

Outcomes of infants and children with inflicted traumatic brain injury

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Inflicted traumatic brain injury (ITBI) or shaken baby syndrome is recognized as a major cause of disability and death in the pediatric population. Although advances have been made in the recognition of the clinical, radiographic, and pathological findings of ITBI, less is known about the long-term outcomes of survivors. Health care providers recognize that these infants and children frequently have poor outcomes. Although an infant or child who sustains an ITBI may look well immediately after the trauma, that child may be left with serious and permanent disabilities.

Here we review articles published since 1975 that discuss the outcomes of infants and children with ITBI. Only articles that had more specific outcome information than just survival were included in the survey. To find the articles, the Medline bibliographic database was searched with the following search terms: shaken baby syndrome, head injury, traumatic brain injury, impact injury, shaking impact syndrome, non-accidental injury, child abuse, inflicted injury, follow-up, outcome, neurological outcome, and vision. A bibliography search of articles was also used.

Data extracted from the articles included: the number of patients studied, the patient's age at injury, the interval to outcome measurement (or age of patient at outcome measurement), the definition and qualification of injury, the outcome measures, and patient outcomes. Patients were categorized into outcome groups when possible (Table I), but ophthalmologic outcomes were not categorized into groups (Table II). Outcome groups include no deficits, mild deficits, moderate deficits, severe deficits, and death. Mild deficits include minor physical, cognitive, or behavioral problems that are still compatible with a normal life. Moderate deficits include more disabling physical, cognitive, or behavioral problems. A severe deficit includes moderate deficits that would require assistance for activities of daily living, and includes patients in a vegetative state. The articles are summarized in Tables I and II.

Overview of acute injuries

The precise type of inflicted trauma is seldom known because

accurate histories are rarely provided, or might be deliberately misleading in cases of abusive head injury. However, there is a constellation of clinical, radiological, and pathological findings associated with ITBI that can help in making the diagnosis. The symptoms and physical signs may include those of head injury and/or increased intracranial pressure: seizures, apnea, decreased level of consciousness, and neurological abnormalities. Often, however, there are only mild neurological signs such as fussiness, vomiting in the absence of diarrhea, or a history of lethargy. Although not always present, bruising of the head or face in an infant or non-mobile child is associated with abusive head injury.¹ Retinal hemorrhages are present in up to 80% of cases; they can be unilateral or bilateral.² Retinal hemorrhages infrequently occur from accidental and non-traumatic events. When retinal hemorrhages do occur after accidental injury they are usually few and confined to the posterior pole, which often differs from the extent of hemorrhages seen with ITBI.² Other ocular findings associated with ITBI include retinal folds or detachments and other internal eye injuries.²

ITBI is usually associated with radiological findings. Subdural and subarachnoid hemorrhages are frequently noted, while skull and other fractures of the skeletal system are seen less frequently. Shaking while holding or squeezing the thorax can result in rib fractures and/or metaphyseal fractures. Parenchymal brain lesions such as shearing injuries, axonal injury, and hypoxic-ischemic changes may be present but are challenging to identify with standard imaging techniques.

Autopsy may reveal brain parenchymal lesions in cases of fatal inflicted brain injury that were not visible radiographically. The autopsy may also show cerebral tissue tears, contusions, subgaleal hemorrhage, and other signs of head impact not noted clinically. Axonal disruptions can be discovered on autopsy, but special stains or immunohistology techniques are often necessary to demonstrate these disruptions. Localized or generalized brain swelling is frequently noted as well. Some infants and children who suffer fatal inflicted brain injuries also have damage of the cervical spinal cord; evidence of this injury is sometimes visible only on post-mortem specimens.³ Acute

skeletal fractures are sometimes not evident radiographically but are picked up on autopsy.

Overview of outcomes

The majority of patients with inflicted head injuries have poor outcomes. In reviewed articles from the past 27 years, almost one-fifth of the patients died, and about half were left with

some impairment. Only 22% of patients had no impairments (see Tables I and II). Most of the surviving children with inflicted head injury were left with impairments in their motor and cognitive abilities, language, vision, and behavior. These impairments affect a child's capacity to interact with the environment, which in turn might contribute to later problems with education and social attainment.

