ARTICLE

Mechanisms, Clinical Presentations, Injuries, and Outcomes From Inflicted Versus Noninflicted Head Trauma During Infancy: Results of a Prospective, Multicentered, Comparative Study

Kent P. Hymel, MD[a,b], Kathi L. Makoroff, MD[c], Antoinette L. Laskey, MD, MPH[d], Mark R. Conaway, PhD[e], James A. Blackman, MD, MPH[a]

[a]Department of Pediatrics and [e]Division of Biostatistics and Epidemiology, University of Virginia, Charlottesville, Virginia; [b]Inova Fairfax Hospital for Children, Falls Church, Virginia; [c]Department of Pediatrics, University of Cincinnati College of Medicine, Mayerson Center for Safe and Healthy Children, Cincinnati Children's Hospital Medical Center, Cincinnati, Ohio; [d]Department of Pediatrics, Indiana University School of Medicine, Indianapolis, Indiana

Dr Hymel made substantial contributions to research project conception and design, acquisition and interpretation of data, drafting of the manuscript, critical revisions of the manuscript for intellectual content, and final approval of the version to be submitted for publication; Dr Hymel takes responsibility for the integrity of this work as a whole, from inception to publication. Dr Makoroff made substantial contributions to research project design, acquisition of data, critical revisions of the manuscript for intellectual content, and final approval of the version to be submitted for publication. Dr Laskey made substantial contributions to research project design, acquisition of data, critical revisions of the manuscript for intellectual content, and final approval of the version to be submitted for publication. Dr Conaway made substantial contributions to research project design, analysis and interpretation of data, and final approval of the version to be submitted for publication. Dr Blackman made substantial contributions to research project conception and design, acquisition of data, critical revisions of the manuscript for intellectual content, and final approval of the version to be submitted for publication.

The authors have indicated they have no financial relationships relevant to this article to disclose.

ABSTRACT

OBJECTIVE. Our goal was to conduct a prospective, multicentered, comparative study that would objectively verify and explain observed differences in short-term neurodevelopmental outcomes after inflicted versus noninflicted head trauma.

METHODS. Children <36 months of age who were hospitalized with acute head trauma confirmed by computed tomography imaging were recruited at multiple sites. Extensive clinical data were captured prospectively, subjects were examined, cranial imaging studies were blindly reviewed, and caregivers underwent scripted interviews. Follow-up neurodevelopmental evaluations were completed 6 months after injury. Head-trauma etiology and mechanisms were categorized by using objective a priori criteria. Thereafter, subject groups with inflicted versus noninflicted etiologies were compared.

RESULTS. Fifty-four subjects who met the eligibility criteria were enrolled at 9 sites. Of 52 surviving subjects, 27 underwent follow-up assessment 6 months after injury. Etiology was categorized as noninflicted in 30 subjects, inflicted in 11, and undetermined in 13. Compared with subjects with noninflicted head trauma, subjects with inflicted head trauma (1) more frequently experienced noncontact injury mechanisms, (2) sustained greater injury depth, (3) more frequently manifested acute cardiorespiratory compromise, (4) had lower initial Glasgow Coma Scale scores, (5) experienced more frequent and prolonged impairments of consciousness, (6) more frequently demonstrated bilateral, hypoxic-ischemic brain injury, (7) had lower mental developmental index scores 6 months postinjury, and (8) had lower gross motor quotient scores 6 months postinjury.

CONCLUSIONS. Compared with infants with noninflicted head trauma, young victims of inflicted head trauma experience more frequent noncontact injury mechanisms that result in deeper brain injuries, cardiorespiratory compromise, diffuse cerebral hypoxia-ischemia, and worse outcomes.

www.pediatrics.org/cgi/doi/10.1542/peds.2006-3111

doi:10.1542/peds.2006-3111

Key Words
head trauma, infants, child abuse, accidents, outcomes

Abbreviations
GCS—Glasgow Coma Scale
PediBIRN—Pediatric Brain Injury Research Network
CT—computed tomography
MDI—mental developmental index
GMQ—gross motor quotient
MVC—motor vehicle crash
SDH—subdural hematoma

Accepted for publication Jan 16, 2007

Address correspondence Kent P. Hymel, MD, Inova Fairfax Hospital for Children, Department of Pediatrics, 3300 Gallows Rd, Falls Church, VA 22042-3300. E-mail: kent.hymel@inova.com

During infancy, inflicted or abusive head trauma is a leading cause of traumatic death and disability,[1] accounting for nearly one quarter of all pediatric hospital admissions for head injury[2] and two thirds of infant homicides.[3] Many survivors of inflicted head trauma during infancy later manifest developmental delays, sensory deficits, feeding difficulties, recurrent seizures, intellectual deficits, motor impairments, attention deficits, and/or educational and behavioral dysfunctions.[4–10]

Published studies that compare outcomes after inflicted versus noninflicted pediatric head trauma are limited in number, largely retrospective, and use a wide variety of definitional criteria for inflicted versus noninflicted etiologies. Some of these definitional criteria are fundamentally flawed, incorporating inappropriate circular logic or inherent biases. Despite their limitations, these studies provide compelling evidence that outcomes after inflicted head trauma are generally worse than outcomes after noninflicted pediatric head trauma.[5,8,9,11–14]

