Fatal Head Injury in Children Younger Than 2 Years in New York City and an Overview of the Shaken Baby Syndrome

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• ***Context.*—Shaken baby syndrome is a controversial topic in forensic pathology. Some forensic pathologists state that shaking alone is insufficient to explain death and that an impact must have occurred even if there is no impact site on the head.**

***Objective.*—To examine a large cohort of fatal, pediatric head injuries for patterns of specific autopsy findings and circumstances that would support or dispute pure shaking as the cause of death.**

***Design.*—We retrospectively reviewed 59 deaths due to head injuries in children younger than 2 years certified in our office during a 9 year period (1998–2006). The review included autopsy, toxicology, microscopy, neuropathology, and police and investigators' reports.**

***Results.*—There were 46 homicides, 8 accidents, and 1 undetermined death from blunt-impact injury of the head. In 10 (22%) of the homicides, there was no impact injury to the head, and the cause of death was certified as whiplash shaking. In 4 (40%) of these 10 deaths, there was a history of shaking. In 5 (83%) of the other 6, there was no history of any purported accidental or homicidal injury. All 8 accidental deaths had impact sites. Of the 59 deaths, 4 (6.7%) had only remote injuries (chronic subdural hematomas, remote long bone fractures) that were certified as undetermined cause and manner. These 4 deaths were excluded from the study.**

***Conclusions.*—We describe a subset of fatal, nonaccidental head-injury deaths in infants without an impact to the head. The autopsy findings and circumstances are diagnostic of a nonimpact, shaking mechanism as the cause of death. Fatal, accidental head injuries in children younger than 2 years are rare.**

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In 1972, John Caffey, a pediatric radiologist, published an article on the theory and practice of the abusive shaking of infants.[1] This was followed, in 1974, with a second article on the ''whiplash shaken baby syndrome.''[2] The shaken baby syndrome (SBS) is defined as a nonaccidental head injury that is characterized by an acute encephalopathy with subdural/subarachnoid and retinal hemorrhages and that occurs in the context of an inappropriate or inconsistent history. It may be accompanied by other injuries. The cause of the injury is due to repetitive, violent, whiplash movement (acceleration-deceleration) of the infant's head by ''shaking.''

There is controversy in the field of forensic pathology as to whether the SBS exists; that is, can an infant be shaken to death without a head impact?[3–9] Two popular forensic pathology textbooks have different opinions.[5,6] In 1 textbook, the authors state that there is no conclusive evidence that the entity exists. These authors agree that the findings are traumatic but state that the injuries are due to impacts and not to shaking.[6] In deaths in which there is no evidence of impact on the scalp or the skull, they state that the ''absence of trauma does not preclude impact.''[6(p361)] In the other textbook, a balanced discussion is given of the issues involved and asks why it is necessary to conclude that an impact must have occurred when there is no evidence of a bruise in the scalp and the caregiver gives a history of shaking without impact.[5]

In 2001, the American Academy of Pediatrics' Committee on Child Abuse and Neglect reported that SBS results from extreme, rotational, cranial acceleration induced by violent shaking or shaking and impact.[10] Two popular pediatric textbooks use the terms *nonaccidental injury* or *intentional head trauma* to describe head injuries in children.[11,12] One notes that ''some so-called 'shaken babies' have some evidence of injuries due to impact.''[12(p465)] Therefore, ''because it is often difficult to be certain whether intracranial bleeding is due to shaking and/or impact, the term shaken baby has been replaced with shaken baby/impact syndrome.''[12(p465)]

In 2001, the National Association of Medical Examiners published a position paper on fatal, abusive head injuries in infants and young children and sidestepped the issue of the SBS.[13] Their original charge was to produce a position paper on the shaken baby syndrome. Instead, ''be-

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cause the term shaken baby has taken on such controversy,'' [13(p112)] they decided only to address the topic of abusive head injury in infants and young children. Recent reports in the neurology and neurosurgical fields have also addressed this controversy.[14,15]

Although this controversy exists, the actual injury is not in dispute. There is little disagreement that these deaths are due to trauma. Those who do not accept the SBS believe that these internal head injuries are due to an impact of the head (not shaking alone). A problem with this approach occurs, however, in infants who die with these same internal findings and circumstances but do not have any impact site at autopsy. The offered explanation is that there was an impact but that it occurred against a soft surface and so it left no impact-site injury. The confessions of caregivers who state they only shook the child are disregarded, and perpetrators are assumed to be more likely to admit to shaking than to ''slamming [the child's] head against an object or throwing [the child] across a room like a football.'' [6(p360)] According to this argument, if there is no evidence of impact, the child's head presumably must have impacted on a soft surface when the child was slammed or thrown across the room. Others, however, question how an infant who is impacted forcefully enough against any surface to result in a subdural hematoma, retinal hemorrhage, or axonal injury can escape having some evidence of a scalp impact.[16] In these instances, violent shaking (without impact) explains the absence of an impact site.

