A Systematic Review of the Diagnostic Accuracy of Ocular Signs in Pediatric Abusive Head Trauma

Gaurav Bhardwaj, BMed,[1] Vivek Chowdhury, MB, BS, PhD,[2] Mark B. Jacobs, MB, BCh, MD,[1,2] Kieran T. Moran, MB, BCh, FRACP,[1,3] Frank J. Martin, FRANZCO, FRACS,[2,4,5] Minas T. Coroneo, MD, FRACS[1,2]

Topic: To review systematically the diagnostic accuracy of various ocular signs for pediatric abusive head trauma (AHT).

Clinical Relevance: Intraocular hemorrhages (IOH), perimacular retinal folds, traumatic retinoschisis and optic nerve sheath hemorrhages have been reported as cardinal signs of AHT. The evidence base supporting the accuracy of this interpretation, however, has not been systematically reviewed.

Methods: A systematic keyword search of MEDLINE, EMBASE, and Evidence-Based Medicine Reviews was conducted for original studies reporting ocular findings in AHT. Articles were graded using a checklist for systematic reviews of diagnostic accuracy.

Results: The initial search yielded 971 articles, of which 55 relevant studies were graded, and 20 studies met inclusion criteria and were included in the review. The overall sensitivity of IOH for AHT was 75% and their specificity was 94%. Intraretinal hemorrhage at the posterior pole was the most common finding, although extensive, bilateral, and multilayered IOH were the most specific for AHT. Optic nerve sheath hemorrhages had a sensitivity and specificity for AHT of 72% and 71%, respectively. Traumatic retinoschisis and perimacular retinal folds were reported in 8% and 14% of AHT, respectively, but were not reported in other conditions.

Conclusions: Prospective, consecutive studies confirm that IOH in infants—particularly bilateral, extensive, and multilayered—are highly specific for AHT. Optic nerve sheath hemorrhages are significantly more common in AHT than in other conditions, in autopsy studies. Traumatic retinoschisis and perimacular folds are present in a minority of AHT, but rarely seen in other conditions.

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Pediatric abusive head trauma (AHT) is a significant cause of morbidity and mortality, with an estimated incidence of 29.7 (95% confidence interval, 22.9–36.7) per 100 000, in infants <1 year old.[1] The spectrum of injury and clinical presentation is broad, and less severe cases are often difficult to diagnose due to unreliability of the history, nonspecific symptoms, and lack of external signs of injury.

The mechanism of injury in AHT is thought to be severe rotational acceleration secondary to violent shaking, direct impact, or a combination of the two. Since the first observation of retinal trauma in an abused infant 45 years ago,[2] intraocular hemorrhages (IOH) have been considered a hallmark of AHT. It has also been thought that perimacular retinal folds, traumatic retinoschisis, and multilayered IOH extending to the retinal periphery were highly correlated with repeated acceleration–deceleration injury.[3–7]

The validity of the diagnostic criteria for AHT and the specificity of any retinal findings for abuse have been called into question. An evidence-based appraisal of the literature up to 1998 reported inadequate scientific evidence to validate most aspects of AHT.[8] Some criticisms of the evidence base were the frequency of circular logic, lack of control groups, inconsistency in definition of cases, and a lack of prospective studies. Although these criticisms may have been valid for the period studied, this article was limited in its scope and did not specifically address retinal findings. Immense interest in this area has subsequently led to a number of detailed prospective studies being conducted.

A recent review[9] reported evidence from prospective studies, supporting the diagnosis of abuse in infants with head injury and IOH, and a correlation between severity of intraocular and intracranial pathology. This review, however, did not mention how articles were considered as being relevant and did not employ a systematic approach to filtering studies or assessing the risk of bias.

The primary objective of this article is to review, systematically, the accuracy of IOH, perimacular retinal folds, traumatic retinoschisis and optic nerve sheath hemorrhages, in diagnosing AHT in infants who do not have a credible history of accident or a medical condition.

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A summary of the findings from relevant studies will be presented, in an attempt to make evidence-based clinical recommendations pertaining to the sensitivity and specificity of these clinical signs for AHT.

Sources and Methods of Literature Search

Inclusion Criteria

Types of Studies. Studies were included that allowed conclusions to be made regarding sensitivity or specificity of ocular findings for AHT. Sensitivity can be calculated from the frequency of ocular findings in a consecutive series of patients with AHT. Specificity can be calculated from the frequency of ocular findings in consecutively recruited control groups. Thus, the types of studies eligible for inclusion were case-control studies, cohort studies, or consecutive case series.

Population. Eligible studies included infants or children with AHT. Although there is no universally accepted reference standard for the diagnosis of AHT, cases that are accompanied by confessions, witnessed by reliable third parties, or confirmed in legal proceedings are commonly accepted as being valid cases. In addition, we accepted as valid, cases that were confirmed by multidisciplinary assessment using standard criteria.

Details. All articles had to include, at a minimum, the frequency of ocular findings in a consecutive series of AHT.

Exclusion Criteria

Studies were excluded if they did not include infants or children with AHT. With respect to study design, case reports and nonconsecutive case series were not included in the analysis, because the frequency and diagnostic accuracy of findings cannot be determined. Case reports of particular significance are, however, mentioned in the relevant sections.

Search Strategy

A comprehensive literature review was performed in September 2009. A broad range of terms encompassing AHT were searched including *shaken baby syndrome, battered child syndrome, child abuse, craniocerebral trauma, whiplash, shaken-impact syndrome, inflicted (traumatic) brain injury, non-accidental (head) injury,* and *abusive head trauma*. These were combined each with *retinal hemorrhage, retinoschisis, retinal fold,* and *optic nerve (sheath) hemorrhage*. The complete search string can be found in Appendix 1 (available on-line at http://aaojournal.org).

The following electronic databases were searched simultaneously via Ovid:

1. MEDLINE (1948–September 4, 2009, including in-process and non-indexed citations);
2. EMBASE (1947–September 4, 2009); and
3. Evidence Based Medicine Reviews, which included:

- Cochrane Collaboration;
- American College of Physicians Journal Club 1991 to September 2009;
- Database of Abstracts of Reviews of Effects; and
- National Health Service Economic Evaluation Database.

To reduce the likelihood that potentially relevant articles were missed, MEDLINE was also searched via PubMed and reference lists from relevant articles were checked. Foreign literature was reviewed for relevance by reading the English abstract. If thought to be relevant, the article was acquired and translation obtained. Foreign literature without an English abstract was not assessed.

Data Extraction

One author (GB) scanned the initial list of articles for relevance to the inclusion criteria, based on title and abstracts. All of the authors then independently assessed the relevant articles for inclusion. Disagreements were resolved by discussion. Grading of studies was performed with a checklist derived from a standard grading tool for systematic reviews of diagnostic accuracy.[10] A detailed explanation of the items that were graded is provided in Appendix 2 (available on-line at http://aaojournal.org).

For each study, tabulated data included (1) study design (i.e., prospective or retrospective, controlled, or case series); (2) study population; (3) cohort size; (4) data on intraocular and cerebral findings; and (5) grading items.

The level of evidence of included studies, and importance of clinical recommendations were rated according to the *Ophthalmology* guidelines. Level I evidence provides strong support for a statement, and is usually composed of well-performed, randomized controlled-trials or meta-analyses of randomized controlled-trials. Level II evidence provides substantial support for the statement but cannot be applied without qualification because the evidence lacks some qualities. This usually includes observational studies, such as cohort studies and case-control studies. Level III indicates a weak body of evidence relying on consensus statements, small noncomparative case series, and individual case reports. In terms of clinical recommendations, Level A is considered very important, level B moderately important, and level C possibly relevant to clinical outcome.

Results

The varied search terms for AHT combined with *retinal hemorrhage, retinal folds*, *retinoschisis* and *optic nerve sheath hemorrhage* returned a total of 971 unique articles. A total of 79 relevant articles from all search sets were reviewed in full. A further 9 articles were added from reference lists of relevant articles, making a total of 88 articles. Of these, 33 articles were excluded for various reasons (Fig 1), leaving 55 studies.

Grading was performed on all 55 eligible studies. Because we wished to assess the diagnostic accuracy of ocular findings for AHT, studies that did not adequately describe the method of ocular examination or used a method that was considered inadequate were also excluded. These were mainly retrospective reviews or prospective studies in which ocular findings were not a main focus.

The 20 studies remaining are the subject of this review (Table 1). Three of these studies only reported patients with retinal findings;

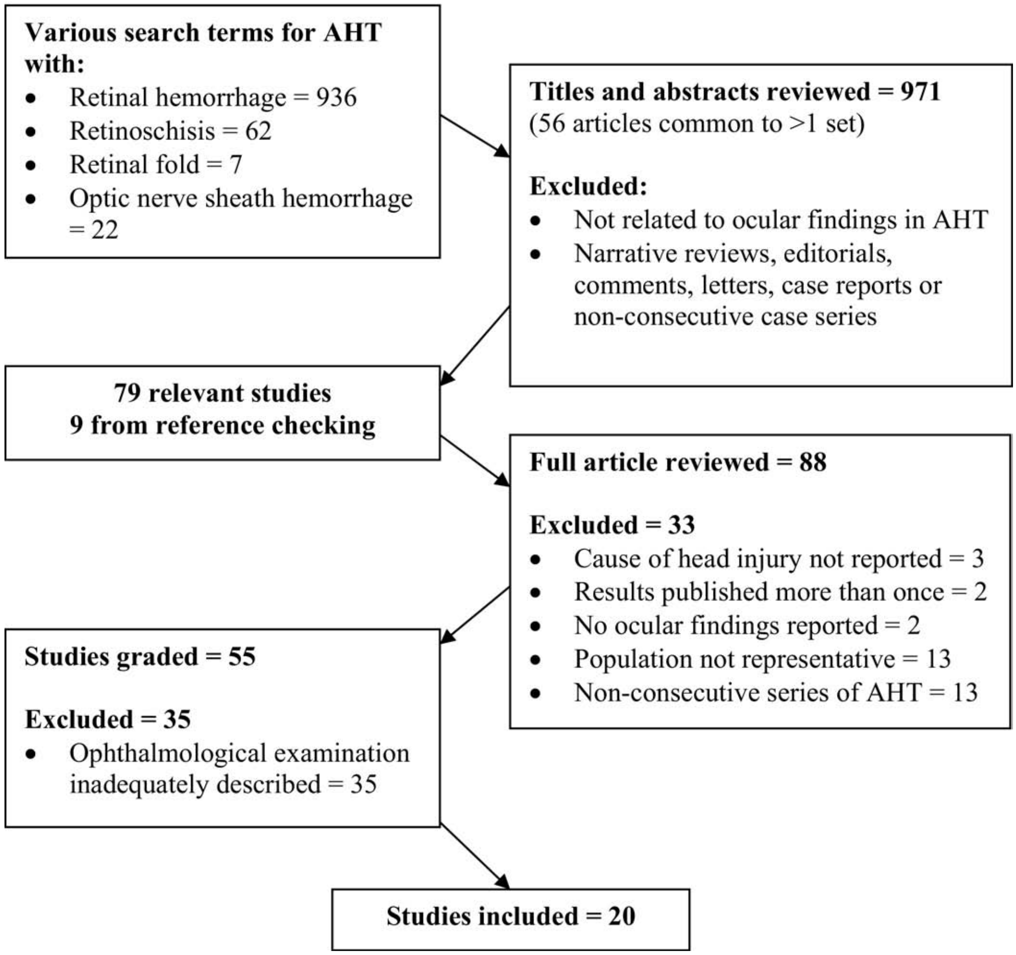

Figure 1. Results of search. AHT = abusive head trauma.

however, they were included because the cases were consecutively recruited and thus the pattern of findings could be analyzed. The overall quality of included studies, with respect to each of the graded items, is shown in Figure 2.

