

Neuroimaging, Physical, and Developmental Findings After Inflicted and Noninflicted Traumatic Brain Injury in Young Children

Linda Ewing-Cobbs, PhD*; Larry Kramer, MD†; Mary Prasad, PhD*; Denise Niles Canales, MA*; Penelope T. Louis, MD§; Jack M. Fletcher, PhD*; Hilda Vollero, MD*; Susan H. Landry, PhD*; and Kim Cheung, MD, PhD*

ABSTRACT. *Objective.* To characterize neuroimaging, physical, neurobehavioral, and developmental findings in children with inflicted and noninflicted traumatic brain injury (TBI) and to identify characteristic features of inflicted TBI.

Methods and Patients. Forty children, 0 to 6 years of age, hospitalized for TBI who had no documented history of previous brain injury were enrolled in a prospective longitudinal study. TBI was categorized as either inflicted ($n = 20$) or noninflicted ($n = 20$) based on the assessment of hospital and county protective services. Glasgow Coma Scale scores and neonatal history were comparable in both groups.

Outcome Measures. Acute computed tomography/magnetic resonance imaging studies and physical findings were evaluated. Glasgow Outcome Scale scores, cognitive development, and motor functioning were assessed an average of 1.3 months after TBI. χ^2 analyses assessed differences in the distribution of findings in the inflicted and noninflicted TBI groups.

Results. Signs of preexisting brain injury, including cerebral atrophy, subdural hygroma, and ex vacuo ventriculomegaly, were present in 45% of children with inflicted TBI and in none of the children with noninflicted TBI. Subdural hematomas and seizures occurred significantly more often in children with inflicted TBI. Intraparenchymal hemorrhage, edema, skull fractures, and cephalohematomas were similar in both groups. Retinal hemorrhage was only identified in the inflicted TBI group. Glasgow Outcome Scale scores indicated a significantly less favorable outcome after inflicted than noninflicted TBI. Mental deficiency was present in 45% of the inflicted and 5% of the noninflicted TBI groups.

Conclusions. Characteristic features of inflicted TBI included acute computed tomography/magnetic resonance imaging findings of preexisting brain injury, extraaxial hemorrhages, seizures, retinal hemorrhages, and significantly impaired cognitive function without prolonged impairment of consciousness. *Pediatrics* 1998;102:300–307; *child abuse, shaken baby syndrome, traumatic brain injury, cognition, outcome, infants, children, neuroimaging, Glasgow Outcome Scale, retinal hemorrhage.*

ABBREVIATIONS. TBI, traumatic brain injury; CT, computed tomography; MRI, magnetic resonance imaging; GCS, Glasgow Coma Scale.

Physical child abuse and traumatic brain injury (TBI) are major public health problems. Injury is the cause of ~40% of fatalities in children from 1 to 4 years of age and ~70% of fatalities in children from 5 to 19 years of age.¹ TBI occurs in ~12% of confirmed cases of physical child abuse; the majority of children are less than 2 years of age.² Homicide is the second most common cause of injury fatalities in children and adolescents; 23% of fatalities caused by inflicted injury occur in children less than 5 years of age.¹ In Kraus and colleagues³ epidemiologic study of pediatric TBI, assault was the external cause of injury in 56% of the cases of serious brain injury in children less than 1 year of age. Although assault was the cause of only 5% of TBI in children ages 1 to 4 years, assault caused 90% of serious brain injury. Studies of consecutively admitted infants and preschoolers with TBI reported rates of inflicted injury ranging from 4% to 24%.^{2,4,5} Given the difficulties in ascertaining the external cause of injury in many cases of pediatric injury, it is likely that some cases reported as falls or with no clear history were actually inflicted injuries, which would increase the incidence of inflicted injury. For example, Duhaime and colleagues⁴ noted that 24% of consecutively admitted children 0 to 24 months of age with head trauma were presumed to have inflicted injuries whereas an additional 32% were suspicious for abuse, neglect, or social and family problems.

Biomechanical forces generated at the time of injury differ in inflicted and noninflicted TBI. Most head injuries involve both contact and inertial forces. Contact forces, which occur either when the head is struck or strikes an object, produce focal injuries to the scalp, skull, and brain such as lacerations, fractures, contusions, and epidural hematomas. Inertial forces, which typically involve acceleration-deceleration forces, result in movement of the brain and yield more diffuse injuries such as concussion, subdural hematoma, and diffuse axonal injury.⁶ In young children, significant rotational acceleration-deceleration forces occur infrequently in noninflicted injuries and commonly in inflicted TBI.⁷ Although the term “shaken baby syndrome” is used to describe the clinical presentation of infants with subdural and

From the Departments of *Pediatrics and †Radiology, University of Texas at Houston Health Science Center; and the §Department of Pediatrics, Baylor College of Medicine, Houston, Texas.

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Reprint requests to (L.E.-C.) Department of Pediatrics, University of Texas at Houston Health Science Center, 6431 Fannin, Houston, TX 77030.

