CME Category 1 credits

Review Article

Inflicted Childhood Neurotrauma (Shaken Baby Syndrome): Ophthalmic Findings

Brian J. Forbes, MD, PhD; Cindy W. Christian, MD; Alexander R. Judkins, MD; and Kasia Kryston, BS

Abstract

Inflicted childhood neurotrauma (shaken baby syndrome) is the term used for violent, nonaccidental, repetitive, unrestrained acceleration–deceleration head and neck movements, with or without blunt head trauma, combined with a unique, age-related biomechanical sensitivity in children typically younger than 3 years. This syndrome is typically characterized by a combination of fractures, intracranial hemorrhages, and intraocular hemorrhages. Retinal hemorrhage is the most common ophthalmic finding, and usually occurs at all levels of the retina. In recent years, increasing pressure has been placed on ophthalmologists to render diagnostic interpretations of the retinal findings in children suspected to be victims, which may have great forensic implications in criminal proceedings. New research has increased our understanding of the pathophysiology of retinal hemorrhages, the importance of specifically characterizing the types, patterns, and extent of these retinal hemorrhages, and the differential diagnosis. ***J Pediatr Ophthalmol Strabismus* 2004;41:80-88.**

Educational Objectives

1. To summarize the ophthalmic literature related to inflicted childhood neurotrauma to review not only the ocular findings, but also the associated systemic and psychosocial findings in the syndrome.
2. To identify the limited differential diagnosis of retinal hemorrhages in the case of a small child or infant.
3. To recognize the important role of the ophthalmologist in the evaluation of victims of inflicted childhood neurotrauma.

See quiz on page 105; no payment required.

Introduction

Homicide is the leading cause of injury and death in infancy, and half of all infant homicides

Dr. Forbes and Ms. Kryston are from the Department of Ophthalmology; Dr. Christian is from the Department of Pediatrics; and Dr. Judkins is from the Department of Pathology, The Children's Hospital of Philadelphia, University of Pennsylvania, Philadelphia, Pennsylvania.

Originally submitted October 26, 2003.

Accepted for publication December 11, 2003.

Address reprint requests to Brian Forbes, MD, PhD, Department of Ophthalmology, Ninth Floor Main Building, The Children's Hospital of Philadelphia, 34th and Civic Center Blvd., Philadelphia, PA 19104.

The authors have no industry relationships to disclose.

In accordance with ACCME policies, the audience is advised that this continuing medical education activity may contain references to unlabeled uses of FDA-approved products or to products not approved by the FDA for use in the United States. The faculty members have been made aware of their obligation to disclose such usage.

The material presented at or in any SLACK Incorporated continuing medical education activities does not necessarily reflect the views and opinions of SLACK Incorporated. Neither SLACK Incorporated nor the faculty endorse or recommend any techniques, commercial products, or manufacturers. The faculty/authors may discuss the use of materials and/or products that have not yet been approved by the U.S. Food and Drug Administration. All readers and continuing education participants should verify all information before treating patients or utilizing any product.

occur during the first 4 months of life.[1,2] Eighty percent of infant homicides are thought to represent child abuse, and each day in the United States, more than 3 children die as a result of child abuse. Most of these deaths are caused by inflicted neurotrauma, which results from violent, nonaccidental shaking, blunt impact to the head, or both. Historically, the injuries resulting from repetitive unrestrained head and neck movements from shaking were termed the "whiplash shaken infant syndrome," which is currently commonly referred to as the "shaken baby syndrome."[3]

The most difficult and controversial aspect of the diagnosis of shaken baby syndrome is the biomechanical implication of this term. Although confessions are obtained in a few cases and the reliability of such confessions must be considered suspect, those perpetrators who have confessed support the prominence of repetitive violent shaking as the key element in the generation of shaken baby syndrome. One landmark study suggested that impact trauma in addition to shaking was required to generate the level of force needed for an infant to sustain brain injury.[4] Most infants with severe brain injury due to abuse have clinical or autopsy evidence of blunt impact trauma, and some biomechanical data suggest that impact is necessary for injury.[4] Ultimately, the contributions of shaking versus impact in the pathogenesis of this syndrome are debated, leading clinicians and researchers to favor more generic terms for the injuries identified from inflicted head trauma. Some have suggested the syndrome be renamed "shaken impact syndrome," whereas others have suggested "inflicted traumatic brain injury" or "inflicted childhood neurotrauma." As inflicted childhood neurotrauma was the favored term at a recent National Institutes of Health conference of leading investigators in the field, it will be used throughout this text.

