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The Infant Whiplash-Shake Injury Syndrome: A Clinical and Pathological Study

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The cases of 13 infants (median age, 3 months) who sustained nonaccidental trauma were reviewed. All presented with profound neurological impairment, seizures, retinal hemorrhages, and intracranial subarachnoid and/or subdural hemorrhages. Of 8 infants who died, autopsy was performed on 6. No patient had a skull fracture, and only one had an extracalvarial contusion. Five of the 6 patients on whom autopsy was performed had injuries at the cervicomedullary junction consisting of sub- or epidural hematomas of the cervical spinal cord with proximal spinal cord contusions. The authors conclude that direct cranial trauma is not an essential element of the injury mechanism in young patients who sustain severe whiplash-shake injuries. In addition to the classic injuries reported to occur with the shaken-baby syndrome, hemorrhages and contusions of the high cervical spinal cord may contribute to morbidity and mortality. (*Neurosurgery* 24:536–540, 1989)

Key words: Cervicomedullary junction, Child abuse, Shaken-baby syndrome, Spinal cord contusion, Whiplash-shake injury

Infant child abuse is not uncommon. The "shaken-baby syndrome," characterized by retinal hemorrhages, subdural and/or subarachnoid hemorrhages, and minimal or absent signs of external craniofacial trauma, has been reviewed in the literature and studied both clinically and in laboratory models (1–8). The issue with respect to the whiplash-shake injury is whether shaking alone can create sufficient acceleration-deceleration forces to cause severe neurological injury and death (4, 7). Several investigators have postulated that rapid and repetitive flexion and extension of the infant head and neck relative to the fixed torso can result in shearing injuries of vascular structures, intracranial hemorrhages, and death. Recent work by Duhaime et al. (3), which included an analysis of a laboratory biomechanical model, disputes this mechanism of injury and indicates that cranial-impact trauma is an essential component of the pathophysiology of the injury syndrome.

To investigate this problem futher, we examined all cases of nonaccidental head injuries in infants treated at this institution during the last 6 years. Of particular interest were those infant child-abuse patients who had neurological symptoms but no evidence of direct cranial trauma.

METHODS

The clinical histories, physical examinations, and radiographic studies of 36 infants who sustained nonaccidental head injuries were reviewed. Outcome and/or autopsy data, where available, were compiled. In recent years, the authors interviewed the perpetrators of these injuries and educated social workers, pediatricians, and psychologists (who also interviewed the perpetrators) involved in the assessment of these cases regarding the nature of our investigation. Their notes on each case were reviewed for presumed cause and mechanism of injury. In addition, the authors and the coroner specifically directed the physicians performing the autopsies to include both the cranial and cervical spine compartments.

RESULTS

Of 36 infants identified as having sustained primary neurological injury from nonaccidental trauma during the last 6

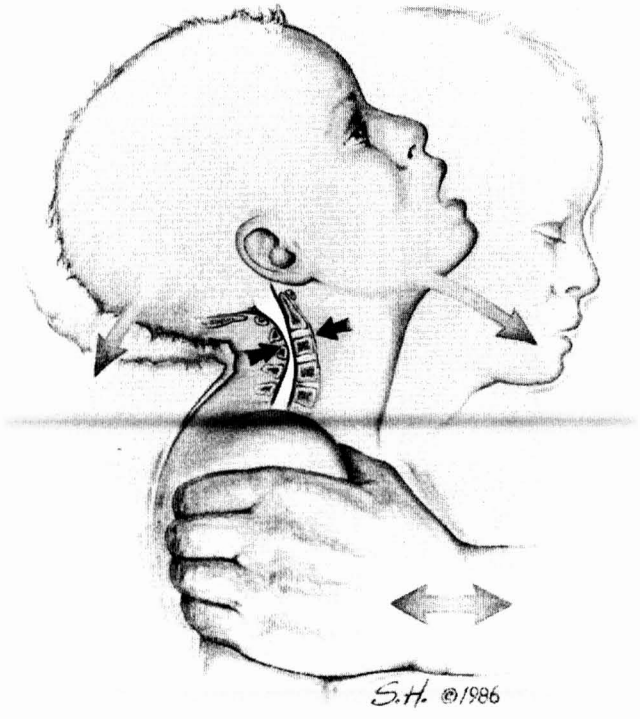

FIG. 1. Artist's representation of the mechanism of whiplash-shake injury with compromise of cervicomedullary neural structures.