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subarachnoid hemorrhage, retinal hemorrhage, and associated long bone changes,⁸ other studies suggest shaking alone may not generate sufficient forces to yield widespread vascular and parenchymal injury.^{9,10} Impact, even against a soft surface, yields significantly more deceleration forces than does shaking. Although contact forces may dissipate over a broad surface area and leave minimal traces of impact, the movement of the brain within the skull is often sufficient to produce significant vascular and parenchymal injury.⁷ Based on this biomechanical data, Bruce and Zimmerman⁹ proposed the term “shaking-impact syndrome” to more comprehensively depict the likely mechanism of inflicted brain injury in infants. However, other investigators inferred that shaking without impact is sufficient to cause severe or fatal intracranial injury.¹¹

Physical and radiologic findings in cases of suspected physical child abuse are commonly seen in the visual, skeletal, and central nervous systems. Duhaime et al⁴ reported retinal hemorrhages in 10% of a consecutive series of 100 patients with head injury who were 24 months of age or younger; 9 of 10 had inflicted injuries. Retinal hemorrhages rarely result from accidental trauma.^{12,13} Although massive bilateral retinal hemorrhages are associated with inflicted brain injury during infancy, slight unilateral retinal hemorrhages may occasionally be noted after severe noninflicted brain injury.¹⁴ In children without a history of major trauma, skeletal surveys have visualized multiple fractures most commonly involving the ribs, metaphyseal long bone fractures or femur fractures during infancy, and multiple fractures in various stages of healing.¹⁵ Similarly, cerebral computed tomography (CT) and magnetic resonance imaging (MRI) findings in children without evidence of major trauma revealed the presence of subdural hematomas, subarachnoid hemorrhage, cortical contusions, cerebral edema, infarction, and white matter injuries.^{16–20}

The different biomechanical forces involved in inflicted and noninflicted TBI may yield characteristic neuroimaging patterns and bodily injuries. Comparison of inflicted and noninflicted groups may yield better discrimination of the characteristics associated with assault. Although neuroimaging and physical findings have been described in assaulted children, there are no studies that assess the impact of these findings on neurobehavioral and developmental outcomes. Therefore, we compared acute CT/MRI findings, physical findings, and early developmental outcomes in children 0 to 6 years of age with inflicted or noninflicted TBI. We hypothesized that inflicted TBI would be associated with a higher frequency of extraaxial hemorrhage, retinal hemorrhage, and mental deficiency than noninflicted TBI.

METHODS

Participants

Physical findings and developmental status were evaluated prospectively in 40 children between the ages of 1 month to 6 years at injury who were hospitalized at Hermann Children's Hospital or Texas Children's Hospital in Houston, Texas, after either inflicted or noninflicted TBI. These children were enrolled

in a prospective, longitudinal investigation of developmental outcome after early acquired brain injury. Inclusionary criteria were moderate to severe TBI, no known preinjury neurologic or metabolic disorders, no history of previous TBI, and gestational age of at least 32 weeks. Of potential participants, ~88% of children in the inflicted group and 71% of children in the noninflicted group were enrolled in the study. Reasons for nonparticipation included parental refusal (21%), residing out of geographic region (12%), and physician refusal to allow patient recruitment (7%).

All children received a detailed physical examination by a pediatrician and/or a trauma surgeon. CT and/or MRI scans of the brain were obtained at or shortly after hospital admission. Skeletal surveys and fundoscopic examinations were performed on children with suspected abusive injuries. Inflicted injury was suspected in cases with inconsistencies in the clinical presentation, history, and neuroimaging findings.²¹ In cases of suspected child abuse, determination of whether an injury was inflicted or noninflicted was based on the assessment of the Child Protection Committee at each hospital and Harris County Children's Protective Services. Similar to the algorithm devised by Duhaime et al⁴ to detect probable inflicted injury, injuries incompatible with the stated mechanism of injury (eg, bilateral subdural hematomas and retinal hemorrhages with a history of falling from a couch; multiple skull fractures, multiple intracranial hemorrhages, and bilateral cephalohematomas with a history of falling 4 feet) and unexplained injuries (eg, no history of trauma in conjunction with intracranial injuries and old skeletal fractures) were presumed to indicate assault. Additional variables associated with inflicted injury such as delay in seeking treatment and inconsistent or changing history were also considered.²²

Demographic and birth history variables for the inflicted (*n* = 20) and noninflicted (*n* = 20) TBI groups are provided in Table 1. The two TBI groups were comparable in terms of ethnicity and

TABLE 1. Demographic and Birth Information for Traumatic Brain Injury (TBI) Groups

	Groups	
	Inflicted TBI (<i>n</i> = 20)	Noninflicted TBI (<i>n</i> = 20)
Demographic variables		
Age at injury (mo)		
<i>M</i>	10.60	35.55*
<i>SD</i>	14.87	25.35
Gender		
Female	17	10
Male	3	10
Ethnicity		
African-American	6	4
Anglo-American	7	10
Hispanic	6	5
Multicultural	1	1
Socioeconomic status (<i>n</i>)†		
Low	5	3
Middle	14	13
High	0	1
Neonatal history		
Gestational age (wk)		
<i>M</i>	39.12	39.12
<i>SD</i>	1.90	2.22
Apgar scores—5 minutes		
<i>M</i>	8.85	9.00‡
Range	8–9	8–10
Complications (<i>n</i>)		
Respiratory distress	1	1
Hyperbilirubinemia	2	0
Hypocalcemia	1	0
Sepsis	3	0
Tachypnea	2	1
Tachycardia	1	1
Other infection	0	2

* *P* < .001.

† Based on the Hollingshead 4 Factor Index of Social Position. Information was available for 17 children in the inflicted group.

