Child's Nerv Syst (2000) 16:25–34

ORIGINAL PAPER

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Acute neuroradiologic findings in young children with inflicted or noninflicted traumatic brain injury

Received: 1 April 1999

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Abstract Acute CT/MRI findings were examined in a prospective, longitudinal study of 60 children 0–6 years of age hospitalized for moderate to severe traumatic brain injury (TBI). TBI was categorized as either inflicted (*n*=31) or noninflicted (*n*=29). Glasgow Coma Scale scores and perinatal history were comparable in both groups. Acute CT/MRI studies were visually inspected by a radiologist blind to group membership. Compared with the noninflicted TBI group, the inflicted TBI group had significantly elevated rates of subdural interhemispheric and convexity hemorrhages as well as signs of pre-existing brain abnormality, including cerebral atrophy, subdural hygroma, and ex vacuo ventriculomegaly. Intraparenchymal hemorrhage, shear injury, and skull fractures were more frequent after noninflicted TBI. Subarachnoid hemorrhage and infarct/edema occurred with comparable frequency in both groups. Characteristic acute neuroimaging findings of inflicted TBI included multiple extraaxial hemorrhages in addition to the mild atrophy, subdural hygromas, and ventriculomegaly that suggest prior brain abnormality.

Key words Child abuse · Shaken baby syndrome · Brain injury · Infants · Children · Neuroimaging

Introduction

Traumatic brain injury (TBI) is the most common cause of injury-related mortality and morbidity in children. Assault is a major external cause of TBI in young children; 23% of homicides in children occur in children younger than 5 years [9]. Epidemiologic studies estimated that assault caused 17% of all brain injuries and 56% of serious brain injury in infants; in children aged 1–4, assault caused 5% of brain injuries and 90% of serious brain injury [24]. Estimates of the occurrence of inflicted TBI range from 4% [20] through 24% [11] to 33% [3] of consecutive hospital admissions of young children with TBI. The most common form of inflicted TBI in young children is shaken baby syndrome [6] or shaking impact syndrome [5]. The large subdural spaces, disproportionately large head, incompletely myelinated brain, and open sutures and fontanels make young children highly susceptible to vascular and parenchymal injury from motion of the brain during rapid acceleration and deceleration of the head [10].

Neuroimaging is critical in the identification of inflicted head injury. Acute computed tomographic (CT) studies of children with inflicted head injury frequently identified the occurrence of interhemispheric or convexity subdural hematomas (SDH) [17, 25, 30, 33, 35, 38], subarachnoid hemorrhage (SAH), cerebral edema [29, 30, 33, 35, 38], and skull fractures [3, 8, 11, 17, 19, 30, 31, 35, 38], and infrequently, of epidural hematomas [30, 34]. Although the incidence of atrophy on acute CT was low, atrophy [35, 38], subdural hygromas, and ventriculomegaly [30] were frequently found on scans obtained at least 20 days after admission or in chronic cases of abuse. Comparison of cranial CT scan findings

in children with inflicted and noninflicted TBI indicated more frequent occurrence of SDH, posterior interhemispheric or parafalcine hemorrhage [17, 22], large (nonacute) extraaxial fluid collection, basal ganglia edema [22] and intracranial hemorrhage [3] after inflicted TBI; the frequencies of posterior fossa hemorrhage and frontal-parietal shear injuries did not differ between groups [22]. Recent examination of acute CT and MRI findings identified higher rates of subdural hematoma, subdural hygromas, cerebral atrophy, and ex vacuo ventriculomegaly after inflicted TBI, whereas edema and intraparenchymal hemorrhage occurred with comparable frequency in patients with inflicted and with noninflicted TBI [14].

The purpose of the present study was to compare prospectively the frequencies of acute CT and MRI findings of extraaxial fluid collection, intraparenchymal hemorrhage or edema, shear injuries, signs of prior brain abnormality, and skull fracture in children aged 0–6 years who were hospitalized with either inflicted or noninflicted TBI.

