# Clinicopathological Findings in Abusive Head Trauma: Analysis of 110 Infant Autopsy Eyes

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• PURPOSE: **To investigate the histopathology in a large series of autopsy eyes from children with abusive head trauma.**

• DESIGN: **Retrospective case-control series.**

• METHODS: **One hundred and ten eyes from 55 autopsies examined at an academic tertiary referral center over 21 years were tabulated for histopathology: subdural hemorrhage in the optic nerve sheath, intrascleral hemorrhage, any retinal hemorrhage, ora-extended hemorrhage, cherry hemorrhage, perimacular ridge, and internal limiting membrane tear. Select tissues with cherry hemorrhage were further examined by transmission electron microscopy.**

• RESULTS: **Sixty eyes were identified as "abusive head trauma" (cases), 46 as "alternative cause" (controls), and 4 as "abusive head trauma survivor". Cases were legally verified or confirmed by confession in all except 1 case. All ocular histopathologic observations from cases were similar or more frequent in infants younger than 16 months of age. When present, a cherry hemorrhage and perimacular ridge were most often found together, and only with a torn internal limiting membrane. Both abusive head trauma survivor cases demonstrated severe optic nerve atrophy and macular ganglion cell loss.**

• CONCLUSIONS: **Younger infants may be even more susceptible to damage from vitreomacular traction by rotational and/or acceleration–deceleration forces. Identifying cherry hemorrhages may aid abusive head trauma diagnosis. Survivor abusive head trauma pathology demonstrates unique, irreversible macular and optic nerve damage. (Am J Ophthalmol 2014;158:1146–1154.** 

SHAKEN BABY SYNDROME, CURRENTLY TERMED abusive head trauma,[1] was first described in 1974 in regard to the physical abuse of children[2] and is characterized by findings such as the perimacular retinal fold.[3]

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Controversy now exists regarding the primary mechanism responsible for the ocular findings found in abusive head trauma, despite the overwhelming evidence in support of the theory of acceleration–deceleration forces solely induced by vigorous shaking.[4] Other hypotheses attribute optic nerve sheath and retinal bleeding to a rise in intracranial pressure from myriad other causes, including intracranial hemorrhage[5] or pressure increases elsewhere in the circulation,[6] such as the abdomen and thorax. These other postulations, however, do not fully consider ocular anatomy, as intense cardiopulmonary resuscitation with presumably high intrathoracic pressures in a relatively large study failed to generate retinal hemorrhages in pediatric patients with a normal coagulation profile and platelet count.[7]

Other viewpoints suggest that the combination of hypoxia, brain swelling, and raised central venous pressure may cause extravasation into the subdural space owing to immaturity rather than direct venous rupture required by considerable force.[8] This complexity of multiple contributing inflammatory factors induced by shaking, then, may account for the subdural bleeding within the brain rather than mechanical forces on the bridging veins alone. It was found that shaking forces, when isolated, are insufficient to cause such documented damage and instead require angular acceleration from impact, albeit in the clinical vacuum of a biomechanical model.[9] However, ocular anatomy and its related biomechanics are not addressed. An extra layer of complexity must be considered given the unique anatomy of the vitreous and retinal tissues.

Perimacular folds, a well-established finding associated with abusive head trauma, are described as white retinal ridges surrounding the macula and have long been attributed to the vitreous traction on the neurosensory retina during shaking episodes.[10] Although they are commonly found in cases of abusive head trauma, there have been 3 documented cases of this retinal ridge clinically that were all attributable to severe crush injury, only 1 of which has histopathologic evidence.[11–13] However, to our knowledge, there are no reports of perimacular ridge formation in instances of minimal trauma or cardiopulmonary resuscitation. Therefore, it is our suspicion that a sufficient amount of acceleration–deceleration forces in conjunction with vitreous traction is required to produce these findings.

Herein, we examine the histopathologic findings of confirmed abusive head trauma cases in the Barbara W. Streeten, MD, Eye Pathology Laboratory. We also describe a unique type of hemorrhage that may be associated with

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abusive head trauma. Finally, we report unique ocular findings on autopsy of 2 survivors who died 2 years after abusive head trauma diagnosis.

## SUBJECTS AND METHODS

THIS MONOCENTER, RETROSPECTIVE, CASE-CONTROL series was reviewed at the Barbara W. Streeten, MD, Eye Pathology Laboratory at the State University of New York, Upstate Medical University in Syracuse, New York over a 21-year period (1994–2014). This study met Health Insurance Portability and Accountability Act requirements for research on decedents. Institutional review board review was waived by the State University of New York, Upstate Medical University Institutional Review Board, as the research did not involve information about living individuals.

