Ocular and Optic Nerve Hemorrhages in Abused Infants with Intracranial Injuries

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Purpose: To ascertain whether the pathology of the eye and optic nerve might be useful in determining the manner of death in infants who died after sustaining apparent nonaccidental intracranial injury.

Methods: Complete autopsies were performed on 13 infants who died of acute intracranial injuries after nonaccidental trauma. The infants were divided into two groups: nine with physical evidence of blunt trauma to the head, and four without detectable scalp or skull injury whose intracranial injuries were attributed to violent shaking (shaken baby syndrome). Six infants with no intracranial injuries who died suddenly from unidentifiable causes (the so-called sudden infant death syndrome) served as controls.

Results: Optic nerve sheath hemorrhage, most prominent in the subdural space, was present in all 13 infants with nonaccidental intracranial injury. Multilayered retinal hemorrhage was present in at least one eye of 11 of these 13 infants. The location and quantity of the intraocular and optic nerve sheath hemorrhages did not differ in those with external head trauma and those without scalp or skull lesions. A single control infant had a few erythrocytes in the nerve fiber layer of the anterior optic nerve of one eye. The remainder of the control infants did not have evidence of hemorrhage in the eye or optic nerve sheath.

Conclusion: Histopathologic analysis of optic nerve sheath and intraocular hemorrhages may be helpful in distinguishing traumatic from non-traumatic causes of death in infants. *Ophthalmology 1994;101:559–565*

Reported fatalities due to child abuse have increased 49% between 1985 and 1992.[1] Since Gilkes and Mann[2] first described retinal hemorrhages in abused children, the clinical association between retinal hemorrhages and child abuse has been well documented.[3–13] This finding becomes particularly important when there is an absence of external signs of trauma, such as in the so-called shaken baby syndrome. In the absence of external signs of trauma, the mechanism of injury is thought to be violent shaking without forceful impact to the head. When there is evidence of blunt trauma, it is difficult to determine whether the intracranial injury resulted from blunt trauma alone or from the trauma plus shaking. The history given by the caretaker is usually unreliable.

Several histopathologic reports have studied the ocular, optic nerve, and intracranial findings in death due to child abuse.[14–21] Three of these reports,[18–20] all from the same series of cases, include control populations for comparison. The current study examines the intracranial, ocular, and intraorbital optic nerve pathology in three groups of patients: child abuse with direct head trauma, child abuse without evidence of direct head trauma (shaken baby syndrome), and a control group of children who died suddenly and unexpectedly of the so-called sudden infant death syndrome (SIDS).

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Methods

From October 1, 1988, to August 30, 1990, a total of 13 autopsies were performed at the Philadelphia Medical Examiner's Office on children younger than 3 years of age whose fatal intracranial injuries were attributed to child abuse. Two groups of patients were identified: infants with fatal intracranial injuries resulting from blunt head trauma, and infants with no evidence of direct head trauma found at autopsy who died of presumed violent shaking injuries (shaken baby syndrome). The distinction between infants with blunt trauma versus pure shaking injury was based on the absence of any scalp injuries or skull fractures in the latter group. As controls, infants were studied who died suddenly and unexpectedly of unidentifiable cause and were placed in the category of the so-called SIDS. The diagnosis of SIDS was one of exclusion based on the absence of external trauma or intracranial hemorrhage or edema.

Standard autopsy procedure included elevation of the floor of the anterior fossa, including the orbital roof, to expose the eye and intraorbital portion of the optic nerve. The optic nerve was transected at the level of the optic canal. The globe and attached optic nerve were removed and fixed in 10% formalin for at least 7 days. After macroscopic examination of the fixed specimen, the optic nerve was cross-sectioned at 5, 8, 11, and 14 mm posterior to its insertion into the globe. The eye and distal 5 mm of the optic nerve were sectioned in the axial plane and processed for paraffin embedding. Multiple representative 5-μm–thick sections were then mounted on slides and stained with hematoxylin–eosin, periodic acid-Schiff, and Perl's iron stains.

