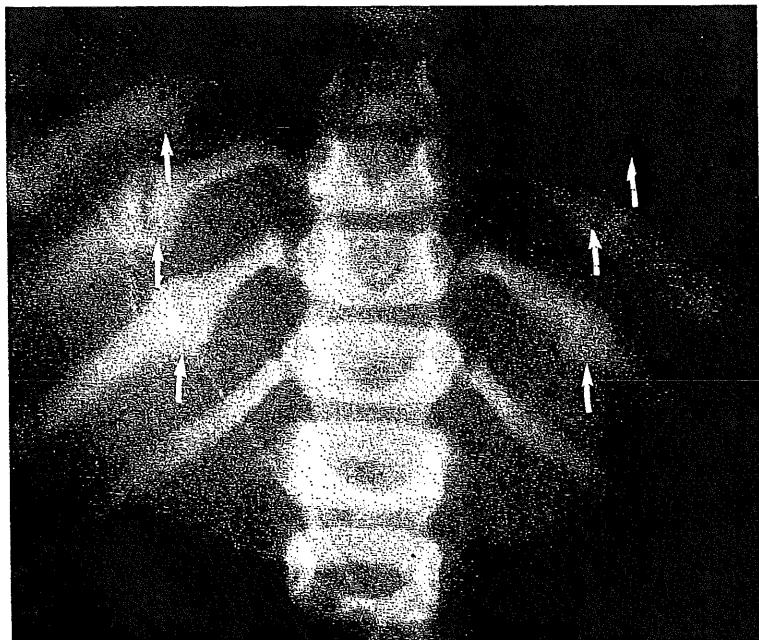


Figure 2. Multiple posterior rib fractures (arrows) in a victim of shaken baby syndrome.

chronic, may be seen in shaken babies. Reported findings have included periorbital bruising,<sup>12</sup> subconjunctival hemorrhage,<sup>79</sup> neovascular glaucoma,<sup>70</sup> cataract,<sup>54,70</sup> and retinal detachment.<sup>39,54,70</sup> The remainder of our discussion of SBS will focus on its most common and significant ophthalmic manifestations: retinal and vitreous hemorrhage with or without associated retinal damage, optic nerve injury, and visual loss secondary to cortical trauma.

#### Retinal and Vitreous Hemorrhage

Retinal hemorrhages (RH) occur in 5% to 23% of all physically abused children<sup>28,49,50,79,85</sup> and in 50% to 80% of shaken babies.<sup>61,100</sup> In more selected populations (e.g., children with ocular manifestations of physical abuse, children with neurologic complications of SBS, or children who die from SBS), the incidence of RH is 70% to 100%.<sup>28,39,65,70,77</sup> These numbers suggest that RH in abused children may have a negative prognostic implication, as it does in adults with subarachnoid hemorrhage.<sup>69</sup> In a recent study, however, the severity of RH correlated only with the severity of acute neurologic manifestations.<sup>96</sup>

These hemorrhages most often involve the posterior pole (Fig. 3), but may extend peripherally to the ora serrata.<sup>55</sup> They occur bilaterally in 58% to 100% of SBS vic-

tims.<sup>39,61,70,77</sup> Strictly unilateral involvement is occasionally seen. Any retinal layer can be involved, but an autopsy study by Rao et al<sup>77</sup> on 14 abused infants, of whom 5 were shaking victims, demonstrated that hemorrhages were most commonly found in the nerve fiber and ganglion cell layers. This pathological finding correlates with the clinical prevalence of flame-shaped hemorrhages. Preretinal hemorrhage occurs frequently (see Figure 3), but subretinal hemorrhage is rare.

Vitreous hemorrhage (VH) may also occur in SBS. Its appearance is usually delayed for 2 days to weeks after shaking injury that acutely is associated with retinal or subhyaloid hemorrhage.<sup>33,70,92</sup> This observation may have forensic importance with regard to establishing the time of injury.

In view of the frequency of retinal and vitreous hemorrhages in shaken babies, dilated indirect ophthalmoscopy is an essential part of the evaluation of any infant with possible SBS. All children under 2 years of age who present with unexplained seizures, altered mental status, or hydrocephalus, as well as those in whom there is specific evidence of trauma or suspicion of abuse, should have such an examination. When there is concern about preserving pupillary reactivity as an indicator of neurologic status, consideration should be given to using weaker, short-acting mydriatic agents. Almost never is the status of the pupils so critical that dilation is absolutely contraindicated. It is of course important to document pupillary size, shape, reactivity, and the presence or absence of a relative afferent defect prior to dilation as up to 33% of abused children with ocular involvement have pupillary abnormalities. For this reason, nurses should not be asked to instill mydratics before the patient is initially seen by the consulting ophthalmologist.

Although some authors have suggested that retinal hemorrhage in children under the age of 2 or 3 years is pathognomonic of child abuse,<sup>24</sup> RH can definitely occur in infants from nontraumatic causes, including severe hypertension, vasculitis, meningitis, endocarditis, generalized sepsis, coagulopathy, and blood dyscrasias. Fundus appearance in such cases may be virtually indistinguishable from that seen in SBS. Intraretinal and subhyaloid hemorrhages have been seen in an



Figure 3. Extensive retinal hemorrhages in a shaken baby.

infant with an intracerebral hematoma secondary to a ruptured arterial aneurysm.<sup>63</sup> Optic disc edema from any cause (e.g., increased intracranial pressure, inflammation) may be associated with peripapillary hemorrhage. Retinal hemorrhages do not result from diabetes or hemoglobinopathies, such as sickle cell disease in infancy.