Several weaknesses were identified in the included studies. Only 2 studies mentioned that examiners were masked to the injury.[11,12] It is unlikely that examination was performed in a masked fashion in the other studies, because this detail was omitted. Confirmation of abuse remained unclear in more than half of studies owing to lack of detail regarding its determination. This also meant that there was the potential for circular logic in all but 4 studies,[12–15] because IOH are often used clinically as a diagnostic sign of abuse.

Individual results of grading for all studies and the references for excluded studies are available in Appendix 3 (available on-line at http://aaojournal.org).

Sources of Heterogeneity

Sources of heterogeneity in the studies included subgroups of patients, inclusion criteria, study design, and method of examination. Importantly, AHT is not a single diagnostic entity; therefore, significant variation in clinical presentation was apparent. Some studies recruited subgroups such as shaken baby syndrome, whereas other studies only recruited patients with subdural hematoma.

All but 1 study[16] included participants <4 years of age, and in this study only 3% were >3. Study designs comprised 3 main types, namely, autopsy series, retrospective chart reviews, and prospective clinical studies.

Intraocular Hemorrhages

The mean sensitivity of IOH for AHT in the included studies was 75% (Table 1). Clinical studies[12–15,17–20] found IOH in 74% (range, 51%–100%) of 560 combined cases and autopsy series found 82% with IOH (range, 63%–100%) in 84 combined cases.[6,7,21–25] This did not include the 3 studies that only reported cases with retinal findings and 2 large autopsy series of all forms of child abuse,[16,26] which found IOH in 46% (range, 45%–47%) of 555 combined cases.

The wide range is likely owing to the small sample sizes in some series[6,18,24] and intracranial hemorrhage as an inclusion criterion in others.[14] In 3 studies,[12–14] which were apparently free from circular logic and included confirmed cases of abuse, IOH were present in 80% of 151 cases of AHT.

Intraocular hemorrhages involved the retinal layers in almost all cases, in both clinical (94% of IOH) and autopsy series (99% of IOH). Most studies found evidence of hemorrhage in all of the retinal layers; however, numerous studies[16,21,23,26,27] reported the superficial layers—including sub-internal limiting membrane, nerve fiber and ganglion cell layers—to be most frequently and heavily involved. Preretinal hemorrhage was more frequently

Table 1. Summary of Studies in Review

Author (year, country)	Cohort Size	Age (yrs)	ICH %	IOH %	Comments
Retrospective chart reviews					
Wilkinson[11] 1989 U.S.A.	14 AHT (SBS)	0–2.3	100	100‡	Significant correlation found between diffuse IOH, large preretinal or vitreous hemorrhage and neurologic injury. Diagnostic accuracy not assessed. IOH part of diagnostic criteria for abuse.
Mills[28] 1998 U.S.A.	10 AHT (SBS)	0–1	100	100‡	Circular retinal folds, retinoschisis, and fixed pupils correlated with fatal outcome. Study evaluated association between ocular findings and neurologic outcome, not diagnostic accuracy.
Kivlin[14] 2000 U.S.A.	111 AHT (SBS)	0–3	100*	83	SDH inclusion criterion, 95 clinical cases, none with peripheral IOH, posterior pole most heavily affected in 24% 16 autopsy cases, mostly diffuse IOH with 77% peripheral involvement. Higher rate of vitreous, subretinal and dome shaped hemorrhage under ILM compared with clinical cases.
Morad[17] 2002 Canada	75 AHT (SBS)	0–4	100	85	83% multiple confluent hemorrhages, and 87% bilateral. Hemorrhage confined to posterior pole in 20%, diffuse IOH 61%. IOH part of diagnostic criteria for abuse.
Gilles[12] 2003 U.S.A.	14 AHT	0–2.4	100	71	Retrospective review of fundus photographs. Laterality and severity of IOH correlated with cerebral hypoattenuation in 70% cases. Diffuse IOH in 50% cases in at least 1 eye.
Binenbaum[20] 2009 U.S.A.	49 AHT	0–1.4	—	51	Reported a positive correlation between incidence and severity of IOH and probability of abuse. Only studied cases which were investigated for AHT.
Prospective clinical studies					
Buys[18] 1992 Canada	3 AHT (SBS) 75 accidental HI	0–3	100 7	100 0	Majority of accidental head injuries mild, recruitment rate of 40%. Small number of cases of AHT for comparison. Excluded infants <1 month old.
Pierre-Kahn[15] 2003 France	224 AHT 7 accidental HI	0–3	100* 100*	68 0	SDH inclusion criterion. Small number of cases of accidental HI. Rate of IOH lower in cases with impact (28%) compared with no sign of impact (78%). No correlation between presence of IOH and age. No pattern of IOH identified.
Bechtel[13] 2004 U.S.A.	15 AHT 67 accidental HI	0–2	80* 27*	60 10	The following were significantly more frequent in AHT: IOH (60% vs 10%), bilateral IOH (40% vs 1%), preretinal hemorrhage (30% vs 0), peripheral IOH (27% vs 0; $P<0.001$ for all), premacular hemorrhage (20% vs 0; $P<0.005$). Vitreous hemorrhage no significant difference (13% vs 0%; $P = 0.03$).
Vinchon[19] 2005 France	57 AHT 93 accidental HI	0–2	81* 28*	75 7	IOH had positive predictive value for AHT 89%, negative predictive value for AHT 83%. IOH part of diagnostic criteria of abuse. Accidental head injury milder in severity.
Autopsy series					
Rao[21] 1988 U.S.A.	14 AHT (5 SBS) 16 controls	0–4 0–3	64 6	63 6	All retinal layers involved especially superficial; sub-ILM, nerve fiber, ganglion cell, inner nuclear. Controls included accidental trauma, SIDS, and others. Unclear whether control cases were consecutively obtained.
Elner[22] 1990 U.S.A.	10 suspected CA	0–4	60	70	Peripapillary scleral hemorrhage 50%. No details given on 3 cases without IOH.
Munger[27] 1993 U.S.A.	12 AHT (SBS)	0–2	92	100‡	75% optic nerve sheath hemorrhage, 42% retinal folds. Hemorrhage most often in nerve fiber and ganglion cell layer.
Budenz[23] 1994 U.S.A.	13 AHT 6 SIDS	0–3 0–0.5	92 0	85 0	Nerve fiber, inner plexiform and inner nuclear layers most involved. Severe optic nerve sheath SDH in all cases of AHT.
Betz[6] 1996 Germany	7 AHT (SBS) 42 control	0–1 0–73	100 71	100 5	All retinal layers involved in AHT. Controls included 24 head injuries, 6 ICH, 2 intravital brain death, 10 SIDS. Two of 24 (8.3%) head injuries had IOH. Age-mismatched controls.
Green[7] 1996 U.K.	16 AHT 7 CA (not primarily HI)	0–3	94 0	81 14	Found hemorrhage at the following sites with increasing force: SDH, preretinal, intraretinal, optic nerve SDH, retinal detachment, choroidal, vitreous, SAH, intracerebral. One case of asphyxia had vitreous, preretinal and intraretinal hemorrhages.

(*Continued*)

Table 1. (Continued.)

Author (year, country)	Cohort size	Age (yrs)	ICH %	IOH %	Comments
Marshall[24] 2001 Canada	6 AHT (SBS)	0–3	100	100	Small series. Unclear as to how diagnosis of SBS was established. IOH part of diagnostic criteria of abuse.
Riffenburgh[26] 2005 U.S.A.	197 confirmed CA 240 suspected CA 401 control	1[†] 0.5[†] 0.3[†]	— — —	47 44 4	Bipolar and ganglion cell-nerve fiber layers most commonly affected in AHT. Controls were unexplained deaths including auto accidents, drowning, SIDS. Odds ratio for abuse ($P<0.001$) with: IOH = 18.9, optic nerve sheath hemorrhage = 32.5, IOH + ICH = 53.0, IOH + ICH + optic nerve sheath hemorrhage = 53.3 True incidence of ICH unknown.
Wygnanski-Jaffe[25] 2006 Canada	18 AHT (SBS) 18 accidental HI	0–3 0–14	94 67	89 17	Focused on optic nerve and orbital pathology. Optic nerve sheath hemorrhage found in 78% AHT and 39% accidental HI ($P<0.04$). Age mismatched controls. No extra details of IOH. IOH part of diagnostic criteria of abuse.
Emerson[16] 2007 U.S.A.	118 CA	0–9 94% <3	53	47	IOH severe in 68% and widespread, confluent in 9%. Associated with ICH or optic nerve sheath hemorrhage in 96% of cases. Peripapillary scleral hemorrhage in 38% always associated with perimacular folds and 98% with optic nerve sheath hemorrhage. Severe retinal involvement seen in 6 of 7 cases with admission of shaking. Nerve fiber layer involved in 97% of IOH, extending to deeper layers in 81%.