Histopathologic examination of the stained slides was performed by two separate examiners (DLB, MGF) without prior knowledge of the cause of death. The amount of hemorrhage within a particular tissue (e.g., intraretinal) or space (e.g., subdural) was quantified according to the following scale: no erythrocytes = 0; a few erythrocytes = 1+; a moderate number of erythrocytes in a tissue or space, but not completely saturated = 2+; and tissue or space completely saturated with erythrocytes = 3+. The location of the hemorrhage in the optic nerve was appraised in the four segments as previously described.

The postmortem examinations of the scalp and cranium included thorough examination for abrasions, lacerations, and contusions after shaving. Galea, periosteum, and bone were examined for evidence of injury. Dura was stripped to examine the entire inner table of the cranium. Intracranial hemorrhage was localized grossly and quantified in milliliters. Detailed gross and microscopic examinations were done on the central nervous system (LBR).

Table 1. Clinical Characteristics

	Blunt Trauma	Shaken Baby Syndrome	Sudden Infant Death Syndrome
No.	9	4	6
Age (mos)			
Mean	16.2	4.3	2.8
Range	2–36	2–9	1–5
Sex			
M	7	3	3
F	2	1	3
Race			
White	3	4	2
African-American	4	0	3
Asian	2	0	1

Table 2. Intracranial Findings

	Blunt Trauma	Shaken Baby Syndrome
	No. (%)	*No. (%)*
Cerebral edema	9 (100)	4 (100)
Subdural hemorrhage	8 (89)	4 (100)
Subarachnoid hemorrhage	6 (67)	1 (25)
Parenchymal hemorrhage	5 (55)	3 (75)

Results

Nine of the 13 abused infants had signs of blunt head trauma and are called the "blunt trauma" group. Four of the abused infants had no signs of blunt head trauma and are regarded as victims of the "shaken baby syndrome." Six infants died suddenly and unexpectedly of unknown cause and were placed in the category of the SIDS. Clinical characteristics of the infants are summarized in Table 1.

The time interval between presentation to the hospital and death was shorter in the shaken baby syndrome group (average, 2.3 days; range, 1–5 days) than in the blunt trauma group (average, 5.4 days; range, 0–15 days). The interval in the infants with SIDS was shortest (average, <1 day; 4 of 6 were dead on arrival). Cardiopulmonary resuscitation was performed in six of nine infants with intentional blunt trauma, one of four with shaken baby syndrome, and four of six with SIDS.

All infants in the blunt trauma group had scalp contusions but only two had skull fractures. Contusions of the trunk were noted in five of nine infants with blunt trauma, and three of nine had rib fractures. Four infants in this group had contusions of the extremities but none had long bone fractures. Three of the nine infants with blunt trauma sustained recent burns. None of the infants in the shaken baby syndrome or SIDS groups had evidence of external head, torso, or extremity injury.

The intracranial findings in the abused infants are summarized in Table 2. All had cerebral edema; intracranial subarachnoid hemorrhage was more common in the blunt trauma than in the shaken baby syndrome

group. None of the six control infants had evidence of intracranial edema or hemorrhage.

Macroscopically, optic nerves with hemorrhage typically appeared blue on the outer surface and dark red within the nerve sheath, either focally or diffusely (Fig 1). The hemorrhages involved only the intraorbital portion of the nerve and could only be seen after elevation of the floor of the anterior cranial fossa (orbital roof). Microscopic abnormalities of the optic nerve are summarized in Table 3. Severe (3+) subdural optic nerve hemorrhage was common in all of the abused children, regardless of mechanism of injury. Subarachnoid, intra-arachnoid, and intradural hemorrhages also were found in the abused children but were not as prominent (1–2+). Optic nerve sheath hemorrhage was more prominent in the anterior portion of the nerve. No hemorrhages were noted in the optic nerve parenchyma. Figure 2 shows the typical appearance of the optic nerve sheath hemorrhages after child abuse. No correlation could be made between the location or amount of hemorrhage in the optic nerve sheath versus hemorrhage in the intracranial meninges. None of the control infants contained evidence of optic nerve sheath hemorrhage, despite the observation that four of the six infants had undergone cardiopulmonary resuscitation. One eye of one patient in the SIDS group contained a few erythrocytes beneath the nerve fiber layer of the anterior optic nerve. This infant had received cardiopulmonary resuscitation.