‡ Information available in 13 and 17 children, respectively.

socioeconomic background. The sample was from predominantly middle to lower socioeconomic backgrounds and included major ethnic groups. In contrast to epidemiologic studies of TBI in young children indicating a higher incidence of TBI in males,²³ the male to female ratio was 1 to 2.1. Because inflicted injuries occur commonly during infancy, the inflicted TBI group was significantly younger at the time of injury ($M = 10.6$ months) than the noninflicted group (Fig 1) ($M = 35.6$ months), $F(1,39) = 14.07$; $P < .0006$. The alleged perpetrators were biological fathers (30%) and mothers (5%), other relatives (10%), boyfriend/girlfriend of parent (15%), baby-sitters (10%), and inconclusive (30%). Regarding birth histories, the groups had comparable duration of hospitalization, gestational ages, 5-minute Apgar scores, and neonatal complications. Birth history was obtained from medical records and parental interview. One child in the noninflicted group was adopted and no birth information was available for review. Although not all variables were available for each child, all children with missing Apgar scores were hospitalized for a maximum of 2 days and had no documented birth or neonatal complications. Three infants in the inflicted group and 4 infants in the noninflicted group had neonatal complications. Information regarding developmental assessment before the brain injury was not noted in either the birth or medical records.

The severity of TBI was determined using the Glasgow Coma Scale (GCS) score,²⁴ the duration of impaired consciousness, and CT/MRI findings. Because the GCS score was developed for adults, the motor and verbal scales were modified to accommodate the behavioral capabilities of children from birth through 35 months of age. Spontaneous movement in infants ages 0 to 6 months and goal-directed movements in children 7 to 35 months were considered comparable to following commands in older children. "Cries" and "cries to indicate need" were regarded as equivalent to the verbal scale items "confused" and "oriented." Duration of impaired consciousness was defined as the number of days a child was unable to follow a one-stage command or engage in goal-directed movements as indicated by the modified motor scale of the GCS score. Moderate TBI was characterized by injuries producing lowest postresuscitation GCS scores from 9 to 12; GCS scores from 9 to 15 with CT/MRI evidence of extraaxial bleed, intraparenchymal hemorrhage, or edema; or impaired consciousness persisting for <24 hours. Severe TBI consisted of lowest postresuscitation GCS scores from 3 to 8 or impaired consciousness persisting for at least 24 hours. Indices of injury severity were comparable in the inflicted and noninflicted TBI groups. The lowest postresuscitation GCS scores and the duration of impaired consciousness (see Table 2) were comparable in both TBI groups.

Procedure

Written informed consent to participate in the study was obtained either during the initial hospitalization or after discharge. For children under the conservatorship of Children's Protective Services, consent to participate was obtained from the agency after placement of the child in foster care or voluntary family placement. The study was approved by and conducted in accordance with the ethical guidelines of the institutional review board at each university.

CT and MRI scans were reviewed by a board-certified radiologist with a fellowship in MRI. The radiologist was blind to the group membership and probable cause of injury for all study

TABLE 2. External Cause of Injury and Indices of Injury Severity

	Group	
	Inflicted TBI (<i>n</i> = 20)	Noninflicted TBI (<i>n</i> = 20)
Reported cause of injury (<i>n</i>)		
Dropped by caregiver	5	0
Falls		
<4 ft	5	2
>4 ft	0	3
Motor vehicle accidents		
Passenger	0	9
Pedestrian	0	3
Hit by moving object	0	3
No history	10	0
Glasgow Coma Scale score (lowest)		
3–8	7	7
9–12	5	8
13–15	8	5
Duration of impaired consciousness (days)		
<i>M</i>	1.90	3.13
<i>SD</i>	3.35	4.98
Glasgow Outcome Scale Score*		
Good recovery	4	11
Moderate disability	13	5
Severe disability	3	4

Abbreviation: TBI, traumatic brain injury.

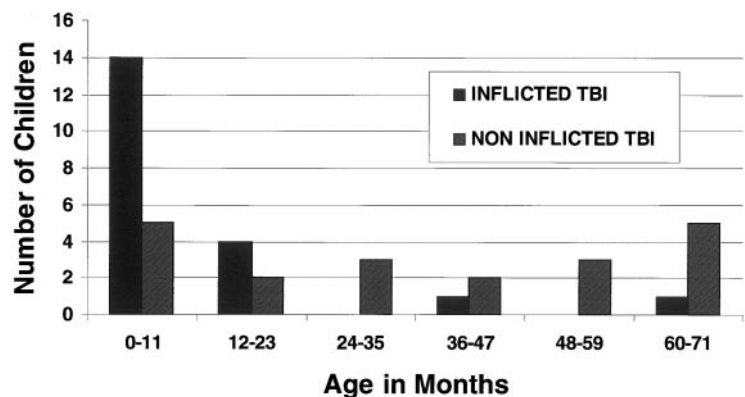
* $P < .05$.

participants. Only scans obtained within 1 week of the injury were included. The major categories of intracranial findings were: 1) extraaxial hematoma or hygroma as indicated by an isodense loculated CSF collection; and 2) parenchymal involvement including edema/infarction, hematoma, diffuse swelling, shear injury, and atrophy. Edema and infarction were included as a single category because of the difficulty reliably distinguishing between them in an acute imaging study. Medical records were reviewed to ascertain the presence of ocular injury, fractures, bruises/lacerations, and neurologic findings.