Materials and methods

Participants

CT and/or MRI findings were investigated prospectively in 60 children aged 1 month to 6 years who were hospitalized for inflicted or noninflicted head injuries at Memorial Hermann Children's Hospital or Texas Children's Hospital in Houston, Tex. All children were enrolled in a prospective, longitudinal study of outcome following early TBI. Table 1 displays demographic and neurologic variables for the two head injury groups. Glasgow Coma Scale (GCS) scores [36] and neuroimaging findings were used to categorize the severity of TBI. GCS scores were modified to accommodate the behavioral capabilities of children from 1 to 35 months of age. The motor scale item of "following commands" was modified to include "spontaneous movements" in infants ages 0–6 months and "goal-directed movements" in children of 7–35 months. On the Verbal scale, "cries" and "cries to indicate

Table 1 Demographic and neurologic variables for inflicted and noninflicted traumatic brain injury (*TBI*) groups (*GCS* Glasgow Coma Scale)

Variable	Inflicted TBI (*n*=31)	Noninflicted TBI (*n*=29)	*P*
Age at injury (months)			
Mean	10.26	34.79	0.0001
SD	13.35	26.36	
Range	01–58	02–73	
Apgar scores			
Mean	8.95	9.00	0.682
SD	0.38	0.40	
Weeks of gestation			
Mean	38.70	39.48	0.172
SD	1.96	1.98	
Range	34–42	33–41	
FOC			
Mean	14.18	13.89	0.813
SD	4.59	3.49	
Admission GCS score (mean)	12.13	10.25	0.070
3–8	05	10	
9–12	07	07	
13–15	19	11	
Lowest GCS score (mean)	10.23	8.83	0.31
3–8	10	15	
9–12	09	05	
13–15	12	09	
Impaired consciousness (days)	3.81	3.22	0.73
Reported cause of injury			
Motor vehicle accident	00	18	
Fall	12	07	
Hit by falling object	00	04	
Dropped	04	00	
Assault	05	00	
No history	10	00	

need" were considered comparable to items "confused" and "oriented," respectively. Severity was categorized as follows: "Moderate" – lowest postresuscitation GCS scores ranging from 9 to 12 or GCS scores of 9–15 with positive CT/MRI findings; "Severe" – lowest postresuscitation GCS scores 3–8. Using established procedures, the best level of response noted for each component was summed to obtain the GCS score [36]. However, if a child deteriorated following the initial resuscitation, the best GCS scores were recorded and summed to provide an index of the reduction in level of consciousness. Postresuscitation GCS scores have been used to characterize the severity of injury and are strongly related to long-term cognitive and behavioral outcome [13]. Scores of 1 were assigned if periorbital swelling or intubation precluded administration of specific items on the eye opening and verbal scales. The use of these assigned scores is not likely to change the total GCS score significantly [28]. In the case of sedation, the child's hourly GCS scores were examined and the best level of response was coded for each 24-h interval. For children experiencing seizures, GCS scores were obtained either prior to evidence of seizure activity or following postictal phases.

Inclusion criteria were (1) hospitalization for moderate or severe TBI, (2) no known preinjury neurological or metabolic disorders, (3) no history of prior TBI, and (4) gestational age of at least 33 weeks. We enrolled a consecutive series of children with inflicted TBI who were under the conservatorship of the state protective agency. When the protective agency did not take conservatorship of a child with putative inflicted TBI, approximately 20% of families participated. Twelve percent of families of children with noninflicted TBI declined to participate. Written informed consent to participate in the study was obtained. For children under the conservatorship of Children's Protective Services, consent was obtained from the agency. The study was approved by and conducted in accordance with the ethical guidelines of the institutional review board at each university.

All children with suspected inflicted injuries underwent skeletal surveys and funduscopic examinations. Determination of inflicted injury was based on the assessment of the state protective agency and the children's protection committee at each hospital. Consistent with the algorithm developed by Duhaime et al. [11] to identify probable inflicted TBI, injuries incompatible with the stated mechanism of injury based on epidemiologic studies of consequences of falls (e.g. bilateral subdural and retinal hemorrhages attributed to falling off a couch; multiple skull fractures, multiple intracranial hemorrhages, and bilateral cephalohematomas resulting from a reported 4-foot fall), and unexplained injuries (e.g. no history of trauma with intracranial injuries and old skeletal fractures) were regarded as indicative of assault. Additionally, delay in seeking treatment and changing history were also considered to be possible indicators of inflicted injuries.