The most common nonabusive cause of RH in infants is the normal birth process. When examined within the first 24 hours of life, 19% to 32% of babies will have RH in one or both eyes.<sup>7,75,82</sup> By 72 hours after birth the frequency drops to 11% to 15%.<sup>7,48</sup> There is one report of a deep macular hemorrhage in a newborn lasting 6 weeks.<sup>82</sup> Flame-shaped hemorrhages, the most common type of birth RH, usually resolve in 3 to 5 days.<sup>7,82</sup> Although more common following difficult vaginal deliveries, it is important to recognize that any delivery (including cesarian) may be associated with RH.<sup>7,48,75,82</sup> One case of choroidal hemorrhage in a newborn has been reported.<sup>75</sup> The hemorrhages associated with birth do not result in sequelae such as pigment irregularity or other scarring, retinal detachment, or loss of vision.<sup>75</sup> Therefore, the presence of any such chronic finding suggests a postnatal etiology. Physical abuse should also be suspected when a flame-shaped retinal hemorrhage is seen beyond the first 2 weeks of life or any RH is observed more than 6 weeks after birth.

It is difficult to answer the question whether trauma other than that resulting from deliberate abuse can cause retinal hemorrhage in infants. Minor falls and blows or jerking resulting from the tonic-clonic movements associated with grand mal seizures do not result in RH or VH. Severe blunt head trauma can definitely cause RH or VH in adults.<sup>69,92</sup> The truth of this statement has not been established for children, however. In fact, no study published to date has documented the presence of RH in a child after accidental head injury. More thorough and controlled investigation of this issue is needed.

Chest compression in cardiopulmonary resuscitation (CPR) is thought by some to be capable of generating RH.<sup>6,57</sup> Relevant data are available only from one study of flawed design.<sup>51,57</sup> Its authors found that among 51

children who had undergone CPR with chest compression only 6 had retinal hemorrhages, of whom 4 were known victims of abuse under the age of 2 years—one was an infant who was cyanotic after a seizure episode of unknown etiology and later had severe systemic hypertension, and one was a pedestrian traffic accident victim. Thus, it appears unlikely that standard CPR alone results in retinal hemorrhage.

I have encountered several cases in which parents or babysitters contended that their overly vigorous and poorly controlled resuscitative shaking of an apneic or unresponsive baby must have caused the child's injuries. In truth, this possibility sometimes cannot be excluded. The work of Duhaime et al<sup>22</sup> suggests that impact of the head against a hard surface may be an important factor in the generation of the large decelerative forces that cause brain injury in SBS. Evidence of such impact (e.g., scalp bruises or skull fracture) clearly implies abuse but may escape detection premortem even with careful external inspection and CT. It is thus of critical importance to obtain an autopsy in every case of sudden unexplained infant death. Such autopsies should always include gross and histologic examination of the eyes and optic nerves, especially when there was no premortem ophthalmological evaluation.

The pathogenesis of retinal hemorrhages in the shaken baby syndrome is a matter of dis-

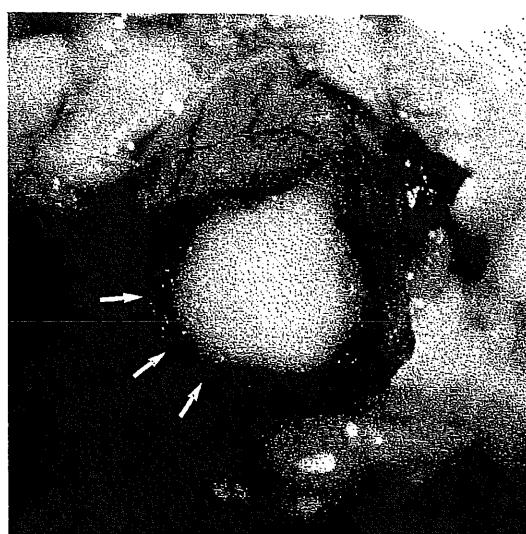


Figure 4. Hemorrhage (arrows) within the optic nerve sheath of a shaken baby.

pute. Many authors continue to believe that they result from hemodynamic forces caused by compression of the chest in the hands of the perpetrator (Purtscher retinopathy),<sup>88</sup> a sudden rise in intracranial pressure due to acute subdural or subarachnoid hemorrhage (Terson syndrome),<sup>89,95</sup> or central retinal vein obstruction from optic nerve swelling or perineural hemorrhage (Fig. 4).<sup>92</sup> Contrecoup trauma from direct ocular compression has also been implicated.<sup>98</sup> Perhaps the most appealing hypothesis is that proposed by Greenwald et al,<sup>33</sup> which holds that hemorrhage is secondary to internal splitting of the retina (traumatic retinoschisis) caused by tractional forces transmitted from the vitreous, which oscillates violently as the head is shaken. There is both clinical and pathological evidence in support of this view.<sup>33,77</sup>

In addition to simple retinal and vitreous hemorrhage, layered blood may be seen within large intraretinal cyst-like cavities, typically in the macular region, which is believed to result from traumatic retinoschisis. Retinal splitting may be so superficial that differentiation from subhyaloid hemorrhage requires very critical indirect ophthalmoscopic inspection. Pathological study of postmortem eyes has in fact revealed that the inner retinal layer often consists only of the internal limiting membrane (E. Torczynski, personal communication). There is frequently a ridge of tented-up retina at the perimeter of the schisis cavity, which gives the entire lesion a crater-like appearance. Disruption of the underlying retinal pigment epithelium may create a curvilinear hypopigmented demarcation at its edge. These findings have not been reported in association with RH or VH from nontraumatic causes.