Abusive acts have been linked to subdural, subarachnoid, and retinal hemorrhaging[14–19]; localized axonal injury in the region of the craniocervical junction and cervical cord; acute respiratory compromise or arrest; loss of consciousness; hypotension[20–24]; and secondary, diffuse, hypoxic-ischemic brain injury with swelling.[7,23,25,26] We suspect that these primary and secondary inflicted traumatic injuries and their clinical manifestations are best explained by noncontact injury mechanisms (ie, rotational cranial acceleration and/or deceleration). Therefore, we hypothesized that compared with infants with noninflicted head trauma, infants with inflicted head trauma experience more frequent noncontact injury mechanisms, greater injury depth on neuroimaging, more frequent acute cardiorespiratory compromise, lower initial Glasgow Coma Scale (GCS) scores, more frequent and prolonged impairments of consciousness, more frequent diffuse, hypoxic-ischemic brain injuries, and worse short-term outcomes.

Our objective was to apply widely acceptable, a priori definitional criteria for etiology and mechanism(s) of injury in a prospective, multicentered, comparative study that would verify and explain observed differences in short-term, neurodevelopmental outcomes after inflicted versus noninflicted head trauma during infancy.

METHODS

The Pediatric Brain Injury Research Network (PediBIRN) is a consortium of clinical investigators who have made a voluntary commitment to conduct collaborative, multicentered, clinical research regarding traumatic brain injuries in young children. The institutional review boards at all 9 participating PediBIRN institutions approved this research study before local subject recruitment. Funded research activities began in March 2003 at the University of Virginia and at Inova Fairfax Hospital for Children. The remaining unfunded PediBIRN sites began subject recruitment at various times thereafter, enrolling a convenience sample of their eligible subjects.

Participants

At every site, hospital admission logs and medical charts were screened to identify eligible study subjects. Inclusion criteria included age <36 months and computed tomography (CT) evidence of any acute, nonpenetrating head or brain injury leading to inpatient evaluation and/or treatment. Children with preexisting brain disease, infection, hypoxia-ischemia, or trauma; birth injury; developmental delays; sensory deficits; bleeding disorder or severe malnutrition were excluded from study participation. Screening for exclusion criteria was accomplished through scripted parental interviews, interpretation of growth parameters, and subsequent blinded reviews of the subjects' initial cranial CT scans.

Procedures

Subjects who met inclusion and exclusion criteria and whose parent(s) consented to study participation were examined serially during the course of their acute hospitalizations. Local researchers reviewed their emergency medical technician, emergency department, and inpatient medical charts to capture extensive demographic, historical, clinical, laboratory, and neurosurgical data. The subjects' primary caregivers (the persons responsible for these children when they each became clearly and persistently ill with clinical signs later linked to their acute, traumatic, cranial injuries) were interviewed extensively by using a scripted interview designed to capture essential historical and clinical data from the scene of injury in a consistent and objective manner. More specifically, every primary caregiver who consented to be interviewed was questioned systematically regarding the specific timing, etiology (inflicted versus noninflicted), and circumstances of injury; the infant's clinical and mental status before and after his/her acute deterioration; and medical interventions at the scene of injury. Qualified neuroradiologists blinded to all other clinical and historical information reviewed the subjects' complete CT and MRI cranial imaging studies to document injuries and to identify the greatest depth of visible injury. Whenever possible, child protection and/or police investigators were interviewed to capture additional historical and investigative information.

Follow-up Neurodevelopmental Assessments

The surviving subjects returned 6 months after injury for an extensive, outpatient, neurodevelopmental assessment that included the Bayley Scales of Infant Development (second edition) and the Peabody Developmental Motor Scales (second edition). The fully qualified physical or occupational therapists, rehabilitation specialists, or developmental pediatricians who conducted these assessments at each participating site were blinded to all

historical, clinical, and radiologic information. The mental developmental index (MDI) and gross motor quotient (GMQ) scores calculated during these follow-up assessments were used as outcome measures of overall cognitive and motor development, respectively. Both are standardized scores like an IQ, with mean values of 100.

Statistical Analyses

All data were captured on data forms that were piloted extensively at the 2 funded PediBIRN institutions. Subsequently, the complete data were transferred into a relational database (Access 2000; Microsoft, Inc, Redmond, WA) designed to facilitate subsequent analyses. To enhance objectivity, database queries were created to sort subjects according to specific, a priori, definitional criteria for etiology (Table 1) and mechanism(s) of injury (Table 2). Other queries were designed to facilitate determinations regarding injury depth and the duration of impaired consciousness. Finally, subject groups were compared and outcome data were analyzed in an attempt to identify predictors of MDI and GMQ scores 6 months after injury.

χ^2 tests were used to compare outcome variables, such as mechanisms of injury, the duration of impaired consciousness, and depth of injury among subject groups defined by etiology, categorized as inflicted, noninflicted, or undetermined. The nonparametric Kruskal-Wallis test was used to compare continuous outcome variables such as the 6-month MDI and GMQ scores among groups defined by etiology, mechanism of injury, or depth of injury. Spearman rank correlations were used to assess the associations between continuous variables. Analyses were conducted in SAS 9.1 (SAS Institute, Inc, Cary, NC) and GAUSS 6.1 (Aptech Systems, Inc, Black Diamond, WA).

