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The shaken baby syndrome

A clinical, pathological, and biomechanical study

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Because a history of shaking is often lacking in the so-called "shaken baby syndrome," diagnosis is usually based on a constellation of clinical and radiographic findings. Forty-eight cases of infants and young children with this diagnosis seen between 1978 and 1985 at the Children's Hospital of Philadelphia were reviewed. All patients had a presenting history thought to be suspicious for child abuse, and either retinal hemorrhages with subdural or subarachnoid hemorrhages or a computerized tomography scan showing subdural or subarachnoid hemorrhages with interhemispheric blood. The physical examination and presence of associated trauma were analyzed; autopsy findings for the 13 fatalities were reviewed. All fatal cases had signs of blunt impact to the head, although in more than half of them these findings were noted only at autopsy. All deaths were associated with uncontrollably increased intracranial pressure.

Models of 1-month-old infants with various neck and skull parameters were instrumented with accelerometers and shaken and impacted against padded or unpadded surfaces. Angular accelerations for shakes were smaller than those for impacts by a factor of 50. All shakes fell below injury thresholds established for subhuman primates scaled for the same brain mass, while impacts spanned concussion, subdural hematoma, and diffuse axonal injury ranges. It was concluded that severe head injuries commonly diagnosed as shaking injuries require impact to occur and that shaking alone in an otherwise normal baby is unlikely to cause the shaken baby syndrome.

KEY WORDS • **shaken baby syndrome** • **head injury** • **child abuse**

THE term "whiplash shaken baby syndrome" was coined by Caffey[3] to describe a clinicopathological entity occurring in infants characterized by retinal hemorrhages, subdural and/or subarachnoid hemorrhages, and minimal or absent signs of external trauma. Because a nursemaid admitted that she had held several such children by the arms or trunk and shaken them, the mechanism of injury was presumed to be a whiplash-type motion of the head, resulting in tearing of the bridging veins. Such an injury was believed to be frequently associated with fatalities in infantile child abuse and has been postulated as a cause of developmental delay in survivors.[4,15]

While the term "shaken baby syndrome" has become well entrenched in the literature of child abuse, it is characteristic of the syndrome that a history of shaking in such cases is usually lacking.[12] Shaking is often assumed, therefore, on the basis of a constellation of clinical findings and on the computerized tomography (CT) picture of subarachnoid and subdural hematomas, particularly in the posterior interhemispheric fissure.[17] Because of the ambiguous circumstances of such injuries, medicolegal questions are particularly troublesome, and the neurosurgeon is often consulted to give an opinion as to whether the findings are consistent with child abuse or accidental injury.

This paper reviews all cases of the shaken baby syndrome seen at the Children's Hospital of Philadelphia (CHOP) between January, 1978, and March, 1985. To better study the mechanism of injury, autopsy results in all fatal cases were reviewed, and the biomechanics of this injury were studied in a series of infant models. Based on these observations, we believe that shaking alone does not produce the shaken baby syndrome.

Clinical Studies

Clinical Material and Methods

All reports submitted to the Suspected Child Abuse and Neglect team were reviewed. Since house officers

TABLE 1

Initial clinical criteria for diagnosis of shaken baby syndrome

Diagnosis*	Cases		No. of Deaths
	No.	Percent	
retinal hemorrhage + SAH or SDH	29	60	5
retinal hemorrhage + SAH & SDH	10	21	5
bilateral chronic SDH	3	6	0
SAH &/or SDH & interhemispheric blood on CT	6	13	3
total	48	100	13

* SAH = subarachnoid hemorrhage; SDH = subdural hemorrhage; CT = computerized tomography.

TABLE 2

Best history in 48 cases of shaken baby syndrome

Etiology	Cases	
	No.	Percent
shaking only	1	2
fall or accidental blunt trauma	15	31
strike or fall plus shaking	10	21
strike only	3	6
trauma or shaking denied, caretakers in attendance	8	17
history unknown, caretakers not in attendance	10	21
cardiopulmonary resuscitation	1	2

TABLE 3

Trauma associated with shaken baby syndrome in 48 cases

Associated Trauma	Cases	
	No.	Percent
no evidence of blunt impact to head	18	37.5
no extracranial trauma	12	25.0
additional extracranial trauma	6	12.5
acute	3	6.25
old trauma only	3	6.25
evidence of blunt impact to head	30	62.5
skull fractures	12	25.0
cranial soft-tissue contusions	18	37.5
additional extracranial trauma	18	37.5
acute	15	31.25
old trauma only	3	6.25

and emergency room personnel are well trained in recognizing the clinical manifestations associated with this syndrome, it is considered that essentially all cases seen at CHOP are reported to this group.