One hundred and ten autopsy eyes from 55 cases suspicious for child abuse were examined. All eyes were formalin-fixed before gross and histopathologic examination (A.B.G.). Their eye pathology reports were retrospectively tabulated (M.P.B., K.H.U.) for the following findings: subdural hemorrhage in the optic nerve sheath, intrascleral hemorrhage, any retinal hemorrhage, hemorrhage extending to the ora serrata, cherry hemorrhage, perimacular ridge, and internal limiting membrane (ILM) tear (separated/detached from retina). Photomicroscopy was performed using the Olympus D28-CB apparatus (Olympus, Tokyo, Japan). Transmission electron microscopy (TEM) was used for 1 autopsy specimen sample. It required fixation in glutaraldehyde, post-fixation in osmium tetroxide, ethanol dehydration, infiltration with propylene oxide, and embedding before imaging by means of a Tecnai 12 BioTwin transmission electron microscope (Field Emission Incorporated, Hillsboro, Oregon, USA). Statistical analysis was performed by hand for odds ratios, proportion calculations, and population estimations, as well as using Microsoft Excel 2011 (Microsoft Inc, Seattle, Washington, USA) for independent *t* tests. The pathologic data and findings were analyzed with respect to the medicolegal and clinical history.

Based on histopathologic, clinical, and legal findings, each case (n = number of eyes) was placed in 1 of 3 causal groups: "abusive head trauma" (n = 60), "abusive head trauma survivor" (n = 4), and "alternative cause" (n = 46). All abusive head trauma cases, except 1, were legally verified by confession or conviction. With abusive head trauma survivor eyes, both cases involved severe, documented, nonaccidental shaking at least 2 years prior to death with significant neurologic and visual deficits; ultimate causes of death were most likely from indirectly related, chronic sequellae of the initial abuse. The alternative cause group was composed of eyes inconsistent with abusive head trauma, including suffocation, drowning, other bodily trauma, and sudden infant death syndrome/unknown. This latter group was treated as controls during relevant statistical analyses.

## RESULTS

PATHOLOGIC OBSERVATIONS WERE FOUND TO BE statistically more frequent with abusive head trauma (cases) than with alternative cause (controls). For each finding in the abusive head trauma group, the percent prevalence, odds ratio between cases and controls, and the corresponding 95% odds ratio confidence interval were as follows: subdural hemorrhage in the optic nerve sheath, 97%, 1305, 114.7–14 851.0; intrascleral hemorrhage, 63%, 79.5, 10.2–616.9; any retinal hemorrhage, 83%, 33.3, 11.2–99.6; hemorrhage extending to the ora, 70%, 107.3, 13.7–839.4; cherry hemorrhage, 40%, 30.7, 4.0–237.6; perimacular ridge, 42%, 15.7, 3.5–70.9; and ILM tear, 85%, 46.5, 14.5–149.4. The odds ratio for cherry hemorrhage, hemorrhage extending to ora, and intrascleral hemorrhage required substituting 1 for 0 in order to avoid indeterminate calculations for control eyes that lacked each of these 3 associated findings, thereby making the corresponding odds ratio estimations conservative. Perimacular ridges were found in only 2 control eyes, both from the same case: a 16-month-old male infant, who was feeding koi fish in a pond with family nearby, drowned and perished despite shaking resuscitative efforts upon rescue from the pond.

The Table shows pathologic observations of the abusive head trauma group organized relative to laterality, sex, and age. Pathologic findings were more commonly seen bilaterally than unilaterally for every observation. Each one had similar or greater frequency in younger infants. Specifically, subdural hemorrhage (2-tailed, unpaired, independent *t* tests, $P = .030$), any retinal hemorrhage ($P = .048$), hemorrhage extending to the ora serrata ($P = .024$), ILM tear ($P = .002$), and formation of the perimacular ridge ($P = .044$) were all significantly more frequent in infant eyes younger than 16 months. There was no significant difference regarding age in findings of intrascleral hemorrhage ($P = .306$) or cherry hemorrhage ($P = .334$). No significant difference with respect to sex was found ($P > .05$). The alternative cause group demonstrated zero to few positive findings for each category (Table).

All 60 abusive head trauma eyes had at least 1 histopathologic finding from the retinal hemorrhages, ocular hemorrhages, or vitreoretinal interface pathology groups, as illustrated in set (Venn) diagrams showing overlapping relationships (Figure 1). Fifty eyes (83%) had retinal hemorrhages, while 10 (17%) did not have a retinal hemorrhage of any kind (Figure 1, Left panel). Of those positive for retinal hemorrhages, 42 (84%) had hemorrhages extending to the ora serrata, and 24 (48%) had a cherry hemorrhage. All 24 eyes (100%) with a cherry hemorrhage had

**TABLE.** Importance of Age in Abusive Head Trauma and Comparison With Alternative Causes Regarding Ocular Histopathology