Assessment of Outcome

The Glasgow Outcome Scale²⁵ was used to characterize global neurobehavioral outcome at the time of the baseline evaluation. Because this scale was developed for adults, the criteria were adjusted for infants and children. Good outcome referred to a return to age-appropriate or preinjury levels of functioning. Moderate disability was assigned if the child had: 1) a significant reduction in cognitive functioning from estimated premorbid levels; 2) motor deficits including hemiparesis interfering with daily living activities; or 3) referral for outpatient rehabilitation therapies. Severe disability was assigned if: 1) cognitive scores were deficient; 2) severe motor deficits were present, such as lack of

Fig 1. The inflicted TBI group was significantly younger at the time of injury than the noninflicted TBI group.



age-appropriate postural control or ambulation; or 3) referral for inpatient rehabilitation. The criteria for persistent vegetative state were unchanged and reflected the presence of day/night cycles and total dependence for daily care.

Initial developmental evaluations were completed an average of 1.3 months after TBI following resolution of posttraumatic amnesia. Children were judged to have emerged from posttraumatic amnesia based on Children's Orientation and Amnesia Test²⁶ scores for 3 to 6 year olds and on return to play activities for 0 to 2 year olds. The Bayley Scales of Infant Development Mental and Motor Scales–Second Edition²⁷ provided standardized measures of cognitive development and motor functions for children ages 0 to 42 months at the time of evaluation. Bayley scores were corrected for prematurity for children with gestational ages of 32 to 37 weeks until they reached a chronologic age of 35 months. For children ages 43 to 71 months at the time of baseline evaluation, the Stanford-Binet Intelligence Scale Fourth Edition²⁸ and the McCarthy Scales of Children's Abilities²⁹ motor scales were administered.

Statistical Methods

χ^2 was used to determine whether the distribution of normal and abnormal CT/MRI and physical findings differed in the inflicted and noninflicted TBI groups. The distribution of cognitive and motor scores was divided into deficient (standard score, ≤ 69 ; <2nd percentile) and nondeficient (standard score, >69) categories.

RESULTS

Neuroimaging Findings

Major categories of brain injury visualized by CT/MRI are depicted in Table 3. Extraaxial collections were visualized in all children with inflicted TBI and in 70% of those with noninflicted TBI, $\chi^2 (1, N = 40) = 4.33, P < .04$. A total of 56 extraaxial bleeds was identified in the inflicted TBI group in comparison to 20 in the noninflicted TBI group. Subdural hematomas ($\chi^2 (1, N = 40) = 5.23, P < .005$) occurred more

frequently in the children with inflicted TBI. Only 1 child had a chronic subdural collection. Epidural hematomas were not visualized in any of the children with inflicted TBI and in 20% of the noninflicted TBI group, $\chi^2 (1, N = 40) = 4.44, P < .04$. Subarachnoid hemorrhage occurred with comparable frequency in both the inflicted (20%) and noninflicted (35%) TBI groups.

Regarding parenchymal involvement, intracerebral hematomas were present in 30% of the noninflicted group and in only 5% of the inflicted TBI group, $\chi^2 (1, N = 40) = 4.33, P < .04$. The occurrence of infarct/edema was distributed comparably across the groups. Two additional children in the inflicted TBI group had late infarcts at 2 and 3 weeks after the injury. In contrast, shear injury was visualized in 20% of the noninflicted TBI group and in none of the inflicted TBI group, $\chi^2 (1, N = 40) = 4.44, P < .04$.

Evidence of preexisting brain injury characterized by cerebral atrophy was visualized only in the inflicted TBI group. On the initial acute CT/MRI scan obtained within 1 week of injury, atrophy ($n = 7$) and encephalomalacia ($n = 1$) were identified in 40% of children with inflicted TBI, $\chi^2 (1, N = 40) = 8.49, P < .004$. Subdural hygromas and atrophy were present on scans obtained on the day of injury in 3 children with inflicted TBI. The presence of subdural hygromas is suggestive of degradation of previous subdural hematomas because there was no evidence of previous infection or recent trauma resulting in a tear in the arachnoid membrane. Ventricular abnormalities were noted in 45% of the inflicted TBI group and in 15% of the noninflicted TBI group, $\chi^2 (1, N = 40) = 4.29, P < .04$. For children with ventricular abnormalities, 8 of 9 children in the inflicted TBI group were rated as having ex vacuo ventriculomegaly; the cause of ventricular enlargement was indeterminate in the remaining case. Three children with noninflicted TBI had small ventricles secondary to mass effect from edema or intracerebral hematoma.

The presence of skull fractures and soft tissue swelling was examined on acute CT scans. The number of children with skull fractures was comparable in the 2 TBI groups. As depicted in Table 3, the distribution of fractures was similar because both groups had children with multiple, linear, depressed, or diastatic fractures. The 4 children in the noninflicted TBI group with diastatic fractures were injured by massive contact forces as follows: unrestrained passenger hit by a tire jack that was propelled in the air when the car hit a tree; the force of the tire jack ejected the child from the car ($n = 1$), hit by a falling television ($n = 2$), and falling four stories onto marble ($n = 1$). The distribution of soft tissue swelling was comparable across the two injury groups: swelling was present in 16 of 20 children with noninflicted TBI and in 11 of 20 children with inflicted TBI. Six of the 11 children with inflicted TBI who had soft tissue swelling also had skull fractures. There was no association between the presence of cranial fractures and/or soft tissue swelling and the occurrence of intracerebral hematoma, infarct/edema, or retinal hemorrhage.