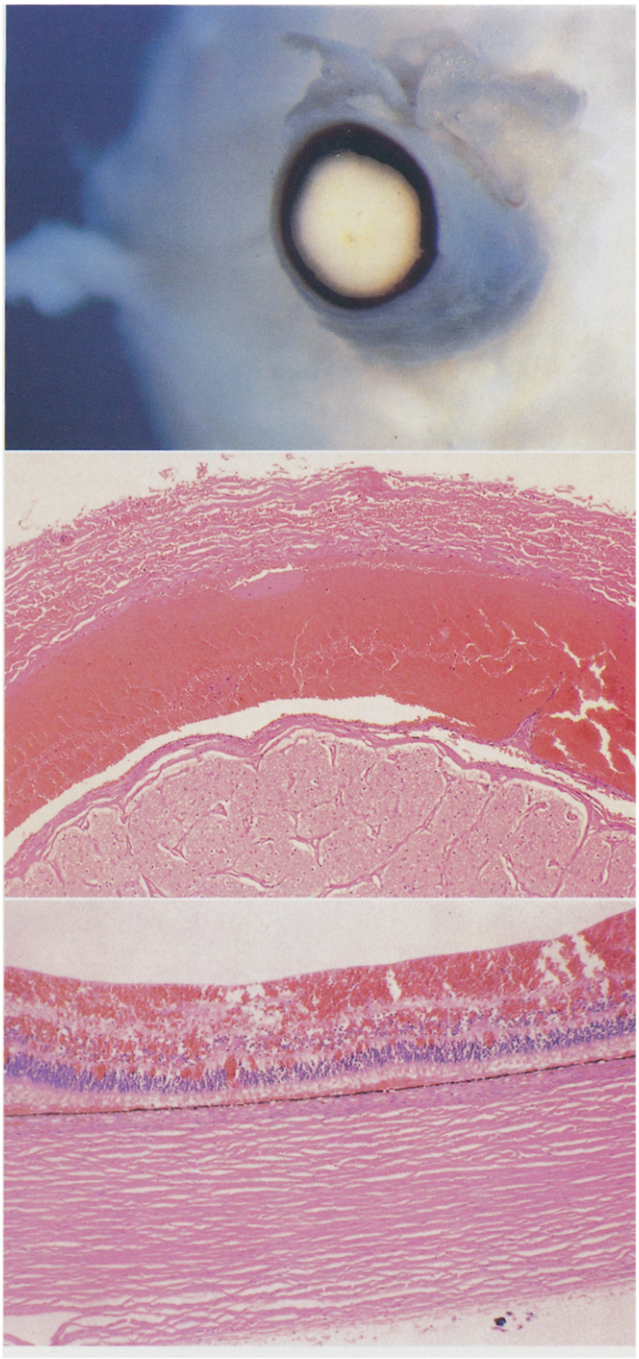
Table 4 summarizes the intraocular hemorrhage findings in the two abuse groups. Multilayered intraretinal hemorrhages were found in 11 of 13 victims of child abuse; none were found in the infants with SIDS. One infant from each group failed to show gross or microscopic evidence of retinal hemorrhage, although both showed bilateral optic nerve sheath hemorrhage. Unilateral retinal hemorrhage was found in three infants in the blunt trauma group. Hemorrhages were most common in the nerve fiber, inner plexiform, and inner nuclear layers. Vitreous, preretinal and subretinal hemorrhages were found in a large proportion of the abused infants, regardless of the mechanism of injury as outlined in Table 4. Figure 3 shows the typical appearance of the multilayered retinal hemorrhages in the victims of child abuse.