AHT = abusive head trauma; CA = child abuse (all forms); CE = cerebral edema; HI = head injury; ICH = intracranial hemorrhage; IOH = intraocular hemorrhage; SBS = shaken baby syndrome; — = not reported.
*Rate of SDH.
[‡]Retinal hemorrhage was an inclusion criterion on.
[†]Median ages reported only.

found in clinical than autopsy series (67% vs 27% of IOH). Vitreous hemorrhage was much more common in autopsy series than clinical series (72% vs 11% of IOH). Clinical studies did not often comment on subretinal or choroidal involvement; however, autopsy series found subretinal involvement in the majority (57%) of cases of AHT (Table 2). Intrascleral hemorrhages were found in 33% of 173 combined cases,[14,16,22,24,27] mainly in the peripapillary region involving the circle of Zinn.

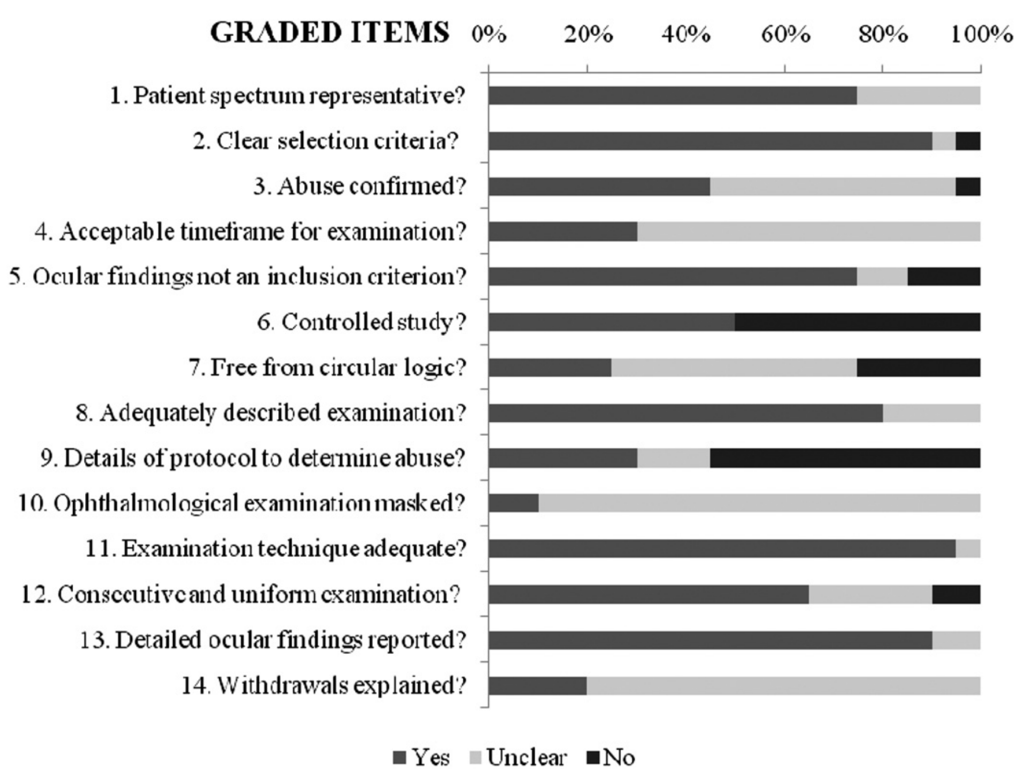

Figure 2. Graded items and overall quality of included studies.

Table 2. Location of Intraocular Hemorrhage in Abusive Head Trauma (AHT)

Clinical Studies			Layer Affected (# cases, % of IOH)			
Study (no. of cases)	*IOH, No. of Cases (%)*	*Age (mos)*	*Vitreous*	*Preretinal*	*Intraretinal*	*Subretinal*
Kivlin[14] (96)	77 (80)	0–36	13 (17)	36 (47)	76 (99)	10 (13)
Morad[17] (75)	64 (85)	2–48	—	55 (86)	55 (86)	44 (69)
Bechtel[13] (15)	9 (60)	0–24	2 (22)	5 (55)	9 (100)	—
Pierre-Kahn[15] (224)	152 (68)	0–29	15 (10)	106 (70)	142 (93)	—
Mills[28] (10)	10 (100)	2–10	3 (30)	7 (70)	10 (100)	—
Total (420)	312 (74)		33 (11)	209 (67)	292 (94)	54 (17)

Autopsy Studies			Layer Affected (# cases, % of IOH)				
Study (# cases)	*IOH (# cases, %)*	*Age (months)*	*Vitreous*	*Preretinal*	*Intraretinal*	*Subretinal*	*Choroid*
Emerson[16] (118)	55 (47)	0.3–108	47 (85)	—	53 (96)	37 (67)	8 (15)
Green[7] (16)	13 (81)	0.5–24	2 (15)	12 (92)	12 (92)	—	5 (38)
Munger[27]* (12)	12 (100)	1–21	10 (83)	3 (25)	12 (100)	11 (92)	6 (50)
Budenz[23] (13)	11 (85)	2–36	7 (54)	7 (54)	11 (100)	8 (73)	—
Kivlin[14] (27)	26 (97)	0–36	17 (65)	11 (42)	26 (100)	10 (38)	—
Total (186)	115 (62)		83 (72)	33 (27)	114 (99)	66 (57)	19 (17)

IOH = intraocular hemorrhage; — = not reported.
*Because IOH were an inclusion criteria, findings may be overrepresented.

The posterior pole of the retina (86% of IOH) was the most frequently reported area of IOH in AHT (Table 3). A similar proportion of hemorrhages (57% of IOH) were reported in the midperiphery and peripheral regions. Studies reporting laterality of ocular findings found, overall, 74% of IOH were bilateral in AHT.[6,11–15,17,18,20,21,24,27]

From the 4 prospective controlled studies, IOH were found in 6% of 242 combined cases of accidental head injuries,[13,15,18,19] and these were all reportedly mild in severity. Thus, when IOH are present after head injury, their specificity for abuse is 94% (range, 90%–100%). One study[19] found that moderate to severe IOH were only found in cases of AHT, and the sensitivity and specificity of these were 66% and 100%, respectively.

Other conditions studied were motor vehicle crashes (MVC),[21,26] sudden infant death syndrome,[6,21,23,26] central nervous system diseases, and other unexplained deaths.[21,26] The studies examining these were mostly small autopsy series and it is unclear whether the control groups were consecutively collected. Nevertheless in 1 large study,[26] IOH were found in just 4% of 401 control cases.

Meta-analysis of these data is unreliable because of the observational nature of studies and their heterogeneity with respect to design, age range, and inclusion criteria.

Retinal Folds and Retinoschisis

Seven studies[14–16,22,24,27,28] included in this review reported perimacular retinal folds in AHT. The frequency of this finding was higher in autopsy series (23%[16,22] of 128 combined cases) than in consecutive clinical studies[14,15] (3% of 347 combined cases). Their overall sensitivity for AHT was 8%. This was not inclusive of studies with retinal findings as an inclusion criterion,[24,27,28] which found retinal folds in 43% of 28 combined cases of AHT.

Traumatic retinoschisis was also reported in seven included studies[15–17,21,22,24,28] and its overall sensitivity for AHT (excluding studies which only reported positive retinal findings[24,28]) was 14%. Autopsy studies[16,21,22] once again demonstrated a higher proportion of cases with retinoschisis (23% of 142 combined cases) than clinical studies[15,17] (9% of 299 combined cases).

There were no studies in this review, which demonstrated retinal folds or retinoschisis in conditions other than AHT. These lesions have only been reported in a few case reports of other conditions relevant to infants, which will be discussed.

Table 3. Distribution of Intraocular Hemorrhage in Abusive Head Trauma (AHT)

Study (no. of cases)	Age (mos)	SDH %	IOH %	Bilateral IOH %	Posterior Pole (%)	Midperiphery (%)	Peripheral (%)
Emerson[16] (118)	0.3–108	53	47	80	85	78	61
Green[7] (16)	0.5–24	94	81	—	25	35	40
Marshall[24] (6)	1–34	100	100	100	100†	83†	67†
Morad[17] (75)	2–48	93	85	69	95	48	48
Gilles[12] (14)	1–54	100	71	57	90	—	90
Total % of IOH					86	57	57

IOH = intraocular hemorrhage; SDH = subdural hematoma; — = not reported.
*SDH was an inclusion criterion.
†Retinopathy of prematurity zones were used in the article. For the purpose of this table, Zone 1 was considered posterior pole, zone 2 midperiphery, and zone 3 peripheral (near ora serrata).

Optic Nerve Sheath Hemorrhage

Eleven included studies[6,7,16,21–28] examined this finding. The overall sensitivity of optic nerve sheath hemorrhage for AHT was 72% in 78 combined cases from consecutive autopsy series of AHT.[6,7,21–23,25] Subdural hematoma of the optic nerve sheath was found in 76% and subarachnoid hemorrhage in 46%. The frequency of involvement varied with inclusion criteria, and case series[16,26] including all forms of child abuse found only 50% of 311 combined cases with optic nerve sheath hemorrhage. Other case series of AHT,[24,27,28] with retinal findings as inclusion criteria, found 75% of cases had optic nerve sheath involvement.

Accidental head injury caused optic nerve sheath hemorrhage in 29% of 42 cases from 2 controlled autopsy studies.[6,25] Thus, its specificity for AHT can be estimated at 71%. The location of hemorrhage in the accidental cases was subdural in 92% and optic nerve subarachnoid hemorrhage was not seen. Riffenburgh[26] found that optic nerve sheath hemorrhage had a higher correlation with abuse than IOH, with an odds ratio of 32.5 ($P<0.001$) compared with 18.9 ($P<0.001$) for IOH.

One study[25] found all forms of orbital pathology more common in the AHT group. Orbital fat hemorrhage was observed in 50% of AHT, as opposed to 22% of accidental head injuries and extraocular muscle hemorrhage was observed in 28% of AHT, but not seen in accidental head injury. These findings were more marked in the posterior orbit.

Discussion

Since the first case report[2] of retinal trauma in an abused infant, level III retrospective studies[14,17] have confirmed that a significant proportion of infants with AHT have IOH. These studies have several potential drawbacks, including incomplete data and interobserver bias. The largest retrospective series[14] found that fatal cases had more severe and diffuse retinal findings extending to the periphery, whereas nonfatal cases had fewer IOH that were confined to the posterior pole.

Numerous level III autopsy series[6,7,16,21–27] have documented the full extent of injuries in severe cases of AHT. The reviewed studies found IOH much more frequently, and with greater severity, in AHT than in other studied conditions, including accidental head injury.