Table I: Outcomes of infants and children with inflicted traumatic brain injury

<i>Reference</i>	<i>n</i>	<i>Age at injury</i>	<i>Interval to [age at] outcome</i>	<i>Definition of injury</i>
4	28	25 pts <6mo; 3 pts >1y	N/A	'Confirmed diagnosis' of non-accidental head injury
5	17	1–20mo; mean: 5.1mo	3–122mo; mean: 33mo	'Non-accidental head injury': head injury plus two of the following: retinal hemorrhages, skeletal fractures, soft tissue injury, inconsistent history
7	28	2–42mo; mean: 9.3mo	3mo	'Inflicted traumatic brain injury': algorithm similar to Duhaime et al. ²⁶
8	20	mean: 10.6mo	mean: 1.3mo	'Inflicted traumatic brain injury': algorithm similar to Duhaime et al. ²⁶
9	14	53d–4.5y; mean: 12.2mo	[4–34mo; mean: 17.4mo] N/A	'Confirmed non-accidental head injury': witnessed event, perpetrator confession, felony conviction or physical evidence of injury without trauma history
10	13	3wk–21mo	4–14y; mean: 7.2y	'Whiplash shaken infant syndrome': intraocular and intracranial hemorrhages in absence of traumatic or non-traumatic mechanism
11	123	99 pts: 0–12mo; 24 pts: 13–36mo	1mo–5y; mean: 26mo	'Shaken baby syndrome': subdural hematomas, skeletal injuries, no history
12	14	2–28mo; mean: 10.5mo	12–56mo	'Shaken baby syndrome': intraocular hemorrhages, intracranial hemorrhages, absence of external signs of head trauma
14	14	1mo–2y; mean: 6.4mo	[5.5–15.5y; mean: 9y]	'Shaken-impact syndrome': extracranial bleed in absence of other diagnosis and other supportive findings
15	4	5mo, 7mo, 9mo, 3mo; mean: 6mo	N/A	'Non-accidental injury': children under two years of age with an unexplained subdural hematoma and determined to be child abuse by case conference

Pt, patient.

The reasons for the poor outcomes in infants and children who sustain ITBI are not known. Some authors have shown that infants and children who suffer non-accidental head injuries have evidence of ischemia and hypoxia, as measured by blood pressure, arterial hypoxia,⁴ and cerebral perfusion pressure.⁵ These authors postulate that early brain hypoxia–ischemia is an important cause of poor outcomes in this

group. Another explanation involves secondary neuronal injury from a metabolic insult. The initial trauma (i.e. shaking) is the initiating event that causes a metabolic cascade of neurotoxic factors which continue to damage neurons once the trauma has ceased. Other investigators have shown that cervical spine injury may influence the degree of injury. Feldman and colleagues demonstrated that four of five children with fatal abusive head injury had extra-axial bleeds at the level of the cervical spinal cord on autopsy.³ This paper and earlier work predicted that cervical spine injury might have a role in the outcomes of these children.^{3,6} Although the mechanisms are not fully understood, it is well recognized by clinicians that most infants and children who sustain ITBI do not do well after the injury.

Table I: continued

<i>Outcome measures</i>	<i>Outcomes/comments</i>
Neurological status	9 pts had no deficits 7 pts had moderate deficits 8 pts had severe deficits 4 pts died
Six-point outcome scale	7 pts had no deficits 2 pts had mild deficits 3 pts had moderate deficits 3 pts had severe deficits 2 pts died
Glasgow Outcome Scale Bayley-II Bayley Behavior Scale	Pts in inflicted group scored lower in all domains than a control group
Glasgow Outcome Scale Bayley-II Stanford-Binet McCarthy	4 pts had no deficits 13 pts had moderate deficits 3 pts had severe deficits
Denver II	2 pts had mild deficits
Neurological examinations	3 pts had moderate deficits
Vision screening	6 pts had severe deficits 3 pts died
<i>Assessments:</i>	1 pt had no deficits
General	11 pts had moderate to severe deficits
Neurological	1 pt died
Psychological	
Social	
<i>Testing:</i>	
Gesell	
WISC	
Terman	
Neurological status	22 pts had no deficits 8 pts had a mild deficit 26 pts had a severe deficit 36 pts died for 31 pts there was no information
Neurological examination	1 pt had no deficits
Late neurological score	2 pts had mild deficits 5 pts had moderate to severe deficits 2 pts died 4 pts were unavailable for follow-up
Grades similar to Glasgow Outcome Scale	2 pts had no deficits 5 pts had moderate deficits 6 pts had a severe deficits 1 pt died
Neurological status	1 pt had mild deficits 1 pt had moderate deficits 2 pts had severe deficits

Developmental and behavioral outcomes

Few studies have systematically studied the effect of ITBI on developmental and behavioral outcomes (Table I). Ewing-Cobbs and colleagues⁷ performed developmental measures on infants and children after ITBI at an average of 1.3 months after the injury: 45% of the patients scored in the mentally deficient range for cognitive testing (group mean: 78.2) and 25% of the patients scored in the mentally deficient range for motor testing (group mean: 80.3).