Other histopathologic ocular findings included optic disc swelling in 7 of 13 abused infants and hemorrhage within the anterior chamber trabecular meshwork in a single eye of one victim of the shaken baby syndrome. No other ocular abnormalities were found.

Perl's iron stain showed iron-laden macrophages in 4 of the 13 abused children. The average time of survival in infants positive for iron was 5.5 days compared with 4.0 days in those negative for iron.

Discussion

Abused infants with intracranial injuries usually present with abnormal central nervous system signs,[9,12,22,23] intracranial hemorrhage,[9,12,24–26] and intraocular hemorrhage.[9–12,26] However, obvious signs of old or recent child



Top, Figure 1. Gross pathologic appearance of the left optic nerve of a 7-month-old infant whose cause of death was attributed to nonaccidental head trauma. Notice characteristic blue discoloration to the outer surface of the nerve secondary to optic nerve sheath hemorrhage.

Center, Figure 2. Histopathologic appearance of the left optic nerve sheath of a 3-month-old infant whose death was attributed to shaking alone. Extensive hemorrhage is present in the subdural space. Minimal hemorrhage can be seen in the dura and within the subarachnoid space (hematoxylin–eosin; original magnification, ×100).

Bottom, Figure 3. Multilayered intraretinal hemorrhage, more prominent in the inner retinal layers, is present in the left eye of this 2-month-old infant whose death was attributed to shaking alone (hematoxylin–eosin; original magnification, ×100).

Table 3. Optic Nerve Hemorrhage

	Blunt Trauma			Shaken Baby Syndrome		
	No. of Patients	*No. of Eyes*	*Amount**	*No. of Patients*	*No. of Eyes*	*Amount**
Subarachnoid	5 (55%)	8	1+	4 (100%)	8	1+
Intra-arachnoid	4 (44%)	7	1+	4 (100%)	7	1+
Subdural	9 (100%)	17	3+	4 (100%)	8	3+
Intradural	9 (100%)	15	2+	4 (100%)	8	2+

* 1+ = few erythrocytes within a space or covering tissue; 2+ = moderate erythrocytes within a space or covering tissue; 3+ = erythrocytes fill space or cover tissue.

abuse, such as skull fractures, scalp contusions, or long bone fractures, may not be evident. Several studies have suggested that the incidence of child abuse is underestimated because of inadequate investigation of childhood fatalities[26] as well as inadequate reporting to state agencies.[27] Therefore, forensic and ophthalmic pathologists must be familiar with the gross and histopathologic findings of abused children as well as other entities that can cause the constellation of clinical findings described above.

Kiffney[14] first reported the pathology of the eye in physical child abuse. He demonstrated retinal detachment but did not find intraocular or optic nerve hemorrhages. Mushin and Morgan[15] reported the histopathology of two somewhat atypical cases of child abuse (1 eye of one patient with a hyphema from direct globe trauma and 1 eye of an infant who was strangled) and described intraocular, but not optic nerve sheath, hemorrhages. Ober[16] observed vitreous, intraretinal, and subretinal hemorrhages in an abused child but found no optic nerve sheath hemorrhages at autopsy. Lambert et al[17] were the first to report optic nerve sheath hemorrhages in association with the shaken baby syndrome. Their patient had a greater amount of blood in the subarachnoid space than the subdural space of the optic nerve. Several reports[18–20] have documented a high proportion of optic nerve sheath hemorrhage although these do not contain an analysis of the location of the optic nerve sheath hemorrhage beyond calling them "submeningeal" or "subdural." Elner et al[21] studied ten fatalities due to child abuse, all with evidence of blunt trauma, and reported hemorrhage in multiple layers of the optic nerve sheath but did not comment on the amount of hemorrhage present in each layer. Our series documents a preponderance of hemorrhage in the subdural space with less hemorrhage in the subarachnoid and intrameningeal tissues of the optic nerve. This was true in every abused child, regardless of the mechanism of injury (e.g., blunt trauma versus shaken baby syndrome).