| | Subdural Hemorrhage | Intrascleral Hemorrhage | Any Retinal Hemorrhage | Hemorrhage Extending to Ora | Cherry Hemorrhage | Perimacular Ridge | Internal Limiting Membrane Tear |
|---|---|---|---|---|---|---|---|
| Abusive head trauma (eyes, n = 60) | 96.7 ± 2.3 | 63.3 ± 6.3 | 83.3 ± 4.9 | 70.0 ± 6.0 | 40.0 ± 6.4 | 41.7 ± 6.4 | 85.0 ± 4.6 |
| Laterality | | | | | | | |
| Unilateral (cases, n = 30) | 0 | 20.0 ± 7.4 | 20.0 ± 7.4 | 26.7 ± 8.2 | 20.0 ± 7.4 | 10.0 ± 5.6 | 3.3 ± 3.3 |
| Bilateral (cases, n = 30) | 96.7 ± 3.3 | 53.3 ± 9.3 | 73.3 ± 8.2 | 56.7 ± 9.2 | 30.0 ± 8.5 | 36.7 ± 8.9 | 83.3 ± 6.9 |
| Neither (cases, n = 30) | 3.3 ± 3.3 | 26.7 ± 8.2 | 6.7 ± 4.6 | 16.7 ± 6.9 | 50.0 ± 9.3 | 53.3 ± 9.3 | 13.3 ± 6.3 |
| Sex | | | | | | | |
| Female (eyes, n = 36) | 94.4 ± 3.9 | 66.7 ± 8.0 | 83.3 ± 6.3 | 69.4 ± 7.8 | 30.6 ± 7.8 | 36.1 ± 8.1 | 88.9 ± 5.3 |
| Male (eyes, n = 24) | 100 | 58.3 ± 10.2 | 83.3 ± 7.8 | 70.8 ± 9.5 | 54.2 ± 10.4 | 50.0 ± 10.4 | 83.3 ± 7.8 |
| *P* value[a] | .2475 | .5199 | 1 | .9103 | .0693 | .2930 | .5431 |
| Age | | | | | | | |
| 1–15 months (eyes, n = 38) | 100 | 65.8 ± 7.8 | 89.5 ± 5.0 | 78.9 ± 6.7 | 42.1 ± 8.1 | 50.0 ± 8.2 | 94.7 ± 3.7 |
| 17–30 months (eyes, n = 22) | 90.9 ± 4.8 | 59.1 ± 7.9 | 72.7 ± 6.9 | 54.5 ± 8.3 | 36.4 ± 8.3 | 27.3 ± 7.9 | 68.2 ± 7.5 |
| *P* value[a] | .0301* | .3055 | .0483* | .0239* | .334 | .0440* | .0025* |
| Alternative cause (eyes, n = 46) | 2.2 ± 2.2 | 0 | 13.0 ± 5.0 | 0 | 0 | 4.3 ± 3.0 | 10.9 ± 4.6 |

The data are expressed as percentage ± standard error.
[a]Two-tailed, unpaired, independent *t* test.
**Significant* P *value* <.05.

hemorrhages extending to the ora serrata. Among the 42 eyes with hemorrhage extending to the ora, 18 (43%) did not have a cherry hemorrhage.

Every abusive head trauma autopsy eye (100%) had at least 1 type of ocular hemorrhage (Figure 1, Middle panel). Thirty-three of the 60 eyes (55%) had all 3 types of ocular hemorrhage: retinal hemorrhage, intrascleral hemorrhage, and subdural hemorrhage. Out of the 50 eyes with retinal hemorrhages, only 1 (2%) lacked either a subdural or intrascleral hemorrhage. Within these, 33 (66%) had both subdural and intrascleral hemorrhages, while 15 (30%) had a subdural without intrascleral hemorrhage, and 1 (2%) had an intrascleral without subdural hemorrhage. Subdural hemorrhage was present in 58 eyes (97%), of which 33 (57%) also had retinal and intrascleral hemorrhages. Only 6 of these eyes (10%) positive for subdural hemorrhage had neither retinal nor intrascleral hemorrhages, while 15 (26%) had retinal hemorrhage of any kind without intrascleral hemorrhage, and 4 (6.9%) had intrascleral hemorrhage without retinal hemorrhage. Therefore, 10 eyes (17%) had subdural hemorrhage without retinal hemorrhage, of which 6 had unilateral retinal hemorrhages and 4 lacked retinal hemorrhages bilaterally. Intrascleral hemorrhage was present in 38 eyes (63%): 33 of those eyes (87%) also had subdural and retinal hemorrhages, 4 (11%) had subdural without retinal hemorrhages, and 1 (2.6%) had retinal without subdural hemorrhage. Intrascleral hemorrhage always accompanied a retinal or subdural hemorrhage.

Vitreoretinal interface abnormalities were seen in 51 abusive head trauma eyes (85%) (Figure 1, Right panel). ILM tear in isolation was the most common observation in 22 eyes (37%). The incidence of ILM tear with a perimacular ridge and cherry hemorrhage was 20 (33%), while incidence of only ILM tear and a perimacular ridge was 5 (8%) and of only cherry hemorrhage with ILM tear was 4 (6.7%). Every eye with a perimacular ridge or cherry hemorrhage had a torn ILM. In eyes with ILM tear, 20 (39%) also had a cherry hemorrhage and a perimacular ridge, 5 (10%) had a perimacular ridge without a cherry hemorrhage, 4 (7.8%) had a cherry hemorrhage without a perimacular ridge, and 22 (43%) did not have an accompanying perimacular ridge or a cherry hemorrhage. In total, 24 (40%) eyes had a cherry hemorrhage: 20 (83%) also had ILM tears and a perimacular ridge, while 4 (17%) had an ILM tear without a perimacular ridge. There were 25 (42%) eyes