CT was obtained on the day of hospital admission in all but 1 child. MRI scans were obtained within 7 days of admission in 24 children with inflicted and in 14 children with noninflicted TBI. MRI scans were completed an average of 2.6 days after admission. For children under the conservatorship of the Children's Protective Services, it occasionally took several days to weeks to receive administrative approval to obtain MRI scans. CT and MRI scans obtained within 7 days of hospitalization were rated by a board-certified radiologist specializing in MRI. The radiologist was blind to the cause of TBI and was informed only of the age of the patient to permit accurate assessment of the degree of myelination. Each scan was clinically evaluated to determine the presence or absence of supratentorial and infratentorial extraaxial fluid collections, intraparenchymal hematoma or edema, shear injury, and chronic changes (atrophy, ex vacuo ventriculomegaly). Owing to difficulty in distinguishing reliably between edema and infarction on early scans, they were included in a single category. Scans were graded clinically in terms of the severity (mild, moderate, severe) and chronicity (acute, subacute, chronic) of the findings. MRI scans were used to identify (1) shear injuries, which were defined using the gradient echo spin sequence that was the most sensitive to blood products, and (2) myelination pattern. CT scans were reviewed to determine the presence of skull fracture and status of the anterior and posterior fontanels. Owing to the greater sensitivity of MRI to contusions, presence and chronicity of SDHs, shear injuries, infarct/edema [33] and myelination pattern, these findings were obtained from MRI scans when both MRI and CT were available. To permit determination of signs of pre-existing brain abnormality, scans that were obtained within 24 h of hospitalization in 59 of the 60 study participants were examined for atrophy, subdural hygroma, and ex vacuo ventriculomegaly.

Chi-square was used to determine whether the number of children with a positive finding on CT/MRI was equally distributed in the inflicted and noninflicted TBI groups. Analysis of variance was used when the total number of clinical findings (e.g., number of extraaxial bleeds) was compared across groups.

Scanning protocol

All MRI and CT scans were performed without contrast. Approximately 96% of the patients required sedation, which was performed using a standard protocol. All MRI scans were performed on a 1.5-T magnet using a quadrature head coil. The research protocol specified: T1-weighted spin-echo 2D images obtained in the sagittal plane using a repetition time of 500 ms and an echo time of 14 ms, FOV (field of view) 24 cm, slice thickness of 5 mm, skip 2.5 mm, matrix 256×192, one repetition; proton density; T2-weighted axial 2D spin echo images using a repetition time of 2000 ms, echo time 34 ms/80 ms, FOV 22 cm, slice thickness 5 mm, skip 2.5 mm, matrix 256×192, 1 repetition; coronal 2D fast spin echo images using a repetition time of 4000 ms, echo time 102 ms, echo train length 8, FOV 22 cm, slice thickness 5 mm, skip 2.5 mm, 2 repetitions; and axial 2D gradient echo using repetition time 800 ms, echo times 11 and 33 ms, FOV 22, thickness 5 mm, skip 2.5 mm, matrix 256×192, 2 repetitions. However, not all the protocols were obtained using the research specifications, which may reduce the ability to identify shear injuries but should not affect other parameters. Since we were not able to enroll all children in the study prior to the initial MRI scan, approximately 10% of the studies were performed using a standard brain protocol for trauma patients. CT scans were performed on a spiral scanner using contiguous 5-mm axial sections.

Results

Patient demographic and neurologic variables

As noted in Table 1, the inflicted TBI group (n=31) was significantly younger than the noninflicted TBI group (n=29). This significant difference in age distribution reflects the epidemiology of TBI in young children. Based on available birth records, the inflicted and noninflicted TBI groups were comparable in terms of neonatal variables including gestational age, days of hospitalization, 5-min Apgar scores, FOC, and length, as well as the frequency of neonatal respiratory, infectious, cardiac, and other complications. Birth weight, the only neonatal variable that differed across groups, was lower in the inflicted TBI group: $F(1,47)=12.85$, $P<0.0008$. Four infants in the inflicted injury group and 2 infants in the noninflicted injury group had birth weights below 2500 g. The inflicted and noninflicted TBI groups had compara-

Table 2 Extraaxial hemorrhage after inflicted and noninflicted TBI

Location of hemorrhage	Inflicted TBI (*n*=31)	Noninflicted TBI (*n*=29)	*P*
Supratentorial	22	14	0.01
Subdural	21	09	0.004
Acute	16	06	
Subacute	03	03	
Chronic	02	00	
Epidural	00	06	0.008
Extradural	01	01	
Subarachnoid	11	11	0.844
Interhemispheric			
Subdural	19	06	0.001
Acute	17	05	
Subacute	01	01	
Chronic	01	00	
Location			0.947
Anterior	06	02	
Posterior	09	03	
Both	04	01	
Cisterns	01	00	
Infratentorial			
Subdural	12	05	0.065
Acute	10	03	
Subacute	02	02	
Chronic	00	00	

ble duration of impaired consciousness and lowest post-resuscitation GCS scores.