Clinical findings in affected infants include subdural hemorrhage, hypoxic–ischemic brain injury, retinal hemorrhages, skeletal injuries, and cutaneous or other injuries. The frequency with which noncranial injuries are identified varies by age and presentation, and skeletal or cutaneous injuries are not necessary for diagnosis. Unlike most other forms of ocular trauma, there are usually minimal external ocular signs of injury and no evidence of direct blows to the eye. Skeletal fractures are found in 30% to 70% of injured children, and retinal hemorrhages are seen in approximately 80%.[5-14]

TABLE
COMMON PRESENTATIONS OF CHILDREN WITH INFLICTED CHILDHOOD NEUROTRAUMA

Upper respiratory infection symptoms
Diarrhea
Fever
Vomiting
Colic
Irritability
Lethargy
Startling episodes
Apnea
Bulging fontanelle
History of minor trauma
Poor feeding
Failure to thrive
Seizures

Victims of inflicted childhood neurotrauma are generally younger than 3 years, and most are infants. The clinical presentation reflects the severity of the injury, and this ranges from mild lethargy or irritability to acute life-threatening events, unexplained seizures, or coma. Falls in childhood are the most common reason for emergency department visits and hospital admissions; the table outlines the most common reasons that children who are eventually diagnosed as having inflicted childhood neurotrauma present to a physician.[15] In a review of missed cases of inflicted childhood neurotrauma, viral gastroenteritis was the most common incorrect diagnosis made, followed by unintentional injury.[15] When physicians misdiagnose inflicted injury as either unintentional trauma or a medical disease, approximately 25% of infants will sustain further injury before the correct diagnosis is made. The consequences of missing abuse are more dangerous to a child than falsely accusing a family of abuse, although underdiagnosis and overdiagnosis have unacceptable consequences for both the children and their families.

Approximately one-third of injured infants are misdiagnosed at the time of initial presentation, especially those who are young, have mild injuries, or live in nonminority, 2-parent households.[16]

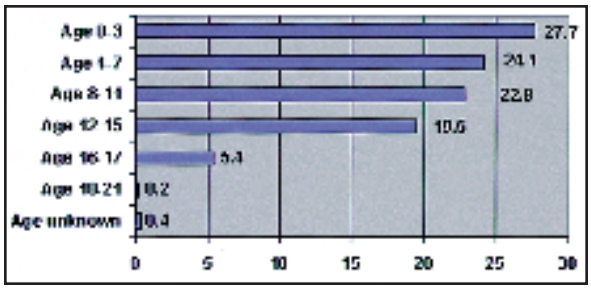

Figure 1. Percentage of victims by age group, 2001.

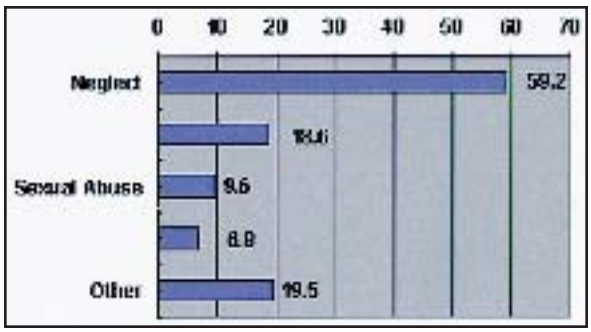

Figure 3. Type of maltreatment of victims, 2001.

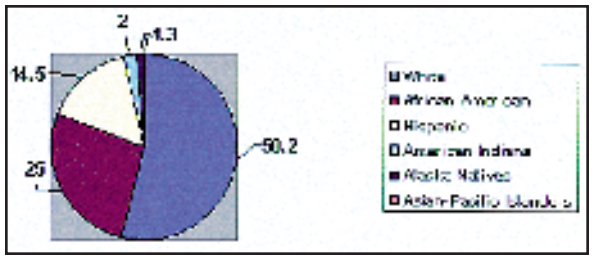

Figure 2. Race of victims, 2001.

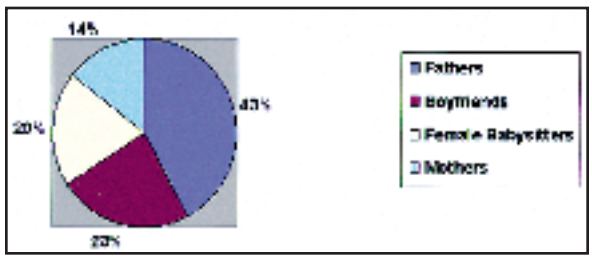

Figure 4. Perpetrators in criminal cases of abusive head trauma.

Victims of inflicted childhood neurotrauma are younger (mean, 12.8 vs 27.5 months), more likely to have a history of medical problems (53% vs 14.1%), and 7 times more likely to have been born prematurely (2.2% vs 0.07%) than are children with accidental neurotrauma.[17] It has also been suggested that racial differences exist in the evaluation and reporting of patients with fractures for child abuse, particularly in toddlers with accidental injuries.[18] In an effort to improve the early identification of abused infants, funduscopy to look for retinal hemorrhages has been advocated with some success in hospitals to evaluate infants who present with acute life-threatening events.[19,20] No medical condition fully mimics the clinical features of inflicted childhood neurotrauma, although intracranial and retinal bleeding can sometimes be seen in accidental injury, coagulopathy, and rare metabolic diseases. A detailed eye examination is necessary to completely assess the presence and extent of intraocular injury in this syndrome and to differentiate it from other medical problems.