Our objective was to offer a broad overview of the types and findings of fatal head injuries in children younger than 2 years in a large medical examiner's office. We selected the 2-year age limit because the incidence of this scenario and the constellation of findings diminish rapidly after the first year of life. We address the autopsy findings, including the presence/absence of impact sites, the circumstances, and mechanisms of death.

MATERIALS AND METHODS

The Office of Chief Medical Examiner investigates all unexpected, violent, and suspicious deaths in the 5 boroughs that comprise New York City, New York. We reviewed all head injury deaths in children younger than 2 years investigated and autopsied at the New York City Office of Chief Medical Examiner between 1998 and mid-2006. The medical examiner records included the autopsy, toxicology, microscopy, neuropathology, police, and investigators' reports. We excluded 4 nonaccidental, delayed, head injury deaths (deaths took place 7–17 years after the initial injury) that occurred during the time interval of the study. In addition, we excluded deaths with concomitant, life-threatening, visceral injuries. Gunshot and stab wounds, intoxications, asphyxiations, and life-threatening traumatic injuries of the trunk or extremities were excluded.

Each brain, spinal cord, and dura had been fixed in formalin for at least 2 weeks before dissection by a board-certified neuropathologist. The Office of Chief Medical Examiner employs a full-time neuropathologist who formally dissects and examines approximately 1500 brains per year. Routine hematoxylin-eosin microscopic examination was performed on all brains. The eyes were removed, at the discretion of the prosector, by a superior approach through the orbital plates. The eyes were opened in the conventional pupil-optic nerve plane, and sections were processed and paraffin-embedded. The histologic sections were examined by the medical examiner and, in some instances, a neuropathologist. Special histochemical and immunohistochemical stains (eg, β-amyloid precursor protein [β-APP] stain[17–20]) were performed in select instances.

β-Amyloid precursor protein is a neuronal transmembrane protein that is transported by axons. Before β-APP studies, histologic stains, including hematoxylin-eosin and silver methods, were able to detect axonal injury (eg, retraction balls, spheroids) if there had been a survival interval of at least 12 hours. The increased sensitivity of β-APP allows detection of axonal injury within a much shorter survival interval (detection following a 35 minute survival interval has been reported).[21] In instances of prolonged survival with marked ischemic brain injury, it may not be possible to distinguish between ischemic and traumatic axonal injury. The stain is useful for identification of damaged axons but not always for determining the etiology of the axonal injury. The pattern and distribution of staining is an important factor, as well as the presence and/or absence of neuronal anoxic-ischemic injury.[17,22] The etiology of axonal injury includes traumatic, vascular, and metabolic.[17] Axonal injury from hypoxic-ischemic injury is designated as *vascular axonal injury*, and injury from acceleration-deceleration forces is *diffuse traumatic axonal injury* (dTAI). Unfortunately, because of the complexity of neurotrauma, multiple types of axonal injury may occur in the same brain.

RESULTS

We detected 55 head-injury deaths in children younger than 2 years: 46 (84%) homicides, 8 (15%) accidents, and 1 (2%) undetermined. There were an additional 4 deaths with chronic, subdural hematomas with or without remote long bone fractures that were certified with an undetermined cause and manner. There were 10 (22%) of the 46 homicides with no impact injury of the head that were certified as whiplash shaking (see Table 1). Three (30%) of these 10 had survival intervals of greater than 10 days. There were 36 (78%) of the 46 homicides and 8 accidental deaths from blunt impact injury of the head (see Table 2). The causes of death for all homicides and accidents in children younger than 2 years investigated between 1998 and 2006 are listed in Table 3.

The 8 accidents included 2 (25%) motor vehicle collisions, 1 (13%) television falling on a child, 1 (13%) adult falling on a child on a dance floor, and 4 (50%) falls from height. One fall involved a 16-month-old infant who fell 5 stories from a fire escape. The second was a 10-month-old infant whom 3 adults witnessed fall from standing height while pushing a stroller. He did not lose consciousness but became lethargic with vomiting during the next several hours. He was brought to the hospital, and a large epidural hematoma was diagnosed and evacuated. There was no other injury. The third was an 8-month-old infant who was in the care of a babysitter. The babysitter turned to get a diaper, and the baby fell off the changing table onto a cement floor covered with carpet. The infant was taken to a clinic and was then brought to an emergency department by ambulance. The infant had a skull fracture with subdural and epidural hematoma. The fourth fall involved a 19-month-old infant who fell 1 story from a staircase landing.

The single death from an acute head injury that was certified with an undetermined manner involved a 23-month-old infant who reportedly fell in a bathtub. The child was in the care of the babysitter's husband when the incident occurred.