Extraaxial hemorrhage

Extraaxial hemorrhage was present in 30 of the 31 children with inflicted injury and in 22 of the 29 children with noninflicted injuries: χ^2 (1, n=60)=8.47, P<0.004. The average number of extraaxial hemorrhages per child was 2.1 in the inflicted TBI group and 1.3 in the noninflicted TBI group: F(1,59)=6.96, P<0.01. The type and location of hemorrhage varied with the external cause of injury. Subdural supratentorial convexity, interhemispheric, and infratentorial hematomas were seen significantly more often after inflicted TBI (mean=1.71) than noninflicted TBI (mean 0.69): F(1,59)=13.58, P<0.0005.

Supratentorial subdural hemispheric convexity hematomas were significantly more common in children with inflicted TBI, while epidural hematomas were only visualized in the noninflicted TBI group (Table 2). Bilateral SDHs were seen in 15 of the 31 children in the inflicted injury group and in 14 of the 29 in the noninflicted TBI group. The majority of interhemispheric hemorrhages were acute. In both groups, posterior interhemispheric hemorrhages occurred more frequently than anterior hemorrhages and the distribution of hemorrhages was comparable. The distribution of SAHs did not differ.

Table 3 depicts the distribution of extraaxial hemorrhages in each TBI group. Multiple hemorrhages were present in 68% of the children with inflicted TBI and in 31% of those in the noninflicted TBI group. The most frequent presentation in the inflicted TBI group was a combination of subdural convexity and interhemispheric hematomas. In the noninflicted TBI group, children tended to present with single SAH, SDH, or epidural hematomas. Interhemispheric SDHs were always associated with other extraaxial hemorrhages and were not seen in isolation in either group.

Table 3 Distribution of extraaxial hemorrhages by TBI group (*EDH* epidural, *IH* interhemispheric, *SAH* subarachnoid, *SDH* subdural, *TBI* traumatic brain injury)

Type of hemorrhage	Inflicted TBI (*n*=31)	Noninflicted TBI (*n*=29)
EDH only	00	04
IH only	00	00
SAH only	05	05
SDH only	04	03
EDH+IH	00	01
SAH+SDH	01	02
SAH+IH	02	01
SDH+IH	15	01
SAH+SDH+IH	03	02
EDH+SAH+SDH+IH	00	01

Parenchymal hemorrhage, edema, and shear injury

Supratentorial and infratentorial parenchymal hematomas occurred more frequently in the noninflicted than

Table 4 Parenchymal findings after inflicted and noninflicted TBI

Location and type of lesion	Inflicted TBI (*n*=31)	Noninflicted TBI (*n*=29)	*P*
Supratentorial			
Hemorrhage	03	10	0.047
Cortical	03	07	0.133
Basal ganglia	00	01	
Thalamus	01	00	
Corpus callosum	00	00	
Infarct/edema	11	09	0.816
Cortical	07	06	
Basal ganglia	03	02	
Thalamus	01	01	
Corpus callosum	00	00	
Shearing	00	09	0.001
Subcortical	00	03	
Basal ganglia	00	00	
Thalamus	00	01	
Corpus callosum	00	05	
Infratentorial			
Hemorrhage	01	03	0.269
Cerebellum	01	02	
Brain stem	00	01	
Infarct/edema	03	03	0.931
Cerebellum	02	03	
Brain stem	01	00	

the inflicted TBI group: $F(1,59)=3.83$, $P<0.05$; see Table 4. Although the majority of children had a single hematoma, 1 child in the inflicted injury group had two hemorrhages while 3 children in the noninflicted group each had two or three hemorrhages. Supratentorial parenchymal hemorrhages were more common following noninflicted injury; in both groups, hemorrhages occurred more frequently in cortical than in subcortical regions. Infratentorial hemorrhages occurred infrequently.