In 2001, an estimated 903,000 children were abused or neglected; Figures 1 to 3 outline the more common characteristics of these children and the type of abuse that occurred.[21] The perpetrators in criminal cases of abusive head trauma are outlined in Figure 4.[22]

Acute Ophthalmic Findings in Inflicted Childhood Neurotrauma

Autopsy and in vivo studies of the acute ocular findings in infants and toddlers younger than 3 years with nonaccidental head injury from inflicted childhood neurotrauma have described a consistent clinical picture. These characteristic ophthalmic findings include intraocular hemorrhage with a reported frequency of 50% to 100%, with most studies having reported approximately 80%.[6-14] Retinal hemorrhage occurs at all levels of the retina, including blot, flame-shaped, and preretinal hemorrhage as well as vitreous hemorrhage. Retinal hemorrhages can be few in number, exclusively intraretinal, and confined to the posterior pole, although they often are too numerous to count, are present at all layers, and extend to the ora serrata (Fig. 5). Dense preretinal or vitreous hemorrhages may obscure underlying retinal hemorrhage.

The frequency of retinal hemorrhage is highest in autopsy cases and lowest in intact survivors, and typically, hemorrhages are present in both eyes, although asymmetry and unilaterality are well recognized (Fig. 6). Papilledema occurs in less than 10% of cases.[23] Both optic nerve sheath and intraocular hemorrhages are frequently

Figure 5. Wide-angle funduscopic photograph showing the ocular fundus of an infant with acute inflicted childhood neurotrauma. Subretinal, intraretinal, and preretinal hemorrhages and optic disc hemorrhages are visible.

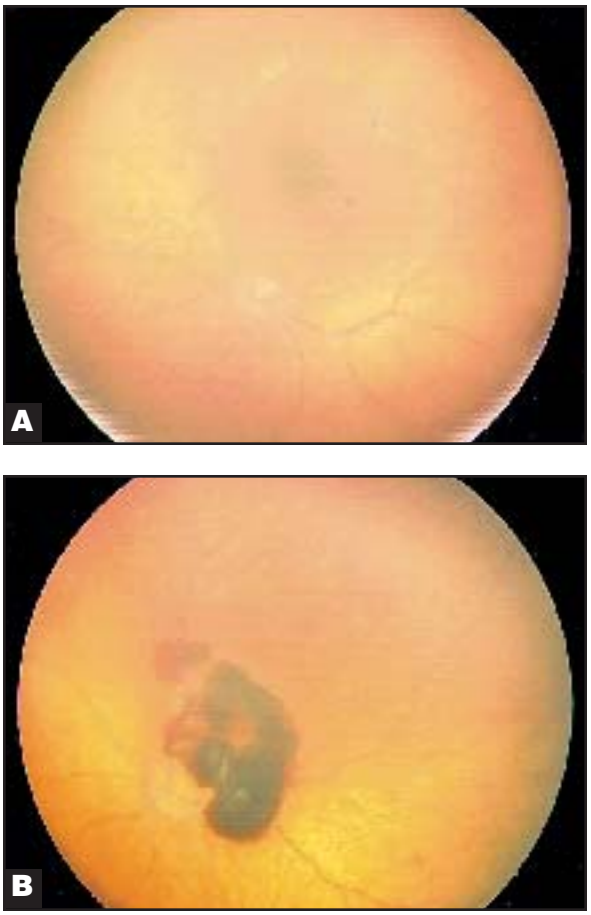

Figure 6. (A) Right and (B) left funduscopic photographs showing marked asymmetry in the degree of hemorrhaging present.

reported findings in postmortem examinations of victims of inflicted childhood neurotrauma (Fig. 7A). Optic nerve sheath hemorrhages frequently involve multiple layers, but often show a preponderance of hemorrhage in the subdural space (Figs. 7B and 7C). Intraocular hemorrhages can involve vitreous, preretinal, intraretinal, and subretinal compartments. Retinal hemorrhages may involve all layers (Fig. 7D) or may be more restricted in distribution depending on the severity of injury.[24]

Retinoschisis may occur, most often in the macular area but also peripherally. Ophthalmoscopically, there is a dense central hemorrhage surrounded by a pale, elevated retinal fold in a circular shape. These lesions, seen both histopathologically and clinically, have also been called "hemorrhagic macula cysts" and "perimacular circular folds,"[25-28] and have a unique and characteristic appearance seen only rarely in other types of head trauma.[26] Macular retinoschisis with or without perimacular folds has been well documented clinically, at postmortem examination, and by electroretinography as a distinctive finding that has not been reported due to any other cause in children younger than 5 years.[28]