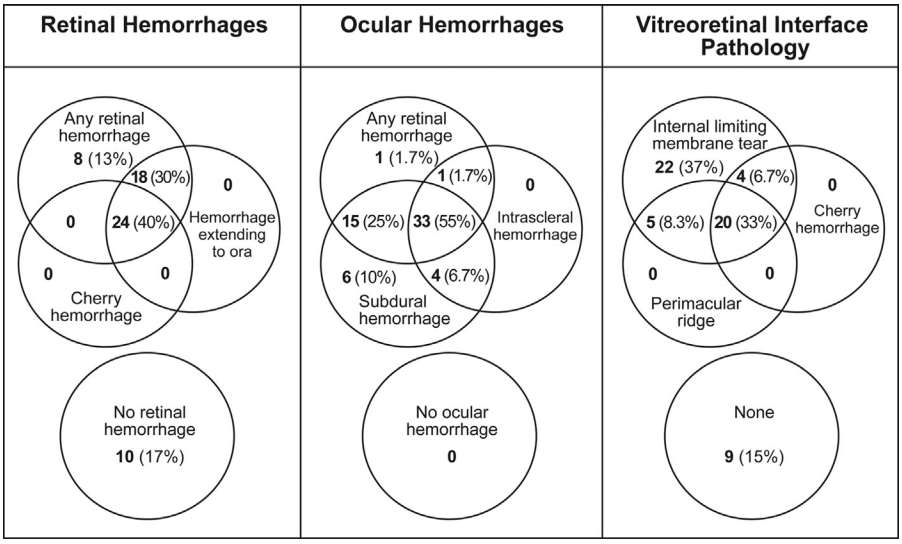


**FIGURE 1. Set (Venn) diagrams showing overlapping associations within clinicopathologic findings of abusive head trauma group (n = 60 eyes). (Left panel) Diagram shows that most abusive head trauma eyes had retinal hemorrhages. Cherry hemorrhages were only found when hemorrhages extending to the ora were also present. (Middle panel) Diagram illustrates that all abusive head trauma eyes had at least 1 type of ocular hemorrhage, and over half had all 3 types. Intrascleral hemorrhage was always accompanied by another type of ocular hemorrhage. (Right panel) Diagram shows that most abusive head trauma eyes had a detached internal limiting membrane. When present, a cherry hemorrhage and perimacular ridge were most often found together, and only with a torn internal limiting membrane.**

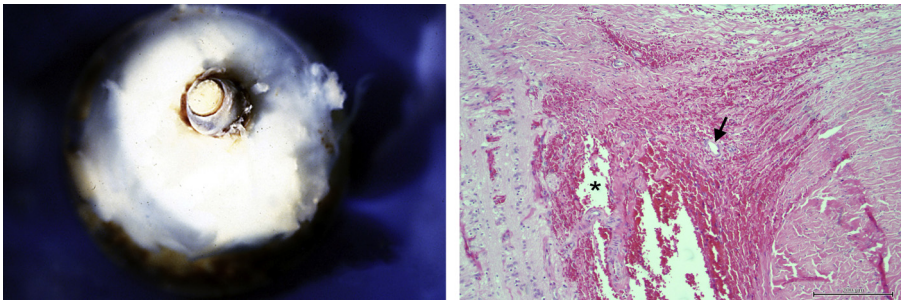

**FIGURE 2. Subdural and intrascleral hemorrhage found on autopsy in abusive head trauma by histopathologic examination. (Left) Cross-section of gross examination from 2-year-old infant with abusive head trauma shows hemorrhage inside the dural sheath of the optic nerve. (Right) Microscopic examination at the optic nerve–scleral junction from 10-week-old infant with abusive head trauma demonstrates massive subdural hemorrhage between optic nerve and meninges (asterisk) and intrascleral hemorrhage. Blood from intrascleral vessel (arrow) infiltrates the surrounding tissue that is continuous with the subdural space (hematoxylin-eosin).**

out of 60 with perimacular ridges: 20 (80%) also had both cherry hemorrhages and ILM tears, while 5 (20%) had a torn ILM without a cherry hemorrhage.

Subdural hemorrhage at the optic nerve has a bluish hue externally. In cross-section, the blood is visible inside the dura (Figure 2, Left). Microscopically, intrascleral hemorrhage is found surrounding ruptured intrascleral vessels at the junction of the optic nerve and sclera (Figure 2, Right). Intrascleral bleeding is often continuous with the subdural space.

Typical perimacular ridges are elevated, circular retinal folds with a canopy of ILM above, torn away from retina, with fibrin-hemorrhage debris below. Often a portion of the perimacular ridge can be seen clinically, surrounding hemorrhage at the macula (Figure 3, Top left). On gross and microscopic examination, retinal ridges have a canopy of detached ILM over the ridge with hemorrhage and fibrin below (Figure 3, Top middle and Bottom).