TABLE 1
Presenting Features

Patient Number	Age (mo)	Sex (M/F)	Level of Consciousness	Seizures	Retinal Hemorrhage	Hematocrit
1	1.5	M	Comatose	Generalized	Bilateral	28
2	1.5	M	Comatose	Generalized	Bilateral	19
3	2	M	Lethargic	Generalized	Unilateral	22
4	2	M	Comatose	Generalized (status)	Bilateral	23
5	2.5	F	Comatose	Generalized	Bilateral	27
6	3	M	Lethargic	Generalized	Unilateral	21
7	3	F	Lethargic	Generalized (status)	Bilateral	29
8	5	M	Lethargic	Generalized	Bilateral	26
9	7	F	Lethargic	Focal motor	Bilateral	25
10	7	F	Comatose	Generalized	Bilateral	31
11	13	F	Comatose	Generalized	Bilateral	34
12	13	M	Lethargic	Generalized (status)	Bilateral	28
13	14	F	Comatose	Generalized	Bilateral	28

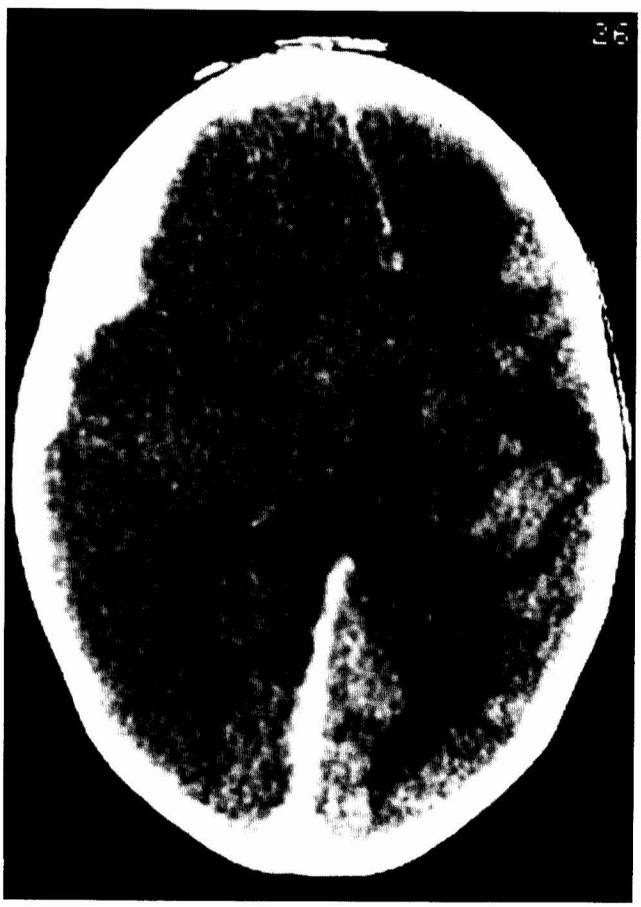

FIG. 2. CT scan revealing marked injury in the right hemisphere with swelling and shift of the midline. A frontal subdural hemorrhage is present.

