Accidental Head Trauma and Retinal Hemorrhage
[Clinical Study]

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ABSTRACT

Retinal hemorrhage and intracranial hemorrhage in a child with little external evidence of trauma and with a poorly documented history are considered pathognomonic child abuse. The mechanism and magnitude of force required to produce the injuries are seldom witnessed or known. This study was designed to determine the incidence of retinal hemorrhage in pediatric head injuries under known accidental circumstances, in association with forces sufficient to cause skull fracture and/or intracranial hemorrhage. Of 525 consecutive hospital admissions for head injuries, 200 children filled these criteria. Thirty children were excluded because of suspected child abuse or gunshot wounds. Of the remaining 170, 140 were evaluated by an ophthalmologist for retinal hemorrhage. Two children, who were both involved in side-impact car accidents, had retinal hemorrhages in associated with severe head injury. Retinal hemorrhage occurs rarely in accidental head injury and is associated with extraordinary force.

More than 2 million children are abused in the United States every year (25). Children can be abused physically, sexually, and emotionally. The spectrum of physical abuse is broad and includes soft tissue laceration and bruising, skeletal fracture, burns, intracranial hemorrhage,

and/or retinal hemorrhage. The most common cause of death in abused children is head injury (3). The syndrome of subdural hemorrhage (SDH) or subarachnoid hemorrhage (SAH), retinal hemorrhage (RH), and metaphyseal fracture is the neurosurgical paradigm of the physically abused child (5). The visual and neurological sequelae of this syndrome of child abuse can be devastating and can include permanent visual impairment, mental retardation, seizures, and tetraparesis. Because the survivors of child abuse are likely to abuse their own children, the tragedy is perpetuated by succeeding generations.

The mechanism of head injury in abused children is seldom witnessed or known and is often concealed behind closed doors. Although the abused child may be visibly battered, bruised, or burned, all too often, there are no outward physical signs of trauma; however, an ophthalmological examination reveals RH and computed tomography demonstrates SDH and/or SAH. Caffey (5) described this form of abuse as the “whiplash shaken infant” syndrome and suggested that the American public is ignorant of the ramifications of punishing a child by “casual” shaking. This opinion has been reflected in “not guilty” verdicts in alleged cases of “shaken baby syndrome” (SBS). No conviction is made because the perpetrator was presumably not aware that death could result from shaking. We hypothesize that SBS is caused by extraordinary force rather than casual shaking.

The purpose of this study is to determine if RH occurs in association with forces that customarily cause accidental skull fracture and/or intracranial hemorrhage.

METHODS AND RESULTS

The Children's National Medical Center serves a population of 4 million in the Washington metropolitan area. From October 1990 to October 1991, 64,400 children were seen in the emergency room; 525 of those children were admitted because of head injuries. The criteria for entry into the study were skull fractures and/or intracranial hemorrhages; 200 children met these criteria. Nine children with gunshot wounds to the head and 21 children, who were referred to Child Protection Service for suspected child abuse, were excluded from the study. Of those suspected of being the victims of abuse, two were known to have been beaten; nine allegedly fell from heights of less than 4 feet; one struck his head on a crib; one fell out of a stroller; and four had no history of trauma. Seven children presented with seizures, and three presented with respiratory arrest. Six patients had SDH and RH. Three had other skeletal fractures, and one suffered a renal laceration. Trials are pending in family court in 17 cases, and 4 cases were referred to the criminal court.

Of the remaining 170 children, 140 were evaluated by an ophthalmologist for RHs. The median age was 4.5 years (range, 3.5 mo to 19.5 yr). Fifty-two children were less than 2 years of age. Motor vehicle accidents accounted for 39% of the injuries; 50% were hurt in falls (Table 1); 6% were assaulted; and 5% suffered miscellaneous injuries (Table 2). Glasgow Coma Scores were tabulated according to the mechanism of the injury (Table 3). RHs were found in two children. Both children were restrained in the back seat of the impacted side of vehicles involved in side-impact crashes; one child died, and the other sustained an ipsilateral cerebral contusion and SAH.