Late Ophthalmic Findings in Inflicted Childhood Neurotrauma

In contrast to the dramatic and relatively specific acute findings, late changes associated with inflicted childhood neurotrauma are neither consistent nor specific to inflicted childhood neurotrauma. Permanent visual impairment is frequent, and central visual impairment related to the hypoxic ischemic brain injury from inflicted childhood neurotrauma and optic atrophy is the most common cause of long-term reduced vision. Amblyopia caused by visual deprivation due to prolonged vitreous hemorrhage may occur.[21] Optic disc pallor, optic atrophy, nonspecific retinal pigmentary changes, macular hole, vitreous opacities, retinal thinning, and high myopia may also be seen in survivors of inflicted childhood neurotrauma (Fig. 8).[21,29,30]

The age of an intraocular hemorrhage is difficult to assess clinically. It is assumed that the hemorrhages occur immediately at the time of injury. Some evolution, including the darkening of the retinal hemorrhages, organization of the vitreous

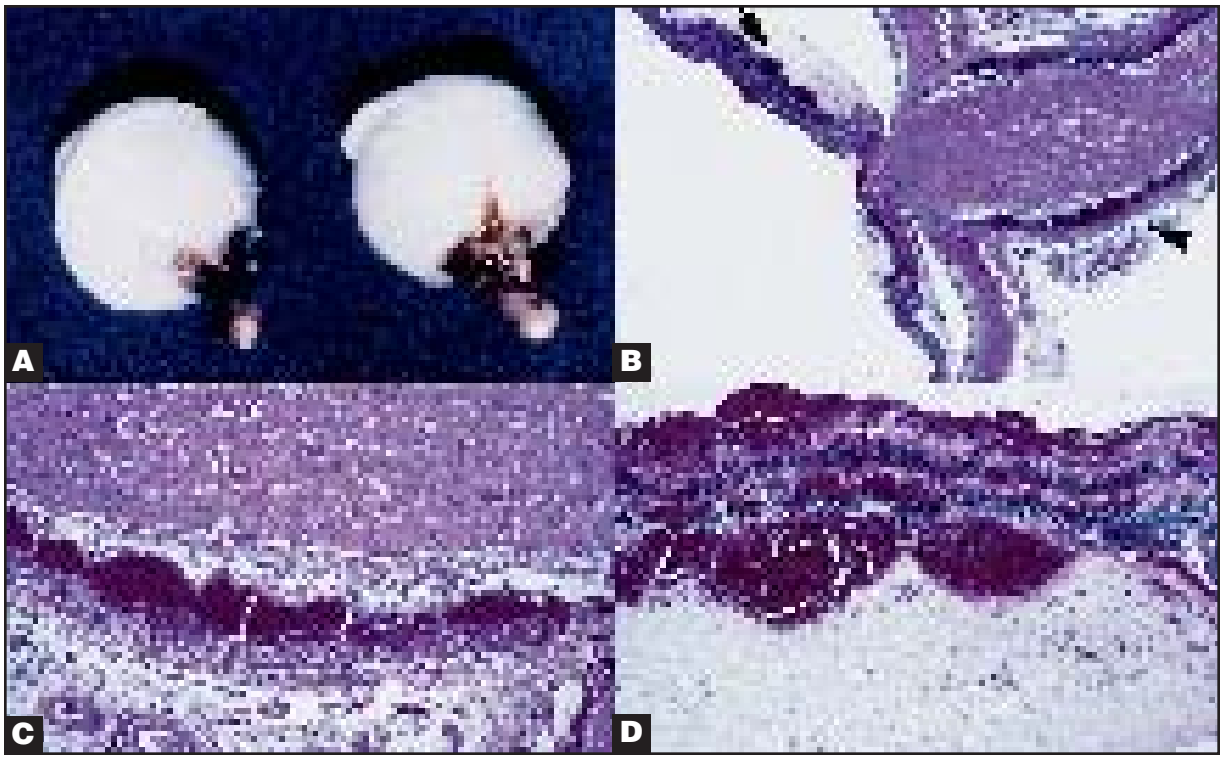

Figure 7. (A) The right and left eyes of a 7-week-old battered infant displaying striking optic nerve sheath hemorrhages, most prominent at the junction of the globe and optic nerve. (B) Low-magnification photomicrograph of a section of the optic nerve coming into the posterior globe. Note the acute hemorrhage in the subdural space on both sides of the optic nerve (arrowhead), projection of the optic nerve head into the posterior chamber, and massive hemorrhage in all layers of the retina (arrow). (C) Higher magnification of the region indicated by the arrow in B, showing acute subdural hemorrhage with intradural extension along the course of the optic nerve. The nerve is in the upper half of the photomicrograph and is cut longitudinally. (D) Higher magnification of the region indicated by the arrow in B, showing massive retinal hemorrhage with bleeding into all layers of the retina with frank clot formation. Also note the accumulation of subretinal edema with scattered acute hemorrhages in the lower half of the figure. (Photographs contributed by Dr. Lucy B. Rorke.)

hemorrhages, and disappearance of the retinal hemorrhages, occurs gradually during 2 to 4 weeks following the acute injury.