TABLE 1 **A Priori Criteria for Categorizing the Etiology of Head Injuries**

Category	Criteria
Noninflicted	Cases in which the child's primary caregiver described an accidental head injury event that was developmentally consistent, historically consistent with repetition over time, could be linked to the child's acute clinical presentation for traumatic cranial injuries, and occurred in the absence of any noncranial injuries considered moderately or highly specific for abuse[a]
	Cases in which an accidental head injury event was witnessed independently and could be linked to the child's acute clinical presentation for traumatic cranial injuries (eg, MVC)
Inflicted	Cases in which the child's primary caregiver admitted abusive acts that could be linked to the child's acute clinical presentation for traumatic cranial injuries
	Cases in which an independent witness verified abusive acts that could be linked to the child's acute clinical presentation for traumatic cranial injuries
	Cases in which a child not yet cruising or walking became clearly and persistently ill with signs of acute cardiorespiratory compromise[b] linked to his/her traumatic cranial injuries while in the care of a primary caregiver who denied any knowledge of a head injury event
	Cases in which the child's primary caregiver provided an explanation for the child's head injury event that was clearly developmentally inconsistent with the parent(s)' description of their child's developmental capabilities
	Cases in which the child's primary caregiver provided an explanation for the child's head injury event that was highly inconsistent with repetition over time
	Cases in which the head-injured child also revealed ≥2 noncranial injuries considered moderately or highly specific for abuse[a]
Undetermined	Cases meeting criteria for both inflicted and noninflicted etiology
	Cases not meeting any criteria for either inflicted or noninflicted etiology

[a] Including classic metaphyseal lesion(s); fractures of the rib(s), scapula, sternum, spinous process(es), or digit(s); vertebral body fracture(s) or dislocation(s); epiphyseal separation(s); noncranial bruising, abrasion(s) or laceration(s) in location(s) other than the knees, shins or elbows; patterned bruise(s) or dry contact burn(s); scalding burns with uniform depth, clear lines of demarcation, and a paucity of splash marks; intra-abdominal injuries; retinal hemorrhages described by an ophthalmologist as dense, extensive, covering a large surface area of the retina, or extending to the periphery of the retina; and retinoschesis diagnosed by an ophthalmologist.
[b] Including breathing difficulty, respiratory distress, infrequent respirations, apnea, or cyanosis; clinical manifestations of shock, delayed capillary refill, or cardiac arrest; any requirement for mouth-to-mouth breathing, bag-mask ventilation, intubation, chest compressions, rapid volume expansion, or epinephrine therapy; occurring at the scene of injury, during transport, in the emergency department, or at the time of hospital admission; documented by medical personnel or reported by the child's primary caregiver.

RESULTS

Twenty-seven eligible subjects were recruited at the University of Virginia or at Inova Fairfax Hospital for Children over a 2½-year period between 2003 and 2006. The remaining PediBIRN sites enrolled a convenience sample of 27 additional subjects during the same time interval. Overall, 54 study subjects meeting eligibility criteria were enrolled.

Etiology was categorized as inflicted in 11, undetermined in 13, and noninflicted in 30 subjects. Among subjects meeting a priori criteria for noninflicted head trauma were 4 children head-injured in a motor vehicle collision and 26 whose injuries were attributed to a fall. These falls included 4 involving stairs, 3 from heights >10 feet, and 2 from heights between 6 and 10 feet. The remaining subjects reportedly fell from heights <6 feet.

Demographic, mechanistic, clinical, neuroimaging, and outcome data regarding our comparative subject groups are summarized in Table 3. Age at the time of injury, gender, and racial and ethnic distributions were similar in subject groups meeting criteria for inflicted and noninflicted head trauma. Compared with our 30 subjects with noninflicted head injuries, the 11 subjects with inflicted head injuries (1) more frequently experienced noncontact injury mechanisms ($P < .001$), (2) revealed greater depth of injury on neuroimaging ($P < .001$), (3) more frequently manifested signs of acute cardiorespiratory compromise ($P < .001$), (4) had lower

TABLE 2 **A Priori Criteria for Categorizing the Mechanism(s) of Head Injury**

Category	Criteria
Contact[a]	Cases with injuries limited to craniofacial bruising, abrasion(s), laceration(s) or swelling; subgaleal hematoma(s); cephalohematoma(s); skull fracture(s) or epidural hematoma
Noncontact[b]	Cases with injuries limited to acute concussion, diffuse axonal injury, or an abnormal subdural collection that extends from the interhemispheric region
Combined	Cases with both contact and noncontact injuries
Undetermined[c]	Cases with injuries limited to subarachnoid hemorrhage, brain contusion(s), brain laceration(s), or an abnormal subdural collection that does not extend from the interhemispheric region

[a] Contact injuries can be viewed as the deformational injuries that result from cranial impact if the head is prevented from moving. Primary brain injuries resulting from isolated contact mechanisms result solely from cerebral tissue distortions induced by skull deformation.

[b] Noncontact injuries result solely from cranial acceleration or deceleration, irrespective of whether or not there is a direct impact to the cranium.