A cherry hemorrhage is an isolated, single, circular, elevated bleed, typically in the equatorial retina, that is observable by gross examination (Figure 4, Top left). Smaller cherry hemorrhages are focal hemorrhagic detachments of the ILM without an obvious break (Figure 3, Top right). Larger ones, microscopically, show a retinal ridge with torn ILM canopy surrounding blood and fibrin

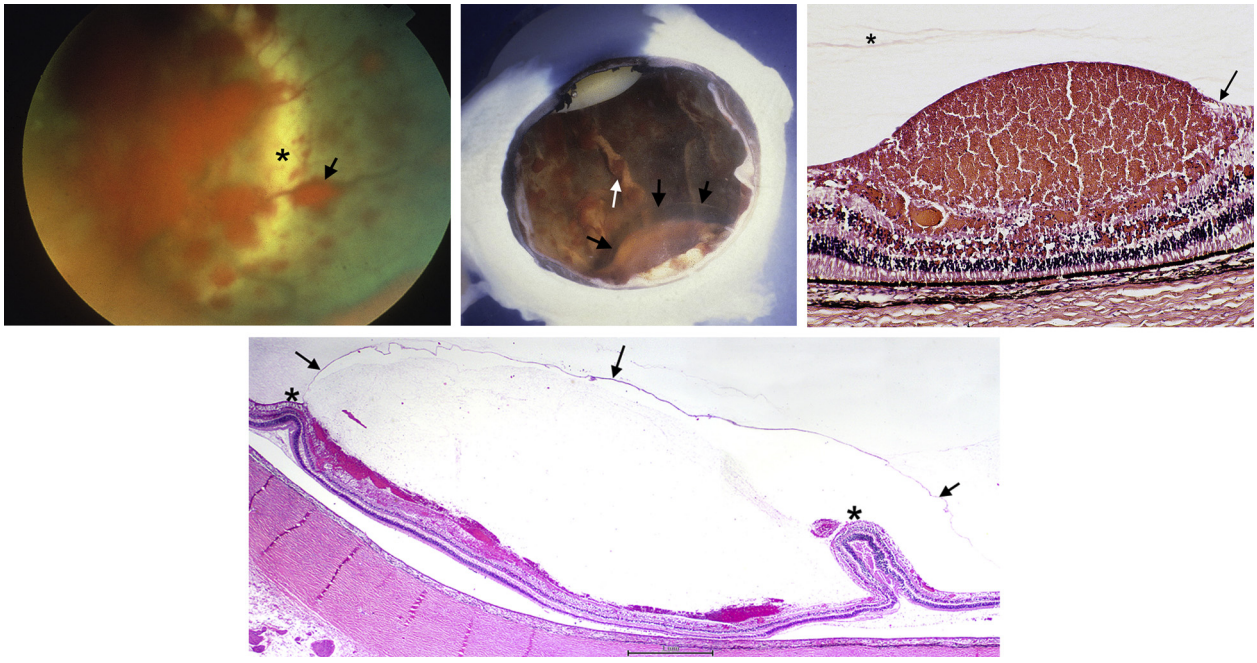

**FIGURE 3. Clinical, gross, and histopathologic features in a 1-year-old infant with abusive head trauma. (Top left) Funduscopic photograph shows retinal hemorrhages and a portion of an elevated perimacular ridge (asterisk) with peripheral cherry hemorrhages (arrow). (Top middle) Gross examination demonstrates a perimacular ridge with a canopy of detached internal limiting membrane (black arrows) above and hemorrhage below, as well as equatorial cherry hemorrhages (white arrow). (Top right) Microscopic examination of a small cherry hemorrhage shows a hemorrhagic detachment of the internal limiting membrane (arrow) with vitreous (asterisk) above (hematoxylin-eosin, ×400). (Bottom) Low-power microscopic examination of the perimacular ridge (asterisks) with its detached internal limiting membrane canopy (arrows) stretching over a crater filled with fibrin and hemorrhage (hematoxylin-eosin).**

beneath (Figure 4, Top right and Bottom left). Ultrastructurally, the basement membrane of the ILM is composed of attached vitreous fibrils on one side and Müller cell remnants on the other (Figure 4, Bottom right). Every eye with a cherry hemorrhage had at least 1 documented ILM tear elsewhere in that eye.

Two patients (4 eyes) in our series survived abusive head trauma 2 years prior to their death (abusive head trauma survivor group). The first patient was a 30-month-old boy who died in bed with vomit around his face and survived shaking at 8 weeks by the confessed biological father, resulting in quadriplegia and cortical blindness until death. The second patient was a 3-year-old girl who survived abusive head trauma at 1 year by the mother's boyfriend, resulting in severe neurological injuries and a severed spinal cord, ultimately succumbing to death from respiratory failure. Histopathologic eye findings were similar in both children; those findings are a thin, cupped optic nerve with bowed lamina cribrosa; macula with torn ILM; and a thin nerve fiber layer with loss of ganglion cells, as well as absent macular/temporal axons consistent with optic nerve and macular ganglion cell degeneration (Figure 5). The optic nerve was demyelinated and no hemorrhage or hemosiderin was detected.