Prognostic Value of Ophthalmic Findings

In addition to the diagnostic significance of acute and late ophthalmic manifestations of inflicted childhood neurotrauma, several ocular findings have prognostic significance. The extent of intraocular hemorrhage, presence of macular retinoschisis lesions, and presence of pupillary abnormalities has been correlated with a fatal outcome and permanent neurologic impairment.[28,31-36] The correlation between the severity of the ocular injury and the neurologic outcome suggests a relationship between the brain and ocular injuries in inflicted childhood neurotrauma.

Differential Diagnosis of Retinal Hemorrhages in Infancy

There are many systemic and ocular conditions that may be associated with retinal hemorrhages, although the absence of supportive findings on ocular examination, physical examination, history, or laboratory evaluation make their consideration equivocal. Retinal hemorrhages are known to be rare in children with the conditions described below. When they do occur, they are few in number, are confined to the posterior pole, or have other recognizable unique features. Again, many of these entities are readily excluded from the differential diagnosis on the basis of history or physical examination.

Idiopathic retinal hemorrhages of newborns, related to obstetric and perinatal hemodynamic

changes, are frequent. Retinal hemorrhages secondary to a normal birth have been extensively studied both retrospectively and prospectively in tens of thousands of infants. From these data, it can be concluded that superficial retinal hemorrhages resolve by 1 week postpartum and deeper retinal hemorrhages resolve by 6 weeks. However, these typically small hemorrhages with relatively few nerve fiber layers are present only during the first 2 to 3 weeks of life, and are distinguished by their exclusively posterior location and small size and number.[37,38]

Retinal hemorrhages have also been reported in association with severe accidental injury. Because many patients with nonaccidental injury present with a history of minor trauma, the threshold for retinal hemorrhage in accidental head trauma is important in consideration of the differential diagnosis. Multiple clinical and postmortem studies of eyes of patients with severe head injury suggest that the rate of retinal hemorrhage is less than 3% of instances.[39-42] When retinal hemorrhages do occur, they are confined to the posterior pole, few in number, and rarely subretinal. The types of accidental trauma that result in retinal hemorrhages are usually severe, life-threatening injuries. Even with severe head and brain injuries sufficient for hospitalization, retinal hemorrhage is quite uncommon.[36,39-42]

Many infants with severe abusive head injury have cardiopulmonary resuscitation including chest compressions and artificial ventilation. Retinal hemorrhages have been seen after prolonged cardiopulmonary resuscitation, but never as numerously or extensively as in inflicted childhood neurotrauma.[43-46] It can be concluded from case reports and prospective studies that retinal hemorrhages occur only rarely from cardiopulmonary resuscitation, and when they do, they are few in number and confined to the posterior pole.

Purtscher's retinopathy may occur following acute compression injuries to the thorax or head with characteristic manifestations including cotton wool spots, retinal hemorrhages, and retinal edema most commonly surrounding the optic disc. Purtscher's retinopathy, which is probably caused by complement-mediated leukoembolization, is uncommon in inflicted childhood neurotrauma, and the retinal hemorrhages in inflicted childhood neurotrauma do not appear to be correlated to the presence or absence of rib fractures, a sign of severe chest compression.[47]

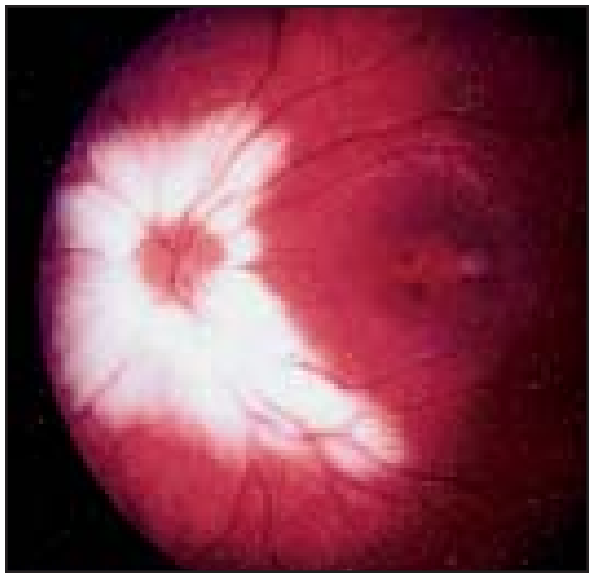

Figure 8. Wide-angle funduscopic photograph showing an ocular fundus. Close inspection of the macula shows a macular hole in an infant with previous inflicted childhood neurotrauma. Myelinated optic nerve fibers are also present.