More recently, level II prospective controlled studies[13,15,19] have confirmed that IOH occur much more frequently in AHT than in accidental head injuries. The studies by Bechtel et al[13] and Vinchon et al[19] found that the specificity of IOH for AHT correlated more with the overall severity of IOH rather than their distribution or depth in the retina. Infants with AHT were also more likely to have multiple, bilateral IOH, involving the preretinal and intraretinal layers, covering the macula, and extending to the retinal periphery. This pattern of involvement was not specifically reported in any control cases in the included studies, but has been reported in fatal MVC[29] and crush head injury.[30–32]

The rate of IOH may be underestimated in most studies of AHT. This is because of delayed presentation in an unknown number of cases and delayed ophthalmologic examination after hospital admission. Only 5 studies[11,12,15,18,28] mentioned the timeframe within which examination was conducted.

The correlation between the incidence of IOH and age at presentation is unclear from the included studies. Riffenburgh et al[33] reported the greatest incidence of IOH in infants <1 year of age, whereas Kivlin et al[14] found that IOH significantly ($P<0.0063$) increased with age. Pierre-Kahn et al[15] found no correlation between age and presence of IOH in their large, prospective study.

Confirming the precise mechanism of injury is one of the major difficulties in studying AHT. Most of the included studies contained a proportion of cases that had evidence of direct cranial impact, in the form of scalp bruising or skull fractures. Pierre-Kahn et al[15] found that cases of AHT with evidence of direct impact had significantly lower rates of IOH compared with those with no evidence of impact. Morad et al[17] found no correlation between signs of direct impact and presence, or severity, of IOH. Budenz et al,[23] in a small autopsy series, found IOH in a higher proportion of cases with injury owing to blunt trauma. Evidence of direct impact, however, does not exclude shaking as a contributing factor. It thus remains difficult to confirm the relative contributions of direct impact and shaking to the overall picture of IOH. It is clear, however, that the full spectrum of ocular injuries can occur in cases without evidence of direct impact.

Emerson et al[16] proposed that peripapillary intrascleral hemorrhage may be characteristic of shaking injury, because evidence of blunt impact was absent in more than half of these cases in their series. Furthermore, all these cases had perimacular retinal folds, and 98% had optic nerve sheath hemorrhages, leading the authors to propose that they may result from torsion at the junction of the optic nerve and the globe during shaking, causing tears in dural bridging veins and the vessels near the circle of Zinn.

Retinal folds and traumatic retinoschisis in AHT were first reported in 1986 by Gaynon et al[3] and Greenwald et al,[4] respectively. Both lesions were reported in a minority of AHT cases in the included studies, although they were more commonly seen in autopsy series. They were also associated with more severe neurologic injury and a high mortality rate, in retrospective clinical series.[11,14,28] The rate of traumatic retinoschisis may be underestimated in clinical studies owing to similarity in appearance and difficulty in differentiation from preretinal hemorrhage.

None of the controlled studies in this review reported these lesions in conditions other than AHT, demonstrating a high specificity. They have previously been reported in 3 cases of crush head injuries.[30–32] A retrospective series[34] of 25 crush head injuries did not find any cases with retinal folds or retinoschisis, indicating that they are an uncommon finding. In the recent autopsy series by Kivlin et al,[29] elevated retinal folds were reported in 3 of 10 victims <3 years of age in fatal MVC. These patients reportedly experienced severe deceleration and rotational whiplash injury.

At our institution, we have also observed a hemorrhagic retinopathy with bilateral macular retinoschisis and elevated retinal folds in a 2-year-old infant after a fatal 11-meter fall onto concrete (Acta Paediatrica 2008;97:149). This fall resulted in multiple, bilateral parietal and occipital skull fractures, acute right subdural hematoma, and diffuse edema of the right cerebral hemisphere with mass effect and tentorial herniation. Police investigation was conducted and no criminal charges were filed. Thus, these lesions have been rarely reported in other circumstances and the history should be the guiding factor in determining their etiology.

The data concerning retinal folds and retinoschisis are mostly derived from level III evidence. The only prospective, controlled study,[15] which mentioned these findings, reported a single case of AHT with a unilateral perimacular retinal fold. Further studies, which accurately and extensively document retinal findings in a wide variety of conditions, are required to better estimate the specificity of these signs for AHT.

Retinal folds and retinoschisis in AHT have been proposed to result from traction and shearing forces applied to the retina, at firm points of attachment to the vitreous during acceleration–deceleration.[3–5,24,28] In infants, the vitreous is especially well-attached to the retina at the posterior pole, over blood vessels and near the ora serrata.[35] This hypothesis does not readily explain the findings in crush head injury. Other authors have proposed that sustained venous stasis,[16] or an abrupt elevation in intracranial pressure transmitted to the optic nerve,[31,36] result in rupture or extravasation of fluid from intraretinal vessels causing separation of the internal limiting membrane[36] or outer nuclear layer[31] from the rest of the retina. Keithahn et al[36] hypothesized that perimacular folds were created as the internal limiting membrane separated from the macula, while remaining attached to the more peripheral retina.

Only one large, controlled study[26] has documented optic nerve findings in a variety of conditions. The incidence of optic nerve sheath hemorrhage was significantly higher in AHT (odds ratio, 32.5; $P<0.001$) than in other conditions including accidental injury, sudden infant death syndrome, and drowning. Other studies[6,25] found almost one third of accidental head injuries also had optic nerve sheath hemorrhage. A recent autopsy series[29] of fatal MVC victims found optic nerve sheath hemorrhages in 9 of 10 cases, all of which were thought to have experienced significant whiplash injury.

The mechanisms underlying optic nerve sheath hemorrhage in AHT are still unresolved. Lambert et al[37] first described the presence of optic nerve sheath hemorrhages in an infant with presumed SBS. They proposed that expansion of the optic nerve sheath, owing to transmission of an acute elevation in intracranial pressure, resulted in rupture of bridging veins. In Riffenburgh's series,[26] however, optic nerve sheath hemorrhage often seemed to be a direct extension of intracranial submeningeal hemorrhage. The predilection of hemorrhage, in some studies,[25] to the optic nerve–globe junction and posterior orbit, have also lead to the hypothesis that these sites may act as fulcrums distributing force during acceleration–deceleration.

Investigation and Follow-up

Given that a diagnosis of abuse carries enormous social and legal ramifications, a comprehensive workup is required to exclude all potential mimics. This is usually undertaken by pediatricians trained in child abuse in collaboration with neurosurgeons, neurologists, radiologists, ophthalmologists, and other subspecialists, such as geneticists and endocrinologists.

A comprehensive history of the presenting illness, including the timeline of symptoms, and any episodes of prior trauma is taken. Detailed past medical history, including neurologic and hematologic disorders, and a family history, including any hereditary conditions, is sought. History of the pregnancy including labor, delivery, and neonatal vitamin K administration is also documented. If there is a history of trauma, a detailed reconstruction of the exact mechanism of injury is performed with assistance from the police.

As well as looking for evidence of cranial trauma, the infant is examined for evidence of extracranial injuries, such as skin, abdominal, and skeletal trauma. The relevant investigations for these injuries are undertaken.

Investigations to rule out other important mimics of child abuse are undertaken in all cases. A full coagulation screen and hematology consult are obtained. Metabolic and genetic conditions such as osteogenesis imperfecta,[38] Menkes,[39] and glutaric aciduria[40] are ruled out with appropriate advice from subspecialists. Cranial imaging is carried out according to recognized international guidelines comprising computed tomography scanning, magnetic resonance imaging, and magnetic resonance angiography, as well as bone scan and skeletal survey. Importantly, psychosocial stressors are not taken into account in making a diagnosis of abusive injury.

Once the diagnosis of AHT is confirmed, ophthalmologic follow-up varies depending on the findings. While a child is still admitted in hospital, weekly follow-up is appropriate. After discharge, the patient should be seen within 2 weeks to monitor the progress of retinal findings and assess the vision. Further follow-up can be lengthened to 4 weeks, 6 weeks, and then 6 months as long as the findings are adequately resolving. The patient's visual acuity, ocular alignment, retinae, and optic discs should be monitored at least until school entry age.

In most cases with extensive IOH, retinal folds, and retinoschisis, the prognosis is poor, and follow-up depends on future clinical management. If retinal detachments are present, referral is made to a vitreoretinal surgeon. If there are associated cranial nerve palsies, weekly follow-up is appropriate. Cortical blindness with severe intracranial injuries is assessed with visual-evoked potentials to estimate the visual outlook. Intracranial hemorrhage with no ocular signs is managed primarily by the neurology and neurosurgery teams with ophthalmologic consultation as required.

Clinical Recommendations

The significance of ocular findings in AHT has been the subject of great controversy. Previously identified[8] weaknesses in the evidence base were the lack of prospective, controlled studies, frequency of circular logic, and inconsistency in definition of cases. Currently, there is level II evidence from prospective controlled studies, supporting a significant relationship between IOH and AHT. Accurate classification of trauma as being from abuse remains difficult, because the trauma is generally unwitnessed and denied by perpetrators. Level I evidence is impossible to achieve in this field, for obvious reasons.

Of the reviewed studies,[6,7,12–15,17–19,21–25] the most sensitive ocular sign for AHT, on clinical examination, is IOH, which is found in approximately 77% of infants (A, II). Combined data from prospective studies of head injury indicate that IOH have a specificity of 94% for abuse[13,15,18,19] (A, II). The specificity of IOH for AHT is further increased when there is bilateral involvement, preretinal hemorrhages, premacular and peripheral involvement, and moderate to severe IOH[13,19] (A, II). Intraocular hemorrhages are not pathognomonic of AHT, and have infrequently been reported in other conditions including severe, accidental head injury[13,19,21,25,26,29] (A, II).

Most frequently, IOH involve the posterior pole in AHT,[16,17,24] although this is not consistent between studies[7] (B, III). The intraretinal layers are most frequently affected in all studies (B, II). Clinical studies[13–15,17,28] report preretinal hemorrhage as the next most common site (B, II), whereas autopsy studies[14,16,23,27] find vitreous and subretinal involvement to be more common (B, III).

Perimacular retinal folds and traumatic retinoschisis have a low sensitivity for AHT, seen overall in approximately 8% and 14% of cases, respectively[14–17,21,22,24,27,28] (A, III). They have a high specificity for AHT and have not been demonstrated in other conditions in controlled studies[15,21] (A, III). They are not, however, pathognomonic for AHT, and have been reported in infants after fatal MVC,[29] crush head injuries,[30–32] and a high altitude fall (A, III). Apart from their diagnostic significance, they are a poor prognostic indicator and have been associated with more severe neurologic injury and mortality[14,28] (A, III).