Greenwald et al<sup>33</sup> observed electroretinographic changes in several infants with deep traumatic retinoschisis who sustained severe retinal injury with permanent visual loss. Absence of a positive B-wave deflection with preservation of the negative A-wave was the characteristic finding. Electroretinography may thus provide a clue to the etiology and prognosis of retinal damage when vitreous hemorrhage prevents ophthalmoscopic visualization of the fundus. Superficial (subinternal limiting membrane) retinal splitting, which does not separate retinal tissue from its

blood supply, leaves the electroretinogram intact and does not prevent complete visual recovery.

The natural history of RH and VH in SBS is quite variable. Retinal hemorrhages may resolve as early as 10 days after injury without sequelae or they may persist for as long as several months.<sup>31,33,70,88</sup> Macular hemorrhage tends to be the slowest to resolve. Vitreous hemorrhage may persist for many months or even years. Harcourt<sup>39</sup> and Levin<sup>58</sup> reported complete resolution of "extensive" retinal and preretinal hemorrhages within 6 weeks of onset in some cases, but in others observed macular scarring or organization of the vitreous with retinal traction.<sup>39</sup> Complete gliotic rings and circumferential ridges around the macula and persistence of intraretinal schisis cavities have also been described.<sup>30,33</sup> While the presence of scarring can be relied on as an indication of chronicity, precise timing of injury in the acute or subacute situation based on the appearance of RH must be considered unreliable.

#### Optic Nerve Involvement

Acute papilledema in abused children has been mentioned relatively infrequently<sup>29,30,39,49,55,73,85</sup> in comparison with the high incidence of optic atrophy in survivors of SBS.<sup>28,29,31,33,39,49,70,79</sup> Perhaps this is due to protection of the optic nerve from exposure to increased intracranial pressure by the expansile unfused calvarium or perhaps it simply reflects the difficulty of assessing the appearance of the optic discs acutely when they are surrounded by RH or obscured by VH. Furthermore, especially in childhood, optic atrophy may result from altered cerebrospinal fluid dynamics without an initial phase of obvious disc edema, or develop secondary to cortical injury with retrograde transsynaptic degeneration. When it is present, papilledema seems to be a poor prognostic sign; Rao's autopsy study showed a 40% incidence.<sup>77</sup> Unilateral or bilateral optic atrophy may occur in up to 45% of survivors.<sup>39</sup> Central retinal artery occlusion (CRAO) has been reported in association with SBS and may be related to the development of optic atrophy.<sup>12,33,49,79</sup> CRAO is usually described in retrospect when survivors of SBS

are found to have optic atrophy plus attenuated retinal vessels; it must be recognized that extensive retinal damage from other mechanisms (e.g., traumatic retinoschisis) could also account for this appearance.

As mentioned above, postmortem examination of the optic nerves in shaken babies often reveals perineural hemorrhage (Fig. 4), which may also contribute, through a mechanism of nerve fiber compression, to the development of optic atrophy in survivors.<sup>55,77</sup> It is not clear whether blood within the optic nerve sheath represents extension from intracranial subarachnoid hemorrhage or accumulates locally due to rupture of bridging veins within the sheath as a result of distension from increased intracranial pressure or shearing forces. Such forces could also cause direct injury to nerve fibers. It is uncertain to what extent either primary or secondary optic nerve damage is responsible for visual loss in SBS.

### Cortical Injury

The most devastating consequence of SBS is cerebral cortical injury, which may leave the survivor blind and/or severely handicapped from motor and cognitive disabilities. Ludwig and Warman<sup>61</sup> reported a 50% incidence of gaze disorders in SBS reflecting central nervous system insults. Various estimates of the frequency of visual deficit secondary to cortical trauma are found in the literature. In an early study, Hollenhorst<sup>44</sup> reported visual loss in 35% of 23 children with subdural or subarachnoid hemorrhage who showed no evidence of retinal damage. Two of 11 SBS survivors in Harcourt and Hopkins' series were bilaterally blind, 3 were unilaterally blind, and 6 had visual deficits attributable to injuries of the visual pathways or cortex.<sup>39</sup> Homonymous hemianopsia has also been reported.<sup>100</sup> Occipital cortical damage with visual loss may result from direct contusion or laceration of the brain, diffuse axonal injury with cerebral edema, intraparenchymal hemorrhage, venous thrombosis, or occlusion of the posterior cerebral arteries.<sup>22,28,100</sup>

High resolution neuroradiological imaging has become an essential element in the evaluation of child abuse victims. Acute occipital

infarcts or edema were evident on CT in 6 of Zimmerman's 21 abused children under the age of 2 years, and an additional 3 showed occipital region pathology on follow-up scans.<sup>100</sup> It was not clear whether all of these victims were shaken. Alexander reported focal occipital lesions in one of four patients studied by MRI.<sup>2</sup> Unfortunately, neither CT nor MRI findings in the acute stage correlate closely enough with chronic functional deficits to permit accurate visual prognosis.

### SEXUAL ABUSE

Sexual abuse of children is a widespread problem that, like most forms of abuse, is underdiagnosed and underreported. It is an uncomfortable and unsettling issue for both medical professionals and lay persons to discuss and confront. Ocular manifestations of sexual abuse are much less common than those of physical abuse, but the ophthalmologist should nevertheless have some familiarity with this area.