Terson's syndrome (ie, retinal hemorrhages associated with subarachnoid hemorrhage) is well recognized in adults, although it appears to be uncommon in children. The lack of correlation between the side of involvement of the subarachnoid hemorrhage and ocular hemorrhage suggests that this is not a sufficient explanation for the retinal hemorrhages seen in inflicted childhood neurotrauma. Retinal hemorrhage has been found to be uncommon in children with intracranial hemorrhage from causes other than inflicted childhood neurotrauma, and the retinal hemorrhages are not in a pattern or quantity consistently found in inflicted childhood neurotrauma.[48,49]

There is no evidence to support a link between immunizations and retinal hemorrhages in children.[50,51]

Coagulopathies and other bleeding disorders, including thrombocytopenia, anemia, leukemia, factor deficiencies, and vitamin K deficiency, as well as metabolic diseases such as glutaric acidemia must be considered in the differential diagnosis of intraocular hemorrhage in infants. In general, retinal hemorrhages related to hematologic abnormalities are less numerous and less extensive and do not extend peripherally in the retina. However, infants with bleeding disorders such as a vitamin K defi-

ciency have been incorrectly diagnosed as victims of abuse. Retinoschisis lesions have not been reported in these conditions. A basic hematologic evaluation including complete blood cell count and coagulation studies should be performed in all suspected cases of inflicted childhood neurotrauma.[52] Other ocular syndromes associated with retinal hemorrhage in childhood, including Norrie's disease, Coats' disease, persistent hyperplastic primary vitreous, hypotony retinopathy, cytomegalovirus retinitis, toxoplasmosis, and retinopathy of prematurity, are usually easily distinguished from nonaccidental head injury by the distinctive clinical appearance as well as the clinical setting.

Pathophysiology of Retinal Hemorrhages in Inflicted Childhood Neurotrauma

Many theories regarding the cause of retinal hemorrhages in inflicted childhood neurotrauma continue to be debated in the literature. One theory postulates that venous obstruction in the retina occurring from increased intracranial pressure due to cerebral edema and subdural hemorrhage is the source of retinal hemorrhages. Sudden increases in chest or head pressure may be contributing factors as well. Another theory postulates that traction of the vitreous on the retina during the acceleration and deceleration of shaking and impact causes circular retinal folds and hemorrhagic retinoschisis cavities, as well as smaller hemorrhages. Subdural hemorrhages in inflicted childhood neurotrauma are thought to be caused by the shearing of small vessels from inertial injury, most likely due to rapid acceleration or deceleration movements.

The correlation between the severity of intraocular and intracranial injury and histopathologic evidence suggests that similar inertial trauma may lead to shearing within the retina and at areas of the retina–vitreous attachment, leading to the funduscopic lesions seen. The role of vitreous shaking in the generation of macular retinoschisis and perimacular folds has strong support in theory and in autopsy findings. The high frequency of hemorrhages at the vitreous base supports a theoretical link to vitreous traction. Orbital shaking injury, including disruption of the autonomic supply to the retinal vessels, may play a role.

The literature suggests that it is the shaking itself, with resultant shearing injury, that is the primary factor in the generation of retinal hemorrhages seen in inflicted childhood neurotrauma. The optic atrophy often seen in survivors is best explained by direct optic nerve injury within the orbit. The role of vitreous traction and tissue shearing must be further explored. Postmortem orbital findings suggest a role for autonomic dysregulation and direct vessel damage that is yet to be explored. The compounding effects of anoxia or hypoxia, anemia, thrombocytopenia, mild coagulopathy, obstruction of retinal venous flow, or possible age-related anatomic variations in the retinal vasculature are not well understood. The adjunctive role of increased intracranial pressure needs further exploration. Although the role of vitamin C deficiency has been suggested exclusively in lay and legal literature, it has not been formally explored, and vitamin C deficiency is currently rare. The minimal forces required to generate retinal hemorrhages or, more specifically, the reason why shaking seems to be unique in the generation of severe retinal hemorrhages is not known.

Controversies in the Diagnosis and Management of Inflicted Childhood Neurotrauma

The most difficult and controversial aspect of the diagnosis of inflicted childhood neurotrauma is the reliability of the designation. Physicians are rarely in a position to make a diagnosis with such profound significance to patients and their families. Ascertainment of child abuse is critical to prevent a potentially fatal recurrence in victims. Extensive intraocular hemorrhage in young infants in the setting of acute brain injury and in the absence of a history of severe accidental trauma or underlying medical cause must be considered to be nonaccidental injury until proven otherwise.

The management of acute intraocular hemorrhages is primarily supportive. Gradual resolution is generally seen in 2 to 6 weeks, although dense preretinal and vitreous hemorrhage may persist much longer. With prolonged vitreous clouding, young children do have a risk of deprivational amblyopia. In rare cases of prolonged vitreous opacity, surgical vitrectomy may be necessary to allow normal visual development. Survivors of inflicted childhood neurotrauma must be reexamined for amblyopia,

refractive errors, and other late complications that require treatment.