Suspicion of shaking was based on history, clinical findings, and CT data. All subjects met the following criteria: presence of retinal hemorrhages with subdural and/or subarachnoid hemorrhages, bilateral chronic subdural hematomas, or a CT scan showing subdural or subarachnoid hemorrhages with interhemispheric blood. In addition, all patients were judged to have histories suggestive of child abuse or neglect; well-documented, witnessed accidental trauma was excluded. Histories were obtained from several interviews with caretakers by physicians, social workers, and in some cases law enforcement agents. Caretakers were routinely asked specifically about shaking.

Associated trauma data were obtained from physical examination, skull radiographs, CT scans, and skeletal surveys. All fatal cases were examined by the Philadelphia Medical Examiner, and pathology data were obtained from that office.

Results

Fifty-seven patients with suspected shake injury were identified. Of these, detailed clinical information was available in 48 cases. These patients ranged in age from 1 month to 2 years (mean 7.85 months). Thirty-one patients were male (65%). There were 13 fatalities (27%). Initial clinical criteria for diagnosis of the shaken baby syndrome are listed in Table 1. Thirty-nine patients (81%) had retinal hemorrhages plus subarachnoid and/or subdural hemorrhages. The remainder had bilateral chronic subdural hematomas (6%) or the above-mentioned CT findings without retinal hemorrhages (13%).

The most common presenting complaints were lethargy, breathing difficulty, irritability, poor feeding, and seizures. Best history is listed in Table 2; the most common histories were accidental blunt trauma (usually a fall) in 15 (31%) and blunt trauma plus shaking in 10 (21%); trauma and shaking were denied in eight (17%). In three cases (6%) the child was struck by the caretaker. In eight additional cases the history was unknown, usually because the child was left alone or with a babysitter. There were two cases (4%) with no history to explain the present findings, but both children were known to have been abused previously or subsequently. One case was associated with cardiopulmonary resuscitation (2%). In only one case was a history of shaking alone obtained; this child was reportedly shaken when she appeared to have difficulty in breathing associated with a respiratory infection.

Associated trauma observed clinically, radiographically, or at autopsy is listed in Table 3. The presence of scalp contusion, subgaleal or subperiosteal hemorrhage, and/or skull fracture was considered evidence of blunt impact to the head. Twelve cases (25%) had intracranial findings associated with the shaken baby syndrome alone, with no findings of associated blunt trauma to the head and no extracranial trauma. Six additional cases (13%) had the syndrome without signs of blunt head trauma but did have associated extracranial trauma. Thirty cases (63%) had findings of blunt impact to the head in addition to the intracranial findings of the shaken baby syndrome. Of these, 12 (25%) had skull fractures and 18 (38%) had significant cranial soft-tissue contusions. Most of the fractures were in the occipital or parieto-occipital region.

Clinical history, physical findings, hospital course, intracranial pressure (ICP, when measured), and pathological findings of the 13 fatalities are listed in Tables

TABLE 4

*Clinical and pathological findings in 13 fatal cases of shaken baby syndrome**

Factor	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6	Case 7	Case 8	Case 9	Case 10	Case 11	Case 12	Case 13
age (mos)	24	7	3	22	11	9	8	5	10	13	24	4	19
sex	F	M	M	M	F	F	F	M	F	M	M	M	F
history													
fall or hit	+		+	+	+			+	+	+	+	+	+
shaking			+								+		+
trauma denied													
unknown		+				+	+						
initial examination													
unresponsive	+	+	+	+	+	+	+	+	+	+	+	+	+
retinal hemorrhages	+	+		+			+	+	+	+	+	+	
cranial impact	+			+						+	+	+	+
extracranial trauma	+			+	+						+	+	
intracranial pressure	↑↑	NM	↑↑	↑↑	↑↑	NM	↑↑	NM	↑↑	NM	↑↑	↑↑	↑↑
survival time (days)	2	2	7	2	3	2	2	1	1	1	4	1	1
pathology													
cranial contusions	+	+	+	+	+	+	+	+	+	+	+	+	+
skull fracture(s)	++			++	++							+	+
subdural hematoma	+	+	+	+	+	+	+	+	+	+	+	+	+
subarachnoid hemorrhage	+	+	+	+	+	+	+	+	+	+	+	+	+
hemispheric contusions	+	+	+			+		+					+
white matter tears	+					+			+				+
diffuse brain swelling	+	+	+	+	+	+	+	+	+	+	+	+	+