Infarct/edema, which was present in 11 of the 31 with inflicted and in 9 of the 29 with noninflicted TBI, was comparable in both groups. The total number of areas of infarction in each group did not differ across groups: $F(1,59)=0.02$, $P<0.9$. Supratentorial infarct/edema was visualized most frequently in the cerebral cortex and second most frequently in the basal ganglia. Multiple areas of infarction/edema were present in 3 children with inflicted and 2 children with noninflicted TBI. Collapsing across groups, the cortical infarcts were mild in 31%, moderate in 54%, and severe in 15%. Infratentorial infarct/edema, which was noted in 3 children in each injury group, was more common in the cerebellum than in the brain stem. Compression of the lateral ventricles was noted in 2 children with inflicted injuries and in 3 children with noninflicted injuries: χ^2 (1, n=60)=0.30, $P<0.6$.

Contrary to expectation, shear injuries were visualized exclusively in children with noninflicted injuries. Shear injuries occurred most often in the corpus callosum and in subcortical regions and were not visualized in deep white matter tracts, periventricular white matter, or the brain stem.

Neuroimaging findings were examined in a subsample of 23 infants with inflicted and 9 with noninflicted TBI who were <13 months of age and had comparable GCS scores [$F(1,31)=0.24$, $P<0.6$] and duration of impaired consciousness: $F(1,31)=0.07$, $P<0.8$). Based on Chi-square analyses, the numbers of infants with SAH and/or SDH ($P<0.1$), edema/infarct ($P<0.3$), and parenchymal hemorrhage ($P<0.2$) were comparable across TBI groups. Thirty-one of 32 children had extraaxial hemorrhages. However, analysis of variance indicated that infants with noninflicted TBI tended to have a higher number of acute lesions overall than infants with inflicted TBI: $F(1,31)=3.41$, $P<0.07$. Although the number of extraaxial hemorrhages ($P<0.4$) was comparable across groups, the noninflicted TBI group had a greater number of regions of infarct/edema [$F(1,31)=5.50$, $P<0.03$] and parenchymal hemorrhage [$F(1,31)=3.76$, $P<0.06$] than the inflicted injury group. Owing to the small sample size and concomitant reduction of power to detect group differences, these findings should be considered preliminary and require replication with a larger sample.

Pre-existing abnormalities

As physical abuse often occurs repetitively, we examined findings suggestive of pre-existing cerebral and ventricular abnormalities on CT and MRI scans obtained within 24 h of admission. As indicated in Table 5, atrophy was identified in 9 of the 30 children in the inflicted TBI

Table 5 Findings suggestive of prior brain injury or abnormality on initial scan

Type of lesion	Inflicted TBI (n=30)	Noninflicted TBI (n=29)	P
Atrophy	09	00	0.001
Subdural hygroma	04	00	0.042
Ventriculomegaly	07	00	0.006
Delayed myelination	04	01	0.185

Table 6 Distribution of skull fractures

Type of fracture	Inflicted TBI (n=31)[a]	Noninflicted TBI (n=29)[a]
Linear	03/03	10/14
Depressed	05/05	08/10
Comminuted	02/02	03/05
Diastatic	03/03	05/06
Multiple	04/08	11/36
Basilar	00/00	01/01

[a] Number of children with fracture/number of fractures

group and in none of the children with noninflicted TBI. Atrophy, which was most commonly visualized in cortical regions, was rated as mild in 8 of 9 children and was bilateral in all cases. Atrophy was restricted to supratentorial regions. Encephalomalacia was not identified in any of our cases.

Subdural hygromas were present in 4 children with inflicted TBI and in none of the children with noninflicted injury. Unilateral subdural hygromas were noted in 13% of children with inflicted and in none of the children with noninflicted TBI. Hygromas were present over the convexity in most cases and in the interhemispheric fissure. Infratentorial hygromas were not visualized in either group.