[c] These undetermined injuries can result from either contact or noncontact injury mechanisms.

initial GCS scores ($P < .001$), (5) experienced more frequent and prolonged impairments of consciousness ($P < .001$), (6) more frequently demonstrated bilateral, hypoxic-ischemic, brain injury, or swelling ($P < .001$), (7) had lower MDI scores 6 months postinjury ($P = .011$), and (8) had lower GMQ scores 6 months postinjury ($P = .002$). One child in each comparative group died.

Twenty-seven of 52 surviving subjects underwent a subsequent follow-up neurodevelopmental assessment. As summarized in Table 4, their MDI scores 6 months postinjury were related significantly to etiology ($P = .011$), depth of injury ($P = .003$), signs of acute cardiorespiratory compromise ($P = .027$), and initial GCS scores ($P = .001$). Their GMQ scores 6 months postinjury were related significantly to etiology ($P = .002$), mechanisms of injury ($P = .05$), depth of injury ($P = .01$), signs of acute cardiorespiratory compromise ($P = .005$), the duration of impaired consciousness ($P = .013$), and initial GCS scores ($P < .001$).

Among the surviving subjects meeting definitional criteria for noninflicted head trauma, none who were evaluated 6 months postinjury had an MDI or GMQ score <60. In contrast, among the subjects meeting definitional criteria for inflicted head trauma, 3 (75%) of 4 who underwent a Bayley assessment had MDI scores <60, and 4 (67%) of 6 who underwent a Peabody assessment had GMQ scores <60. Our study included too few subjects with follow-up data to conduct additional multivariable analyses.

DISCUSSION

Poor outcomes after pediatric head trauma have been linked to low initial GCS scores, increased depth and duration of impaired consciousness, diffuse cerebral edema, cerebral hypoperfusion, brain infarction, and increased depth of parenchymal injury.[9,24,25,27–31] Our results confirm specifically all of these conclusions from previous studies (Tables 3 and 4).

Neuropathological studies of abused infants have identified localized injuries in the region of the craniocervical junction that can trigger acute concussion, apnea, respiratory insufficiency, bradycardia, shock, or cardiac arrest.[20–22] We suspect that these deep injuries result from noncontact (that is, rotational cranial acceleration or deceleration) injury mechanisms resulting from abusive acts. If not promptly resolved or treated, the resulting cardiorespiratory compromise will initiate or accelerate the development of secondary, diffuse, hypoxic-ischemic brain injuries that have been linked to poor outcomes. Through these pathophysiological processes, etiology significantly influences outcome.

On the basis of these considerations, we hypothesized that, compared with infants with noninflicted head trauma, infants with inflicted head trauma experience more frequent noncontact injury mechanisms, greater injury depth, more frequent signs of acute cardiorespiratory compromise, lower initial GCS scores, more frequent and prolonged impairments of consciousness, more frequent diffuse hypoxic-ischemic brain injuries, and worse short-term outcomes. Our results confirm these hypotheses (Table 3) and provide preliminary evidence that young victims of inflicted head trauma have poor outcomes because they frequently experience noncontact injury mechanisms.

Compared with our subjects with noninflicted head injuries, our surviving subjects with inflicted head trauma revealed MDI and GMQ scores 6 months postinjury that were significantly and markedly lower. In light of so many other significant differences summarized earlier, this result is not surprising. Although there are limitations on the use of MDI scores and other Bayley results for prediction of future cognitive function, such large discrepancies suggest that clinicians should be cautious regarding prognosis after inflicted head trauma, and perhaps more optimistic after noninflicted head trauma. The same could be said for motor prognosis, even more confidently, as evaluation of motor skills in this age group is more reliable.

Controversy persists in the medical literature regarding the dangers of shaking an infant. Although published case reports and series have attributed pediatric intracranial injuries to isolated shaking,[32,33] some authors contend that shaking is not injurious and/or that perpetrator admissions of shaking are unreliable.[34,35] In this multicentered study, we used a scripted interview to capture historical data from caregivers in a consistent manner. None of the caregivers interviewed during this study disclosed abusive acts. Nevertheless, it is interesting to note that 8 of 11 subjects meeting a priori criteria for inflicted pediatric head trauma (Table 1) manifested cranial injuries indicative of an isolated noncontact mechanism of injury.

TABLE 3 **Demographic, Mechanistic, Clinical, Neuroimaging, and Outcome Data of Comparative Subject Groups**