Optic nerve sheath hemorrhages are found overall in approximately 72% of cases of AHT at autopsy[6,7,21–23,25] (A, III). In contrast, controlled studies have found optic nerve sheath hemorrhages in up to 29% of accidental head injury.[6,25] The diagnostic accuracy for abuse is improved when multiple pathologies including IOH, optic nerve sheath hemorrhage and intracranial hemorrhage are present[26] (A, III).

References

1. Keenan HT, Runyan DK, Marshall SW, et al. A population-based study of inflicted traumatic brain injury in young children. JAMA 2003;290:621–6.
2. Kiffney GT. The eye of the “battered child”. Arch Ophthalmol 1964;72:231–3.
3. Gaynon MW, Koh K, Marmor MF, Frankel LR. Retinal folds in the shaken baby syndrome. Am J Ophthalmol 1988;106:423–5.
4. Greenwald MJ, Weiss A, Oesterle CS, Friendly DS. Traumatic retinoschisis in battered babies. Ophthalmology 1986;93:618–25.
5. Massicotte SJ, Folberg R, Torczynski E, et al. Vitreoretinal traction and perimacular retinal folds in the eyes of deliberately traumatized children. Ophthalmology 1991;98:1124–7.
6. Betz P, Puschel K, Miltner E, et al. Morphometrical analysis of retinal hemorrhages in the shaken baby syndrome. Forensic Sci Int 1996;78:71–80.
7. Green MA, Lieberman G, Milroy CM, Parsons MA. Ocular and cerebral trauma in non-accidental injury in infancy: underlying mechanisms and implications for paediatric practice. Br J Ophthalmol 1996;80:282–7.
8. Donohoe M. Evidence-based medicine and shaken baby syndrome: part I: literature review, 1966–1998. Am J Forensic Med Pathol 2003;24:239–42.
9. Togioka BM, Arnold MA, Bathurst MA, et al. Retinal hemorrhages and shaken baby syndrome: an evidence-based review. J Emerg Med 2009;37:98–106.
10. Whiting P, Rutjes AWS, Reitsma JB, et al. The development of QUADAS: a tool for the quality assessment of studies of diagnostic accuracy included in systematic reviews. BMC Med Res Methodol [serial online] 2003;3:25. Available at: http://www.biomedcentral.com/1471-2288/3/25. Accessed August 25, 2009.
11. Wilkinson WS, Han DP, Rappley MD, Owings CL. Retinal hemorrhage predicts neurologic injury in the shaken baby syndrome. Arch Ophthalmol 1989;107:1472–4.
12. Gilles EE, McGregor ML, Levy-Clarke G. Retinal hemorrhage asymmetry in inflicted head injury: a clue to pathogenesis? J Pediatr 2003;143:494–9.
13. Bechtel K, Stoessel K, Leventhal JM, et al. Characteristics that distinguish accidental from abusive injury in hospitalized young children with head trauma. Pediatrics 2004;114:165–8.
14. Kivlin JD, Simons KB, Lazoritz S, Ruttum MS. Shaken baby syndrome. Ophthalmology 2000;107:1246–54.
15. Pierre-Kahn V, Roche O, Dureau P, et al. Ophthalmologic findings in suspected child abuse victims with subdural hematomas. Ophthalmology 2003;110:1718–23.
16. Emerson MV, Jakobs E, Green WR. Ocular autopsy and histopathologic features of child abuse. Ophthalmology 2007;114:1384–94.
17. Morad Y, Kim YM, Armstrong DC, et al. Correlation between retinal abnormalities and intracranial abnormalities in the shaken baby syndrome. Am J Ophthalmol 2002;134:354–9.
18. Buys YM, Levin AV, Enzenauer RW, et al. Retinal findings after head trauma in infants and young children. Ophthalmology 1992;99:1718–23.
19. Vinchon M, Defoort-Dhellemmes S, Desurmont M, Dhellemmes P. Accidental and nonaccidental head injuries in infants: a prospective study. J Neurosurg 2005;102(suppl):380–4.
20. Binenbaum G, Mirza-George N, Christian CW, Forbes BJ. Odds of abuse associated with retinal hemorrhages in children suspected of child abuse. J AAPOS 2009;13:268–72.
21. 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.
22. 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.
23. Budenz DL, Farber MG, Mirchandani HG, et al. Ocular and optic nerve hemorrhages in abused infants with intracranial injuries. Ophthalmology 1994;101:559–65.
24. Marshall DH, Brownstein S, Dorey MW, et al. The spectrum of postmortem ocular findings in victims of shaken baby syndrome. Can J Ophthalmol 2001;36:377–83.
25. Wygnanski-Jaffe T, Levin AV, Shafiq A, et al. Postmortem orbital findings in shaken baby syndrome. Am J Ophthalmol 2006;142:233–40.
26. Riffenburgh RS. Ocular hemorrhage in autopsies of child abuse victims. Clin Surg Ophthalmol 2005;23:178–86.
27. Munger CE, Peiffer RL, Bouldin TW, et al. Ocular and associated neuropathologic observations in suspected whiplash

shaken infant syndrome: a retrospective study of 12 cases. Am J Forensic Med Pathol 1993;14:193–200.
28. Mills M. Funduscopic lesions associated with mortality in shaken baby syndrome. J AAPOS 1998;2:67–71.
29. Kivlin JD, Currie ML, Greenbaum VJ, et al. Retinal hemorrhages in children following fatal motor vehicle crashes: a case series. Arch Ophthalmol 2008;126:800–4.
30. Lantz PE, Sinal SH, Stanton CA, Weaver RG Jr. Perimacular retinal folds from childhood head trauma. BMJ 2004;328:754–6.
31. Lueder GT, Turner JW, Paschall R. Perimacular retinal folds simulating nonaccidental injury in an infant. Arch Ophthalmol 2006;124:1782–3.
32. Watts P, Obi E. Retinal folds and retinoschisis in accidental and non-accidental head injury. Eye 2008;22:1514–6.
33. Riffenburgh RS, Sathyavagiswaran L. Ocular findings at autopsy of child abuse victims. Ophthalmology 1991;98:1519–24.
34. Gnanaraj L, Gilliland MGF, Yahya RR, et al. Ocular manifestations of crush head injury in children. Eye 2007;21:5–10.
35. Levin AV. Retinal haemorrhages and child abuse. In: David TJ, ed. Recent Advances in Paediatrics. vol 17. London: Churchill-Livingstone, 1999:151–219.
36. Keithahn MA, Bennett SR, Cameron D, Mieler WF. Retinal folds in Terson syndrome. Ophthalmology 1993;100:1187–90.
37. Lambert SR, Johnson TE, Hoyt CS. Optic nerve sheath and retinal hemorrhages associated with the shaken baby syndrome. Arch Ophthalmol 1986;104:1509–12.
38. Ganesh A, Jenny C, Geyer J, et al. Retinal hemorrhages in type I osteogenesis imperfecta after minor trauma. Ophthalmology 2004;111:1428–31.
39. Nassogne M-C, Sharrard M, Hertz-Pannier L, et al. Massive subdural haematomas in Menkes disease mimicking shaken baby syndrome. Childs Nerv Syst 2002;18:729–31.
40. Kafil-Hussain NA, Monavari A, Bowell R, et al. Ocular findings in glutaric aciduria type 1. J Pediatr Ophthalmol Strabismus 2000;3:289–93.

Footnotes and Financial Disclosures

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[1] Faculty of Medicine, University of New South Wales, Randwick, NSW, Australia.

[2] Department of Ophthalmology, Sydney Children's Hospital, Randwick, NSW, Australia.

[3] Child Protection Unit, Sydney Children's Hospital, Randwick, NSW, Australia.

[4] Department of Ophthalmology, The Children's Hospital at Westmead, Westmead, NSW, Australia.

[5] Faculty of Medicine, University of Sydney, NSW, Australia.

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Correspondence:
Dr. Gaurav Bhardwaj, Department of Ophthalmology, 2nd floor, South Wing, Edmund Blacket Building, Prince of Wales Hospital, Randwick, NSW, 2031. E-mail: gauravb23@gmail.com.

APPENDIX 1

Search String

1 shaken baby syndrome.mp. or exp Shaken Baby Syndrome/

2 battered child syndrome.mp. or exp Battered Child Syndrome/

3 child abuse.mp. or exp Child Abuse/

4 craniocerebral trauma.mp. or exp Craniocerebral Trauma/

5 exp Whiplash Injuries/ or shaken impact syndrome.mp.

6 (inflicted adj3 brain adj3 injury).mp. [mp=title, original title, abstract, name of substance word, subject heading word]

7 (non-accidental adj2 injury).mp. [mp=title, original title, abstract, name of substance word, subject heading word]

8 abusive head trauma.mp. [mp=title, original title, abstract, name of substance word, subject heading word]

9 exp Retinal Hemorrhage/ or retinal h?emorrhage*.mp.

10 retinoschisis.mp. or exp Retinoschisis/

11 retinal fold.mp.

12 (optic nerve adj2 h?emorrhage*).mp. [mp=title, original title, abstract, name of substance word, subject heading word]

13 (1 or 2 or 3 or 4 or 5 or 6 or 7 or 8)

14 (9 or 10 or 11 or 12)

15 (13 and 14)

16 remove duplicates from 15

17 limit 16 to humans

APPENDIX 2

Explanation of grading items

This checklist is modified from the QUADAS[1] checklist for systematic reviews of diagnostic accuracy. Some items required substitution, since our aim was to assess the accuracy of ocular findings in the diagnosis of AHT, not the accuracy of ophthalmological examination (the index test) in detecting abnormalities. Each item was scored as "yes", "no" or "unclear". Studies which were more comprehensive with respect to retinal findings and had a lower risk of bias, scored "yes" for more items. An overall quality score was not calculated, as this can be unreliable due to the variable importance of each item.

1. Was the spectrum of patients representative?

Assessment of this item included the population and condition studied. Studies which examined infants or children with AHT in an inpatient setting were considered representative. Studies including adults, in which the data on children could not be extrapolated, were considered non-representative. Studies of child abuse, not exclusively with head injury, were graded "unclear".

2. Were selection criteria clearly described?

This item was scored "yes" if the study reported the institution(s) where recruitment took place and an adequate definition of the inclusion and exclusion criteria.