TABLE 3. Neuroimaging Findings From Acute CT/MRI Scans

	Group	
	Inflicted TBI ($n = 20$)	Noninflicted TBI ($n = 20$)
Extraaxial collection		
Hematoma		
Subdural	16	9†
Epidural	0	4†
Subarachnoid	4	7
Hygroma		
Subdural	3	0
Parenchymal involvement		
Edema/infarction	5	6
Hematoma	1	6*
Diffuse swelling	2	2
Shear injury	0	4†
Atrophy	7	0†
Soft tissue swelling		
Present	11	16
Absent	9	4
Skull fracture		
Linear	3	2
Comminuted	0	1
Diastatic	2	4
Depressed	3	3
Multiple	1	5
Basilar	0	1

Abbreviations: CT, computed tomography; MRI, magnetic resonance imaging; TBI, traumatic brain injury.

* $P < .05$.

† $P < .005$.

Retinal Hemorrhage and Physical Findings

The distribution of retinal hemorrhages and physical findings is presented in Table 4. Retinal hemorrhages, which were present in 70% of the inflicted TBI group, were not noted after noninflicted TBI, $\chi^2 (1, N = 40) = 21.54, P < .001$. The distribution of skeletal fractures and bruises/lacerations was similar across groups. In the inflicted TBI group, fractures were most often seen in the ribs, tibia/fibula, and femur whereas facial fractures predominated in the noninflicted TBI group. Similar to previous findings,³⁰ bruises and lacerations were most commonly present in the face in both groups. Lower extremity bruises and lacerations and damage to internal organs were only identified in the noninflicted TBI group.

Neurologic findings are depicted in Table 5. The distribution of hemiparesis was similar across groups: 50% of the noninflicted TBI group and 30% of the inflicted TBI group were hemiparetic. Cranial nerve findings were positive in 4 children with inflicted TBI; 1 child had involvement of 4 nerves, 1 child had involvement of 2 nerves, and 2 children had damage to 1 nerve. Similarly, 5 children in the noninflicted TBI group had positive cranial nerve findings. Seizures occurred in significantly more children with inflicted TBI, $\chi^2 (1, N = 40) = 10.42, P < .001$.

Early Neurobehavioral and Developmental Outcomes

The distribution of Glasgow Outcome Scale²⁵ scores differed significantly in the TBI groups, $\chi^2 (1, N = 40) = 6.97, P < .05$. Good recovery was present in more children with noninflicted TBI (55% vs 20%) whereas moderate disability was present in more children with inflicted TBI (65% vs 20%). Deficits in children with a moderate disability included

TABLE 4. Bodily Injuries in Inflicted and Noninflicted TBI Groups

	Group	
	Inflicted TBI (n = 20)	Noninflicted TBI (n = 20)
Ocular injury		
Retinal hemorrhage		
Bilateral	13	0*
Unilateral	1	0
Corneal abrasion/cataracts	2	2
Fractures		
Clavicle	1	0
Scapula	1	0
Ulna/radius	1	1
Humerus	0	1
Tibia/fibula	4	0
Femur	2	1
Rib	3	1
Facial	0	3
C-spine	0	3
Bruises/lacerations		
Facial	11	14
Torso	6	2
Upper extremities	2	3
Lower extremities	0	4
Genitalia/buttocks	2	0

Abbreviation: TBI, traumatic brain injury.

* $P < .001$.

TABLE 5. Neurological Findings in Inflicted and Noninflicted TBI Groups

	Group	
	Inflicted TBI (n = 20)	Noninflicted TBI (n = 20)
Seizures		
Present	13	3*
Absent	7	17
Hemiparesis		
Present	6	10
Absent	16	10
Cranial nerve abnormality		
III	2	3
IV	1	1
V	1	0
VI	2	2
VII	2	2

Abbreviation: TBI, traumatic brain injury.

* $P < .001$.

hemiparesis, cognitive scores in the borderline range, and/or requiring more than one rehabilitation therapy or placement in a self-contained classroom (eg, early childhood intervention). Severe disability, characterized by total dependence for daily care inappropriate for chronologic age, severe motor deficits, or cognitive deficiency, occurred in 15% of the inflicted TBI group and in 20% of the noninflicted TBI group.

Cognitive test scores differed across groups; 45% of children with inflicted TBI and 5% of children with noninflicted TBI scored in the mentally deficient range, $\chi^2 (1, N = 40) = 8.53, P < .005$. The group means were 78.2 and 87.7, respectively. The distribution of motor scores was similar in both groups, with 25% of each group scoring in the deficient range; group means were 80.3 and 84.3, respectively.

Outcome was examined in patients with and without infarct/edema visualized on the acute scans. An acceptable outcome was defined as either a good recovery or moderate disability on the Glasgow Outcome Scale. The presence of infarct/edema was associated with a less favorable outcome, $\chi^2 (1, N = 40) = 6.54, P < .01$. In comparison to patients without CT or MRI scan evidence of infarct/edema, patients with infarct/edema had significantly lower mental ($M = 74.7$ vs 86.2), $F(1,38) = 4.83, P < .05$, and motor scores ($M = 72.8$ vs 85.7), $F(1,38) = 4.27, P < .05$. Of the 11 children with infarct/edema, 1 child with inflicted injury and 1 child with noninflicted injury had diffuse edema, 1 had involvement of a single lobe, and the remaining children had either unilateral or bilateral edema in multiple lobes and subcortical structures.