Peripheral retinal hemorrhages were detected in 6 (86%) of 7 homicides with no impact site where children had survival intervals less than 14 days (the seventh child had bilateral optic nerve hemorrhages and was pronounced dead in the emergency department). Of the 3 delayed homicides without an impact site, 1 child (33%) had peripheral retinal hemosiderin deposits at autopsy (Table 1, case

6), the second had ''massive bilateral pre- and intraretinal hemorrhages'' described at the hospital and confirmed at autopsy (Table 1, case 7), and the third had 5 peripheral retinal hemorrhages of the left eye noted at the hospital but not at autopsy (Table 1, case 10). The eyes of all 10 deceased children without an impact site were examined microscopically.

COMMENT

All experienced forensic pathologists have autopsied persons who had fatal blunt head impacts without externally visible injury, often explicable based on thick head hair or a hat. However, such persons almost invariably have contusions underneath sites of impact in the subscalpular soft tissues, demonstrated by reflecting the scalp and galea (Figure 1, a and b). In fact, it is difficult to imagine a circumstance in which a blunt impact to the head transmitted enough force to tear bridging veins, subarachnoid and retinal blood vessels, and axons deep in the cerebral hemispheres without breaking a few capillaries in the subscalpular soft tissues. Hence, we submit that routinely explaining away the fatal head injuries of children inflicted by shaking as a consequence of blunt impact without subscalpular contusion is an extension of the denial of reality in the name of objectivity.

We detected 10 (18%) of the 55 infants who died of head injuries and had no contact injury (impact) at autopsy (ie, no subscalpular hemorrhage, skull fracture, cerebral contusion/laceration, or scalp or facial injury). Three (30%) of the 10 deceased infants with no evidence of impact had survival intervals of more than 2 weeks. These 10 homicides were 22% (10 of 46) of the nonpenetrating, head-injury homicides and 9% of all homicides in this cohort. Some have stated that an impact may not be reflected on the scalp of an infant because a young child's scalp is elastic and may stretch on impact.[4,6] If this were the case, it may be a reasonable explanation for the absence of a scalp laceration but does not explain the absence of damaged blood vessels. All 8 of the accidental deaths had impact sites of the head. The absence of an impact site was seen exclusively in the homicide group.

Geddes et al[23] reported a detailed neuropathologic study of 37 infants who died of nonaccidental head injury. Among those, there were 8 (22%) ''shaken-only'' infants who had no signs of impact. The clinical presentations of 7 (19% of all study infants; 88% of shaken-only study infants) were collapse or respiratory arrest. Gilliland and Folberg[24] found no scalp or skull injuries in 9 (11%) of 80 children who died of fatal head injuries. They summarized 8 other postmortem studies on abused children that found no impact injuries at autopsy in 25 (31%) of those 80 deaths.[16,24–26]

Head injuries may involve contact and/or acceleration-deceleration forces. Contact forces cause focal injuries (scalp contusion, skull fracture, epidural hemorrhage), whereas acceleration-deceleration (primarily rotational forces) result in diffuse injuries (eg, subdural hemorrhage, diffuse axonal injury, intermediate contusion). Diffuse brain injury results in widespread, global disruption of brain function, and loss of consciousness is a consistent feature.[27] Diffuse brain injury may be produced without an impact, although impacts often complicate these events and augment the forces of acceleration-deceleration.[27] In infants, impacts typically cause cerebral tears instead of cerebral cortical contusions because of the immaturity of the brain.[28] Focal lesions are impact related and are not associated with immediate loss of consciousness unless there is associated diffuse brain injury. An expanding, space-occupying lesion (eg, epidural hematoma) from a focal injury may eventually result in unconsciousness, following a lucid interval, because of increasing intracranial pressure.[4,27]

Subdural hemorrhages typically are caused by rotational acceleration-deceleration forces initiated or arrested by an impact that tear bridging veins between the dura and the brain.[29] They have been described in adults riding high-speed roller coasters and have been reported to occur ''spontaneously'' (most likely because of an unrecognized minor injury, often seen in conjunction with anticoagulation therapy, hemodialysis, and hemophilia).[30–36] Microscopic examination of subdural hematomas may provide useful dating information that may be used to corroborate the circumstances of the reputed injury.[27] Extraction artifacts are potential causes of acute hemorrhage in the central nervous system. Direct observation of the brain and spinal cord during postmortem exposure, and immediate removal by the prosector should be done to avoid misinterpreting autopsy artifacts.

The effects of all subdural hematomas are not equal. Small, noncompressive, subdural hemorrhages (the kind typically seen in SBS) are not acutely dangerous, cause few signs or symptoms, and, alone, do not impair consciousness (Figure 2). If a subdural hematoma expands and results in compression of the brain with increased intracranial pressure and herniation, then it becomes a life-threatening process. Because the subdural hematomas in the SBS are small and noncompressive, they do not adequately explain the loss of consciousness (or death) of these infants. In some instances, an acute subdural hemorrhage also may have a chronic component.