Acts of sexual abuse may range from exhibitionism to penile penetration of the vagina or rectum. A majority of cases involve only fondling and genital manipulation.<sup>56</sup> The perpetrators are usually teenagers or adults who seem to obtain at least part of their sexual excitation merely from being in a position of sexual and emotional control over the child. They are most often men who are well known to the child (fathers, step-fathers, paramours of the mother),<sup>15,56</sup> but women also may abuse children of either sex. The child is usually victimized over an extended period of time in a nonviolent fashion, perhaps facilitated by threats or other forms of emotional coercion by the perpetrator. The victim may be of any age from infancy to adolescence.<sup>16</sup>

In most cases of sexual abuse there are no physical signs of injury to the genitalia or other body parts. Periorbital ecchymosis, retinal and vitreous hemorrhages, papilledema, and subconjunctival hemorrhage have been reported in a few children who were beaten in conjunction with sexual abuse.<sup>29,80</sup> RH, VH, or SCH may also result from intense Valsalva maneuvers as the child resists penetration. A more likely ophthalmological manifestation

of sexual abuse is the subsequent onset of functional (nonorganic) visual loss or other functional symptoms such as excessive blinking, photophobia, micropsia, or transient visual obscurations.<sup>17,93</sup> These are common occurrences in childhood, typically developing in response to such emotional stresses as school difficulties, the birth of a new sibling, or the death of a family member.<sup>93</sup> However, such symptoms may also be a manifestation of sexual conflicts or stress.<sup>17,76</sup> The ophthalmologist who diagnoses a functional disorder should be alert to this possibility. Open-ended questions about stress in the child's life may allow the parent to express concern about factors that may include sexual abuse. The primary care physician should always be notified so that further investigations can be conducted if indicated.

Sexual abuse may also be responsible for ocular infection by sexually transmitted diseases (STD) in childhood. The presence of *Neisseria gonorrhoeae* (GC) in the vagina, urethra, rectum, or pharynx of a non-neonatal prepubertal child is considered diagnostic of sexual abuse.<sup>20,47,71</sup> No child who has been diagnosed as a victim of sexual abuse has been reported to have gonorrhea conjunctivitis (GCC), either in isolation or when GC was present at other body sites. However, in a number of reported cases where GCC was attributed to transmission by other means, sexual abuse appears to have been a strong possibility.<sup>4,11,18,21,27,59,83</sup> Auto-inoculation of the conjunctiva via the hands or fomites from other infected body sites is the likely route of infection in adults.<sup>38</sup> Conjunctival infection resulting from the use of urine as a folk remedy for ocular inflammation has been reported in children.<sup>3,91</sup>

Evaluation of the child with GCC must include a complete physical examination, including genital and anal inspection, culturing of the rectum, vagina or male urethra, and throat for GC and other STD, and interviews with the child and family by specially trained professionals. When available, other family members should be referred for GC culturing. The ophthalmologist, in compliance with state law, must report the occurrence of GCC in the non-neonatal prepubertal age range as suspicious for child sexual abuse. At Children's Hospital of Philadelphia, we have

identified three cases of isolated childhood GCC in which extensive "state of the art" medical and social evaluation by physicians and social workers with expertise in child abuse disclosed no evidence of sexual abuse. Infected adults who had nonsexual contact with the child were identified in each case. It appears therefore that nonsexual transmission of GC to the conjunctiva may rarely occur. Should the report of suspected abuse be labelled unfounded after investigation is completed, state laws usually provide for the expunging of records.

Chlamydia, syphilis, herpes simplex (HSV) types 1 and 2, acquired immunodeficiency syndrome (AIDS), and human papilloma virus (HPV) infections have all been reported to occur in children as a result of sexual abuse.<sup>71</sup> Chlamydia conjunctivitis beyond the neonatal period may be a result of prolonged carriage of the infectious microorganism rather than newly acquired sexually transmitted infection.<sup>36,62</sup> Although nonsexual transmission of these diseases (except syphilis) may occur, their ocular manifestations (with the possible exception of nongenital HSV) should nevertheless be treated as possible indicators of covert sexual abuse. Viral typing of HPV<sup>8</sup> and HSV may be helpful in determining the source of the infection, although both HSV 1 and HSV 2 can be transmitted to the genitals or oropharynx as a result of abuse.

## NEGLECT

Abusive neglect of children may involve the withholding of basic general needs or care, or failure to seek necessary medical attention or comply with treatment recommendations. The syndrome of nonorganic failure to thrive (NOFTT)<sup>45</sup> can be an important indicator of general neglect, usually in infancy. This consists of failure to grow and develop normally in the absence of any identifiable disease state (e.g., gastrointestinal malabsorption, congenital infection). Dietary inadequacy may be a factor, but many such infants have sufficient food intake. It is believed that NOFTT results from abnormal emotional bonding and interaction between parent and child that somehow affects the hormonal sys-

tems that regulate growth. Affected infants may appear quite emaciated or just small for their age. They are withdrawn and poorly interactive. Head circumference remains normal. Ophthalmological manifestations of this syndrome have not been reported.