* ↑↑ = increased; NM = not measured; + = factor present; ++ = severe.

4 and 5. Mean age in this group was 12.23 months; 54% were male. All of these children arrived at the hospital in an essentially unresponsive state, and all died from the effects of uncontrollably increased ICP associated with massive brain swelling. In only one case was a subdural hematoma thought to be of significant size to warrant surgical intervention, and drainage was ineffective in controlling elevated ICP.

Pathological examination showed that all of the children who died had evidence of blunt head trauma. Eight had soft-tissue contusions and five had contusions and skull fractures. In seven cases, however, impact findings were noted only at autopsy, and had not been apparent prior to death. All fatal cases had subdural and subarachnoid bleeding. Focal cerebral contusions and lacerations occurred in six. Microscopic examination was performed in three cases and showed corpus callosum hemorrhages, cortical laminar necrosis, or white matter hemorrhages. All children had diffuse and usually massive brain swelling.

Biomechanical Studies

Whole Infant Models

To test the hypothesis that infants are particularly susceptible to injury from shaking because of a relatively large head and weak neck, we constructed models of 1-month-old infants that were implanted with an accelerometer to measure the results of shaking or impact manipulations. Since the mechanical properties of the infant neck have not been studied, three models were built with different neck structures in order to include the range of limiting conditions that might exist in the live infant. Both a fixed center of rotation with zero resistance (hinge model) and moving centers of rotation with low and moderate resistance (rubber neck models) were tested.

Experimental Methods

The heads and bodies of the models were adapted from Just Born dolls. Head circumference was 36 cm, coronal width was 10 cm, anteroposterior diameter was 10.75 cm, and height from vertex to base (calculated from a line drawn from chin to caudal occiput) was 9.0 cm; values were comparable to human infants. Brain weight for an infant of this size was assumed to be 500 gm.[1] The ideal weight of the head was estimated by balance-weight measurements of several infants with an average age of 1 month, and was 770 to 870 gm. The heads of the models were tightly filled with cotton, with water added until the desired weight range was reached. The water was absorbed by the cotton and distributed so that no sloshing of the contents occurred. The heads were reweighed after neck insertion and sealing and at the end of all experiments.

Neck length from the skull base to the T-1 vertebra

TABLE 5

Summary of findings in 13 fatal cases of shaken baby syndrome

Factor	Finding
age (mos)	
mean	12.23
range	3–24
sex M/F	7/6
history	
fall or hit (three with shaking)	10
unknown	3
initial examination	
unresponsive	13
retinal hemorrhages	9
cranial impact	6
extracranial trauma	5
intracranial pressure	
measured, unable to control	9
not measured	4
survival time (days)	
range	1–7
mean	2.2
pathology	
cranial contusions	13
skull fractures(s)	5
subdural hematomas (one requiring surgery)	13
subarachnoid hemorrhage	13
unilateral	3
diffuse	3
multifocal	7
hemispheric contusions	6
diffuse, multiple	3
focal, coup-contrecoup	3
white matter tears	4
gross	2
microscopic	2
diffuse brain swelling (11 with herniation evident)	13

TABLE 6

Mean acceleration and time course of shakes and impacts in all models

Manipulation	No.	Peak Tangential Acceleration (G)	Time (msec)	Angular Velocity (radians/sec)	Angular Acceleration (radians/sec^2)
shakes	69	9.29	106.6	60.68	1138.54
impacts	60	428.18	20.9	548.63	52,475.70

TABLE 7

Effects of neck condition and "skull" on mean peak tangential acceleration and time course of shakes and impacts