The issue that arises with acute and chronic subdural hematomas is whether the acute hemorrhage is due to a spontaneous rebleed or due to a second injury.[3] The pathogenesis of chronic subdural hematomas is variable and relatively minor trauma may initiate rebleeding that enlarges gradually.[27] In this study, there were 3 children in the nonimpact group who were dead on arrival and had acute and chronic subdural hematomas at autopsy. These acute (and chronic) bleeds were small, without mass effect, which confirms that there were at least 2 traumatic events, with the more recent causing the fatal neurologic injury. In these 3 children, the acute hemorrhage was not due to a spontaneous bleed or a second ''minor'' trauma because this small bleeding, alone, would not explain the deaths. Because these were not compressive hematomas, a different mechanism is required to explain these deaths (see below).

Rapidly developing, diffuse swelling of the entire brain (*malignant brain edema*) occurs mainly in children and adolescents.[37] It is apparent on a computed tomography (CT) scan as small symmetrical ventricles and effacement of the basal cisterns.[38] Some children with diffuse brain edema have been described with lucid intervals.[37,39,40] A head injury occurs, and, analogous to the expanding subdural, the child may be conscious as the brain swells. Once it swells enough to raise intracranial pressure, the child becomes unconscious. Therefore, if the child survives long enough to undergo a CT scan, one would expect to see a swollen brain with small symmetric ventricles and effaced basal cisterns.[38] If an unconscious child with a closed head

Table 1. Homicides With No Impact Site*

Case No.	History
1	9-mo-old infant: Became unresponsive after mother's boyfriend shook child. He stated, "I shook my baby when she didn't want to eat. I didn't know how hard I shook her. I placed her down. The phone rang. I got up to answer the phone. When I went back to the baby she was not breathing so I called 911." He pled guilty to manslaughter in the second degree. Survival interval: 6 d
2	6-mo-old infant: The foster-mother gave conflicting accounts of what happened. To the first team of emergency medical technicians, she said the child had a seizure because of a high fever. To the second team, she said that the infant had been fine in the morning and was placed on the couch to sleep. She found infant unresponsive at 10:30 am. Survival interval: DOA
3	2-mo-old infant: The child started making "strange" breathing noises while in the care of the father that had lasted 1–2 h. Upon return from work, the mother brought the child to the hospital. At the emergency department, he was noted to be grunting. Head CT showed SDH with normal ventricles and open basal cisterns. Ophthalmology consultation noted unilateral retinal hemorrhages. As the clinical course deteriorated, a follow-up CT (13 h after the first) showed diffuse bilateral cerebral edema. During questioning with the detectives, the father admitted to shaking the child. Survival interval: 4 d
4	4-mo-old infant: 27-y-old boyfriend of mother (and father of child) came to babysit, so the mother could go shopping. The child was in his usual state of health. After bottle feeding, he was placed in a car seat in the living room. The father heard the baby scream, and then, the child started shaking and vomited. The infant had trouble breathing, and he shook the child. Survival interval: 9 d
5	6-mo-old infant: Grandmother found infant unresponsive. Survival interval: DOA
6	6-mo-old infant: Father found child unresponsive at home. Subsequent investigation revealed that 10-y-old sibling had shaken the child. No external trauma seen at hospital. Survival interval: 2 mo
7	2-mo-old infant: Father was carrying baby and dropped her on the way back to bed. No external trauma. Healing femur and tibia fractures. Survival interval: 19 d
8	6-mo-old infant: Father found child unresponsive in crib. Attempted "CPR" that involved shaking. Survival interval: 5 d
9	4-mo-old infant: Boyfriend of mother stated baby was crying in crib. He noted a bulge on the child's L chest that he pushed. After this, the child stopped breathing and became unresponsive. Survival interval: DOA
10	3-mo-old infant: The boyfriend of the mother was caring for the infant. The infant was "fussy," and he tried to feed him. He placed the child in the crib, and when he returned in 10 min, the child was unresponsive. The child was admitted to the hospital, and healing rib and tibial metaphyseal fractures were noted. No external impact injury seen at hospital. CT (>4 h after presentation) showed cerebral edema. Ophthalmology consultation noted focal peripheral retinal hemorrhages. Survival interval: 4 wk

* No impact site indicates no external or internal scalp or facial injury; SAH, subarachnoid hemorrhage; SDH, subdural hemorrhage; HIE, hypoxic-ischemic encephalopathy; Y, yes; N, no; DOA, dead on arrival; CT, computed tomography scan; UK, unknown; and CPR, cardiopulmonary resuscitation.

† Small, no mass effect.

‡ At hospital.

§ Hospital: "Massive bilateral pre- and intraretinal hemorrhages"; autopsy: bilateral retinal, peribulbar adipose tissue, nerve sheath hemorrhage, and hemosiderin.