Four of the 13 fatalities in the current series showed no clinical or pathologic evidence of blunt head trauma. The constellation of clinical, intracranial, optic nerve, and intraocular findings described previously[9–12,22,24,25] led to the diagnosis of death by shaking, the so-called shaken baby syndrome. Duhaime et al[13] reviewed the pathologic findings in 13 child abuse fatalities and were able to identify autopsy evidence of blunt trauma in all infants. In the same article, the authors reported an experimental model for head injury in infants and concluded that shaking injury alone is unlikely to cause severe intracranial damage in infants. However, our observations, as well as those of others,[17–20,28] suggest that pure shaking injury may indeed lead to fatal intracranial injury. On the other hand, we support the conclusion of Duhaime et al that severe cerebral edema is a common finding in fatalities due to child abuse involving head injury and may be of major significance in causing death in these infants.

Other possible causes of retinal hemorrhages in infants include birth trauma, cardiopulmonary resuscitation, accidental head trauma, hematologic disorders, and rup-

Table 4. Intraocular Hemorrhage

	Blunt Trauma			Shaken Baby Syndrome		
	No. of Patients	*No. of Eyes*	*Amount**	*No. of Patients*	*No. of Eyes*	*Amount**
Vitreous	4 (44%)	4	1+	3 (75%)	6	2+
Preretinal	4 (44%)	5	2+	3 (75%)	6	2+
Intraretinal	8 (89%)	11	1+	3 (75%)	6	2+
Subretinal	5 (56%)	6	1+	3 (75%)	6	1+

* 1+ = few erythrocytes within a space or covering tissue; 2+ = moderate erythrocytes within a space or covering tissue; 3+ = erythrocytes fill space or cover tissue.

tured cerebral aneurysm or vascular malformations. Therefore, the diagnosis of child abuse is one of exclusion based on careful investigation for other causes of retinal hemorrhages in this age group, particularly when there is no evidence of blunt trauma.

Birth trauma is a well-recognized cause of retinal hemorrhage in newborn infants. These may occur in 2.6%[29] to 40%[30] of newborns, depending on the time after birth that patients are examined. Retinal hemorrhages are most common after vaginal rather than Caesarean delivery[29,31] and may persist from 24 hours to up to 6 weeks after delivery.[29] Optic nerve sheath hemorrhages have not been reported in newborns, although intraventricular hemorrhage in premature infants with accompanying increased intracranial pressure theoretically could cause such hemorrhages.

Results of clinical studies vary regarding the frequency of retinal hemorrhages after cardiopulmonary resuscitation (CPR) is administered. Kanter[32] found no examples of retinal hemorrhages in children after CPR was administered unless some other reason for hemorrhages existed, such as head trauma or severe systemic hypertension. However, Goetting and Sowa[33] reported 2 of 20 children (ages, 2 weeks to 17 years) who underwent CPR and had retinal hemorrhages in the absence of other trauma or other known cause of hemorrhage. Our study showed microscopic evidence of optic disc hemorrhage in one eye of a child who received CPR and who was placed in the category of SIDS. This amount of hemorrhage would not be detectable clinically. Larger clinical studies are needed to determine the prevalence of retinal hemorrhage after CPR in this age group. Optic nerve sheath hemorrhage has not been reported after CPR alone.