Ventriculomegaly was present in 7 of the 30 children with inflicted and in none of those with noninflicted TBI. The ventricular enlargement was considered ex vacuo in all cases except 1, in whom it was of indeterminate origin. Delayed myelination, which was comparable in both groups, was visualized in 4 of 30 in the inflicted and in 1 of 29 in the noninflicted injury groups. All 9 children with atrophy had other cerebral abnormalities, and 4 of the 9 also had ventriculomegaly, while the remaining 4 had ventriculomegaly and hygromas. One child had normal ventricles but subdural hygromas. Delayed myelination was associated with atrophy in 2 children and with ventriculomegaly in 1 child. The remaining 2 children had no other pre-existing abnormalities.

Skull fractures and status of fontanels

Skull fractures occurred more frequently in children with noninflicted TBI: $F(1,59)=15.93$, $P<0.004$. The mean number of fractures was 1.48 in the noninflicted and 0.45 in the inflicted TBI groups. As seen in Table 6, the distribution of fractures was similar in the two injury groups. Linear fractures and comminuted fractures with depressed fragments were commonly seen. Diastatic fractures were identified in 3 out of 30 children in the inflicted injury group and 5 out of 29 in the noninflicted group. Frontal bone fractures were only visualized in children with noninflicted injuries: $\chi^2(1,N=60)=5.83$, $P<0.05$ and occipital fractures were only present in children with inflicted injury: $\chi^2(1,N=60)=1.94$, $P<0.4$. Temporal and parietal bone fractures occurred infrequently. Bilateral fractures were visualized rarely; biparietal fractures were seen only in 1 child with inflicted TBI. Fractures crossing sutures were present in 2 cases of inflicted and 3 of noninflicted injury. Multiple fractures were present in 4 of 31 children with inflicted and in 11 of the 29 children with noninflicted TBI. The higher rate of multiple, diastatic, and frontal bone fractures and of fractures crossing the sutures in the noninflicted injury group may be related to the high number of children with noninflicted injuries who sustained crush injuries to the skull. There was no relationship between the presence of a skull fracture and intracranial lesions: $F(1,59)=2.4$, $P<0.2$. Patency of the anterior and posterior fontanels was not related to the overall number of intracranial lesions [$F(2,58)=0.04$, $P<0.9$], but was related to the number of extraaxial hemorrhages: $F(2,58)=4.35$, $P<0.02$. Extraaxial hemorrhages occurred more frequently when the anterior fontanel was open [$F(1,58)=7.65$, $P<0.008$], but did not vary with the patency of the posterior fontanel: $F(1,58)=1.05$, $P<0.4$.

Discussion

Neuroradiologic findings differed in young children who sustained TBI from different external causes. Severe traumatic intracranial injury is related primarily to acceleration/ deceleration forces [16]. Compared with typical

acceleration/deceleration forces, inflicted TBI is associated with distinct sudden angular deceleration of the brain and cerebral vessels, which is often repetitive [10]. Extraaxial hemorrhage was present in 30 of 31 children with inflicted TBI and in 22 of 29 children with noninflicted TBI. In keeping with other studies comparing rates of intracranial injury in children with inflicted and noninflicted TBI [3, 22], the inflicted TBI group had significantly higher rates of supratentorial subdural interhemispheric and convexity hemorrhages [17] and tended to have more infratentorial hemorrhages. Although the inflicted TBI group had a higher rate of interhemispheric hemorrhage, interhemispheric hemorrhages were visualized in the posterior region or both anterior and posterior regions in 65–70% of the children with interhemispheric hemorrhage in each TBI group. SAH and infratentorial SDH were comparable between the TBI groups. The most frequent presentation in the inflicted TBI group was a combination of subdural convexity and interhemispheric hematomas, while single hemorrhages predominated in the noninflicted TBI group. Similar to the results recorded in other series of patients, suggesting that epidural hematomas result from brief linear contact forces rather than repetitive rotational acceleration/deceleration forces [34], epidural hematomas were only found in children with noninflicted injury.

Parenchymal injury varied across the injury groups. Infarct/edema, which was the most frequently noted parenchymal abnormality, was comparable across TBI groups. Although Hymel et al. [22] noted that basal ganglia edema occurred with greater frequency in inflicted than in noninflicted TBI, we identified basal ganglia edema only in 3 cases of inflicted and 2 of noninflicted injury. Intracerebral hemorrhage occurred most frequently after noninflicted TBI. Supratentorial parenchymal hemorrhages occurred primarily in cortical regions. Infratentorial hemorrhages were visualized infrequently in both groups. These findings contrast with those of Goldstein et al. [17], who found comparable rates of parenchymal hemorrhage in inflicted and noninflicted TBI cases, and Billmire and Meyers [3], who found intracranial damage almost exclusively in abused infants. The low rate of intracranial findings in the children with noninflicted injuries evaluated by Billmire and Meyers is unusual. Variation in rates of hemorrhage across studies may be due to differences in age ranges evaluated and to variability caused by sampling artifacts.