‖ Scattered β-amyloid precursor protein-positive axons in the spinal nerve roots.

injury has no expanding intracranial hematoma at presentation (or autopsy) and the cisterns are open with normal ventricles, then there would be no reason to have had a lucid interval. In these instances, the neurologic impairments are due to other mechanisms.

In the context of infant head injury, these other mechanisms include marked dTAI or immediate hypoxic-ischemic cerebral injury because of apnea and/or bradycardia from the injury. Diffuse traumatic axonal injury results in immediate unconsciousness. Concussion or injury of the cervical spinal cord and/or brainstem may result in apnea and other cardiopulmonary dysfunction.[19,23,26,41–43] If the apnea is prolonged, the infant may sustain ischemic brain injury because of cardiac compromise and hypoperfusion. Both dTAI and cerebral ischemia eventually may result in swelling of the brain. In either of these scenarios, one would not expect an infant to have a lucid interval (ie, act healthy and then suddenly become unresponsive). If these mechanisms are in action, the neurologic decline would occur immediately following the injury. The reverse is also true. If there were a large expanding hematoma (eg, subdural, epidural, and/or contusional) or a markedly swollen brain at presentation (ie, at the emergency department or autopsy table), then a lucid interval may have occurred. If there is no pathologic explanation for a lucid interval, however, then it should not be speculated to have occurred.

In reputed SBS deaths (with or without impact), dTAI

Table 1. Extended

Neuropathology Findings (Brain Weight, g)	Ischemic Brain Injury	Retinal Hemorrhage	History of Shaking	Spinal Cord Findings
SAH, bilateral cerebral hemispheres, L frontal; organizing SDH†, loosely adherent to R falx, R cerebral convexity, and L frontal area; HIE; sinus thrombosis (1050)	Y	Y	Y	Ischemic changes
Acute† and chronic SDH, bilateral subdural membranes, and fresh L cerebral hemisphere; SAH, L frontal; no sinus thrombosis (490)	N	Bilateral optic nerve hemorrhage without retinal hemorrhage	N	Grossly normal
Bilateral SDH†/SAH; HIE (640)	Y	Y	Y	Gross and microscopic exam unremarkable
Acute and chronic bilateral SDH†; acute bilateral SAH; HIE; no sinus thrombosis (1100)	Y	Y	Y	Nerve root hemorrhage (thoracic)
Acute and chronic SDH†, subdural membrane with patchy fresh R cerebral convexity; focal SAH, R parietal, and L frontal (650)	Y	Y	UK	Grossly normal
Bilateral chronic SDH† with foci of acute; HIE (293)	Y	Y‡ (hemosiderin at autopsy)	N	Gross and microscopic exam unremarkable
Organizing SDH†, bilateral convexities and lateral thoracic and dorsal lumbar spine; SAH, bilateral cerebral hemispheres, posterior cerebellum, and spinal cord (no weight)	Y	Y§	N	Ischemic changes
SAH, R cerebrum (1170)	Y	Y	Y	Acute spinal nerve root hemorrhage (thoracic, lumbar) and focal axonal injury‖
Acute and chronic SDH†/SAH, R frontal parietal (variably adherent), and L parietal (not adherent) with membrane on R convexity; no sinus thrombosis (702)	N	Y	N	Grossly normal
HIE; no sinus thrombosis (520)	Y	Y‡ (no hemosiderin at autopsy)	N	Ischemic changes

Table 2. Summary of Head Injury Deaths

Findings	Cause of Death			Total
	Pure Shaken Baby Syndrome	Blunt Impact* (Homicide)	Blunt Impact (Accident)	
No. of cases	10	36	8	**54**
Age range (average), mo	2–9 (4.8)	Newborn to 23 (7.5)	8–20 (13.5)	
External injury, No. (%)	0 (0)	26 (72)	7 (88)	**33** (61)
Impact site, No. (%)	0 (0)	36 (100)	8 (100)	**44** (81)
Skull fracture, No. (%)	0 (0)	17 (47)	7 (88)	**24** (44)
Subdural hemorrhage, No. (%)	8 (80)	24 (67)	3 (38)	**35** (65)
Subarachnoid hemorrhage, No. (%)	7 (70)	23 (64)	3 (38)	**33** (61)
Hypoxic-ischemic encephalopathy, No. (%)	8 (80)	11 (31)	3 (38)	**22** (41)
Retinal hemorrhages, No. (%)	9† (90)	16 (50‡)	1 (33‡)	**27** (60‡)

* Includes head-impact deaths and shaken-impact deaths.

† One death showed bilateral optic nerve hemorrhages and no retinal hemorrhages.

‡ Some retinal examinations were not reported or were not performed, so percentages for homicidal blunt trauma were based on 16/32 deaths; for accidental blunt trauma, 1/3 deaths; and for the total, 27/45 deaths.