Accidental head trauma in infants most commonly occurs after falls.[34–38] These infants present in a manner similar to child abuse victims, with neurologic signs secondary to intracranial edema and hemorrhage[34–39] and retinal hemorrhages.[34,37–39] Severe intracranial injury[35–37] and retinal hemorrhages[37] are more likely to occur after significant and easily identifiable accidents, such as motor vehicle accidents. In a recent comparison of 75 infants with accidental head injury to 3 infants with nonaccidental injury, Buys et al[38] found retinal hemorrhages in all of the nonaccidental head trauma patients but in none of the infants with accidental head trauma. In contrast to this is an isolated report from Japan[34] in which the authors claim that 26 infants sustained acute subdural hematomas secondary to minor falls; all had retinal and preretinal hemorrhages. In reviewing necropsy findings of infants who died accidentally during the study period, we identified one 8-month-old whose carriage was struck by a truck. This infant sustained severe intracranial, optic nerve sheath, and intraocular hemorrhages identical to those seen in the abused infants.

Hematologic disorders can cause retinal hemorrhages secondary to anemia,[40–42] thrombocytopenia,[40,42] and hyperviscosity.[42,43] In children, acute leukemia is the most common hematologic cause of retinal hemorrhages. While more common in adults with leukemias than in children,[44] the finding of retinal hemorrhages warrants investigation of hematologic status. Thrombocytopenia and, to a lesser extent, severe anemia appear to be associated with retinal hemorrhages in this group of patients.[44] Two large histopathologic series[45,46] have demonstrated infiltration of the optic nerve by leukemic cells but not erythrocytes.

Ruptured vascular malformation or cerebral aneurysm can present with a similar complex of clinical and pathologic findings as the shaken baby syndrome, namely central nervous system symptoms with intracranial and intraocular hemorrhage in the absence of signs of external head trauma.[47–50] Although the prevalence of ruptured cerebral aneurysms and ruptured vascular malformations in the infant age group is extremely low,[47,48] these entities should be excluded before making the diagnosis of shaken baby syndrome because they may be confused clinically. Although not described in infants, the histopathologic appearance of the intraocular and optic nerve sheath findings in adults with increased intracranial pressure due to ruptured cerebral aneurysm[51,52] is similar to that found in the shaken baby syndrome. We have observed optic nerve sheath hemorrhages in a 7-week-old infant who died after rupture of a vascular malformation of the frontal lobe. The optic nerve sheath hemorrhage was unilateral and confined to the subarachnoid space. No subdural or retinal hemorrhages were found.

The significance of positive iron stain in the eyes of abused children has been the subject of recent debate. Gilliland et al[53] have pointed out that the presence of hemosiderin in the eyes of abused children must be interpreted with caution because hemosiderin-laden macrophages may appear within 48 to 72 hours after injury. Therefore, the interval between injury and death must be taken into account rather than simply attributing the hemosiderin to prior physical abuse. Lambert and colleagues[17] found no iron in the eyes of an infant who had been shaken 5 days before her death. Rao et al[18] demonstrated hemosiderin in 1 of 14 abused children. The interval between injury and death was not recorded as part of their investigation. Elner et al[21] found hemosiderin deposits in three of their abused infants; two of these three infants survived less than 3 days. Only one of Riffenburgh and Sathyavagiswaran's patients[19,20] had a positive hemosiderin stain. We found iron-laden macrophages in 4 of 13 infants, with a slightly greater survival time in patients whose eyes tested positive for iron. It is critical to consider the time interval between injury and death when interpreting the results of iron stains in pathologic specimens because this is not necessarily indicative of repeated trauma.

The diagnosis of shaken baby syndrome remains one of exclusion in infants whose cause of death is suspicious. Even in the absence of retinal hemorrhages, one may demonstrate intracranial and optic nerve sheath abnormalities, in which case histopathologic evaluation of the optic nerve and globe is warranted.