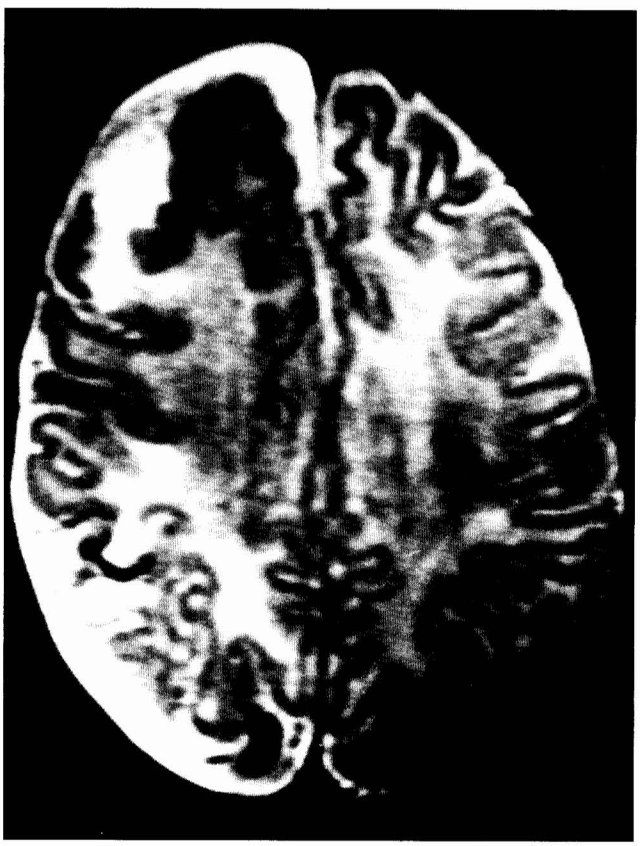

FIG. 3. Patient 12. MRI scan showing multiple contusions and accumulation of subarachnoid and subdural blood.

years, 13 patients were suspected of having a clinical history of shake injury without direct cranial trauma, as determined by multiple interviewers and cross-referenced notes made by those individuals investigating each case. In addition, these 13 patients had no physical evidence of external craniofacial trauma or skull/facial fractures, as determined by standard radiographs or computed tomographic (CT) studies. The median age of the patients was 3 months (range, 1.5 to 14 months). There were 7 boys and 6 girls. All 13 patients in this presumed isolated whiplash-shake subgroup (Fig. 1) presented with a decreased level of consciousness (severe), seizures, and retinal hemorrhages (Table 1). All had a low hematocrit at the time of presentation, and all 13 had evidence of subarachnoid and/or subdural hemorrhages on CT scans (Fig. 2). Nine had contusions in the cerebral hemispheres (Fig. 3 and Table 2).

Eight of the 13 patients died. The 5 surviving patients had a clinical pattern of profound encephalopathy. Complete autopsies were performed on 6 of the 8 infants who died (Table 3). All 6 patients on whom autopsy was performed had cranial subdural hemorrhages, cerebral contusions, and marked swelling with herniation. Only one patient had evidence of an extracalvarial contusion. This was identified in the fascia and

musculature of the temporalis muscle, but was not large and was not associated with a scalp injury or skull frature. No patient had a skull fracture. Five of the 6 patients had epidural and/or subdural hematomas of the spinal cord at the cervicomedullary junction (Fig. 4), and 4 of 6 patients had evidence of ventral spinal contusions at high cervical levels, (Fig. 5).

DISCUSSION

Although the "shaken-baby syndrome" is well known to neurological surgeons, the precise mechanisms of neurological injury in this syndrome remain elusive. It is often difficult to obtain a reliable history of infant abuse. The absence of a specific history of infant shaking in many cases has hindered clinicopathological correlation. Laboratory studies of acceleration injury have revealed that the angular acceleration and velocity associated with shaking are below the thresholds that cause intracranial injury (2, 3). Several investigators have concluded that direct cranial impact trauma is an essential component of the injury mechanism that leads to intracranial-extracranial hemorrhage, axonal injury, and cerebral contusions (3, 5, 6).

Our review identified 36 infants who sustained nonaccidental head injuries, 13 of whom met two specific criteria: (1) a documented history of infant shaking as admitted by the parent-boyfriend-babysitter perpetrator, and (2) no historical, clinical, or radiographic evidence of direct impact trauma to the craniofacial region. We consider this select population of nonaccidental cranial trauma patients (36% of the total group) to be an isolated whiplash-shake injury subgroup.

All of our patients were very young infants (14 months of age or younger; median, 3 months). Several anatomical and biomechanical features of young infants increase the susceptibility of these patients to neurological injury caused by the

TABLE 2
Radiographic Findings

Patient Number	Computed Tomographic Findings[a]			Death
	SAH	SDH	Contusions	
1	+	++	−	Yes
2	−	+++	+	Yes
3	+	+	−	No
4	+	++	+	Yes
5	+	++	+	Yes
6	+	+	+	No
7	−	++	−	No
8	+	++	+	No
9	+	+	+	No
10	−	+++	+	Yes
11	+	++	+	Yes
12	+	+++	+	Yes
13	+	++	−	Yes
Total	10	13	9	8

[a] SAH, subarachnoid hemorrhage; SDH, subdural hemorrhage; +, present; −, absent; ++, moderate; +++, severe.