Role of the Ophthalmologist in the Diagnosis and Management of Inflicted Childhood Neurotrauma

Inflicted childhood neurotrauma is a clinical pattern of nonaccidental injuries including intracranial and intraocular hemorrhage occurring in infants and toddlers younger than 3 years. Examination of the eyes through undilated pupils with a direct ophthalmoscope is inadequate for a complete evaluation of the ocular findings in inflicted childhood neurotrauma. The primary role of the ophthalmologist in the care of these young children is to provide a complete evaluation of the intraocular hemorrhages. Ophthalmic consultation allows complete assessment and documentation of the ocular findings frequently with retinal photography, an essential component of the diagnosis of inflicted childhood neurotrauma. In addition to establishing the diagnosis, examination provides prognostic information related to the ocular findings. Ophthalmologists are able to coordinate the long-term management of frequent visual complications. In the case of a fatal outcome, postmortem examination must include both gross and histopathologic evaluation of the eyes and optic nerves. Finally, physicians who treat infants and children are mandated to report suspected child abuse to child welfare agencies for investigation, and ophthalmologists who encounter children with ophthalmic manifestations of abuse need to ensure that the proper steps are taken to protect their patients from further harm. Ophthalmologists are in a crucial position to detect signs of child abuse. Abused children may have no other advocate.

References

1. Brenner RA, Overpeck MD, Trumble AC, DerSimonian R, Berendes H. Deaths attributable to injuries in infants, United States, 1983-1991. *Pediatrics* 1999;103:968-974.
2. Overpeck RA, Brenner AC, Trumble LB, Trifilette LB, Berendes HW. Risk factors for infant homicide in the US. *N Engl J Med* 1998;339:1211-1216.
3. 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.
4. Duhaime A, Gennarelli TA, Thibault LE, et al. The shaken baby syndrome: a clinical, pathological, and biomechanical study. *J Neurosurg* 1987;66:409-415.
5. Lazoritz S, Baldwin S, Kini N. The whiplash shaken infant syndrome: has Caffey's syndrome changed or have we changed his syndrome? *Child Abuse Negl* 1997;21:1009-1014.
6. Merten DF, Osborne DRS, Radkowski MA, et al. Craniocerebral trauma in the child abuse syndrome: radiological observations. *Pediatr Radiol* 1984;14:272-277.
7. Levin A. Ocular manifestations of child abuse. *Ophthalmol Clin North Am* 1990;3:249-264.
8. Harcourt B, Hopkins D. Ophthalmic manifestations of the battered-baby syndrome. *Br Med J* 1971;3:398-401.
9. Ober RR. Hemorrhagic retinopathy in infancy: a clinicopathologic report. *J Pediatr Ophthalmol Strabismus* 1980;17:5-13.
10. Rao N, Smith RE, Choi JH, et al. Autopsy findings in the eyes of fourteen fatally abused children. *Forensic Sci Int* 1988;39:293-299.
11. 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;80:282-287.
12. Altman RL, Kutscher MO, Brand DA. The "shaken-baby syndrome." *N Engl J Med* 1998;339:1329-1330.
13. Duhaime AC, Christian CW, Rorke LB, Zimmerman RA. Nonaccidental head injury in infants: the "shaken baby syndrome." *N Engl J Med* 1998;338:1822-1829.
14. Kivlin JD, Simons KB, Laxoritz A, Ruttum MS. Shaken baby syndrome. *Ophthalmology* 2000;107:1246-1254.
15. Rivera F. Population-based study of fall injuries in children and adolescents resulting in hospitalization or death. *Pediatrics* 1993;92:61-63.
16. Jenny C, Hymel KP, Ritzen A, et al. Analysis of missed cases of abusive head trauma. *JAMA* 1999;281:621-626.
17. DiScala C, Sege R, Li G, Reece RM. Child abuse and unintentional injuries: a ten-year retrospective. *Arch Pediatr Adolesc Med* 2000;154:16-22.
18. Lane WG, Rubin DM, Monteith R, Christian CW. Racial differences in the evaluation of pediatric fractures for physical abuse. *JAMA* 2002;288:1603-1609.
19. Altman RL, Kutscher ML, Brand DA. The "shaken baby syndrome." *N Engl J Med* 1988;339:1329-1330.
20. Pitetti RD, Maffei F, Chang K, Hickey R, Berger R, Pierce MC. Prevalence of retinal hemorrhages and child abuse in children who present with an apparent life-threatening event. *Pediatrics* 2002;110:557-562.
21. U.S. Department of Health and Human Services, Administration on Children, Youth and Families. *Child Maltreatment 2001*. Washington, DC: U.S. Government Printing Office; 2003.
22. Starling SP, Holden JR, Jenny C. Abusive head trauma: the relationship of perpetrators to their victims. *Pediatrics* 1995;95:259-262.
23. Gleckman AM, Evans RJ, Bell MD, Smith TW. Optic nerve damage in shaken baby syndrome: detection by beta-amyloid precursor protein immunohistochemistry. *Arch Pathol Lab Med* 2000;124:251-256.
24. Budenz DL, Farber MG, Mirchandani HG, Park H, Rorke LB. Ocular and optic nerve hemorrhages in abused infants with intracranial injuries. *Ophthalmology* 1994;101:559-565.
25. Greenwald MJ, Weiss A, Oesterle CS, Friendly DS. Traumatic retinoschisis in battered babies. *Ophthalmology* 1986;93:618-625.
26. Gaynon M, Koh K, Marmor M, Frankel LR. Retinal folds in the shaken baby syndrome. *Am J Ophthalmol* 1988;106:423-425.
27. Keithahn MAZ, Bennett SR, Cameron D, Mieler WF. Retinal folds in Terson syndrome. *Ophthalmology* 1993;100:1187-1190.
28. Mills MD. Association of funduscopic lesions with fatal outcome in Shaken Baby syndrome. *J AAPOS* 1998;2:67-71.
29. Fishman G, Dasher W, Lambert S. Electroretinographic findings in infants with the shaken baby syndrome. *J Pediatr Ophthalmol Strabismus* 1998;35:22-26.
30. Han DP, Wilkinson WS. Late ophthalmic manifestations of the shaken baby syndrome. *J Pediatr Ophthalmol Strabismus* 1990;27:300-302.
31. Williams DF, Swengel RM, Scharre DW. Posterior segment manifestations of ocular trauma. *Retina* 1995;10:535.
32. Haviland J, Russell RI. Outcomes after severe non-accidental