	Inflicted ($N = 11$)	Noninflicted ($N = 30$)	Undetermined ($N = 13$)
Age at the time of injury, mo			
Median	5	4	6
Mean	10.5	9	11.5
SD (range)	10 (0.5–27)	10 (0.5–28)	11 (2–31)
Gender, *n* (%)			
Female	5 (45)	14 (47)	7 (54)
Male	6 (55)	16 (53)	6 (46)
Race, *n* (%)			
White	8 (73)	27 (90)	7 (54)
Black or African American	0 (0)	2 (7)	2 (15)
Other	3 (27)	1 (3)	4 (31)
Ethnicity, *n* (%)			
Hispanic or Latino	3 (27)	2 (7)	0 (0)
Non-Hispanic, non-Latino, or unknown ethnicity	8 (73)	28 (93)	13 (100)
Mechanism(s) of injury, *n* (%)[a]			
Isolated contact mechanism	1 (3)	19 (70)	8 (62)
Isolated noncontact mechanism	8 (73)	1 (4)	1 (8)
Combined contact and noncontact mechanism(s)	2 (18)	7 (26)	3 (23)
Undetermined mechanism(s)	0 (0)	3 (10)	1 (8)
Greatest depth of injuries visible on neuroimaging, *n* (%)[a]			
Scalp	0 (0)	0 (0)	1 (8)
Skull	0 (0)	13 (43)	3 (23)
Epidural space	0 (0)	1 (3)	2 (15)
Extra-axial spaces	1 (9)	8 (27)	4 (31)
Cortical brain	2 (18)	6 (20)	2 (15)
Subcortical brain	8 (73)	2 (7)	1 (8)
Brain stem or cerebellum	0 (0)	0 (0)	0 (0)
Signs of acute cardiorespiratory compromise, *n* (%)[a,b]			
Absent	4 (36)	27 (90)	12 (92)
Present	7 (64)	3 (10)	1 (8)
Initial GCS[a]			
N	11	29	13
Mean	8.0	13.2	13.7
SD	4.4	2.9	13.7
Duration of impaired consciousness, *n* (%)[a]			
None reported or documented	0 (0)	24 (80)	11 (85)
Only at the scene of injury	5 (45)	3 (10)	1 (8)
At hospital admission but persistent for <6 h	0 (0)	0 (0)	0 (0)
6–24 h, responsive by 24 h	1 (9)	1 (3)	0 (0)
>24 h, never decerebrate, decorticate, or flaccid	1 (9)	1 (3)	0 (0)
>24 h, decerebrate, decorticate, or flaccid	4 (36)	1 (3)	1 (8)
Traumatic injuries visible on neuroimaging, *n* (%)			
Soft tissue injuries	3 (27)	23 (77)	9 (69)
Skull fracture(s)	2 (18)	23 (77)	8 (62)
Epidural hematoma	1 (9)	2 (7)	2 (15)
Subdural hemorrhage	8 (73)	9 (30)	3 (23)
Subarachnoid hemorrhage	3 (27)	9 (30)	5 (38)
Brain contusion(s) or laceration(s)	7 (64)	8 (27)	2 (15)
Diffuse axonal injury	3 (27)	0 (0)	0 (0)
Unilateral hypoxia, ischemia, or swelling	2 (18)	2 (7)	1 (8)
Bilateral hypoxia, ischemia, or swelling[a]	7 (64)	1 (3)	1 (8)
Death from head injuries, *n* (%)			
No	10 (91)	29 (97)	13 (100)
Yes	1 (9)	1 (3)	0 (0)
MDI standardized score 6 mo postinjury[c]			
N	4	16	6
Mean	60.0	94.4	107.3
SD	20.0	11.8	11.7
GMQ standardized score 6 mo postinjury[c]			
n	6	11	6
Mean	59.8	101.8	102.2
SD	21.5	10.2	6.9

[a] Observed differences between inflicted ($n = 11$) and noninflicted ($n = 30$) subject groups were highly significant with $P < .001$ by χ^2 testing.

[b] Including breathing difficulty, respiratory distress, infrequent respirations, apneal or cyanosis; clinical manifestations of shock, delayed capillary refill, or cardiac arrest; any requirement for mouth-to-mouth breathing, bag-mask ventilation, intubation, chest compressions, rapid volume expansion, or epinephrine therapy; occurring at the scene of injury, during transport, in the emergency department, or at the time of hospital admission; documented by medical personnel or reported by the child's primary caregiver.

[c] Observed differences between inflicted ($n = 11$) and noninflicted ($n = 30$) subject groups were significant with $P < .05$ by Kruskal-Wallis testing.

TABLE 4 **Predictors of Outcome 6 Months After Pediatric Head Trauma**

	MDI Scores 6 mo Postinjury				GMQ Scores 6 mo Postinjury			
	Mean	SD	*N*	*P*	Mean	SD	*N*	*P*
Etiology of injury[a]				.011				.002
Noninflicted	94.4	11.8	16		101.8	10.2	11	
Inflicted	60.0	20.0	4		59.8	21.5	6	
Mechanism(s) of injury[b]				.66				.05
Isolated contact mechanism	94.8	11.8	16		100.6	10.0	14	
Isolated noncontact mechanism	73.0	39.8	3		67.6	28.4	5	
Combined contact and noncontact mechanism(s)	90.5	30.2	4		79.0	33.0	3	
Greatest depth of injuries visible on neuroimaging				.003				.01
Scalp	—	—	0		—	—	0	
Skull	94.1	10.3	8		95.8	10.0	6	
Epidural space	92.0	9.9	2		103.0	—	1	
Extra-axial spaces	110.0	9.0	7		104.4	6.3	5	
Cortical brain	89.5	10.8	6		103.7	10.2	6	
Subcortical brain	50.0	0.0	3		54.0	17.9	5	
Brain stem or cerebellum	—	—	0		—	—	0	
Signs of acute cardiorespiratory compromise[c]				.027				.005
Absent	95.8	16.1	23		98.4	17.4	18	
Present	63.3	23.1	3		64.2	20.8	5	
Duration of impaired consciousness				.12				.013
None reported or documented	98.1	13.2	20		102.1	9.2	16	
Only at the scene of injury	73.0	27.6	4		64.0	31.6	3	
In the emergency department	—	—	0		—	—	0	
At hospital admission but persistent for <6 h	90	—	1		89	—	1	
6–24 h, responsive by 24 h	—	—	0		—	—	0	
>24 h, never decerebrate, decorticate, or flaccid	—	—	0		—	—	0	
>24 h, decerebrate, decorticate, or flaccid	50	—	1		59.3	22.6	3	
	Correlation	95% CI	*N*	*P*	Correlation	95% CI	*N*	*P*
Initial GCS score	0.64	(0.32–0.83)	26	.001	0.74	(0.46–0.89)	22	<.001