Variant	Shakes		Impacts	
	Acceleration (G)	Time (msec)	Acceleration (G)	Time (msec)
hinge neck	13.85	92.7	423.42	18.6
flexible rubber neck	5.70	93.3	427.78	21.4
stiff rubber neck	7.02	130.5	433.33	22.8
skull	9.86	107.4	436.12	20.2
no skull	8.89	103.5	427.04	21.6

TABLE 8

Effect of impact surface on mean peak tangential acceleration and time course

Surface of Impact	Acceleration (G)	Time (msec)
padded surface	380.60	24.22
metal bar	489.51	17.13

was measured from lateral neck films of several normal infants with an average age of 1 month and ranged from 3.5 to 4.5 cm; all models were therefore given neck lengths of 4.0 cm. Necks were embedded in Castolite resin* superiorly, which was also used to seal the head. The interior part of the neck was secured in dental stone.† The stuffed body was then replaced around the dental stone "thorax," with lead weights added as necessary to the thorax to reach a total body weight of 3 to 4 kg. Arms and legs were not weighted, so the slightly low total weight for age reflects an attempt to approximate trunk:head weight ratios.

Model 1 had a hinge neck made from a 360° steel hinge, 3.6 cm in width, placed in the horizontal plane to allow complete anteroposterior angulation of the head. The center of rotation was 3.3 cm below the estimated level of the skull base (approximating at the C-6 vertebral level). Model 2 had a 1.9-cm diameter hollow rubber neck with a 0.8-cm lumen. This neck did not support the weight of the head in the upright position but did not kink when the head was allowed to fall unsupported. Model 3 had a 2.9-cm rubber neck with a 1.2-cm lumen. This neck was able to support the head in the vertical position but allowed full passive movement of the head. In all models, head motion was limited in the anteroposterior direction by the occiput striking the upper back and the chin striking the chest.

To test for the effect of the deformability of the model heads on impact, all models were tested with and without an external "pseudoskull" made from thermoplastic.‡ This "skull" was 1/8 in. thick and was molded to the occipital, parietal, temporal, and posterior frontal areas, with the facial area uncovered. The "skulls" weighed 170 to 200 gm.

Data were recorded from a piezoelectric accelerometer§ embedded in a small piece of thermoplastic and attached to the vertex in a coronal plane through the

* Resin manufactured by Buehler Ltd., Evanston, Illinois.

† Dental stone, Glastone Type IV, manufactured by Ransom and Randolph Co., Toledo, Ohio

‡ Polyform thermoplastic manufactured by Rolyan Medical Products, Menomonee Falls, Wisconsin.

§ Accelerometer manufactured by Endevco Corp., San Juan Capistrano, California.

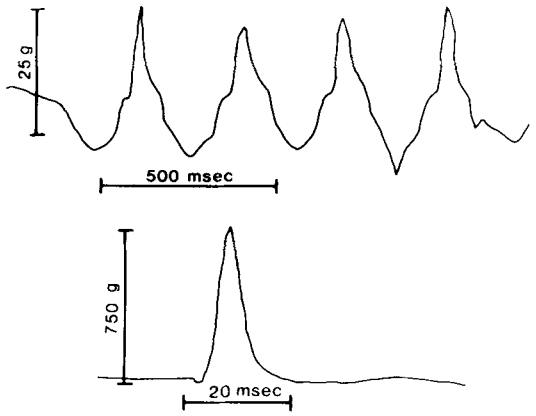

FIG. 1. Representative tangential acceleration traces for infant models undergoing shake *(upper)* and impact *(lower)* manipulations. While manipulations of the infant models were performed as described, with a series of shakes followed by an impact, the magnitude of the impact accelerations was so much greater than that associated with the shakes that different scales are used to display the respective acceleration traces.