Extreme neglect of basic physical needs resulting in frank malnutrition or life-threatening exposure may of course lead to ocular problems, but the common form of neglect encountered by the ophthalmologist involves medical noncompliance. Failure to obtain glasses, adhere to patching regimens, or keep follow-up appointments is a frequent and frustrating experience in the practice of pediatric ophthalmology. Parents also may not seek medical attention for their child when obvious ocular or visual symptoms are present or after receiving notice of failure on a screening visual acuity test, and in extreme cases may refuse necessary surgery or hospitalization. It is often difficult to determine whether noncompliance is abusively neglectful or whether inadequate financial or social resources are responsible. The parent also may not understand the seriousness of the problem, the importance of the prescribed treatment, or the long-term implications of noncompliance. Some disadvantaged families even avoid medical contact out of fear that a child's problem will be attributed to poor parenting and result in the loss of the child to foster care.

The ophthalmologist confronted with a serious compliance problem must seek out and attempt to remedy such contributing factors. Assistance from social workers, nurses, school teachers, and the primary care pediatrician or family physician may be very helpful in this regard. Only after these efforts have been made should reporting of abuse be considered, unless there is an urgent need for treatment (e.g., in the case of newly diagnosed congenital glaucoma). When it appears that a report may be necessary, the gravity of the situation should be discussed with the parent and a clear statement written in the medical record (and signed by the ophthalmologist, the parent, and a witness if possible) indicating the degree of compliance that will obviate the need for reporting. Should the parent then fail to meet the stated requirements, reporting must proceed.

#### MUNCHAUSEN SYNDROME BY PROXY

Munchausen syndrome by proxy (MBP) is one of the most disturbing of all forms of child abuse. Although it is relatively unknown to ophthalmologists, it may occasionally have ocular manifestations. In this disorder, a parent (almost always the mother) causes her child to come under excessive and chronic medical scrutiny by covertly creating the appearance of illness or even inducing illness in the child.<sup>64,78</sup> This is accomplished by falsification of history (e.g., claiming falsely that a seizure occurred at home), tampering with laboratory testing procedures (e.g., adding sugar to the child's urine specimen), or creating factitious physical findings by actually harming the child (e.g., causing respiratory arrest by suffocation). The resulting illusion of the child's having a bizarre disease leads well-intentioned physicians down a seemingly endless path of medical testing and even invasive measures in an attempt to arrive at a diagnosis.

The child victim may spend long periods in the hospital, which rewards the perpetrator with desired attention, a sense of importance, or perhaps escape from an adverse home situation. She gives the appearance of being a "perfect" parent, constantly at the child's bedside and volunteering to assume nursing duties, which in fact provides opportunity to perpetuate the deception she has created. There is sometimes a history of self-induced illness in the mother during childhood or adolescence. Prior paramedical or nursing training may have given her the knowledge to create convincing evidence of disease. The father is typically passive and shows little involvement in or knowledge of the situation. The child may appear depressed and overly bonded with the perpetrator. The victim is typically preverbal at the time of onset, and may refuse to disclose the terrible secret even when verbal communication becomes possible.

The clinical manifestations of MBP appear to be limited only by the imagination of the perpetrator and have ranged from excessive doctor shopping to fatal injection of insulin. Most commonly, the presenting "illness" involves apnea, cyanosis, seizures, bleeding from an orifice, vomiting, or diarrhea.<sup>78</sup>

Table 3. Warning Signs: Munchausen Syndrome by Proxy

Persistent unexplainable symptoms
Discrepancies between clinical findings and history
A clinical picture that "just doesn't make sense"
Child resistant or inconsistently responsive to all treatment attempts
Caretaker with a history of factitious illness
Caretaker who refuses to leave child alone in the hospital yet is resistant to outpatient treatment
Caretaker who establishes a "social network" within the hospital during child's hospitalization
Caretaker who is inexplicably calm considering the clinical situation
Caretaker who has had some medical/paramedical/nursing training
Clinical manifestations that always have their onset in the presence of the caretaker and are never witnessed by any other person, especially during hospitalization
Caretaker's spouse visits infrequently and is poorly informed as to the child's medical status
Caretaker adamantly refuses to accept the possibility that the child's illness is nonorganic

Adapted from Meadow R: Munchausen syndrome by proxy. Arch Dis Child 57:92-98, 1982; and Guandolo VL: Munchausen syndrome by proxy: An outpatient challenge. Pediatrics 75:526-530, 1985; with permission.

There have been three reports of ocular involvement. Two siblings were the victims of repetitive instillation of a toxic chemical into the eyes that resulted in chronic conjunctivitis leading to scarring and corneal opacification.<sup>87</sup> In another case, unilateral mydriasis was attributed to the covert instillation of atropine.<sup>99</sup> Factitious periorbital cellulitis was caused by subcutaneous injection of a metallic substance in the third reported case.<sup>25</sup>

The key to diagnosis of MBP is suspicion in appropriate circumstances. Table 3 lists a number of factors that should lead the ophthalmologist at least to consider the possibility that a puzzling chronic disorder that does not respond to treatment may be the result of covert acts on the part of a parent. When evidence is regarded as sufficient to make a presumptive diagnosis, the child must be separated from the perpetrator (with the assistance of state authorities if necessary). Immediate cessation of all clinical manifestations then confirms the suspicion.

## THE OPHTHALMOLOGIST'S ROLE

With any clinical symptom or sign, the skilled physician creates a mental list of diagnoses that could explain the complaint or finding, then considers in turn each condition on the list, quickly discarding some possibilities while seriously entertaining others, moving closer and closer to a final diagnosis through a planned series of evaluative steps. In some cases this process can be completed in minutes; in others it extends over months or years. We have seen in this article that virtually any visual or ocular abnormality may be related to child abuse. The ophthalmologist must therefore be careful to place this etiology in the tentative differential diagnosis, to be ruled in or out on the basis of information acquired through historical inquiry, ocular or general physical examination, or social evaluation.