3. Were the cases of abuse confirmed?

Since there is no reference standard for the diagnosis of abuse, we assessed the studies' definition of what was classified as AHT, to consider whether they were likely to be true cases. Cases which were accompanied by confessions, witnessed by reliable third parties or confirmed in legal proceedings were considered to be true cases. Cases which were confirmed by multidisciplinary assessment using stated criteria were also considered likely to be true cases. This item was scored as "yes" if any of these conditions were met. Studies which stated the occurrence of AHT without detailing the method of determination were graded as "unclear". Studies with only suspected cases of AHT were scored "no" for this category.

4. Was the timeframe for examination acceptable?

For this review, we assessed the timeframe in which retinal examination was performed in relation to the injury. A time delay in retinal examination may render the results unreliable as a certain proportion of findings may have resolved. We considered retinal examination within five days of the injury as acceptable.

5. Was the inclusion of participants independent to the presence of ocular findings?

This extra item was assessed separately since the presence of retinal hemorrhages have been an inclusion criteria in some studies. While these types of studies illustrate patterns of retinal involvement in AHT, diagnostic accuracy of findings cannot be assessed.

6. Was it a controlled study?

This extra item was added to the checklist to reflect whether more than one cohort was assessed and compared i.e. whether it was a case series of AHT or a comparative study. If the control group was inappropriate, or not comparable, e.g. adults or adolescents compared with infants, this item was graded "unclear".

7. Was the diagnosis of abuse made independent of the ocular findings? (i.e. free from circular reasoning)

We assessed whether the definition of abuse in the study was based partly on retinal findings, i.e. the presence of circular logic. If insufficient details were provided, this item was graded "unclear".

8. Were sufficient details provided on the method of ophthalmological examination?

We verified whether details were provided on the experience of the examiner, use of mydriasis, and the method of examination i.e. direct or indirect ophthalmoscopy, fundus photography, autopsy. This item was scored "yes" if all of the former aspects were mentioned, or the autopsy protocol was sufficiently described. If some of the details were not mentioned, the grading was "unclear", and if no details were provided, it was graded "no".

9. Was the protocol used in determination of abusive injuries described in sufficient detail?

We verified whether the protocol used for determination of abusive injuries was detailed in the methods section in the way of text or a flow diagram, detailing specific criteria. Multidisciplinary assessment with no stated criteria was graded "unclear".

10. Was ophthalmological examination performed in a masked fashion?

It is possible that retinal examination may be more rigorous and well documented by an examiner in a case of suspected AHT. Therefore, we wished to assess whether examiners were masked to the mode of injury. This item was graded "unclear" if no details were provided.

11. Was the method of ophthalmological examination sufficiently sensitive to detect abnormalities?

This extra item assessed whether the method of examination was adequate to detect all possible positive findings. The method was considered sufficient if performed in mydriasis by an ophthalmologist. Autopsy examinations were also considered sufficiently sensitive. If insufficient details were available, this item was graded "unclear". If examination was performed by non-ophthalmologists in a significant number of cases, this item was scored "no".

12. Was ophthalmological examination performed consecutively and uniformly across the cohorts?

This extra item assessed whether the frequency of retinal examination was consecutive and uniform across all cohorts i.e. selection bias. For case series, we assessed whether retinal examination was performed consecutively. This item was graded "unclear" if this information was not mentioned.

13. Were further details included on the nature and distribution of ocular findings?

This item differentiated between studies which only reported the incidence of ocular findings from those which presented detailed findings. Studies which provided details which were not quantitative or only mentioned whether findings were bilateral or unilateral were graded "unclear".

14. Were withdrawals from the study explained?

We wished to assess, in prospective studies, whether withdrawals from the study were mentioned. Studies of a retrospective nature were graded as "unclear" since consent is usually not obtained from each individual case.

Substituted items

Did the whole sample or a random selection of the sample, receive verification using the reference standard? This item did not relate to AHT since there is no reference standard. Furthermore, in practice, extensive investigation to exclude AHT is only performed where there is an absence of a credible history, or other factors leading to doubt regarding the history.

Did patients receive the same reference standard regardless of the index test result?
Were the reference standard results interpreted without knowledge of the results of the index test? These two items were assessed by item 7.

Were the same clinical data available when test results were interpreted as would be available when the test is used in practice? This item does not relate to studies of AHT.

Were uninterpretable/intermediate test results reported? We regarded that results of retinal examination would always be interpretable and would never be intermediate i.e. retinal hemorrhages are either present or absent. We did not therefore assess this item in studies.

Reference

1. Whiting P, Rutjes AWS, Reitsma JB, et al. The development of QUADAS: a tool for the quality assessment of studies of diagnostic accuracy included in systematic reviews. BMC Med Res Methodol [serial online] 2003;3:25. Available at: http://www.biomedcentral.com/1471-2288/3/25. Accessed August 25, 2009.

APPENDIX 3

Included studies **Items for grading**

First Author	Year	Type	Cases of AHT	Controls	Max age (months)	ICH (%)	SDH (%)	IOH (%)	1	2	3	4	5	6	7	8	9	10	11	12	13	14
Bechtel[1]	2004	P	15	67 head injury	24	-	80	60	Y	Y	Y	U	Y	Y	Y	U	Y	U	Y	Y	Y	Y
Betz[2]	1996	A	7	42 other causes	9	100	100	100	U	N	Y	U	U	Y	U	Y	N	U	Y	U	Y	U
Binenbaum[3]	2009	R	49	61 other causes	15	-	-	51	Y	Y	Y	Y	Y	Y	Y	Y	Y	U	Y	N	U	U
Budenz[4]	1994	A	13	6 SIDS	36	-	92	85	Y	Y	U	U	Y	Y	U	Y	N	U	Y	U	Y	U
Buys[5]	1992	P	3	75 head injury	36	100	66	100	Y	Y	U	Y	Y	Y	U	Y	N	U	Y	N	Y	Y
Elner[6]	1990	A	10		48	60	60	70	U	Y	N	U	Y	N	U	Y	N	U	Y	Y	Y	U
Emerson[7]	2007	A	118†		108	53	-	47	U	Y	U	U	Y	N	U	Y	N	U	Y	Y	Y	U
Gilles[8]	2003	R	14		29	-	100	71	Y	Y	Y	Y	Y	N	Y	U	Y	Y	U	U	Y	U
Green[9]	1996	A	16		36	94	94	81	Y	Y	Y	U	Y	N	U	Y	U	U	Y	Y	Y	U
Kivlin[10]	2000	R	123		36	100	100*	83	Y	Y	Y	U	Y	N	Y	U	U	U	Y	Y	Y	U
Marshall[11]	2001	A	6		34	100	100	100	Y	Y	U	U	U	N	N	Y	N	U	Y	Y	Y	U
Mills[12]	1998	R	10		10	100	-	100	Y	Y	Y	Y	N	N	N	Y	Y	U	Y	Y	Y	U
Morad[13]	2002	R	75		48	-	93	85	Y	Y	U	U	Y	N	N	Y	Y	U	Y	Y	Y	U
Munger[14]	1993	A	12		21	92	83	100	Y	Y	U	U	N	N	U	Y	N	U	Y	Y	Y	U
Pierre-Kahn[15]	2003	P	224	7 head injury	29	100	100*	68	Y	Y	U	Y	Y	Y	Y	Y	N	U	Y	Y	Y	Y
Rao[16]	1988	A	14	16 other causes	48	64	43	63	U	Y	U	U	Y	Y	U	Y	N	U	Y	Y	Y	U
Riffenburgh[17]	2005	A	197†	396 other causes	-	-	-	47	U	U	U	U	Y	Y	U	Y	N	U	Y	U	U	U
Vinchon[18]	2005	P	57	93 head injury	24	-	81	75	Y	Y	Y	U	Y	Y	U	U	U	U	Y	Y	Y	Y
Wilkinson[19]	1989	R	14		28	-	-	100	Y	Y	U	Y	N	N	N	Y	N	Y	Y	Y	Y	U
Wygnanski-Jaffe[20]	2006	A	18	18 head injury	36	100	94	89	Y	Y	Y	U	Y	Y	N	Y	Y	U	Y	U	Y	U

Excluded studies **Items for grading**

First Author	Year	Type	Cases of AHT	Controls	Max age (months)	ICH (%)	SDH (%)	IOH (%)	1	2	3	4	5	6	7	8	9	10	11	12	13	14
DiScala[21]	2000	R	1997†	16831 trauma	48	43	-	28	U	Y	U	U	Y	Y	U	N	N	U	U	U	N	U
Eisenbrey[22]	1979	R	26	24 head injury	-	-	-	67	U	N	U	U	Y	Y	U	N	N	U	U	Y	N	U
Ewing-Cobbs[23]	1998	P	20	20 head injury	72	100	80	70	U	Y	Y	U	Y	Y	U	N	Y	U	U	U	N	Y
Frank[24]	1985	R	4		4	50	25	100	U	N	U	U	N	N	U	N	N	U	U	Y	U	U
Graupman[25]	2006	R	35		120	100	89	81	U	Y	U	U	Y	N	U	N	N	U	U	Y	N	U
Smith[26]	1974	P	134†	53 other causes	60	35	22	6	U	N	U	U	U	Y	U	N	N	U	U	U	N	U
Atwal[27]	1998	A	24		32	100	100	96	Y	Y	U	U	Y	N	U	N	N	U	U	Y	N	U
Barlow[28]	2005	P	25		34	-	84	64	Y	Y	Y	U	N	N	N	N	Y	U	U	U	N	Y
Billmire[29]	1985	R	30	54 head injury	12	64	-	30	Y	Y	Y	U	Y	Y	Y	N	U	U	U	U	N	U
Biousse[30]	2002	R	26		24	100	100	69	Y	Y	Y	U	Y	N	Y	N	Y	U	U	Y	N	U
Bonnier[31]	2003	R	23		13	96	-	87	Y	Y	Y	U	Y	N	N	N	Y	U	U	U	U	U
Cheah[32]	1994	R	41		48	100*	80	23	Y	Y	U	U	Y	N	U	N	N	U	U	U	N	U
Dashti[33]	1999	R	32	68 head injury	120	-	69	53	Y	Y	Y	U	Y	Y	Y	N	Y	U	U	U	N	U
Duhaime[34]	1992	P	24	76 head injury	24	54	54	38	Y	Y	Y	Y	Y	Y	Y	N	Y	U	N	Y	N	Y
Duhaime[35]	1987	R	48		24	100	-	81	Y	Y	Y	U	N	N	N	N	Y	U	U	U	N	U
Feldman[36]	2001	P	39	15 SDH	36	100	100*	72	Y	Y	Y	U	Y	Y	Y	N	Y	U	U	N	N	Y
Fujiwara[37]	2008	R	28	232 head injury	24	-	64	52	Y	Y	Y	U	Y	Y	Y	N	Y	U	U	N	U	U
Fung[38]	2002	R	4		24	100	100*	63	Y	Y	U	U	Y	N	U	N	N	U	U	U	N	U
Ghahreman[39]	2005	R	65		46	-	82	59	Y	Y	U	U	Y	N	U	N	N	U	U	U	N	U
Golden[40]	2004	R	39		5	100	100*	17	Y	Y	U	U	Y	N	Y	N	Y	U	U	N	N	U
Goldstein[41]	1993	P	11	29 other causes	43	-	57	57	Y	Y	Y	U	Y	U	U	N	N	U	N	U	N	Y
Hadley[42]	1989	R	13		14	100	100	100	Y	Y	Y	U	Y	N	U	N	N	U	U	U	N	U
Hoskote[43]	2002	R	14		7	100	100*	85	Y	Y	U	U	Y	N	U	N	N	U	U	U	N	U
Jacobi[44]	1986	R	62		30	-	-	52	Y	U	U	U	Y	N	U	N	N	U	U	U	U	U
Jayawant[45]	1998	R	27		24	100	100*	-	Y	Y	Y	U	Y	N	U	N	N	U	N	U	U	U
Keenan[46]	2004	P	80	72 head injury	24	100	94	76	Y	Y	U	U	Y	Y	U	N	N	U	U	N	N	U
King[47]	2003	R	364		58	98	86	76	Y	Y	U	U	N	N	N	N	N	U	U	U	N	U