In a second study, Ewing-Cobbs and colleagues⁸ evaluated infants and young children with ITBI at an average of 4.6 months after injury. Mean scores for both the cognitive and motor domains were in the low average range. Almost half of the infants and young children also had impaired scores for emotional regulation and motor quality. Other studies have demonstrated that most children with non-accidental head injury show developmental delays and mental retardation*.^{9,10} Impairments such as learning disabilities and behavior problems might not manifest until a child attends school, and so continuing developmental evaluations are important to ensure that children with ‘late’ sequelae from traumatic brain injury are recognized.

Visual outcomes

Visual impairment and blindness are documented problems after ITBI (Table II). In one large study, over 70% of patients were reported to see well, as measured by visual acuity and visual fields testing, at their last follow-up visit; 25% of patients had some visual impairment, as measured by visual acuity and visual fields testing.¹¹

Cortical injury, and not retinal hemorrhage, is thought to be the most significant cause of long-term visual impairment and blindness after ITBI. Studies that examine the visual outcomes of infants and children who sustain ITBI show a variety of causes for the impairment, including occipital lobe injury, occipital lobe atrophy, optic atrophy, retinal fibrosis, and retinal scarring. Visual impairments can also contribute to developmental deficits, learning problems, and social attainment.^{11–13}

Age at time of injury and outcomes

In the literature for the past 27 years that we reviewed, the mean age of all patients at time of injury (in studies in which the age at time of injury was reported) was 6.2 months (SD 2.7). Mean age of the patients at time of injury who died as a result of their injuries was 7.7 months (SD 7.2). Mean age of patients at

*UK usage: learning disability.

time of injury with no impairment was 5.2 months (SD 3.2) and the mean ages of patients at time of injury with mild and moderate to severe impairments were 6.5 months (SD 2.8) and 5.9 months (SD 8.0) respectively (see Tables I and II). There was

no statistical difference in age at the time of injury among these outcome groups. Although no difference in age was identified in this review sample, future investigators should be alert to the possibility that age at injury might influence outcomes.

Table I: continued

<i>Reference</i>	<i>n</i>	<i>Age at injury</i>	<i>Interval to [age at] outcome</i>	<i>Definition of injury</i>
16	15	1–30mo; mean: 5.6mo	3mo–3y; mean: 14.8mo	‘Non-accidental head injury’: clinical, radiological and/or ophthalmologic evidence or if inconsistent history
17	38	15d–34mo; mean: 6.4mo	N/A	‘Shaken baby syndrome’: all presented with neurological or cardiorespiratory symptoms
18	5	3–8mo; mean: 5.5mo	N/A	‘Confirmed shaken baby syndrome’: intraocular hemorrhages, subdural or subarachnoid hemorrhages with a history of suspected abuse
19	10	1–12mo; mean: 4.1mo	[8–15y; mean: 10.1y]	‘Shaken baby syndrome’: intracranial and retinal hemorrhages in absence of head trauma
20	6	3wk–4mo; mean: 10.6wk	4mo–1y; mean: 6.4mo	‘Non-accidental head injury’: (also had cortical tears by ultrasound)
21	17	1–20mo; mean: 4.6mo	N/A	‘Whiplash shaken infant syndrome’: history of shaking or intracranial and intraocular hemorrhages in the absence of signs of external trauma
22	4	3mo, 5wk, 6wk, 4mo	N/A, N/A, 5mo, [10mo]	‘Shaking injuries’: neurological and ophthalmologic abnormalities
23	20	1–15mo; mean: 5.8mo	N/A	‘Shaken baby syndrome’: perpetrator confession or no other diagnosis for findings
24	3	3mo, 3.5mo, 7mo	[7y, 5.2y, 4.2y]	‘Brain damage’: by shaking, swinging, hitting, and throwing

Pt, patient.