## DISCUSSION

PERIMACULAR FOLDS, FIRST DESCRIBED BY GREENWALD and associates[14] in 1986, are considered a specific finding for abusive head trauma in the appropriate clinical situation, but not pathognomonic. We found perimacular folds in nearly half of abusive head trauma eyes. Although not a sensitive finding, they are specific for high-acceleration trauma. Two eyes from 1 accidentally drowned infant case showed perimacular folds; it is highly probable that these resulted from frantic resuscitative shaking efforts by family members. Consistent with our previous hypothesis, perimacular folds were found only in situations suspicious for severe acceleration–deceleration motion to a child's head, including the above case. Otherwise, no cases with relatively minor trauma had associated perimacular ridges. Though alternative causes like suffocation did not demonstrate pathology similar to abusive head trauma, it is important to note that these other mechanisms can be part of an abusive picture without being detected by histopathology.

Intrascleral hemorrhages were not found in any controls but were highly prevalent and specific for abusive head trauma, nearing two thirds of all cases. This figure is similar

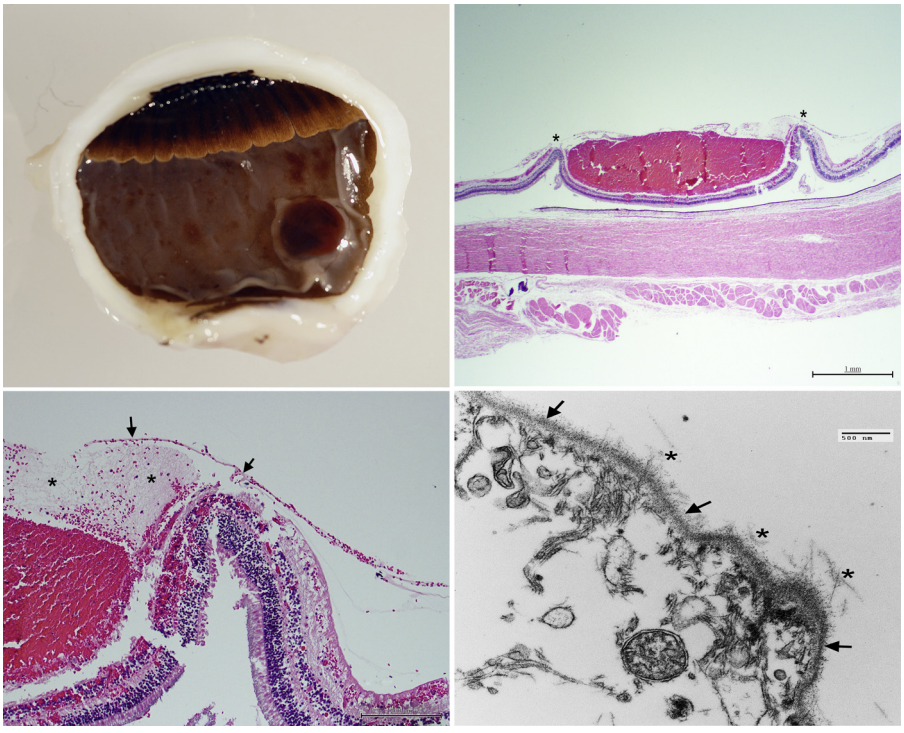

**FIGURE 4. Large cherry hemorrhage from a 17-month-old infant with abusive head trauma. (Top left) Cherry hemorrhage is 3.5 mm across at the equator. Small retinal hemorrhages extend to the ora serrata. (Top right) Histopathology of same lesion at low magnification demonstrates severe hemorrhage surrounded by an equatorial (asterisks) retinal ridge (hematoxylin-eosin). (Bottom left) High magnification shows a detached internal limiting membrane (arrows) with hemorrhage and fibrin (asterisks) below (hematoxylin-eosin). (Bottom right) Transmission electron microscopy demonstrates vitreous fibrils (asterisks) attached to the outer internal limiting membrane (arrows) with many Müller cell organelles attached to the inner portion of the internal limiting membrane.**

to Elner and associates' findings, which we calculated as 71%.[15] Our estimate of 63% and its 95% confidence interval range (50.4%–75.6%) for the population mean is no different. Subdural hemorrhages in the optic nerve sheath were detected bilaterally in all but 1 case. An intrascleral hemorrhage was found in 1 of these 2 eyes without subdural hemorrhage. Similarly, in Elner and associates' study,[15] subdural hemorrhage was found in all but 1 case, which, like ours, was positive for intrascleral hemorrhage. These exceptional cases illustrate that subdural hemorrhages are likely neither sufficient nor necessary for an intrascleral hemorrhage. It is our suspicion that scleral shearing forces are necessary to rupture the intrascleral vessels. In yet another study, optic nerve sheath hemorrhages were found to be statistically more frequent in 18 abusive head trauma "cases" compared to 18 fatal, accidental, and traumatic "controls."[16] These findings align with our own and support the theory that shaking forces are likely critical for creating subdural and intrascleral hemorrhages.

The acceleration–deceleration cycles responsible for causing vitreoretinal traction and intraocular trauma are likely similar to those that create damage at the scleral–optic nerve junction. This theory of tight tethering at this junction is consistent with other reports of intrascleral hemorrhages adjacent to the optic nerve.[17] In the literature, only 2 cases of peripapillary intrascleral hemorrhage have occurred in the absence of abusive head trauma.[18] Both of these cases involved neonates in utero of mothers involved in a motor vehicle accident, underscoring the requirement of intense acceleration–deceleration forces.