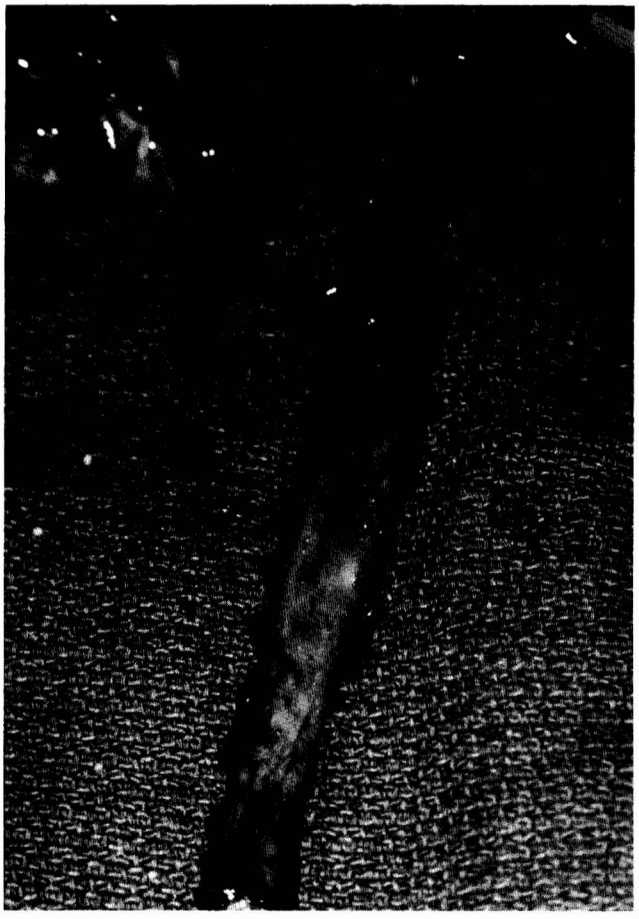

FIG. 4. Autopsy photo revealing subdural hemorrhages in the high dorsal cervical cord (the cerebellum has been retracted upward).

TABLE 3
Autopsy Data[a]

Patient Number	Extracranial Contusions	Skull Fracture	Cranial SDH	Cerebral Contusions/ Herniation	Cervical Spinal Cord Hematoma		
					Epidural	Subdural	Contusions
1	−	−	+	+	+	+	+
2	−	−	+	+	+	−	−
5	−	−	+	+	+	+	+
10	−	−	+	+	−	−	−
11	−	−	+	+	+	+	+
13	+	−	+	+	+	+	+
Total 6	1	0	6	6	5	4	4

[a] SDH, Subdural hematoma; −, absent; +, present.

FIG. 5. Subarachnoid blood and spinal cord contusions (*arrows*) at the ventral cervicomedullary junction

whiplash-shake mechanism without the addition of direct calvarial impact trauma. These include the large size of the infant's head with respect to its torso; elastic, underdeveloped ligaments; weak, underdeveloped paraspinous/neck musculature; shallow, horizontally oriented cervical facet joints; incompletely ossified cervical vertebrae; a soft, compliant calvaria; and large subarachnoid spaces (1, 7, 8).

Our data do not conclusively demonstrate that severe neurological trauma can occur with rapid, forceful, repetitive acceleration-deceleration of the head and neck with respect to the torso. Because most of these assaults are not witnessed, doubt remains about the true mechanism of these injuries. However, to the best of our abilities and those of the other professionals investigating these cases, we were able to document that each of the 13 patients had a history of whiplash-shake injury without direct impact trauma. Supporting the history in each case is the fact that none of the patients had clinical signs or radiographic evidence of craniofacial trauma. That 5 of 6 patients on whom an autopsy was performed had no postmortem evidence of direct cranial trauma further supports this conclusion.

The discrepancies between this review and that recently published by Duhaime et al. (3) may be explained in part by the younger age of the patients in our series (median age, 3 months; range, 1.5 to 14 months) versus theirs (median age, 10 months; range, 3 to 24 months). The high incidence of subdural hematoma in our 13 whiplash-shake injury patients and the high incidence of cervical cord injury in the 6 patients in whom autopsy was performed presumably also relates to the very young, immature patients in the population in our study.