Table II: Ophthalmologic outcomes of infants and children with inflicted traumatic brain injury

<i>Reference</i>	<i>n</i>	<i>Age at injury</i>	<i>Interval to outcome</i>	<i>Definition of injury</i>
11	123	99 pts: 0–12mo; 24 pts: 13–36mo	1mo–7y; mean: 21mo	‘Shaken baby syndrome’: subdural hematomas, skeletal injuries, no history
13	30	1–39mo; mean: 9.3mo	1–36mo; mean: 6.7mo	‘Shaken baby syndrome’: bilateral retinal hemorrhages, inconsistent history, other signs of abuse or previous suspicious episode of abuse
25	6	mean: 9.1mo	12–55mo; mean: 29.8mo	‘Shaken baby syndrome’: retinal, vitreous or subhyaloid hemorrhage with intracranial injury and without external signs of head trauma

Pt, patient.

Some investigators have demonstrated that age less than or equal to 6 months is an indicator of poor prognosis.^{12,14}

The mean interval to the time of outcome measures (in studies in which interval to the time of outcome measures

was reported) was 15 months (SD 13.6). Some deficits might not have been apparent at that time because some learning disabilities and behavior problems might not manifest until a child is older and is attending school.

Table I: continued

<i>Outcome measures</i>	<i>Outcomes/comments</i>
Neurological status	1 pt had no deficits
Developmental status	1 pt had mild deficits
Behavioral status	4 pts had moderate deficits 7 pts had severe deficits 2 pts died
Neurological status	14 pts had no deficits
Visual status	4 pts had mild deficits 6 pts had severe deficits 12 pts died for 2 pts there was no information
Neurological examinations	2 pts had no deficits 2 pts had moderate to severe deficits 1 pt had severe deficits
Neurological status	3 pts had no deficits
Cognitive status	7 pts had some deficits
Behavioral status	
Neurological assessment	5 pts had moderate to severe deficits
Developmental assessment	1 pt died
Developmental evaluations	1 pt had no deficits
Psychological testing	3 pts had mild deficits 4 pts had moderate deficits 7 pts had severe deficits 2 pts died
Assessment of neurol. status	4 pts had severe deficits
Neurological status	7 pts had no deficits
Developmental status	10 pts had moderate to severe deficits 3 pts died
Neurological status	3 pts had severe deficits
Developmental status	

Table II: continued

<i>Outcome measures</i>	<i>Outcomes/comments</i>
Ophthalmologic examination	49 pts had 'good' vision 19 pts had 'poor' vision (only 68 pt records could be assessed)
Ophthalmologic examination	17 pts had a good visual outcome 5 pts were left with poor vision 8 pts died
Ophthalmologic examination	1 pt had a minor visual deficit 2 pts had moderate visual loss in one eye 3 pts had profound visual loss

Conclusions

Because the outcomes of many children who have sustained ITBI are poor, efforts should be directed at improving the identification of the specific deficits and improving outcomes. Although research has been directed toward the rehabilitation of children with traumatic brain injury, more investigation is needed in the specific rehabilitation of infants and children with ITBI, because the type of injury sustained by these two groups and, therefore, their outcomes, might differ. Neurological, behavioral, and developmental impairments can be seen after ITBI even when there is apparent early, good recovery. Because of this, developmental assessments of these children should be continued throughout the school years.

Finally, given the high incidence of death and disabilities that follows an ITBI, the negative psychosocial effects on the children, and the high health care costs required for diagnosis and treatment, prevention programs should be increased. Research into prevention should be encouraged, and parents, future parents, and caregivers must be educated about the dangers of shaking infants and children.