Although subdural hemorrhages are one of the most sensitive findings for abusive head trauma, reaching 100% in 1 report,[19] they are not always present in shaking trauma, as demonstrated by the 97% proportion in our own cases. No specific histopathologic finding, including subdural hemorrhage or any retinal hemorrhage, is sufficient or necessary for a diagnosis of abusive head trauma.[20] Rather, it is the presence or absence of several findings, with clinical clues from the history, that collectively lead to a reliable, valid, and correct diagnosis.

In 100 hospitalized patients younger than 2 years, retinal hemorrhages were exclusively found in patients with inflicted injury, and only occasionally from serious accidental head injury.[21] In the absence of other reasonable medical explanation, retinal hemorrhages most often require severe physical trauma.

The proportion of retinal hemorrhages, 83% in all our abusive head trauma cases, is a figure that is essentially equivalent to the 85% found and summarized previously.[22] Out of the 17% that did not have retinal

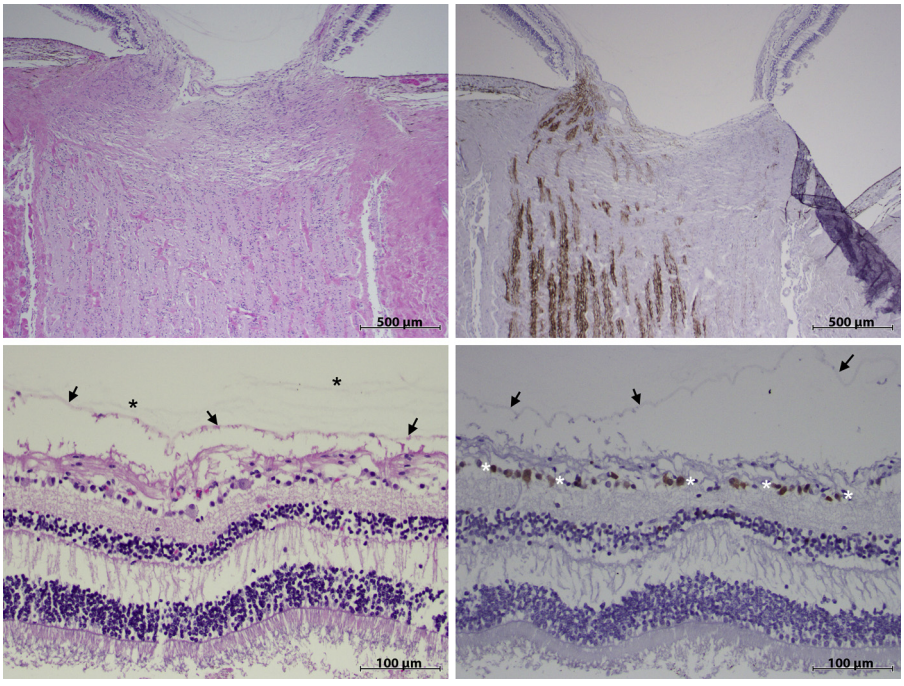


**FIGURE 5. Histopathology of retina and optic nerve from a child survivor of abusive head trauma at age 1 year who died 2 years later from respiratory infection. (Top left) Low magnification demonstrates cupping and atrophy of the optic nerve with bowing of the lamina cribrosa (hematoxylin-eosin). (Top right) Immunohistochemistry of optic nerve head shows axons traversing the nerve from the nasal retina, but not from the macular fibers of the temporal retina (neurofibrillary protein). (Bottom left) High magnification at the macular region shows vitreous fibrils (asterisks) superiorly and a shallowly detached internal limiting membrane (arrows). Presumed prior hemorrhage is no longer present and macular ganglion cells are reduced to a single layer (hematoxylin-eosin). (Bottom right) Immunohistochemistry demonstrates the sparse nuclei of ganglion cells (white asterisks) and an internal limiting membrane (black arrows) detachment (NeuN).**

hemorrhages, all but 4 eyes (2 cases) were unilateral and, therefore, detectable in the fellow eye. These other 4 eyes (6.7%) were devoid of retinal hemorrhage bilaterally but had subdural hemorrhage, consistent with abusive head trauma. Our findings suggest that clinicians may not always find retinal hemorrhages in abused children. Moreover, our study perhaps underestimated the incidence of such findings since we focused on injuries found to be severe enough to cause death. The survivors may have had subdural hemorrhages detectable by magnetic resonance imaging (MRI). The MRI can be a vital tool, with great sensitivity and specificity, for identifying those infants who have brain subdural hemorrhage but lack retinal hemorrhages and who would otherwise be overlooked for abusive head trauma.[23]

Retinal hemorrhages in our study were also found to be proportionately more frequent in children younger than 16 months of age compared to infants older than 16 months. Our study is similar to one in which children younger than 1 year were found more likely to have retinal hemorrhages.[24] This same study also demonstrated a "dome-shaped hemorrhagic lesion" in the macula "that elevates the internal limiting membrane," essentially describing the perimacular ridge. This is similar in appearance to cherry hemorrhages typically located peripherally. To our knowledge, the cherry hemorrhage has not been previously described. Found in 40% of our abusive head trauma eyes and demonstrated using gross, histopathologic, and TEM examinations (Figure 4), the cherry hemorrhage is a distinct hemorrhagic lesion often confined to the equatorial retina that can be seen by indirect ophthalmoscopy. Microscopically, it is similar to the perimacular ridge with a dome of torn ILM over a large hemorrhage. Furthermore, this lesion was found only in eyes that had a torn ILM and concurrent retinal hemorrhages extending to the ora serrata. The threshold of acceleration–deceleration forces necessary to produce bleeding throughout the retina (ora-extended) is likely lower than that for creating the cherry hemorrhage. Neither a cherry hemorrhage nor an ora-extended hemorrhage was found in control eyes. Thus, the cherry hemorrhage is one more robust criterion for identifying abusive head trauma.