References

1. McCurdy K, Daro D. Current Trends in Child Abuse Reporting and Fatalities: The Results of the 1992 Annual Fifty

State Survey. The National Committee for Prevention of Child Abuse: Chicago 1993.
2. Gilkes MJ, Mann TP. Fundi of battered babies. Lancet 1967;2:468–9.
3. Friendly DS. Ocular manifestations of physical child abuse. Trans Am Acad Ophthalmol Otolaryngol 1971;75:318–32.
4. Harcourt B, Hopkins D. Ophthalmic manifestations of the battered-baby syndrome. Br Med J 1971;3:398–401.
5. Mushin AS. Ocular damage in the battered-baby syndrome. Br Med J 1971;3:402–4.
6. Jensen AD, Smith RE, Olson MI. Ocular clues to child abuse. J Pediatr Ophthalmol 1971;8:270–2.
7. Eisenbrey AB. Retinal hemorrhage in the battered child. Childs Brain 1979;5:40–4.
8. Harley RD. Ocular manifestations of child abuse. J Pediatr Ophthalmol Strabismus 1980;17:5–13.
9. Greenwald MJ, Weiss A, Oesterle CS, Friendly DS. Traumatic retinoschisis in battered babies. Ophthalmology 1986;93:618–25.
10. Caffey J. On the theory and practice of shaking infants. Its potential residual effects of permanent brain damage and mental retardation. Am J Dis Child 1972;124:161–9.
11. 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.
12. Ludwig S, Warman M. Shaken baby syndrome: a review of 20 cases. Ann Emerg Med 1984;13:104–7.
13. Duhaime AC, Gennarelli TA, Thibault LE, et al. The shaken baby syndrome. A clinical, pathological, and biomechanical study. J Neurosurg 1987;66:409–15.
14. Kiffney GT Jr. The eye of the "battered child." Arch Ophthalmol 1964;72:231–3.
15. Mushin A, Morgan G. Ocular injury in the battered baby syndrome. Report of two cases. Br J Ophthalmol 1971;55: 343–7.
16. Ober RR. Hemorrhagic retinopathy in infancy: a clinicopathologic report. J Pediatr Ophthalmol Strabismus 1980;17:17–20.
17. Lambert SR, Johnson TE, Hoyt CS. Optic nerve sheath and retinal hemorrhages associated with the shaken baby syndrome. Arch Ophthalmol 1986;104:1509–12.
18. Rao N, Smith RE, Choi JH, et al. Autopsy findings in the eyes of fourteen fatally abused children. Forensic Sci Int 1988;39:293–9.
19. Riffenburgh RS, Sathyavagiswaran L. The eyes of child abuse victims: autopsy findings. J Forensic Sci 1991;36: 741–7.
20. Riffenburgh RS, Sathyavagiswaran L. Ocular findings at autopsy of child abuse victims. Ophthalmology 1991;98: 1519–24.
21. Elner SG, Elner VM, Arnall M, Albert DM. Ocular and associated systemic findings in suspected child abuse. A necropsy study. Arch Ophthalmol 1990;108:1094–101.
22. Frank Y, Zimmerman R, Leeds NMD. Neurological manifestations in abused children who have been shaken. Dev Med Child Neurol 1985;27:312–16.
23. Zimmerman RA, Bilaniuk LT, Bruce D, et al. Computed tomography of craniocerebral injury in the abused child. Radiology 1979;130:687–90.
24. Giangiacomo J, Khan JA, Levine C, Thompson VM. Sequential cranial computed tomography in infants with retinal hemorrhages. Ophthalmology 1988;95: 295–9.
25. Wilkinson WS, Han DP, Rappley MD, Owings CL. Retinal hemorrhage predicts neurologic injury in the shaken baby syndrome. Arch Ophthalmol 1989;107:1472–4.
26. Mitchel L. Child Abuse and Neglect Fatalities: A Review of the Problem and Strategies for Reform. National Committee for Prevention of Child Abuse: Chicago 1987.
27. California Office of the Auditor General. Report by the Office of the Auditor General. California's Records on the Incidence of Child Abuse Are Incomplete and Inaccurate. Sacramento, CA. August, 1988.