Table 3. Causes of Death for All Children Younger Than 2 Years Investigated by the New York City Office of Chief Medical Examiner Between 1998 and 2006

Cause	Deaths, No. (% of Accident/ Homicide Category, % of Total)
Accidents	
Asphyxial deaths	31 (36, 16)
Smoke inhalation ± burns	18 (21, 9)
Blunt torso ± head	14 (16, 7)
Drowning	9 (10, 5)
Blunt head trauma	8 (9, 4)
Thermal injury	4 (5, 2)
Maternal cocaine	2 (2, 1)
Electrocution	1 (1, 0.5)
Total	**87** (100, 44)
Homicides	
Blunt impact to the head	36 (32, 18)
Blunt impact to the torso ± head	22 (20, 22)
Pure shaken baby syndrome	10 (9, 5)
Asphyxia	9 (8, 5)
Drowning	8 (7, 4)
Sharp injury	5 (5, 3)
Smoke/thermal injury	5 (5, 3)
Scald burn	5 (5, 3)
Gunshot wound	3 (3, 2)
Neglect	3 (3, 2)
Intoxication	3 (3, 2)
Hyper/hypothermia	2 (2, 1)
Total	**111** (100, 56)
Total No. of all deaths investigated	**198**

has been considered the mechanism of death. Based on injury threshold studies in primates, however, some authors[25,44] have concluded that an impact is needed for dTAI to result and that pure shaking is insufficient to cause it. Adult primate anatomy, however, is different from a human infant, and the studied primates were not shaken (they were subjected to a single acceleration).[44] This does not take into account the effects of repeated 3-dimensional stresses (anterior-posterior, side to side, and rotational). Even if the force produced by a single shake is insufficient to produce the injuries typically seen, one must consider that each movement adds some quantum of damage to the axons and blood vessels.[27]

A study by Duhaime et al[25] is often cited to ''prove'' that an impact is required to explain the neurologic compromise (ie, traumatic diffuse axonal injury) of these infants. The argument is that if dTAI is the mechanism of injury, and if one cannot reach the injury threshold by shaking alone, then there must have been an impact. Instead of dTAI as the sole mechanism of brain injury in reputed shaken-baby deaths, several studies have examined the role of apnea and/or ischemia in these deaths.[19,23,41–43,45,46] In these studies, the pathology does not support dTAI. There is minimal, if any, evidence of dTAI, which is, however, consistent with what is known about concussion. In head-injured and neck-injured patients, the causes of hypoxia and ischemia are multiple.[47,48] Apnea transiently occurs, and the more severe the injury, the longer the apnea.[48] Grubb et al[49] used apnea, irregular-slow respirations, and bradycardia as criteria for concussive injury in animal experiments. Hypotension, which may result in ischemia, is an even more frequent complication of head injury than is hypoxia.[49]

During whiplash motion, there are widespread internal movements of the brain, which may be more pronounced in the infant skull because of the large subarachnoid