Our findings most strongly corroborate the role of vitreoretinal traction. Other, less-substantiated hypotheses include increased intrathoracic pressure, increased intracranial pressure, and retinal hypoxia.[22] Indeed, animal models have determined a limited role for retinal hypoxia in the presence of retinal hemorrhages.[25] This finding

parallels the absence of retinal hemorrhages found clinically in hypoxic children.[22]

Laterality of findings is an important consideration when faced with a diagnosis of abusive head trauma. All eyes in our series were proportionately more likely to have bilateral than unilateral pathology. However, at least 1 unilateral presentation for each finding, except subdural hemorrhage, was found in all cases. These findings corroborate previous studies[26] that should alert the clinician to the significant, non-zero incidence of abusive head trauma cases with unilateral findings, including retinal hemorrhages.

These dramatic clinicopathologic findings show that vitreomacular attachments most likely are needed for transmitting intense acceleration–deceleration forces throughout the eye. The characteristic pathology of the perimacular ridge, described as a "dome-like lesion" filled as a "traumatic bloody cavity" at the macula with fibrin deposition and an elevated, peeled ILM, is the logical consequence of these traumatic forces.[27] Observing these findings in their abusive head trauma "cases" but not "controls" is again consistent with our histopathology.

Perimacular ridge formation is often minimized as an unreliable finding in abusive head trauma, partially because of its presence in 2 seemingly accidental cases,[11,12] rather than considering them as outliers that deviate from the norm.[28] Though it may not be pathognomonic, it is important to emphasize the perimacular ridge in diagnosing abusive head trauma, by recognizing the vitreomacular traction involved in its formation. Every perimacular ridge in our study, like the cherry hemorrhage, was found in association with an ILM tear. Roughly half of all ILM tears were associated with perimacular ridge formations, and still, the majority of cherry hemorrhages were found concurrently with a perimacular ridge and an ILM tear. This evidence points strongly towards a linked mechanism of vitreoretinal traction for creating the perimacular ridge and cherry hemorrhage.

Vitreomacular attachments become weaker by as early as 20 years of age.[29–31] Furthermore, clinically relevant effects of this diminishing vitreomacular connection may be seen at as early as 1 and 2 years of age, based on our results. Specifically, retinal hemorrhages, hemorrhages extending to the ora, perimacular ridges, and ILM tears all occurred more frequently in infants less than 16 months of age compared to those older than 16 months. While controlling for other confounding variables may be necessary, it seems most plausible that the age-related change in the vitreomacular interface plays at least some part in this proportional difference in findings between 1- and 2-year-old abused children. Thus, the youngest eyes may be the most vulnerable to violent forces.

Our 2 cases of "survivor" abusive head trauma after inflicted trauma 2 years prior to death demonstrate unique histopathologic features. The remarkable optic nerve cupping and atrophy with macular ganglion cell scarcity, in addition to the perpetually torn ILM, demonstrate the long-term consequences of ocular changes in previously shaken infants. The lack of hemorrhage and the negative iron stain may both indicate that blood and hemosiderin alike had long been resorbed earlier during the 2-year period. To the authors' knowledge, neither chronic ocular changes demonstrated by autopsy of abusive head trauma eyes years after the abuse nor an expected time of hemosiderin resorption has been reported in the literature. However, ischemic and neovascular retinal changes secondary to abusive head trauma have been described in 3 live children in whom preretinal fibrovascular proliferation was found in a several-month time course after shaking.[32] We hypothesize that the shaking trauma may have been more severe in our 2 cases, leading to the loss of inner retinal vessels rather than healed vessels. The dramatic optic nerve atrophy and ganglion cell decrease may not have made fibrovascular membrane formation viable for the inner retina in our 2 cases. Further pathologic and clinical investigation of the chronic effects of abusive head trauma, along with its related, and more frequent, acute presentation, will be necessary for clarification.

The diagnosis of abusive head trauma can be challenging and involves a multidisciplinary approach. Ocular histopathology, combined with the clinical picture, is often essential for establishing abusive head trauma in suspected infant autopsies. The findings described in this study, including the perimacular ridge, further illustrate the physical mechanism of violent forces transmitted by vitreoretinal traction that embodies abusive head trauma based on age-related, anatomical vulnerability. Future studies, including biomechanical models, regarding the perimacular ridge, cherry hemorrhage, and the unique pathology of surviving abusive head trauma children may hopefully shed further light on this disease.

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## Biosketch

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