Loh[48]	2002	R	11		12	100	100*	100	Y	Y	U	U	Y	N	N	N	N	U	U	U	N	U
Ludwig[49]	1984	R	20		15	-	50	60	Y	Y	U	U	U	N	U	N	N	U	U	U	U	U
Makaroff[50]	2005	P	11		6.5	100	100	73	Y	Y	U	U	Y	N	Y	N	N	U	U	Y	N	Y
McCabe[51]	2000	R	30		39	-	70	100	Y	Y	Y	U	N	N	N	N	U	U	U	Y	U	U
Reece[52]	2000	R	54	233 head injury	36	-	50	35	Y	Y	Y	U	Y	Y	Y	N	Y	U	U	U	N	U
Sun[53]	2006	R	15		60	100	100*	91	Y	Y	U	U	Y	N	U	N	N	U	U	N	N	U
Talvik[54]	2006	R/P	26		12	-	77	62	Y	U	U	U	N	N	U	N	N	U	U	Y	N	U
Tzioumi[55]	1998	R	21	17 SDH	24	100	100*	84	Y	Y	U	U	Y	Y	U	N	N	U	U	N	N	U

* SDH was an inclusion criterion

† Study included all forms of child abuse (not restricted to head injury)

- Not reported

A – Autopsy study

P – Prospective study

R – Retrospective study

AHT – abusive head trauma

ICH – intracranial hemorrhage

SDH – subdural hematoma

IOH – intraocular hemorrhage

Studies excluded prior to grading

First Author	Year	Reason for exclusion
Arlotti[56]	2008	Only included cases of unilateral IOH
Biron[57]	2007	Examined perpetrator accounts from cases with IOH. Not a consecutive series of AHT.
Biron[58]	2005	Examined cases of AHT for evidence of time delay before onset of symptoms. Not a consecutive series of AHT.
Cantani[59]	1990	Proportion of injuries related to abuse unknown.
Drack[60]	1999	Non-consecutive case series of unilateral IOH in AHT
Fishman[61]	1998	Non-consecutive series of AHT. Investigated the use of electroretinography in IOH
Geddes[62]	2001	Cohort comprised personal database of cases of two authors. Not a consecutive series.
Geddes[63]	2003	Compared dural findings from non-traumatic intrauterine and infant deaths with three cases of AHT which were not consecutive.
Giangiacomo[64]	1988	Investigated the role of sequential computed tomography in five infants with IOH. Diagnostic accuracy of IOH not assessed.
Gilliland[65]	1994	Patient recruitment based on voluntary contributions from participating pathologists.
Gleckman[66]	2000	Investigated the use of ß-amyloid precursor protein in detection of optic nerve diffuse axonal injury.
Greenes[67]	1998	Proportion of injuries related to abuse unknown.
Greenwald[68]	1986	First case series describing traumatic retinoschisis as a specific lesion. Diagnostic accuracy not assessable.
Han[69]	1990	Selected case reports regarding late ophthalmic manifestations of AHT.
Harcourt[70]	1971	Selected case reports based on referral to ophthalmology service.
Hendeles[71]	1985	Data on infant age group not able to be extrapolated. Significant proportion of cases older than four years.
Holloway[72]	1994	Frequency of IOH not reported.

Kapoor[73]	1997	Selected case reports of white centered IOH.
Laskey[74]	2004	Studied a subgroup of patients which were neurologically asymptomatic for the frequency of ophthalmology consults.
Leestma[75]	2005	Retrospective study of cases with a perpetrator confession of shaking. Not based on consecutive recruitment.
Luersson[76]	1991	Proportion related to child abuse not known.
Massicotte[77]	1986	Selected case reports of perimacular retinal folds with evidence of vitreoretinal traction.
Morad[78]	2003	Study of the accuracy of non-ophthalmologists in detecting IOH.
Morad[79]	2004	Recruitment based on contributions from an email based listserv.
Morad[80]	2004	Recruitment based on contributions from an email based listserv.
Morris[81]	2000	Only studied cases of SDH with no other injuries including IOH.
Mushin[82]	1971	Selected case reports of infants with IOH with visual compromise.
Naidoo[83]	2000	No quantitative data on ocular findings presented.
Riffenburgh[84]	1991	More recent publication[17], which includes the data from this study, was already included.
Rubin[85]	2003	Only studied a subgroup of neurologically asymptomatic patients.
Schmidt[86]	1997	Selected case reports of infants with IOH.
Tung[87]	2006	No data on IOH.
Vinchon[88]	2004	Subset of cases presented in another publication.

REFERENCES

1. Bechtel K, Stoessel K, Leventhal JM, et al. Characteristics that distinguish accidental from abusive injury in hospitalized young children with head trauma. Pediatrics 2004;114:165-8.
2. Betz P, Puschel K, Miltner E, et al. Morphometrical analysis of retinal hemorrhages in the shaken baby syndrome. Forensic Sci Int 1996;78:71-80.
3. Binenbaum G, Mirza-George N, Christian CW, Forbes BJ. Odds of abuse associated with retinal hemorrhages in children suspected of child abuse. J AAPOS 2009;13:268-72.
4. Budenz DL, Farber MG, Mirchandani HG, et al. Ocular and optic nerve hemorrhages in abused infants with intracranial injuries. Ophthalmology 1994;101:559-65.
5. Buys YM, Levin AV, Enzenauer RW, et al. Retinal findings after head trauma in infants and young children. Ophthalmology 1992; 99:1718-23.
6. Elner SG, Elner VM, Arnall M, et al. Ocular and associated systemic findings in suspected child abuse: A necropsy study. Arch Ophthalmol 1990;108:1094-101.
7. Emerson MV, Jakobs E, Green WR. Ocular autopsy and histopathologic features of child abuse. Ophthalmology 2007;114:1384-94.
8. Gilles EE, McGregor ML, Levy-Clarke G. Retinal hemorrhage asymmetry in inflicted head injury: A clue to pathogenesis? J Pediatr 2003;143:494-9.
9. Green MA, Lieberman G, Milroy CM, et al. Ocular and cerebral trauma in non-accidental injury in infancy: underlying mechanisms and implications for paediatric practice. Br J Ophthalmol 1996;80:282-7.
10. Kivlin JD, Simons KB, Lazoritz S, et al. Shaken baby syndrome. Ophthalmology 2000;107:1246-54.

11. Marshall DH, Brownstein S, Dorey MW, et al. The spectrum of postmortem ocular findings in victims of shaken baby syndrome. Can J Ophthalmol 2001;36:377-83.

12. Mills M. Funduscopic lesions associated with mortality in shaken baby syndrome. J AAPOS 1998;2:67-71.

13. Morad Y, Kim YM, Armstrong DC, et al. Correlation between retinal abnormalities and intracranial abnormalities in the shaken baby syndrome. Am J Ophthalmol 2002;134:354-9.

14. Munger CE, Peiffer RL, Bouldin TW, et al. Ocular and associated neuropathologic observations in suspected whiplash shaken infant syndrome. A retrospective study of 12 cases. Am J Forensic Med Pathol 1993;14:193-200.

15. Pierre-Kahn V, Roche O, Dureau P, et al. Ophthalmologic findings in suspected child abuse victims with subdural hematomas. Ophthalmology 2003;110:1718-23.

16. 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.

17. Riffenburgh RS. Ocular hemorrhage in autopsies of child abuse victims. Clinical and Surgical Ophthalmology 2005;23:178-86.

18. Vinchon M, Defoort-Dhellemmes S, Desurmont M, et al. Accidental and nonaccidental head injuries in infants: a prospective study. J Neurosurg 2005;102:380-4.

19. Wilkinson WS, Han DP, Rappley MD, et al. Retinal hemorrhage predicts neurologic injury in the shaken baby syndrome. Arch Ophthalmol 1989;107:1472-4.

20. Wygnanski-Jaffe T, Levin AV, Shafiq A, et al. Postmortem orbital findings in shaken baby syndrome. Am J Ophthalmol 2006;142:233-40.

21. DiScala C, Sege R, Li G, et al. Child abuse and unintentional injuries: a 10-year retrospective. Arch Pediatr Adolesc Med 2000;154:16-22.

22. Eisenbrey AB. Retinal hemorrhage in the battered child. Childs Brain 1979;5:40-4.

23. 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;102:300-7.

24. Frank Y, Zimmerman R, Leeds NM. Neurological manifestations in abused children who have been shaken. Dev Med Child Neurol 1985;27:312-6.