DISCUSSION

Children with inflicted and noninflicted TBI had different patterns of neuroimaging, physical, and cognitive findings. Although both groups had negative histories of previous brain injury, signs of pre-existing brain injury, characterized by cerebral atrophy and ex vacuo ventriculomegaly, were apparent in 40% to 45% of children with inflicted TBI and in none of the children with noninflicted injuries. Subdural hematomas occurred in a greater number of

children and with greater frequency in children with inflicted versus noninflicted TBI. In contrast, epidural hematomas and shear injuries were only visualized in the noninflicted injury group. Intraparenchymal hemorrhages and edema/infarction were distributed comparably throughout the two groups. Our findings differ from those of Billmire and Myers,²¹ who compared infants with accidental versus nonaccidental injuries and found that 95% of children with serious intracranial injury or hemorrhage had inflicted injuries. Neurobehavioral and motor outcomes were poorer in patients with edema/infarction. Skull fractures, soft tissue swelling throughout the skull, skeletal fractures, and bruises/lacerations occurred with comparable frequency in both groups. Damage to internal organs occurred significantly more often after noninflicted TBI. Retinal hemorrhages were present in 70% of the inflicted TBI group. Direct comparison of the rates of hemorrhages across groups was limited because ophthalmologic assessment was not performed in all children with noninflicted TBI. Seizures occurred with significantly greater frequency after inflicted TBI although other signs of neurologic injury, including cranial nerve injury and hemiparesis, were distributed equally across groups. Glasgow Outcome Scale scores indicated greater disability in the inflicted TBI group: fewer children had a good recovery and more had a moderate disability. Severe disability was distributed equally across groups. Significantly more children with inflicted TBI scored in the mentally deficient range than did children with noninflicted TBI. Motor scores did not differ across groups. The significant disability identified after inflicted TBI is consistent with the high incidence of major permanent morbidity documented by long-term follow-up of children with the shaking-impact syndrome.³¹ Because the TBI groups did not differ on indices of neonatal complications, injury severity, or acute parenchymal injury, the high frequency of deficient cognitive scores and unfavorable outcome ratings in children with inflicted TBI likely reflects the interaction of the current injury with previous neurologic injury and adverse environmental conditions. The poorer cognitive and motor outcomes in assaulted than nonassaulted children may account for the worse neurobehavioral outcomes in children sustaining TBI at 0 to 2 years of age than in older children.^{5,32-34}

The division of children into inflicted and noninflicted TBI groups was based in part on histories incompatible with the type, severity, and/or pattern of injuries. The congruence between the injury and history was based on empirical studies of consequences of falls,³⁵ witnessed falls,³⁶⁻³⁹ and stairway injuries.^{40,41} Although retinal hemorrhage was not a variable independently considered, the fact that 70% of the inflicted group and none of the children in the noninflicted group were noted to have retinal hemorrhages supports the selection criteria. Subdural and subarachnoid hemorrhages, which occurred in both groups, did not independently indicate the presence or absence of assault. However, in noninflicted TBI, subdural hematomas were most common

in motor vehicle accidents and were not associated with either falls or crush injuries.

The occurrence of skull fractures in 40% and soft tissue swelling over the cranium in 55% of children in the inflicted TBI group provides some support for the "shaking-impact" mechanism of injury proposed by Bruce and Zimmerman.⁹ However, the remaining children did not show overt signs of assault involving the cranium. Because accurate histories of the assault are difficult or impossible to obtain, it is unclear whether children with no overt signs of assault involving the cranium received contact trauma to the head. There was no association between skull fracture and/or soft tissue swelling and either parenchymal involvement or retinal hemorrhage.

The age difference between the inflicted and noninflicted TBI groups complicates group comparisons. Although the occurrence of inflicted injury peaks during infancy, the age distribution of noninflicted injury is fairly constant during infancy and the preschool years.^{3,23} Therefore, the age distributions of the inflicted and noninflicted groups differ significantly. Longitudinal outcome studies are needed to follow children with early inflicted and noninflicted TBI to see whether neuropsychologic deficits are similar in children injured during infancy and preschool years when assessed during middle childhood.

Assessment of injury severity in infants and young children is complicated by the lack of well-validated measures of level of consciousness. Although the GCS²⁴ is effective in determining injury severity in older children who are verbal, assessment of injury severity is difficult in preverbal children. Despite modifications in the GCS to accommodate the behavioral capabilities of infants, none of the modified versions is widely used. Some investigators question the association between coma scale scores and outcome in children because the relationship between coma scores and outcome is not invariant.⁴² Clinical evaluation in infants may be misleading because significant parenchymal injury may be present in a child with spontaneous eye opening and spontaneous movements; infants may also withdraw from painful stimuli applied to the limbs based on primitive motor patterns.⁷ Because level of consciousness may be either misleading or difficult to assess in infants, neuroimaging findings provide essential data for assessment of injury severity during infancy.⁴³ The combination of coma rating scales and neuroimaging findings may provide the best assessment of injury severity.

Assessment of outcome is also problematic in young children. Widely used outcome scales, such as the Glasgow Outcome Scale, which were developed for adults, do not reflect the functional issues related to young children. For example, because all young children are dependent on others for daily care, it is difficult to ascertain quantitative changes in level of dependency after brain injury. Moreover, the burden of caring for dependent adults is likely perceived as more troublesome than caring for dependent children. After severe TBI, children may function adequately in a school environment because of the pres-

ence of special educational programs and curriculum modifications. Consequently, the functional change in cognition and daily living skills may be underestimated in infants and young children. Outcome measures that assess behaviors relevant at different developmental stages need to be developed and validated to assess functional outcome after early brain injury.