CI indicates confidence interval.

[a] See Table 1 for specific, prospective, definitional criteria.

[b] See Table 2 for specific, prospective, definitional criteria.

[c] Including breathing difficulty, respiratory distress, infrequent respirations, apnea, or cyanosis; clinical manifestations of shock, delayed capillary refill, or cardiac arrest; any requirement for mouth-to-mouth breathing, bag-mask ventilation, intubation, chest compressions, rapid volume expansion, or epinephrine therapy; occurring at the scene of injury, during transport, in the emergency department, or at the time of hospital admission; documented by medical personnel or reported by the child's primary caregiver.

The 30 subjects who met definitional criteria for noninflicted head injury included 4 very young children injured during a motor vehicle crash (MVC). Two of these subjects were not properly restrained at the time, including a 1-month-old who was in his mothers' arms and a 7-month-old restrained in a car seat that was facing forward rather than backward. Both presented with acute, life-threatening clinical signs and with alterations of consciousness lasting >24 hours. Both experienced noncontact injury mechanisms—1 in isolation and the other with associated contact injuries. Both demonstrated deep brain injuries involving their subcortical brains. The 1-month-old infant died, the only fatality among our 30 subjects with noninflicted head injury. In contrast, the other 2 young children who sustained their cranial injuries during a MVC were still properly restrained facing backward in their car seats after their MVC. Both sustained a skull fracture without any visible intracranial or brain injuries. Neither experienced an impairment of consciousness.

A large body of literature supports a conclusion that short-distance, pediatric falls are generally benign.[36–41] Our results also support this conclusion. Six months after injury, our 26 subjects with noninflicted head injury attributed to a fall demonstrated normal MDI and GMQ scores (mean: 93.7 [SD: 11.8] and 102 [SD: 10.8], respectively). On the other hand, our data revealed a few interesting exceptions.

A 19½-month-old black child fell 6 to 10 feet onto concrete. His caregiver heard him fall, responded rapidly, and reported that he cried immediately but was readily consolable. Thirty minutes later, he developed an alteration of consciousness that lasted >6 hours and was associated with brief periods of flaccidity, unresponsiveness, seizure activity, and apnea without cyanosis. His cranial CT scan revealed only skull fracture without any visible, underlying intracranial or brain injury. He improved dramatically and was discharged on his second hospital day, but did not return 6 months postinjury for a follow-up assessment. This case seems to demonstrate

that delayed and significant clinical deterioration can occur after closed pediatric head trauma, even in the absence of visible intracranial injuries.

Four of our 26 subjects with noninflicted head injuries attributed to a fall manifested an impairment of consciousness. Seven of 26 revealed subdural hematoma (SDH) on neuroimaging studies. These 7 subjects included 3 with SDH that extended or originated from the interhemispheric region, indicative of a noncontact mechanism of injury. All 3 of these young children fell from a height of only 3 to 6 feet; manifested associated contact injuries; revealed only superficial, focal, cortical brain injuries; and experienced no alterations of consciousness. These cases seem to demonstrate that noncontact (ie, interhemispheric) SDH of minimal clinical significance can result from a short-distance fall and that the injury thresholds or biomechanical requirements for noncontact SDH and acute concussion, also a noncontact injury, are different.

Our study's primary limitations include its small sample size, a potential for sampling bias, and the limited number of subjects with follow-up neurodevelopmental assessments. Despite these limitations, our data revealed highly significant differences between comparison groups. With additional MDI and GMQ scores, we could have conducted the appropriate multivariable analyses to identify which variables are independent and significant predictors of neurodevelopmental outcome 6 months after head injury in this young age group.

In previously published comparative studies, a wide variety of research criteria were used to differentiate between inflicted and noninflicted pediatric head trauma. Some of these definitional criteria are fundamentally flawed. By incorporating elements that refer to specific cranial injuries, some authors introduced inappropriate circular logic (for example, many young victims of abuse manifest subdural bleeding. Therefore, this child with subdural bleeding must have been abused.) By incorporating definitional elements that compared a child's specific cranial injuries to the caregiver's specific explanation for those injuries, some authors introduced inappropriate biases (eg, Short falls do not cause subdural bleeding).