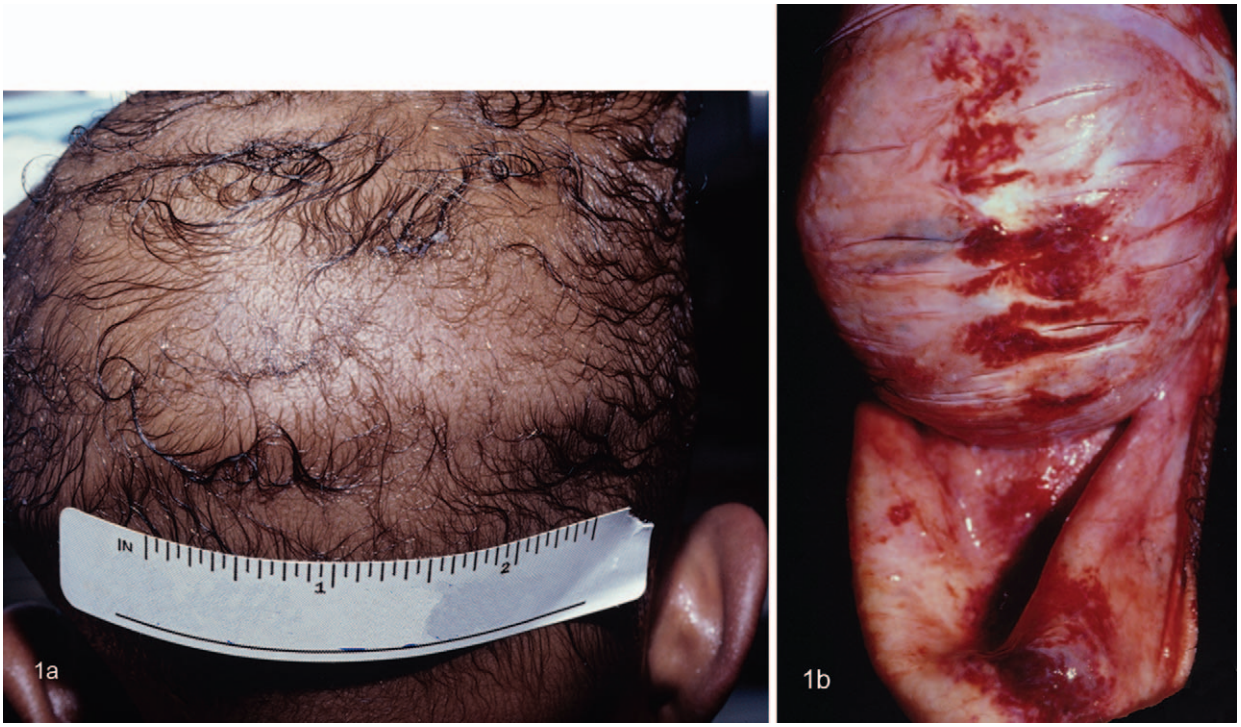

Figure 1. *Posterior scalp with no external evidence of injury (a), and subscalpular contusion observed following reflection of the scalp (b).*

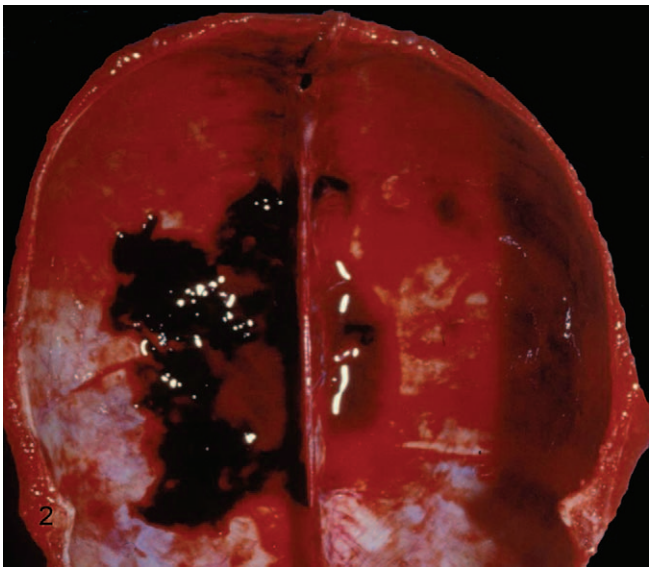

Figure 2. *Small, noncompressive subdural hematoma.*

space, incomplete myelination, and relatively weak neck muscles and large head. The structure of the infant neck is a key component of the injury mechanism in whiplash shaking, and yet, there are few studies that have examined the mechanical properties.[25] Relative fixation of the cerebellum by the tentorium, with repetitive pivoting at the incisura, may cause distortion in the brainstem at the floor of the fourth ventricle with neuraxis damage during these internal brain movements. This anatomic location contains cardiopulmonary regulatory centers.

Violent shaking (without impact) that causes cardiopulmonary abnormalities (eg, apnea, bradycardia, hypotension) may result in hypoxic-ischemic cerebral injury if the compromise is prolonged. Instead of the brain swelling occurring as a secondary effect of dTAI, it is due to a primary hypoxic-ischemic event from the trauma-induced cardiopulmonary pathology. Of our 10 infants without an impact, all but 2 had ischemic injury. The 2 (20%) infants that did not have ischemic injury were pronounced dead on arrival. The variations in the length of these cardiopulmonary deficits would explain the clinical observations of various degrees of ischemic brain injury and survival intervals. The clinical courses and cerebral findings of these survivors are similar to those seen with perinatal hypoxic-ischemic encephalopathy (so-called cerebral palsy).[50]

But how does a posttraumatic cardiopulmonary mechanism explain the subdural hemorrhages? According to the scaled primate studies, one would not expect subdural hemorrhage without an impact.[25,44] As Ommaya, Goldsmith, and Thibault[51(p222)] stated, ''Repetitive and prolonged episodic shaking by mechanical means without head impact and over specific time periods has not been experimentally tested.'' They recommended a large animal study to correlate the biomechanics and physiopathology of repetitive and prolonged whiplash shaking, particularly to investigate the cumulative effect of repeated shaking.[51] A basic tenet of injury analysis is that repetitive injury of a constant force on the same structure (with constant surface area and time durations) will cause more injury than that obtained from a single application of that same force. The damage summates, as with any repetitive injury occurrence (eg, carpal tunnel syndrome), until the movement stops or the injuries develop.[27] Recent neurosurgical studies have examined this issue with regard to the SBS.[52]

Raghupathi et al[52(p307)] found evidence of a graded response to rotational injury and an increased vulnerability with ''repeated, mild, nonimpact loading conditions.'' Therefore, one would expect that repetitive, subthreshold injury over time could result in subdural hemorrhage. In regard to the 2003 study using anthropomorphic simulations,[53] Margulies stated that ''what is missing are studies of repeated low-level acceleration-decelerations, like shaking—without that data, we cannot yet interpret doll studies results and predict injuries.''[14(p17)] In addition to these small animal studies, there have been human reports of this phenomenon. A report of a prisoner who was repeatedly shaken during a prolonged interrogation and died from a large subdural hematoma supports this mechanism.[54] Two neurosurgical studies from Japan have incidentally examined this issue.[55,56]