head injury. *Arch Dis Child* 1997;77:504-507.
33. Matthews GP, Das A. Dense vitreous hemorrhages predict poor visual and neurological prognosis in infants with shaken baby syndrome. *J Pediatr Ophthalmol Strabismus* 1996;33:260-265.
34. McCabe CF, Donahue SP. Prognostic indicators for vision and mortality in shaken baby syndrome. *Arch Ophthalmol* 2000;118:373-377.
35. Wilkenson WS, Han DP, Rappley MD, Owings CL. Retinal hemorrhage predicts neurologic injury in the shaken baby syndrome. *Arch Ophthalmol* 1989;107:1472-1474.
36. Duhaime AC, Alario AJ, Lewander WJ, Schut L, et al. Head injury in very young children: mechanisms, injury types and ophthalmic findings in 100 hospitalized patients younger than 2 years of age. *Pediatrics* 1992;90:179-185.
37. Kaur B, Taylor D. Fundus hemorrhages in infancy. *Surv Ophthalmol* 1992;37:1-17.
38. Emerson MV, Pieramici DJ, Stoessel KM, Berreen JP, Gariano RF. Incidence and rate of disappearance of retinal hemorrhage in newborns. *Ophthalmology* 2001;108:36-39.
39. Ewing-Cobbs L, Kramer L, Prasad M, et al. Neuroimaging, physical, and developmental findings after inflicted and noninflicted traumatic brain injury in young children. *Pediatrics* 1998;102:300-307.
40. Feldman KW, Bethel R, Shugerman RP, et al. The cause of infant and toddler subdural hemorrhage: a prospective study. *Pediatrics* 2001;108:636-646.
41. Buys YM, Levin AV, Enzenauer RW, Elder JE, et al. Retinal findings after head trauma in infants and young children. *Ophthalmology* 1992;99:1718-1723.
42. Christian CW, Taylor AA, Hertle RW, Duhaime AC. Retinal hemorrhages caused by accidental household trauma. *J Pediatr* 1999;135:125-127.
43. Goetting MG, Sowa B. Retinal hemorrhage after cardiopulmonary resuscitation in children: an etiologic reevaluation. *Pediatrics* 1990;85:585-588.
44. Kramer K, Goldstein B. Retinal hemorrhages following cardiopulmonary resuscitation. *Clin Pediatr* 1993;32:366-368.
45. Gilliland MGF, Luckenbach MW. Are retinal hemorrhages found after resuscitation attempts? A study of the eyes of 169 children. *Am J Forensic Med Pathol* 1993;14:187-192.
46. Odom A, Christ E, Kerr N, et al. Prevalence of retinal hemorrhages in pediatric patients after in-hospital cardiopulmonary resuscitation: a prospective study. *Pediatrics* 1997;99:E3.
47. Duane T, Osher R, Green W. White centered hemorrhages: their significance. *Ophthalmology* 1980;87:66-69.
48. Mills M. Terson Syndrome. *Ophthalmology* 1998;105:2161-2162.
49. Schloff S, Mullaney MD, Armstrong DC, et al. Retinal findings in children with intracranial hemorrhage. *Ophthalmology* 2002;109:1472-1475.
50. Scheibner V. Shaken baby syndrome: the vaccination link. *Nexus* 1998;87:35-38.
51. Friedlander E. Opposition to immunization: a pattern of deception. *Scientific Reviews of Alternative Medicine* 2001;5:18-23.
52. Gayle MO, Kisson N, Gerd RW, et al. Retinal hemorrhage in the young child: a review of etiology, predisposed conditions, and clinical implications. *J Emerg Med* 1995;13:233-239.