28. Hadley MN, Sonntag VKH, Rekate HL, Murphy A. The infant whiplash-shake injury syndrome: a clinical and pathological study. Neurosurgery 1989;24:536–40.
29. Sezen F. Retinal haemorrhages in newborn infants. Br J Ophthalmol 1970;55:248–53.
30. Giles CL. Retinal hemorrhages in the newborn. Am J Ophthalmol 1960;49:1005–11.
31. Jain IS, Singh YP, Grupta SL, Gupta A. Ocular hazards during birth. J Pediatr Ophthalmol Strabismus 1980;17:14–16.
32. Kanter RK. Retinal hemorrhage after cardiopulmonary resuscitation or child abuse. J Pediatr 1986;108:430–2.
33. Goetting MG, Sowa B. Retinal hemorrhage after cardiopulmonary resuscitation in children: an etiologic reevaluation. Pediatrics 1990;85:585–8.
34. Aoki N, Masuzawa H. Infantile acute subdural hematoma. Clinical analysis of 26 cases. J Neurosurg 1984;61:273–80.
35. Billmire ME, Myers PA. Serious head injury in infants: accident or abuse? Pediatrics 1985;75:340–2.
36. Rivara FP, Kamitsuka MD, Quan L. Injuries to children younger than 1 year of age. Pediatrics 1988;81:93–7.
37. Duhaime AC, Alario AJ, Lewander WJ, et al. Head injury in very young children: mechanisms, injury types, and ophthalmologic findings in 100 hospitalized patients younger than 2 years of age. Pediatrics 1992;90:179–85.
38. Buys YM, Levin AV, Enzenauer RW, et al. Retinal findings after head trauma in infants and young children. Ophthalmology 1992;99:1718–23.
39. Till K. Subdural haematoma and effusion in infancy. Br Med J 1968;3:400–2.
40. Rubenstein RA, Yanoff M, Albert DM. Thrombocytopenia, anemia, and retinal hemorrhage. Am J Ophthalmol 1968;65: 435–9.
41. Merin S, Freund M. Retinopathy in severe anemia. Am J Ophthalmol 1968;66:1102–6.
42. Holt JM, Gordon-Smith EC. Retinal abnormalities in diseases of the blood. Br J Ophthalmol 1969;53:145–60.
43. Carr RE, Henkind P. Retinal findings associated with serum hyperviscosity. Am J Ophthalmol 1963;56:23–31.
44. Guyer DR, Schachat AP, Vitale S, et al. Leukemic retinopathy. Relationship between fundus lesions and hematologic parameters at diagnosis. Ophthalmology 1989;96:860–4.
45. Robb RM, Ervin LD, Sallan SE. A pathological study of eye involvement in acute leukemia of childhood. Trans Am Ophthalmol Soc 1978;76:90–101.
46. Kincaid MC, Green WR. Ocular and orbital involvement in leukemia. Surv Ophthalmol 1983;27:211–32.
47. Nishio A, Sakaguchi M, Murata K, et al. Anterior communicating artery aneurysm in early childhood. Report of a case. Surg Neurol 1991;35:224–9.
48. Crisostomo EA, Leaton E, Rosenblum EL. Features of in-

tracranial aneurysms in infants and report of a case. Dev Med Child Neurol 1986;28:68–72.
49. McLellan NJ, Prasad R, Punt J. Spontaneous subhyaloid and retinal haemorrhages in an infant. Arch Dis Child 1986;61:1130–2.
50. Vapalahti PM, Schugk P, Tarkkanen L, Bjorkesten G. Intracranial arterial aneurysm in a three-month-old infant. Case report. J Neurosurg 1969;30:169–71.
51. Walsh FB, Hedges TR Jr. Optic nerve sheath hemorrhage. The Jackson Memorial Lecture. Am J Ophthalmol 1951;34: 509–27.
52. Muller PJ, Deck JHN. Intraocular and optic nerve sheath hemorrhage in cases of sudden intracranial hypertension. J Neurosurg 1974;41:160–6.
53. Gilliland MGF, Luckenbach MW, Massicotte SJ, Folberg R. The medicolegal implications of detecting hemosiderin in the eyes of children who are suspected of being abused [letter]. Arch Ophthalmol 1991;109:321–2.