These 2 neurosurgical studies[55,56] involved the treatment of subarachnoid hematomas caused by ruptured cerebral artery aneurysms in adults. A ventricular and prepontine-cisternal irrigation system with urokinase infusion, and a head-shaking device was used in an attempt to wash out the clot to prevent vasospasm.[55] In the first study, 17 patients were placed in a head oscillating device (Neuroshaker, Mizuho, Tokyo, Japan), which included a sliding table that made a reciprocal, side-to-side, motion with an amplitude of 4 cm. It slowly swung the patients, who were fixed on the table with a soft pillow. The frequency of the motion could be adjusted from 0.5 to 3 cycles/s. In this first study, the Neuroshaker was operated continuously at a frequency of 1.5 cycles/s for 48 to 72 hours. There were several patients with neurologic complications, including an acute interhemispheric subdural hematoma. The second study[56] involved 114 patients who also received the Neuroshaker therapy, with the swinging frequency set at 1 cycle/s and the duration of each session lasting only 15 minutes. There were no subdural complications, and the authors hypothesized that the continuous operation at a relatively high-swinging frequency might have been the cause of the adverse effects in the previous study.[55]

Spinal cord injury has been demonstrated in infants who die of a suspected shaking mechanism.[19,23,26,41,42] In the study by Geddes et al,[23] 2 (25%) of 8 infants in the shaken-only group had spinal cord injury. Of their 37 infants, 11 (30%) had localized injury at the craniocervical junction or the cervical cord.[23] Whiplash type injuries of the neck (even those seen in motor vehicle collisions) may be subtle, may only involve ligamental sprains without fracture, and may be missed without meticulous dissection of the anterior and posterior neck.[47,57,58] Injury of the neck and spinal cord has been described in instances of reputed SBS deaths.[19,23,26,41,42] Microscopic spinal nerve root hemorrhage was detected in 2 (29%) of 7 of our pure shaking deaths (Table 1).

Anatomically, the dura is anchored at the foramen magnum and the nerve root dural sheaths are tethered within the intervertebral foramen by distinct ligaments.[59] These roots have been found to be 10% as strong as the nerve and relatively little applied force may produce obvious damage in the nerve roots.[60] Therefore, nerve roots may be more susceptible to damage by tensile forces, such as from spinal flexion-induced deformations of the spinal

cord within the spinal canal.[60] Repetitive shaking results in flexion-induced deformations, and the resultant spinal nerve root hemorrhages are signs of this distraction injury. Identification of spinal cord injury may require microscopic and immunohistochemical (β-APP) examination (Table 1).

Our study is in agreement with other reports[61–65] that show that fatal, accidental head injuries in children younger than 2 years are rare. Of the 55 infant deaths, there were 4 deaths (7%) because of falls, 2 (4%) because of motor vehicles, and 2 (4%) by a person or object falling on a child. Children, even infants, accidentally fall. Kravitz et al[63] estimated that more than 1.75 million infants have sustained at least 1 fall during their first year of life. Several studies have examined injuries following these falls. Helfer studied 246 children who fell fewer than 3 feet and found 3 (1%) with skull fractures and none with other serious injury.[64] Duhaime et al[62] studied 100 children younger than 2 years with head injuries who were admitted to the hospital. Although falls were the most common circumstance, they found that most household falls in this patient population were neurologically benign.[62]

It would be extremely unusual for an infant to sustain a fatal head injury without the cause being apparent to a caregiver. Therefore, an infant who presents with a traumatic brain injury, where there is no history of any preceding accidental injury, is highly suspicious for nonaccidental injury.[66] One does not expect to see accidental blunt injuries without an obvious cause in an infant who cannot yet walk. In all the accidental deaths, the child was ambulatory and had an impact-site injury. Circumstantial-anatomic disconnect is suspicious for nonaccidental injury. In some instances, there may be a clinical history of a preceding, reputed accidental injury. In these instances, the forensic pathologist must evaluate the head injury with the proposed accidental mechanism to determine whether the injury is consistent with the alleged circumstances.

CONCLUSION

Forensic pathologists continue to see infants who present with small subdural hemorrhages, subarachnoid hemorrhages, and optic hemorrhages *without* evidence of head impact after a thorough and competent postmortem examination.[27] This study is similar to previously published cohorts demonstrating this distinctive constellation of findings. In addition, some caregivers admit to shaking,[67] and this syndrome diminishes rapidly after the first year of life. Whiplash shaking without impact is the cause of death of this subset of infant homicides.

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