On the acute scans, shearing injury was only visualized in 9 children with noninflicted TBI. Examination of MRI scans obtained within 3 months of injury in 27 of 31 children in the inflicted TBI group revealed 1 child with shearing injury. This child was 4 years old at the time of injury, and the exact mechanism of injury remains unknown. Although autopsy studies of infants with inflicted TBI have documented cortical tears and shearing injuries, particularly in infants less than 5 months of age [7, 27, 37], MRI evaluation in our sample of children surviving assault, including 17 children less than 5 months of age, did not disclose shearing injury. Vowles et al. [37] inferred that recurrent inflicted TBI in infants may result in diffuse axonal injury without tearing blood vessels. As the MRI protocol in this study relied on the presence of hemorrhage to identify shear injury, it may have underestimated the actual occurrence of shear injuries without obvious hemorrhage. Identification of these primarily nonhemorrhagic lesions is difficult [23]. Moreover, myelination in infants under 5 months of age extends to the corona radiata and is beginning to occur in the centra semiovale [4]. The rudimentary degree of myelination in the infants with inflicted TBI may have reduced the likelihood of shear injury at junctures of white and gray matter.

Skull fractures were more common following noninflicted TBI, with each child averaging nearly 1.5 fractures. Linear and depressed fractures were identified most frequently. Multiple fractures occurred in 11 of the 29 children with noninflicted and in only 4 of the 31 with inflicted TBI. This finding differs from studies that identified either a higher rate of multiple fractures following inflicted TBI [3, 31] or reported equal numbers of skull fractures in inflicted and noninflicted injury groups [17]. Hobbs [21] identified several configurations of fractures, including depressed, diastatic, nonparietal, complex, multiple, and bilateral fractures and fractures that crossed sutures, that were more characteristic of inflicted than of noninflicted injury. In our sample and that of Meservy and colleagues [31], rates of depressed, diastatic, and complex fractures did not differ according to the mechanism of injury. Meservy et al. [31] noted that multiple or bilateral fractures or fractures crossing sutures were more common after inflicted TBI. However, the distribution of bilateral fractures or fractures crossing sutures did not differ in our groups, while multiple fractures were significantly more common in the noninflicted injury group. The rate of multiple fractures may be elevated in our sample, since 5 children in the noninflicted TBI group sustained crush injuries. In contrast to previous studies [3, 20], fractures of the parietal bone were infrequent in both groups. The distribution of fractures across studies may vary considerably based on the mechanisms for recruitment of patients (prospective enrollment versus retrospective chart review), type of patient population (ICU admissions, TBI patients, general samples of physically abused children), severity of injury (fatalities versus survivors), and demographic variables such as age. In keeping with the observations reported by Cohen et al. [8], there was no significant association between the presence of skull fractures and the presence of intracranial lesions. As reported by Raimondi and Hirschauer [32], extraaxial hemorrhage occurred more frequently in infants with open anterior fontanels.

Owing to the repetitive nature of physical child abuse [2], signs of possible occult preinjury brain abnormality