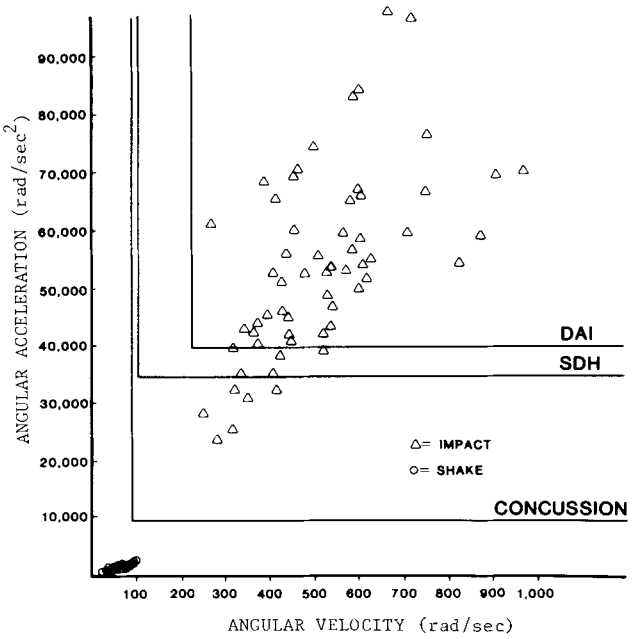

FIG. 2. Angular acceleration versus angular velocity for shakes and impacts, with injury thresholds from primate experiments scaled to 500-gm brain weight. DAI = diffuse axonal injury; SDH = subdural hematoma.

center of the neck. Each model was subjected to repetitive violent shaking, allowing the head to travel its full excursion several times, by adult male and female experimenters. The models were held by the thorax facing the experimenter and were shaken in the anteroposterior plane, since this is the motion most commonly described in the shaken baby syndrome. At the end of each series of shakes the occiput was impacted against either a metal bar or a padded surface. Each model was tested at least 20 times. Acceleration traces were amplified and recorded.‖

Angular accelerations were calculated from the measured peak tangential accelerations by using C-6 as the center of rotation in all cases. Angular velocity was calculated as the time integral of the acceleration curve. Translational forces were assumed to be minimal.

Results

The data were collected from 69 shaking episodes ("shakes") and 60 "impacts." Typical tangential acceleration traces for shake and impact manipulations are shown in Fig. 1. The criterion for significant difference was $p < 0.01$ in all cases.

Shakes Versus Impacts. Angular acceleration and angular velocity for each shake and impact are shown in Fig. 2. Mean peak tangential acceleration for 69 shaking episodes was 9.29 G; mean peak tangential acceleration for 60 impacts was 428.18 G (Table 6). The accelerations due to impact are significantly greater than those obtained by shaking ($p < 0.0001$); on the average, impact accelerations exceed shake accelerations by a factor of nearly 50 times. Mean time interval for shakes was 106.6 msec and for impacts was 20.9 msec. This difference is significant at the $p = 0.001$ level.

Effects of Neck Condition. Mean tangential accelerations and time courses for shakes and impacts for each neck condition are presented in Table 7. There is no significant difference between the hinge neck, the flexible rubber neck, and the stiff rubber neck in the mean acceleration resulting from impacts (423.4, 427.8, and 433.3 G, respectively) or in the mean time course (18.6, 21.4, and 22.8 msec, respectively). With shakes, the more flexible hinge neck is associated with higher accelerations (mean 13.85 G) than the two rubber neck models (mean 5.7 and 7.0 G) ($p < 0.001$). There is an inverse relationship between neck stiffness and time duration of a shake: the stiff rubber neck was associated with a longer time course than the more flexible rubber neck (130.5 msec and 93.3 msec, respectively) ($p < 0.001$).

Effects of "Skull." The presence of a hard thermoplastic "skull" did not change the magnitude or time course of accelerations associated with shaking of the models. The acceleration magnitude and time course were also unchanged when the models were impacted. These data are shown in Table 7.

Effects of Impact Surface. Impact against a padded surface was associated with significantly smaller acceleration (mean 380.6 G) and longer time course (mean 24.22 msec) than that against a metal bar (mean 489.5 G and 17.13 msec) ($p < 0.001$). Data are shown in Table 8.

‖ Shock amplifier, Model 2740 A, and pulse memory unit, Model 2743, manufactured by Endevco Corp., San Juan Capistrano, California.