25. Graupman P, Winston KR. Nonaccidental head trauma as a cause of childhood death. J Neurosurg 2006;104:245-50.

26. Smith SM, Hanson R. 134 battered children: A medical and psychological study. Br Med J 1974;3:666-70.

27. Atwal GS, Rutty GN, Carter N, et al. Bruising in non-accidental head injured children; a retrospective study of the prevalence, distribution and pathological associations in 24 cases. Forensic Sci Int 1998;96:215-30.

28. Barlow KM, Thomson E, Johnson D, et al. Late neurologic and cognitive sequelae of inflicted traumatic brain injury in infancy. Pediatrics 2005;116:174-85.

29. Billmire ME, Myers PA. Serious head injury in infants: accident or abuse? Pediatrics 1985;75:340-2.

30. Biousse V, Suh DY, Newman NJ, et al. Diffusion-weighted magnetic resonance imaging in Shaken Baby Syndrome. Am J Ophthalmol 2002;133:249-55.

31. Bonnier C, Nassogne M-C, Saint-Martin C, et al. Neuroimaging of intraparenchymal lesions predicts outcome in shaken baby syndrome. Pediatrics 2003;112:808-14.

32. Cheah IGS, Kasim MS, Shafie HM, et al. Intracranial haemorrhage and child abuse. Ann Trop Paediatr 1994;14:325-8.

33. Dashti SR, Decker DD, Razzaq A, et al. Current patterns of inflicted head injury in children. Pediatr Neurosurg 1999;31:302-6.

34. 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.

35. Duhaime AC, Gennarelli TA, Thibault LE, et al. The shaken baby syndrome. A clinical, pathological, and biomechanical study. J Neurosurg 1987;66:409-15.

36. Feldman KW, Bethel R, Shugerman RP, et al. The cause of infant and toddler subdural hemorrhage: A prospective study. Pediatrics 2001;108:636-46.

37. Fujiwara T, Okuyama M, Miyasaka M. Characteristics that distinguish abusive from nonabusive head trauma among young children who underwent head computed tomography in Japan. Pediatrics 2008;122:841-7.

38. Fung ELW, Sung RYT, Nelson EAS, et al. Unexplained subdural hematoma in young children: is it always child abuse? Pediatr Int 2002;44:37-42.

39. Ghahreman A, Bhasin V, Chaseling R, et al. Nonaccidental head injuries in children: a Sydney experience. J Neurosurg 2005;103:213-8.

40. Golden N, Maliawan S. Clinical analysis of non-accidental head injury in infants. J Clin Neurosci 2005;12:235-9.

41. Goldstein B, Kelly MM, Bruton D, et al. Inflicted versus accidental head injury in critically injured children. Crit Care Med 1993;21:1328-32.

42. Hadley MN, Sonntag VK, Rekate HL, et al. The infant whiplash-shake injury syndrome: a clinical and pathological study. Neurosurgery 1989;24:536-40.

43. Hoskote A, Richards P, Anslow P, et al. Subdural haematoma and non-accidental head injury in children. Childs Nerv Syst 2002;18:311-7.

44. Jacobi G. Damage patterns in severe child abuse with and without fatal sequelae. Monatsschr Kinderheilkd 1986;134:307-15.

45. Jayawant S, Parr J. Outcome following subdural haemorrhages in infancy. Arch Dis Child 2007;92:343-7.

46. Keenan HT, Runyan DK, Marshall SW, et al. A population-based comparison of clinical and outcome characteristics of young children with serious inflicted and noninflicted traumatic brain injury. Pediatrics 2004;114:633-9.

47. King WJ, MacKay M, Sirnick A, Canadian Shaken Baby Study G. Shaken baby syndrome in Canada: clinical characteristics and outcomes of hospital cases. CMAJ 2003;168:155-9.

48. Loh J-K, Lin C-L, Kwan A-L, et al. Acute subdural hematoma in infancy. Surg Neurol 2002;58:218-24.

49. Ludwig S, Warman M. Shaken baby syndrome: a review of 20 cases. Ann Emerg Med 1984;13:104-7.

50. Makoroff KL, Cecil KM, Care M, et al. Elevated lactate as an early marker of brain injury in inflicted traumatic brain injury. Pediatr Radiol 2005;35:668-76.

51. McCabe CF, Donahue SP. Prognostic indicators for vision and mortality in shaken baby syndrome. Arch Ophthalmol 2000;118:373-7.

52. Reece RM, Sege R. Childhood head injuries: accidental or inflicted? Arch Pediatr Adolesc Med 2000;154:11-5.

53. Sun DTF, Zhu XL, Poon WS. Non-accidental subdural haemorrhage in Hong Kong: incidence, clinical features, management and outcome. Childs Nerv Syst 2006;22:593-8.

54. Talvik I, Metsvaht T, Leito K, et al. Inflicted traumatic brain injury (ITBI) or shaken baby syndrome (SBS) in Estonia. Acta Paediatrica 2006;95:799-804.

55. Tzioumi D, Oates RK. Subdural hematomas in children under 2 years. Accidental or inflicted? A 10-year experience. Child Abuse Negl 1998;22:1105-12.

56. Arlotti SA, Forbes BJ, Dias MS, et al. Unilateral retinal hemorrhages in shaken baby syndrome. J AAPOS 2007;11:175-8.

57. Biron D, Shelton D. Functional time limit and onset of symptoms in infant abusive head trauma. J Paediatr Child Health 2007;43:60-5.

58. Biron D, Shelton D. Perpetrator accounts in infant abusive head trauma brought about by a shaking event. Child Abuse Negl 2005;29:1347-58.

59. Cantani A, Bamonte G. Epidemiology of ocular complications of childhood head trauma. Pediatr Emerg Care 1990;6:271-4.

60. Drack AV, Petronio J, Capone A. Unilateral retinal hemorrhages in documented cases of child abuse. Am J Ophthalmol 1999;128:340-4.

61. Fishman CD, Dasher WB, 3rd, Lambert SR. Electroretinographic findings in infants with the shaken baby syndrome. J Pediatr Ophthalmol Strabismus 1998;35:22-6.

62. Geddes JF, Hackshaw AK, Vowles GH, et al. Neuropathology of inflicted head injury in children. I. Patterns of brain damage. Brain 2001;124:1290-8.

63. Geddes JF, Tasker RC, Hackshaw AK, et al. Dural haemorrhage in non-traumatic infant deaths: does it explain the bleeding in 'shaken baby syndrome'? Neuropathol Appl Neurobiol 2003;29:14-22.

64. Giangiacomo J, Khan JA, Levine C, et al. Sequential cranial computed tomography in infants with retinal hemorrhages. Ophthalmology 1988;95:295-9.

65. Gilliland MG, Luckenbach MW. Are retinal hemorrhages found after resuscitation attempts? A study of the eyes of 169 children. Am J Forensic Med Pathol 1993;14:187-92.

66. Gleckman AM, Evans RJ, Bell MD, et al. Optic nerve damage in shaken baby syndrome: detection by beta-amyloid precursor protein immunohistochemistry. Arch Pathol Lab Med 2000;124:251-6.

67. Greenes DS, Schutzman SA. Occult intracranial injury in infants. Ann Emerg Med 1998;32:680-6.

68. Greenwald MJ, Weiss A, Oesterle CS, et al. Traumatic retinoschisis in battered babies. Ophthalmology 1986;93:618-25.

69. Han DP, Wilkinson WS. Late ophthalmic manifestations of the shaken baby syndrome. J Pediatr Ophthalmol Strabismus 1990;27:299-303.

70. Harcourt B, Hopkins D. Ophthalmic manifestations of the battered-baby syndrome. Br Med J 1971;3:398-401.

71. Hendeles S, Barber K, Willshaw HE. The risk of ocular involvement in non-accidental injury. Child Care Health Dev1985;11:345-8.

72. Holloway M, Bye AM, Moran K. Non-accidental head injury in children. Med J Aust1994;160:786-9.

73. Kapoor S, Schiffman J, Tang R, et al. The significance of white-centered retinal hemorrhages in the shaken baby syndrome. Pediatr Emerg Care1997;13:183-5.

74. Laskey AL, Holsti M, Runyan DK, et al. Occult head trauma in young suspected victims of physical abuse. J Pediatr 2004;144:719-22.

75. Leestma JE. Case analysis of brain-injured admittedly shaken infants: 54 cases, 1969-2001. Am J Forensic Med Pathol 2005;26:199-212.

76. Luersson TG, Huang JC, McLone DG, et al. Retinal hemorrhages, seizures, and intracranial hemorrhages: Relationships and outcomes in children suffereing traumatic brain injury. Concepts Pediatr Neurosurg 1991;11:87-94.

77. Massicotte SJ, Folberg R, Torczynski E, et al. Vitreoretinal traction and perimacular retinal folds in the eyes of deliberately traumatized children. Ophthalmology 1991;98:1124-7.

78. Morad Y, Kim YM, Mian M, et al. Nonophthalmologist accuracy in diagnosing retinal hemorrhages in the shaken baby syndrome. J Pediatr 2003;142:431-4.

79. Morad Y, Avni I, Benton SA, et al. Normal computerized tomography of brain in children with shaken baby syndrome. J AAPOS 2004;8:445-50.

80. Morad Y, Avni I, Capra L, et al. Shaken baby syndrome without intracranial hemorrhage on initial computed tomography. J AAPOS 2004;8:521-7.

81. Morris MW, Smith S, Cressman J, et al. Evaluation of infants with subdural hematoma who lack external evidence of abuse. Pediatrics 2000;105:549-53.

82. Mushin AS. Ocular damage in the battered-baby syndrome. Br Med J 1971;3:402-4.

83. Naidoo S. A profile of the oro-facial injuries in child physical abuse at a children's hospital. Ch Abuse Negl 2000;24:521-34.

84. Riffenburgh RS, Sathyavagiswaran L. Ocular findings at autopsy of child abuse victims. Ophthalmology 1991;98:1519-24.

85. Rubin DM, Christian CW, Bilaniuk LT, et al. Occult head injury in high-risk abused children. Pediatrics 2003;111:1382-6.

86. Schmidt US, Mittelviefhaus K, Hansen LL. Retinal hemorrhage in the infant as an indication of shaken baby trauma. Klin Monatsbl Augenheilkd 1997;211:354-8.

87. Tung GA, Kumar M, Richardson RC, et al. Comparison of accidental and nonaccidental traumatic head injury in children on noncontrast computed tomography. Pediatrics 2006;118:626-33.

88. Vinchon M, Defoort-Dhellemmes S, Noule N, et al. Accidental or non-accidental brain injury in infants: Prospective study of 88 cases. Presse Medicale 2004;33:1174-9.