To overcome these deficiencies, in this prospective comparative study, we categorized the etiology of each subject's head trauma as inflicted, noninflicted, or undetermined by using the a priori criteria summarized in Table 1. These criteria incorporate the results of a recent physician survey[42] designed to identify widely acceptable definitional criteria for inflicted and noninflicted pediatric head trauma. Furthermore, these criteria judiciously avoid circular logic and other inherent biases. For example, in the absence of any other specific criteria for inflicted or noninflicted etiology, if the child's primary caregiver at the time of injury could not be interviewed, then cases with consistent documentation of an accidental head injury event were nevertheless classified as undetermined with respect to etiology. Similarly, in the absence of any other specific criteria for inflicted or noninflicted etiology, cases with a single, isolated, noncranial injury considered moderately or highly specific for abuse (see Table 1, footnote 1) were also classified as undetermined with respect to etiology.

Our study's primary strengths include its prospective, multicentered design and the a priori application of widely acceptable, definitional criteria for inflicted versus noninflicted etiology that are free of circular logic and other inherent biases. Applied to our data set, our definitional criteria for etiology categorized more cases as undetermined ($n = 13$) than inflicted ($n = 11$). This result serves to reinforce our impression that our a priori criteria for etiology are appropriately objective and conservative.

CONCLUSIONS

Compared with infants with noninflicted head trauma, young victims of inflicted head trauma experience more frequent noncontact injury mechanisms that can trigger deeper brain injuries, acute cardiorespiratory compromise, secondary diffuse cerebral hypoxia-ischemia, and worse outcomes. During infancy, etiology is a significant predictor of short-term neurodevelopmental outcome after head injury. Young victims of inflicted head trauma require thorough neurodevelopmental assessment and monitoring.

ACKNOWLEDGMENTS

This work was supported, in part, with resources provided by the National Institutes of Health (grants R21HD043351-01 and R24HD39361-05), the American Academy for Cerebral Palsy and Developmental Medicine, the University of Virginia Children's Hospital Research Fund, and PediBIRN.

In addition to the authors, the following PediBIRN participants and sites also made substantial contributions to research project design and acquisition of data: Michael A. Stoiko, MD, and Amy S. Coombs, RN (Michigan State University); Deborah E. Lowen, MD (University of Oklahoma); Nancy S. Harper, MD, and Carole Jenny, MD, MBA (Brown University); Bruce E. Herman, MD (University of Utah); and Suzanne M. Edmunds, MD (Wake Forest University). We thank Patrick Hartsfield and Teresa Olsen for vital assistance with data entry and management.

REFERENCES

1. Duhaime AC, Christian CW, Rorke LB, Zimmerman RA. Nonaccidental head injury in infants: the "shaken-baby syndrome." *N Engl J Med.* 1998;338:1822–1829
2. DiScala C, Osberg JS, Savage RC. Children hospitalized for traumatic brain injury: transition to postacute care. *J Head Trauma Rehabil.* 1997;12:1–10
3. Overpeck MD, Brenner RA, Trumble AC, Trifiletti LB, Beren-