Discussion

Clinical head injury can be classified into two major categories according to the distribution of pathological damage, whether focal or diffuse.[10] Such a distinction is important for treatment and prognosis, as well as for establishing the biomechanical conditions necessary to produce a given injury type. It has been established both experimentally and clinically that most focal injuries are associated with impact loading, resulting in contact phenomena, while diffuse injuries are associated with impulsive loading conditions resulting from acceleration-deceleration phenomena.[6] Damage to the brain occurs as a result of these biomechanical forces and from the secondary effects of ischemia due to altered autoregulation or brain swelling.

The shaken baby syndrome has been postulated to result from the effects of nonimpact acceleration-deceleration forces. It has been suggested that the back and forth movement of the head alone is sufficient to cause tearing of bridging veins, resultant subdural hematomas, and death.[8,13] The relatively large size of an infant's head, weakness of the neck musculature, softness of the skull, relatively large subarachnoid space, and high water content of the brain have been postulated to contribute to the susceptibility of shaking injuries in infants.[4,14]

While shaking alone has been considered sufficient to cause a fatal injury, the usual lack of history of the true mechanism of injury in these cases has hampered accurate clinicopathological correlations. It is of interest, however, that in a recent series of fatal cases of infantile head injuries from suspected child abuse,[5] white matter tears were found similar to those described by Lindenberg and Freytag[11] in blunt trauma in infancy. In addition, lesions in the distribution typical of diffuse axonal injury, like those found in adult head injury and in subhuman primates subjected to high acceleration-deceleration injury,[7] were described in some cases. In fact, at least one of Caffey's original cases[3] included "lacerations of the cerebral parenchyma." Shaking alone was the presumed mechanism of these injuries.

As experience has accumulated in experimental angular acceleration injury it has become clear that, besides the magnitude of the acceleration, another important biomechanical factor influencing injury type is the time interval over which the acceleration occurs. Thus, large angular accelerations occurring over shorter time periods tend to result in subdural hematoma, while longer intervals are associated with diffuse axonal injury.[6] A tolerance scale relating these two factors to resultant injury has been developed for the subhuman primate by Thibault and Gennarelli.[16] Values above certain critical limits result in a particular type of injury such as concussion, subdural hematoma, or diffuse axonal injury. When such a curve is scaled for the brain mass of an infant the size of our models, it can be seen that the angular acceleration and velocity associated with shaking occurs well below the injury range, while the values for impacts span concussion, subdural, and diffuse axonal injury ranges (Fig. 2). This was true for all neck conditions with and without skulls. A padded surface decreases the magnitude of acceleration and lengthens the time course to some extent, but these impacts also fall in the injury range.

These results are consistent with the observation that the fatal cases of the shaken baby syndrome in this series were all associated with evidence of blunt impact to the head. This preponderance of blunt trauma has also been found in at least one other series of nonaccidental head trauma in childhood in which the mechanism of injury was investigated.[9] It is of interest that in more than half of our fatal cases, no evidence of external trauma was noted on the initial physical examination, which helped to contribute to the diagnosis of "shaken baby syndrome." Skull fractures and scalp contusions were found at autopsy, however, most often in the occipital or parieto-occipital region. In addition, several babies had parenchymal lesions in a distribution consistent with diffuse axonal injury.[11]

While some reports on the shaken baby syndrome mention brain swelling, in most reports the subdural collections themselves have been postulated as the cause of death. In this series, all fatalities were consequent to uncontrollable brain swelling, and it is clear that drainage of the small collections present would have been useless in controlling the ICP. The problem of acute brain swelling is particularly common in the pediatric population, and its cause is poorly understood.[2] Whether high accelerations in the anteroposterior direction have some particular association to this complication remains to be investigated.

It is our conclusion that the shaken baby syndrome, at least in its most severe acute form, is not usually caused by shaking alone. Although shaking may, in fact, be a part of the process, it is more likely that such infants suffer blunt impact. The most common scenario may be a child who is shaken, then thrown into or against a crib or other surface, striking the back of the head and thus undergoing a large, brief deceleration. This child then has both types of injury — impact with its resulting focal damage, and severe acceleration-deceleration effects associated with impact causing shearing forces on the vessels and parenchyma. Unless a child has predisposing factors such as subdural hygromas, brain atrophy, or collagen-vascular disease, fatal cases of the shaken baby syndrome are not likely to occur from the shaking that occurs during play, feeding, or in a swing, or even from the more vigorous shaking given by a caretaker as a means of discipline.

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