Ophthalmologists who care for children must be ready to act as a part of a multidisciplinary team that works with abusive families and child victims. Involvement may take the form of primary reporting when the initial manifestation of abuse is an ocular abnormality, providing confirmatory evidence through consultation in cases where others have suspected abuse. Specific contributions by the consulting ophthalmologist might include documentation of the presence (and estimation of chronicity) of retinal hemorrhage or other signs of shaking injury or blunt trauma (which can be done by means of postmortem indirect ophthalmoscopy when necessary), and in fatal cases assisting the forensic pathologist by obtaining vitreous specimens for postmortem toxicology or removing the globes and optic nerves for gross and microscopic study.<sup>34,101</sup> Other members of the team (social workers, law enforcement officers, nurses, primary care physicians) can assist the ophthalmologist by investigating the social situation and addressing concerns regarding the history, using nonconfrontational techniques of inquiry that minimize the anxiety and hostility of parents.

Like other physicians, ophthalmologists tend to be underreporters of child abuse. This may be due to unfamiliarity with its clinical manifestations, disbelief that seemingly caring parents could commit abusive acts, reluc-

tance to deal with matters that may be unpleasant, time-consuming and financially unrewarding, feelings of inadequacy in dealing with such matters, fear of provoking verbal or even physical expression of hostility through accusations, lack of faith in the effectiveness of the reporting system, or concern that overzealous agencies will separate a child from its parents inappropriately. Physicians who take the time to educate themselves about their own state laws and local social work support systems usually find that many of their fears and concerns are unfounded. For example, it is extraordinarily rare for a reporting physician to be asked to appear in court as a witness.

It must always be remembered that reporting suspicion of abuse is mandated by law and that the physician is protected against legal recrimination for such action. But beyond our legal obligation, it is morally incumbent upon us to act as advocates on behalf of the innocent victims of child abuse. In so doing we help to protect our society's most vital resource: its children.

## REFERENCES

- Aikman J: Cerebral hemorrhage in infant, aged eight months: Recovery. *Arch Pediatr* 45:56, 1928
- Alexander RC, Schor DP, Smith WL: Magnetic resonance imaging of intracranial injuries from child abuse. *J Pediatr* 109:975, 1986
- Alfonso E, Friedland B, Hupp S, et al: *Neisseria gonorrhoeae* conjunctivitis: An outbreak during an epidemic of acute hemorrhagic conjunctivitis. *JAMA* 250:794, 1983
- Allue X, Rubio T, Riley HD: Gonococcal infections in infants and children: Lessons from fifteen cases. *Clin Pediatr* 12:584, 1973
- Aron JJ, Marx P, Blanck MF, et al: Signes oculaires observés dans le syndrome de Silverman. *Ann Oculist* 203:533, 1970
- Bacon CJ, Sayer GC, Howe JW: Extensive retinal haemorrhages in infancy—An innocent cause. *Br Med J* 1:281, 1978
- Baum JD, Bulpitt CJ: Retinal and conjunctival haemorrhage in newborn. *Arch Dis Child* 45:344, 1970
- Bender ME: New concepts of condyloma acuminata in children. *Arch Dermatol* 122:1121, 1986
- Bennett HS, French JH: Elevated intracranial pressure in a whiplash-shaken infant syndrome detected with normal computerized tomography. *Clin Pediatr* 19:633, 1980
- Blinder KJ, Scott W, Lange MP: Abuse of cyanoacrylate in child abuse. *Arch Ophthalmol* 105:1632, 1987
- Branch G, Paxton R: A study of gonococcal infections among infants and children. *Public Health Rep* 80:347, 1965
- Caffey J: Multiple fractures in the long bones of infants suffering from chronic subdural hematoma. *AJR* 56:163, 1946
- Caffey J: On the theory and practice of shaking infants. *Am J Dis Child* 124:161, 1972
- 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* 54:396, 1974
- Cameron JM, Johnson HR, Camps FE: The battered child syndrome. *Med Sci Law* 6:2, 1966
- Cantwell HB: Sexual abuse of children in Denver, 1979: Reviewed with implications for pediatric intervention and possible prevention. *Child Abuse Neglect* 5:75, 1981
- Catalano RA, Simon JW, Krohel GB, et al: Functional visual loss in children. *Ophthalmology* 93:385, 1986
- Cooperman MB: Gonococcus arthritis in infancy: A clinical study of forty-four cases. *Am J Dis Child* 33:932, 1927
- Cox MS, Schepens CL, Freeman HM: Retinal detachment due to ocular contusion. *Arch Ophthalmol* 76:678, 1966
- DeJong AR: Sexually transmitted diseases in sexually abused children. *Sex Transm Dis* 13:123, 1986
- Doyle JO: Accidental gonococcal infection in the eyes of children. *Br Med J* 1:88, 1972
- Duhame A, Gennarelli TA, Thibault LE, et al: The shaken baby syndrome: A clinical, pathological, and biomechanical study. *J Neurosurg* 66:409, 1987
- Dykes LJ: The whiplash shaken infant syndrome: What has been learned? *Child Abuse Neglect* 10:211, 1986
- Eisenbrey AB: Retinal hemorrhage in the battered child. *Child Brain* 5:40, 1979
- Feeenstra J, Merth IT, Traffers PD: A case of Munchausen syndrome by proxy. *Tijdschrift Voor Kindergeneeskunde* 56:148, 1988
- Feldman KW, Schaller RT, Feldman JA, et al: Tap water scald burns in children. *Pediatrics* 62:1, 1978
- Folland DS, Burke RE, Hinman AR, et al: Gonorrhoea in preadolescent children: An inquiry into source of infection and mode of transmission. *Pediatrics* 60:153, 1977
- Frank Y, Zimmerman R, Leeds NM: Neurologic manifestation in abused children who have been shaken. *Dev Med Child Neurol* 27:312, 1985
- Friendly DS: Ocular manifestations of physical child abuse. *Trans Am Acad Ophthalmol Otolaryngol* 75:318, 1971
- Gaynor MW, Koh K, Marmor MF, et al: Retinal folds in the shaken baby syndrome. *Am J Ophthalmol* 106:423, 1988
- Giangiacomo J, Barkett KJ: Ophthalmoscopic findings in occult child abuse. *J Pediatr Ophthalmol Strabismus* 22:234, 1985
- Gilkes MJ, Mann TP: Fundi of battered babies. *Lancet* 2:468, 1967
- Greenwald MJ, Weiss A, Oesterle CS, et al: Traumatic retinoschisis in battered babies. *Ophthalmology* 93:618, 1986