des HW. Risk factors for infant homicide in the United States. *N Engl J Med.* 1998;339:1211–1216
4. Anderson V, Fenwick T, Manly T, Robertson I. Attentional skills following traumatic brain injury in childhood: a componential analysis. *Brain Inj.* 1998;12:937–939
5. Barlow K, Thompson E, Johnson D, Minns RA. The neurological outcome of non-accidental head injury. *Pediatr Rehabil.* 2004;7:195–203
6. Bonnier C, Nassogne MC, Evrard P. Outcome and prognosis of whiplash shaken infant syndrome; late consequences after a symptom-free interval. *Dev Med Child Neurol.* 1995;37:943–956
7. Duhaime AC, Christian C, Moss E, Seidl T. Long-term outcome in infants with the shaking-impact syndrome. *Pediatr Neurosurg.* 1996;24:292–298
8. Ewing-Cobbs L, Kramer L, Prasad M, et al. Neuroimaging, physical, and developmental findings after inflicted and noninflicted traumatic brain injury in young children. *Pediatrics.* 1998;102:300–307
9. Ewing-Cobbs L, Prasad M, Kramer L, Landry SH. Inflicted traumatic brain injury: relationship of developmental outcome to severity of injury. *Pediatr Neurosurg.* 2000;31:251–258
10. Hymel KP. Inflicted traumatic brain injury in infants and young children. *Infants Young Child.* 2002;15:57–65
11. Goldstein B, Kelly MM, Bruton D, Cox C. Inflicted versus accidental head injury in critically injured children. *Crit Care Med.* 1993;21:1328–1332
12. Haviland J, Russell RI. Outcome after severe non-accidental head injury. *Arch Dis Child.* 1997;77:504–507
13. Prasad MR, Ewing-Cobbs L, Swank PR, Kramer L. Predictors of outcome following traumatic brain injury in young children. *Pediatr Neurosurg.* 2002;36:64–74
14. Reece RM, Sege R. Childhood head injuries: accidental or inflicted? *Arch Pediatr Adolesc Med.* 2000;154:11–15
15. Bechtel K, Stoessel K, Leventhal JM, et al. Characteristics that distinguish accidental from abusive injury in hospitalized young children with head trauma. *Pediatrics.* 2004;114:165–168
16. Duhaime AC, Alario AJ, Lewander WJ, et al. Head injury in very young children: mechanisms, injury types, and ophthalmologic findings in 100 hospitalized patients younger than 2 years of age. *Pediatrics.* 1992;90:179–185
17. Feldman KW, Bethel R, Shugerman RP, Grossman DC, Grady MS, Ellenbogen RG. The cause of infant and toddler subdural hemorrhage: a prospective study. *Pediatrics.* 2001;108:636–646
18. Keenan HT, Runyan DK, Marshall SW, Nocera MA, Merten DF. A population-based comparison of clinical and outcome characteristics of young children with serious inflicted and noninflicted traumatic brain injury. *Pediatrics.* 2004;114:633–639
19. Levin AV. Retinal haemorrhage and child abuse. In: David TJ, ed. *Recent Advances in Pediatrics.* London, United Kingdom: Churchill Livingstone; 2000:151–219
20. Geddes JF, Whitwell HL, Graham DI. Traumatic axonal injury: practical issues for diagnosis in medicolegal cases. *Neuropathol Appl Neurobiol.* 2000;26:105–116
21. Geddes JF, Vowles GH, Hackshaw AK, Nickols CD, Scott IS, Whitwell HL. Neuropathology of inflicted head injury in children. II. Microscopic brain injury in infants. *Brain.* 2001;124:1299–1306
22. Geddes JF, Hackshaw AK, Vowles GH, Nickols CD, Whitwell HL. Neuropathology of inflicted head injury in children. I. Patterns of brain damage. *Brain.* 2001;124:1290–1298
23. Johnson DL, Boal D, Baule R. Role of apnea in nonaccidental head injury. *Pediatr Neurosurg.* 1995;23:305–310
24. Karandikar S, Coles L, Jayawant S, Kemp AM. The neurodevelopmental outcome in infants who have sustained a subdural haemorrhage from non-accidental head trauma. *Child Abuse Rev.* 2004;13:178–187
25. Duhaime AC, Bilaniuk L, Zimmerman R. The "big black brain": radiographic changes after severe inflicted head injury in infancy [abstract]. *J Neurotrauma.* 1993;10(suppl 1):S59
26. Zimmerman RA, Bilaniuk LT, Bruce D, Schut L, Uzzell B, Goldberg HI. Computed tomography of craniocerebral injury in the abused child. *Radiology.* 1979;130:687–690
27. Adelson PD, Clyde B, Kochanek PM, Wisniewski SR, Marion DW, Yonas H. Cerebrovascular response in infants and young children following severe traumatic brain injury. *Pediatr Neurosurg.* 1997;26:200–207
28. Bonnier C, Nassogne MC, Saint-Martin C, Mesples B, Kadhim H, Sebire G. Neuroimaging of intraparenchymal lesions predicts outcome in shaken baby syndrome. *Pediatrics.* 2003;112:808–814
29. Hans-Joachim F, Drommer S, Heyer R. Severe head injury in children: impact of risk factors on outcome. *J Trauma.* 1999;47:33–38
30. Jones PA, Andrews PJD, Easton VJ, Minns RA. Traumatic brain injury in childhood: intensive care times series data and outcome. *Br J Neurosurg.* 2003;17:29–39
31. Levin HS, Mendelsohn D, Lilly MA, et al. Magnetic resonance imaging in relation to functional outcome of pediatric closed head injury: a test of the Ommaya-Genarelli model. *Neurosurgery.* 1997;40:432–440
32. Biron D, Shelton D. Perpetrator accounts in infant abusive head trauma brought about by a shaking event. *Child Abuse Negl.* 2005;29:1347–1358
33. Starling SP, Patel S, Burke BL, Sirotnak AP, Stronks S, Rosquist P. Analysis of perpetrator admissions to inflicted traumatic brain injury in children. *Arch Pediatr Adolesc Med.* 2004;158:454–458
34. Donohoe M. Evidence-based medicine and shaken baby syndrome: part I—literature review, 1966–1998. *Am J Forensic Med Pathol.* 2003;24:239–242
35. Geddes JF, Plunkett J. The evidence base for shaken baby syndrome. *BMJ.* 2004;328:719–720
36. Chang A, Lugg MM, Nebedum A. Injuries among preschool children enrolled in day-care centers. *Pediatrics.* 1989;83:272–277
37. Helfer RE, Slovis TL, Black M. Injuries resulting when small children fall out of bed. *Pediatrics.* 1977;60:533–535
38. Landman PF, Landman GB. Accidental injuries in children in day-care centers. *Am J Dis Child.* 1987;141:292–293
39. Leland NL, Garrard J, Smith DK. Injuries to preschool-age children in day-care centers: a retrospective record review. *Am J Dis Child.* 1993;147:826–831
40. Nimityongskul P, Anderson LD. The likelihood of injuries when children fall out of bed. *J Pediatr Orthop.* 1987;7:184–186
41. Warrington SA, Wright CM. Accidents and resulting injuries in premobile infants: data from the ALSPAC study. *Arch Dis Child.* 2001;85:104–107
42. Laskey AL, Sheridan MJ, Hymel KP. Physicians' initial forensic impressions of hypothetical cases of pediatric traumatic brain injury. *Child Abuse Negl.* 2007; In press