were examined on the admission CT scan. We identified elevated rates of cortical atrophy, ventriculomegaly, and subdural hygroma only in children with inflicted TBI. The radiologic signs of chronic cerebral abnormalities may reflect the effects of repeated maltreatment and are consistent with the reported 33% recidivism rate for physical child abuse [2]. Mild cortical atrophy with accompanying ex vacuo ventriculomegaly was apparent in 30% of the inflicted and in none of the noninflicted TBI cases. Several of the scans rated as atrophic were read as within normal limits upon routine radiological diagnosis. However, closer inspection revealed the presence of subtle atrophic changes characterized by sulcal and ventricular enlargement inconsistent with the child's age. This discrepancy suggests that radiologists evaluating imaging studies of young children, even those in whom there is no suspicion of physical abuse, should routinely assess subtle signs of chronic brain damage in addition to acute findings. Subdural hygromas were visualized exclusively in children with inflicted TBI with atrophy. Subdural hygromas result from dural tears or infectious processes, or represent residual by-products of partially reabsorbed SDHs [18]. Given the co-occurrence of atrophy and ventriculomegaly, it is possible that the hygromas resulted from earlier, previously undetected brain injury. Similar to findings in the elderly, the presence of atrophy may contribute to the development of SDHs in children with repetitive inflicted TBI. Since the bridging veins are stretched as the parenchyma shrinks, the vessels are rendered more vulnerable to damage by rotational acceleration forces. With the exception of birth weight, the birth histories and neonatal course of the inflicted and noninflicted groups were comparable. Therefore, the pre-existing neuroradiologic abnormalities are unlikely to result from perinatal or neonatal risk factors. Our findings are in concordance with neuroradiologic findings of subdural hygroma, atrophy or encephalomalacia, and ventriculomegaly associated with chronic abusive head injury [8, 30, 35]. Hymel et al. [22] also recorded findings similar to ours: they identified nonacute extraaxial fluid collections in 21% of children with inflicted head injury and in none of the children with noninflicted injuries. Although delayed myelination was observed in both groups, the inflicted injury cases outnumbered the noninflicted injury cases by 4 to 1. Delayed myelination was not related systematically to other indices of acute or chronic injury. Of the 5 children with delayed myelination, 2 had no indices of prior injury, 1 had ventriculomegaly, and 2 had mild bilateral atrophy and ventriculomegaly.

Our findings are generally consistent with the previous literature suggesting a high rate of extraaxial hemorrhage and infarct/edema after inflicted TBI. Even on admission CT scans, signs of pre-existing brain abnormality were present in nearly one-third of children with acute inflicted brain injury who had no history of prior injury. Children with inflicted TBI have unfavorable long-term cognitive and motor outcomes [12, 15] and are more severely disabled than children with noninflicted TBI of comparable severity [14]. The poorer developmental outcomes in children with inflicted TBI probably reflect a combination of prior occult brain injury, the current brain injury, and unfavorable environmental factors. Our findings are also consistent with those of Alexander and colleagues [2], who identified signs of previous abuse, neglect or both in 71% of children with shaken baby syndrome. Neuroimaging is essential in the diagnosis of inflicted injury. In particular, subtle signs of possible preexisting brain abnormalities may be important indicators of prior injury and should raise the threshold of suspicion of inflicted injury. Because children with inflicted injury are at great risk for subsequent and possibly fatal injuries, medical specialists must intervene early by identifying possible abusive injuries and providing liaison with children's protective agencies to minimize the cycle of repetitive child abuse.

The present study highlights the difficulty of completing research studies in children with inflicted and noninflicted injury. Specific limitations include circularity in the definition of inflicted TBI, which is problematic in all studies of inflicted injury, and disparity in age at injury. Because there are no definite guidelines for distinguishing inflicted from noninflicted TBI, differentiation of cases is based largely on discrepancies between the history, clinical presentation, and findings from epidemiologic studies delineating the expected consequences of witnessed injuries from falls. As in Hymel et al.'s [22] study and in epidemiologic studies [24, 26], children with inflicted TBI were younger than children with noninflicted TBI. The disparity in age complicates direct comparison of intracranial findings owing to significant differences in brain maturation [4] and physiologic response to injury [1]. Although limited by reduced power, our analysis of the distribution of neuroimaging findings in children <12 months of age suggested similar distribution of extraaxial hemorrhage in the inflicted and noninflicted TBI groups in conjunction with a higher frequency of edema/infarction and parenchymal hemorrhage in the noninflicted cases. The success of recent injury prevention programs, particularly legislation requiring car seats for infants and young children, has reduced the occurrence of significant TBI in young children. Multicenter studies are needed to obtain sufficient numbers of young children with noninflicted TBI for additional comparisons of injury characteristics and outcome.

Acknowledgements Preparation of this paper was supported in part by National Institute of Neurological Disorders and Stroke grant 29462: Accidental and Nonaccidental Pediatric Brain Injury, to Dr. Ewing-Cobbs and by National Institutes of Health grant no. M01-RR-02558. The assistance of the Harris County Children's Protective Services and the University Clinical Research Center at Memorial Hermann Hospital is gratefully acknowledged.

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