Despite their conclusion that direct-impact cranial trauma is required to create the injuries associated with the shaken-baby syndrome, only 60% of the 50 patients Duhaime et al. (3) chronicled had clinical or radiographic evidence of cranial impact trauma. Of the 13 patients in whom autopsies were performed, only 5 had evidence of skull fracture. We do not dispute their conclusion—based on a thorough clinical and pathological review accompanied by laboratory acceleration analysis—that most infants diagnosed with the shaken-baby syndrome have sustained some form of direct cranial trauma. We do, however, underscore the point that direct cranial impact is *not always* required. We believe that a subgroup of patients, particularly the very young, may sustain severe neurological morbidity and mortality from rapid acceleration-deceleration injuries incurred from a whiplash-shake mechanism alone.

Our autopsy results revealed two new pathological findings seen in virtually every patient: sub- or epidural hematomas on the high cervical spinal cord and cervical spinal cord contusions. These findings also support a whiplash-shake mechanism of injury (i.e., rapid acceleration-deceleration of a heavy mobile mass (the head) affixed to a relatively fixed torso).

We conclude that a whiplash-shake injury can cause severe neurological injury without direct-impact cranial trauma, at least in a small subgroup of patients. These patients are typically very young infants who demonstrate profound neurological impairment and seizures. In addition to the classic injuries reported to occur with the shaken-baby syndrome (retinal hemorrhages, subarachnoid and/or subdural hematomas), hemorrhages and contusions of the high cervical spinal cord may contribute to morbidity and mortality in the injured patient.

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REFERENCES

1. 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 54:396–403, 1974.
2. 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 124:161–9, 1972.
3. Duhaime AC, Gennarelli TA, Thibault CE, Bruce DA, Margulies SS, Wiser R: The shaken baby syndrome. A clinical, pathological, and biomechanical study. J Neurosurg 66:409–15, 1987.
4. Guthkelch AN: Infantile subdural haematoma and its relationship to whiplash injuries. Br Med J 2:430–1, 1971.
5. Hahn YS, Raimondi AJ, McLone DG, Yamanouchi Y: Traumatic mechanisms of head injury in child abuse. Childs Brain 10:229–41, 1983.
6. Hanigan WC, Peterson FA, Njus G: Tin ear syndrome: Rotational acceleration in pediatric head injuries. Pediatrics 80:618–22, 1987.
7. McClelland CQ, Rekate H, Kaufman B, Persse L: Cerebral injury in child abuse: A changing profile. Childs Brain 7:225–3, 1980.
8. Merten DF, Osborne DRS: Craniocerebral trauma in the child abuse syndrome. Pediatr Ann 12:882–7, 1983.

COMMENTS

The authors have very carefully examined a group of infants with trauma. In spite of their detailed analysis, I do not feel that they have conclusively answered the question of whether severe neurological injury can occur without direct cranial impact trauma. The authors do, however, add the significant observation that there can be damage at the cervicomedullary region. Too often, the brain is removed at the cervicomedullary junction and this region is not given gross or histological examination. This area should be routinely examined in all children with inflicted trauma.

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Proof of assault in suspected incidents of child abuse is a frustrating business. Extrapolation of data to determine mechanisms of injury from clinical evidence superimposes another degree of difficulty. Under these conditions, it is not surprising that clinical descriptions, syndromes, and their interpretation require perpeptual amendment. Drs. Hadley, Sonntag, Rekate, and Murphy have forthrightly and simply marshalled some evidence that further adds to the expanding knowledge of the neurosurgical aspect of child abuse.

Aside from the mild controversy on the issue of the mechanism of head injury in such patients, this small series has some interesting features. The high incidence of subdural hematoma is somewhat surprising, and probably reflects careful review of scans and autopsy material. The striking preponderance of pathological indications at the cervicomedullary junction and cervical spine has previously received little attention in the literature. These injuries may be associated with apnea and may contribute to sudden infant death syndrome, which often escapes the attention of neurosurgical services and hospital care systems.

Although substantiation of the mechanism of injury and lack of direct blows to the head is sparse in this paper, the case material and interpretations of the authors are reasonably compatible with previous descriptions of the infant whiplash-shake injury syndrome. My clinical experience supports the conclusion that, at least in a small subgroup of very young patients, severe neurological morbidity and mortality can result from rapid acceleration-deceleration injuries without direct blows to the head. Neither this nor previous publications represent the final word on this subject for neurosurgeons.

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