34. Griest KJ, Zumwalt RE: Child abuse by drowning. *Pediatrics* 83:41, 1989
35. Guandolo VL: Munchausen syndrome by proxy: An outpatient challenge. *Pediatrics* 75:526-530, 1985
36. Hammerschlag MR: Chlamydial infections. *J Pediatr* 114:727, 1989
37. Hampton RL, Newberger EH: Child abuse incidence and reporting by hospitals: Significance of severity, class, and race. *Am J Public Health* 75:56, 1985
38. Hansen T, Burns RP, Allen A: Gonorrhreal conjunctivitis: An old disease returned. *JAMA* 195:178, 1966
39. Harcourt B, Hopkins D: Ophthalmic manifestations of the battered-baby syndrome. *Br Med J* 3:398, 1971
40. Harley RD: Ocular manifestations of child abuse. *J Pediatr Ophthalmol Strab* 17:5, 1980
41. Heins M: Pediatrics. *JAMA* 261:2874, 1989
42. Hight DW, Bakalar HR, Lloyd JR: Inflicted burns in children: Recognition and treatment. *JAMA* 242:517, 1979
43. Hobbs CJ: When are burns nonaccidental? *Arch Dis Child* 61:357, 1986
44. Hollenhorst RW, Stein HA: Ocular signs and prognosis in subdural and subarachnoid bleeding in young children. *Arch Ophthalmol* 60:187, 1958
45. Homer C, Ludwig S: Categorization of etiology of failure to thrive. *Am J Dis Child* 135:848, 1981
46. Hovland KR, Schepens CL, Freeman HM: Developmental giant retinal tears associated with lens colobomas. *Arch Ophthalmol* 80:325, 1968
47. Ingram DL, White ST, Durfee MF, et al: Sexual contact in children with gonorrhea. *Am J Dis Child* 136:994, 1982
48. Jain IS, Singh YP, Gupta SL, et al: Ocular hazards during birth. *J Pediatr Ophthalmol Strab* 17:14, 1980
49. Jensen AD, Smith RE, Olson MI: Ocular clues to child abuse. *J Pediatr Ophthalmol* 8:270, 1971
50. Johnson CF, Showers J: Injury variables in child abuse. *Child Abuse Neglect* 9:207, 1985
51. Kanter RK: Retinal hemorrhage after cardiopulmonary resuscitation or child abuse. *J Pediatr* 180:430, 1986
52. Keen JH, Lendrum J, Wolman B: Inflicted burns and scalds in children. *Br Med J* 4:268, 1975
53. Kempe CH, Silverman FN, Steele BF, et al: The battered-child syndrome. *JAMA* 181:17, 1962
54. Kiffney GT: The eye of the "battered child." *Arch Ophthalmol* 72:231, 1964
55. Lambert SR, Johnson TE, Hoyt CS: Optic nerve sheath hemorrhages associated with the shaken baby syndrome. *Arch Ophthalmol* 104:1509, 1986
56. Leventhal JM: Have there been changes in the epidemiology of sexual abuse of children during the 20th century? *Pediatrics* 82:766, 1988
57. Levin AV: Retinal hemorrhages after cardiopulmonary resuscitation: Literature review and commentary. *Pediatr Emerg Care* 2:269, 1986
58. Levin AV, Magnusson MR, Rafto SE, et al: Shaken baby syndrome diagnosed by magnetic resonance imaging. *Pediatr Emerg Care* 5:181, 1989
59. Low RC, Cho CT, Dudding BA: Gonococcal infections in young children. *Clin Pediatr* 16:626, 1977
60. Ludwig S: Child abuse. In Fleisher G, Ludwig S (eds): *Textbook of Pediatric Emergency Medicine*. Baltimore, Williams & Wilkins, 1983, p 1027
61. Ludwig S, Warman M: Shaken baby syndrome: A review of 20 cases. *Ann Emerg Med* 13:51, 1984
62. Markham RH, Richmond SJ, Walsh NW, et al: Severe persistent inclusion conjunctivitis in a young child. *Am J Ophthalmol* 83:414, 1977
63. McLellan NJ, Prasad R, Punt J: Spontaneous subhyaloid and retinal haemorrhages in an infant. *Arch Dis Child* 61:1130, 1986
64. Meadow R: Munchausen syndrome by proxy. *Arch Dis Child* 57:92-98, 1982
65. Miziara CS, Serrano VA, Kok F, et al: (The battered child syndrome: Neurologic aspects in 7 cases). *Arq Neuropsiquiatr* 46:359, 1988
66. Montrey JS, Barcia PJ: Nonaccidental burns in child abuse. *South Med J* 78:1324, 1985
67. Moritz AR, Henriques FC: Studies of thermal injury: II. The relative importance of time and surface temperature in the causation of cutaneous burns. *Am J Pathol* 23:695, 1947
68. Moritz AR: Studies of thermal injury: III. The pathology and pathogenesis of cutaneous burns. *Am J Pathol* 23:915, 1947
69. Muller PJ, Deck JH: Intraocular and optic nerve sheath hemorrhage in cases of sudden intracranial hypertension. *Neurosurgery* 41:160, 1974
70. Mushin AS: Ocular damage in the battered-baby syndrome. *Br Med J* 3:402, 1971
71. Neinstein LS, Goldenring J, Carpenter S: Nonsexual transmission of sexually transmitted diseases: an infrequent occurrence. *Pediatrics* 74:67, 1984
72. Nelson LB, Maumenee IH: Ectopia lentis. *Surv Ophthalmol* 27:143, 1982
73. Ober RR: Hemorrhagic retinopathy in infancy: A clinicopathologic report. *J Pediatr Ophthalmol Strabismus* 17:17, 1980
74. O'Neill JA, Meacham WF, Griffin PP, et al: Patterns of injury in the battered child syndrome. *J Trauma* 13:332, 1973
75. Planten JT, Schaaf PC: Retinal hemorrhage in the newborn. *Ophthalmologica* 162:213, 1971
76. Rada RT, Meyer CG, Kellner R: Visual conversion reaction in children and adults. *J Nerv Ment Dis* 166:580, 1978
77. Rao N, Smith RE, Choi JH, et al: Autopsy findings in the eyes of fourteen fatally abused children. *Forensic Sci Int* 39:293, 1988
78. Rosenberg DA: Web of deceit: A literature review of Munchausen syndrome by proxy. *Child Abuse Neglect* 11:547, 1987
79. Roussey M, Betremieux P, Journe H, et al: L'ophtalmologue et les victimes de s'veces. *J Fr Ophthalmol* 10:201, 1987
80. San Martin R, Steinkuller PG, Nisbet RM: Retinopathy in the sexually abused battered child. *Ann Ophthalmol* 13:89-91, 1981
81. Sato Y, Yuh WT, Smith WL, et al: Head injury in child abuse: Evaluation with MR imaging. *Radiology* 173:653, 1989
82. Sezen F: Retinal hemorrhage in newborn infants. *Br J Ophthalmol* 55:248, 1970
83. Shore WB, Winkelstein JA: Nonvenereal transmission of gonococcal infections to children. *J Pediatr* 79:661, 1971
84. Simpson K, Knight B: *Forensic Medicine*, ed 9. London, Edward Arnold, 1985, p 55
85. Smith SM, Hanson R: 134 battered children: A