Psychometric evaluation of outcome in young children is essential to characterize the quality of outcome. Despite comparable GCS scores and similar duration of impaired consciousness, significantly more children in the inflicted TBI group than the noninflicted TBI group scored in the mentally deficient range on age-adjusted measures of cognitive ability. Although the inflicted TBI group had more early seizures and extraaxial hemorrhages, indices of acute parenchymal damage involving hemorrhage or infarct/edema were comparable across groups. Cognitive scores were lowest in children with hemispheric infarct and evidence of preexisting brain injury. The presence of signs of earlier brain injury on acute CT/MRI may partially explain the high number of children with inflicted TBI who were mentally deficient. Our findings are consistent with Caffey's⁸ hypothesis that occult brain injury may account for the reductions in cognitive development commonly noted in physically abused children. Neurologic and cognitive deficits reflecting a combination of adverse environmental and central nervous system variables have been reported in maltreated children with and without known brain injuries.^{44,45} Longitudinal outcome studies that evaluate physical, radiologic, neurologic, neuropsychologic, and social/emotional outcomes are essential to identify the developmental course and full range of sequelae of maltreated children with and without brain injury.

Although chronic changes were noted on acute imaging studies in 45% of children with inflicted TBI, these children had no reported history of previous brain injury. Chronic changes, which were present in children as young as 6 to 8 weeks of age, suggest previous assault and cumulative brain injury. As noted by Alexander and colleagues,¹¹ inflicted injuries in young children are frequently preceded by other forms of maltreatment. Given the repetitive nature of child maltreatment, it is essential that pediatricians and other professionals follow the duty to report statutes and report cases of suspected child maltreatment. Without intervention, the perpetrators are likely to engage in increasingly more violent assault.

Children with inflicted TBI present complex diagnostic issues. Because of the lack of accurate history of the trauma and misrepresentation of previous medical history, physicians must be attuned to subtle aspects of injury that might contribute to accurate diagnosis and detection of assault. Because the history provided by caregivers is likely to be unreliable, neuroimaging is essential for identification of acute intracranial findings as well as subtle evidence of previous brain injury. The use of neuroimaging techniques is particularly important in cases with no external trace of injury because shaking-impact injuries

can be lethal without external evidence of injury. Ophthalmologic evaluation and skeletal surveys also provide cardinal information to corroborate or allay suspicions of inflicted injury. Given the poor developmental outcomes of children with inflicted TBI compared with children with noninflicted TBI who have similar indices of injury severity, early identification of abuse, neuropsychologic assessment, initiation of rehabilitation, and family intervention are essential. Abused brain-injured children typically require sequential neuropsychologic evaluations and long-term rehabilitation services. Because the consequences of early brain injury may become more prominent as children develop during time, sequential evaluations are needed to assess the rate of development of new skills, to identify areas of deficiency requiring intervention, ensure referral for rehabilitation services, and monitor the family environment.

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REFERENCES

1. Division of Injury Control, Center for Environmental Health and Injury Control, Centers for Disease Control. Childhood injuries in the United States. *Am J Dis Child.* 1990;144:627-646
2. Hahn YS, Raimondi AJ, McLone DG, Yamanouchi Y. Traumatic mechanisms of head injury in child abuse. *Childs Brain.* 1983;10:229-241
3. Kraus JF, Rock A, Hemyari P. Brain injuries among infants. *Am J Dis Child.* 1990;144:684-691
4. 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 two years of age. *Pediatrics.* 1992;90:179-185
5. Levin HS, Aldrich EF, Saydjari C, et al. Severe head injury in children: experience of the traumatic coma data bank. *Neurosurgery.* 1992;31:435-444
6. Gennarelli TA, Thibault LE. Biomechanics of head injury. In: Wilkins RH, Rengachary SS, eds. *Neurosurgery, II.* New York, NY: McGraw-Hill; 1985
7. Ewing-Cobbs L, Duhaime AC, Fletcher JM. Inflicted and noninflicted traumatic brain injury in infants and preschoolers. *J Head Trauma Rehab.* 1995;10:13-21
8. Caffey J. The whiplash shaken infant syndrome: manual shaking by the extremities with whiplash-induced intracranial and intraocular bleedings, linked with residual permanent brain damage and mental retardation. *Pediatrics.* 1974;54:396-403
9. Bruce DA, Zimmerman RA. Shaken impact syndrome. *Pediatr Ann.* 1989;18:482-489
10. Duhaime AC, Gennarelli TG, Thibault LE, Bruce DA, Margulies SS, Wiser R. The shaken baby syndrome. A clinical, pathological, and biomechanical study. *J Neurosurg.* 1987;66:409-415
11. Alexander R, Sato Y, Smith W, Bennett T. Incidence of impact trauma with cranial injuries ascribed to shaking. *Am J Dis Child.* 1990;144:724-726
12. Elder JE, Taylor RG, Klug GL. Retinal haemorrhage in accidental head trauma in childhood. *J Paediatr Child Health.* 1991;27:286-289
13. Johnson DL, Braun D, Friendly D. Accidental head trauma and retinal hemorrhage. *Neurosurgery.* 1993;33:231-235
14. Betz P, Puschel K, Miltner E, Lignitz E, Eisenmenger. Morphometrical analysis of retinal hemorrhages in the shaken baby syndrome. *Forensic Sci Int.* 1996;78:71-80