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References

- Sugar NF, Taylor JA, Feldman KW. (1999) Bruises in infants and toddlers: those who don't cruise rarely bruise. *Arch Pediatr Adolesc Med* **153**: 399–403.
- Levin AV. (2000) Retinal hemorrhages and child abuse. In: David TJ, editor. *Recent Advances in Pediatrics*. London: Churchill Livingstone. p 151–219.
- Feldman KW, Weinberger E, Milstein J, Fliener CL. (1997) Cervical spine MRI in abused infants. *Child Abuse Neglect* **21**: 199–205.
- Johnson DL, Boal D, Baule R. (1995) Role of apnea in nonaccidental head injury. *Pediatr Neurosurg* **23**: 305–10.
- Barlow KM, Minns RA. (1999) The relationship between intracranial pressure and outcome in non-accidental head injury. *Dev Med Child Neurol* **4**: 220–5.
- Hadley M, Sonntag V, Reke H, Murphy A. (1989) The infant whiplash-shake injury syndrome: a clinical and pathological study. *Neurosurgery* **24**: 536–9.
- Ewing-Cobbs L, Kramer L, Prasad M, Canales DN, Louis PT, Fletcher JM, Vollero H, Landry SH, Cheung K. (1998) Neuroimaging, physical, and developmental findings after inflicted and noninflicted traumatic brain injury in young children. *Pediatrics* **102**: 300–7.
- Ewing-Cobbs L, Prasad M, Kramer L, Landry S. (1999) Inflicted traumatic brain injury: relationship of developmental outcome to severity of injury. *Pediatr Neurosurg* **31**: 251–8.
- Gilles EE, Nelson MD. (1998) Cerebral complications of nonaccidental head injury in childhood. *Pediatr Neurol* **19**: 119–28.
- Bonnier C, Nassogne MC, Evrard P. (1995) Outcome and prognosis of whiplash shaken infant syndrome: late consequences after a symptom-free interval. *Dev Med Child Neurol* **37**: 943–56.
- Kivlin JD, Simons KB, Lazowitz S, Ruttum MS. (2000) Shaken baby syndrome. *Ophthalmology* **107**: 1246–54.
- Wilkerson WS, Han DP, Rappley MD, Owings CL. (1989) Retinal hemorrhage predicts neurologic injury in the shaken baby syndrome. *Arch Ophthalmol* **107**: 1472–4.
- McCabe F, Donahue SP. (2000) Prognostic indicators for vision and mortality in shaken baby syndrome. *Arch Ophthalmol* **118**: 373–7.

14. Duhaime AC, Christian C, Moss E, Seidl T. (1996) Long-term outcome in infants with the shaking-impact syndrome. *Pediatr Neurosurg* **24**: 292-8.
15. Fung ELW, Sung RYT, Nelson EAS, Poon WS. (2002) Unexplained subdural hematoma in young children: is it always child abuse? *Pediatr Int* **44**: 37-42.
16. Haviland J, Russel RI. (1997) Outcome after severe non-accidental head injury. *Arch Dis Child* **77**: 504-7.
17. Swenson J, Levitt C. (1997) Shaken baby syndrome diagnosis and prevention. *Minn Med* **80**: 41-4.
18. Matthews GP, Das A. (1996) Dense vitreous hemorrhages predict poor visual and neurological prognosis in infants with shaken baby syndrome. *J Pediatr Ophthalmol Strabismus* **33**: 260-5.
19. Fischer H, Allasio D. (1994) Permanently damaged: long-term follow-up of shaken babies. *Pediatrics* **33**: 696-8.
20. Jaspan T, Narborough G, Punt JAG, Lowe J. (1992) Cerebral contusional tears as a marker of child abuse-detection by cranial sonography. *Pediatr Radiol* **22**: 237-45.
21. Sinal SH, Ball MR. (1987) Head trauma due to child abuse: serial computerized tomography in diagnosis and management. *South Med J* **80**: 1505-12.
22. Frank Y, Zimmerman R, Leeds NMD. (1985) Neurological manifestations in abused children who have been shaken. *Dev Med Child Neurol* **27**: 312-6.
23. Ludwig S, Warman M. (1984) Shaken baby syndrome: a review of 20 cases. *Ann Emerg Med* **13**: 105-7.
24. Oliver JE. (1975) Microcephaly following baby battering and shaking. *BMJ* **2**: 262-4.
25. Han DP, Wilkinson WS. (1990) Late ophthalmic manifestations of the shaken baby syndrome. *J Pediatr Ophthalmol Strabismus* **27**: 299-303.
26. Duhaime AC, Alario AJ, Lewander WJ, Schut L, Sutton LN, Seidl TS, Nudelman S, Budnez D, Hertle R, Tsiaras W, Loporchio S. (1992) Head injury in very young children: mechanisms, injury types, and ophthalmologic findings in 100 hospitalized patients younger than 2 years of age. *Pediatrics* **90**: 179-85.

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