- medical and psychological study. *Br Med J* 3:666, 1974
86. Stone NH, Rinaldo L, Humphrey CR, et al: Child abuse by burning. *Surg Clin North Am* 50:1419, 1970
  87. Taylor D, Bentovim A: Recurrent nonaccidentally inflicted chemical eye injuries to siblings. *J Pediatr Ophthalmol* 13:238, 1976
  88. Tomasi LG, Rosman P: Purtscher retinopathy in the battered child syndrome. *Am J Dis Child* 93:1435, 1986
  89. Toosi SH, Malton M: Terson's syndrome—Significance of ocular findings. *Ann Ophthalmol* 19:7, 1987
  90. Tseng SS, Keys MP: Battered child syndrome simulating congenital glaucoma. *Arch Ophthalmol* 94:839, 1976
  91. Valenton MJ, Abendanjo R: Gonorrhreal conjunctivitis: Complication after contamination with urine. *Can J Ophthalmol* 8:421, 1973
  92. Vanderlinden RG, Chisolm LD: Vitreous hemorrhages and sudden increased intracranial pressure. *J Neurosurg* 41:167, 1974
  93. Vrabec TR, Levin AV, Nelson LB: Functional blinking in childhood. *Pediatrics* 83:967, 1989
  94. Weidenthal DT, Levin DB: Retinal detachment in a battered infant. *Am J Ophthalmol* 81:725, 1976
  95. Weingeist TA, Goldman EJ, Folk JC, et al: Terson's syndrome: Clinicopathological correlations. *Ophthalmology* 93:1435, 1986
  96. Wilkinson WS, Han DP, Rappley MD, et al: Retinal hemorrhage predicts neurologic injury in the shaken baby syndrome. *Arch Ophthalmol* 107:1472, 1989
  97. Wilson EF: Estimation of the age of cutaneous contusions in child abuse. *Pediatrics* 60:750, 1977
  98. Wolter JR: Coup-contrecoup mechanism of ocular injuries. *Am J Ophthalmol* 56:785, 1975
  99. Wood PR, Fowlkes J, Holden P, et al: Fever of unknown origin for six years: Munchausen syndrome by proxy. *J Fam Pract* 28:391, 1989
  100. Zimmerman RA, Bilaniuk LT, Bruce D, et al: Computed tomography of craniocerebral injury in the abused child. *Radiology* 130:687, 1979
  101. Zurnwalt RE, Hirsch CS: Subtle fatal child abuse. *Hum Pathol* 11:167, 1980

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