15. Wissow LS. Child abuse and neglect. *N Engl J Med*. 1995;332:1425–1431
16. Alexander RC, Schor DP, Smith WL. Magnetic resonance imaging of intracranial injuries from child abuse. *J Pediatr*. 1986;109:975–979
17. Ellison PH, Tsai FY, Largent JA. Computed tomography in child abuse and cerebral contusion. *Pediatrics*. 1978;62:151–154
18. Levin AV, Magnusson MR, Rafto SE, Zimmerman RA. Shaken baby syndrome diagnosed by magnetic resonance imaging. *Pediatr Emerg Care*. 1989;5:181–186
19. McClelland CQ, Reke H, Kaufman B, Persse L. Cerebral injury in child abuse: a changing profile. *Childs Brain*. 1980;7:225–235
20. Sato Y, Yuh WTC, Smith WL, Alexander RC, Kao SCS, Ellerbrock CJ. Head injury in child abuse: evaluation with MR imaging. *Radiology*. 1989;173:653–657
21. Billmire ME, Myers PA. Serious head injury in infants: accident or abuse? *Pediatrics*. 1985;75:340–342
22. Caffey J. On the theory and practice of shaking infants: its potential residual effects of permanent brain damage and mental retardation. *Am J Dis Child*. 1972;124:161–169
23. Kraus JF, Fife D, Cox P, Ramstein K, Conroy C. Incidence, severity, and external causes of pediatric brain injury. *Am J Dis Child*. 1986;140:687–693
24. Teasdale G, Jennett B. Assessment of coma and impaired consciousness: a practical scale. *Lancet*. 1974;2:81–84
25. Jennett B, Bond M. Assessment of outcome after severe brain damage. *Lancet*. 1975;1:480–487
26. Ewing-Cobbs L, Levin HS, Fletcher JM, Miner ME, Eisenberg HM. The children's orientation and amnesia test: relationship to severity of acute head injury and to recovery of memory. *Neurosurgery*. 1990;27:683–691
27. Bayley N. *Bayley Scales of Infant Development*. 2nd ed. San Antonio, TX: Psychological Corporation; 1993
28. Thorndike RL, Hagen EP, Sattler JM. *Stanford-Binet Intelligence Scale*. 4th ed. Chicago, IL: Riverside Publishing Co; 1986
29. McCarthy D. *McCarthy Scales of Children's Abilities*. New York, NY: Psychological Corporation; 1972
30. Jessee SA. Physical manifestations of child abuse to the head, face, and mouth: a hospital survey. *J Dent Child*. 1995;60:245–249
31. Duhaime AC, Christian C, Moss E, Seidl T. Long-term outcome in infants with the shaking-impact syndrome. *Pediatr Neurosurg*. 1996;24:292–298
32. Luerssen TG, Klauber NR, Marshall LF. Outcome from head injury related to patient's age: a longitudinal prospective study of adult and pediatric head injury. *J Neurosurg*. 1988;68:409–416
33. Michaud LJ, Rivara FP, Grady MS, Reay DT. Predictors of survival and severity of disability after severe brain injury in children. *Neurosurgery*. 1991;31:254–264
34. Raimondi AJ, Hirschauer J. Head injury in the infant and toddler: coma scoring and outcome scale. *Childs Brain*. 1984;11:12–35
35. Musemeche CA, Barthel M, Cosentino C, Reynolds M. Pediatric falls from heights. *J Trauma*. 1991;31:1347–1349
36. Helfer RE, Slovis TL, Black M. Injuries resulting when small children fall out of bed. *Pediatrics*. 1977;60:533–535
37. Lyons TJ, Oates RK. Falling out of bed: a relatively benign occurrence. *Pediatrics*. 1993;92:125–127
38. Nimityoungskul P, Anderson LD. The likelihood of injuries when children fall out of bed. *J Pediatr Orthop*. 1987;7:184–186
39. Williams RA. Injuries in infants and small children resulting from witnessed and corroborated free falls. *J Trauma*. 1991;31:1350–1352
40. Chiaviello CT, Christoph RA, Bond RG. Stairway-related injuries in children. *Pediatrics*. 1994;94:679–681
41. Joffe M, Ludwig S. Stairway injuries in children. *Pediatrics*. 1988;82:457–461
42. Lieh-Lai MW, Theodorou AA, Sarniak AP, Meert KL, Maylan PM, Canady AI. Limitations of the Glasgow Coma Scale in predicting outcome in children with traumatic brain injury. *J Pediatr*. 1992;120:195–199
43. Ewing-Cobbs L, Fletcher JM, Levin HS, Francis DJ, Davidson K, Miner ME. Longitudinal neuropsychological outcome in infants and preschoolers with traumatic brain injury. *J Int Neuropsychol Soc*. 1997;3:581–591
44. Carrey NJ, Butter HJ, Persinger MA, Bialik RJ. Physiological and cognitive correlates of child abuse. *J Am Acad Child Adolesc Psychiatry*. 1995;34:1067–1075
45. Green AH, Voeller K, Gaines R, Kubie J. Neurological impairment in maltreated children. *Child Abuse Neglect*. 1981;5:129–134

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