Falls	n (%)
<5 feet (e.g., couch, table, grocery cart)	17 (24)
5–10 feet (e.g., porch, landing, wall)	11 (16)
Stairs	17 (24)
Falls from buildings	12 (17)
1 story	5
2 stories	6
3 stories	1
Playground (e.g., swing, monkey bars, sliding board)	13 (19)
Total	70 (100)

Table 1. Types of Falls

Mechanism	n (all)	%	n (eye exams)	%	% (no abuse or GSW)
MVA	65	32.5	55	33	39
Falls	86	43	70	42	50
Abuse	21	10.5	20	12	
Assault	17	8.5	13	8	9
Other	11	5.5	8	5	5
Total	200	100	166	100	100

[a] MVA, motor vehicle accident; GSW, gunshot wound.

Table 2. Patient Data[a]

Mechanism	n (%)	13–15	9–12	3–8
MVA[a]	55 (39)	33	8	14
Falls	70 (50)	62	3	5
Assault	8 (6)	5	1	2
Other	7 (5)	5	1	1
Total	140	105	13	22

[a] MVA, motor vehicle accident.

Table 3. Mechanism of Injury by Glasgow Coma Score

DISCUSSION

The clinical association between intracranial hemorrhage and RH was first recognized by Terson (41) in 1900 and has come to be associated with spontaneous SAH and RH in adults. In 1946, Caffey (4) reported the association of SDH, RH, and radiographic evidence of metaphyseal avulsions and subperiosteal hemorrhage. Neither trauma nor abuse was suspected. Matson and Ingraham (27) first acknowledged that intracranial hemorrhage, especially SDH, was nearly always associated with trauma in children. The traumatic origin of the long bone radiographic changes was soon recognized (39), but another decade went by before the suspicion was raised that the traumatic injuries were nonaccidental (44). In 1962, (21) Kempe captured national attention with the description of the “battered child syndrome” SDH and the skeletal changes described above were the key components of the syndrome. With the help of the American Academy of Pediatrics, Kempe launched an educational campaign to enlighten pediatricians

who, he anticipated, could in turn recognize poor parenting practices and intervene on the part of the child. In 1974, Caffey (5) reexamined the syndrome that he had identified 26 years earlier: SDH, RH, and radiographic changes in the long bones. He drew attention to the incongruity between the severity of brain injury and the frequent absence of signs of external trauma and concluded that the injuries were caused by “habitual, moderate, casual manual whiplash shaking.” Caffey also presumed that public education could prevent nonaccidental injuries and that early intervention, prompted by routine regular examinations of the ocular fundi to detect RHs, would put an end to habitual casual shaking and SBS. To the contrary, public education has had no effect on the incidence of SBS (12,23).

The concept that “moderate casual shaking” can cause SDH, SAH, and RH was disputed by Woolley who asserted that “malicious violence” was necessary, especially in light of the rare occurrence of SDH in accidental falls (43). Kravitz et al. (22) subsequently reported only one subdural hematoma in children less than 2 years of age who had suffered accidental falls. More recently, several studies have reported the absence of SDH in children who have experienced accidental head trauma (7,8,10,17,18). Ommaya (31) provided an experimental basis for SBS in his studies of head injury in nonhuman primates, but the forces created were violent rather than moderate or casual. Monkeys were seated in a carriage that was impacted from behind by a piston. Acceleration, especially rotational acceleration, of unrestrained animals' heads produced subarachnoid and SDH, cerebral contusions, and cerebral edema without direct impact to the head. The animals were rendered unconscious, and corneal reflexes were abolished.

In an experimental model of a 1-month-old baby, Duhaime et al. (7) were unable to duplicate the forces created in Ommaya's experimental animal model. The forces could be reproduced only if sudden impact was combined with shaking. Acceleration or deceleration forces of only 10 G were produced by just shaking, but when the model was impacted even against a soft mattress, 300 G could be generated. In the clinical section of the study, direct evidence of head impact was found in most of the abused children. The term “shaken impact syndrome” was adopted. The importance of impact to SBS has been recently disputed. In a study of 24 infants diagnosed by a multidisciplinary child protection team as being victims of shaking, only half had signs of external head trauma. The basis for the diagnosis was not detailed, and the incidence of RH was not documented (1). Hadley et al. (15) described 13 of 36 (36%) infants with SBS who had no evidence of external trauma and concluded that impact was not an integral component of the syndrome.

In an autopsy study of eyes from children who had died in auto accidents, 2 of 12 (17%) had RHs, but no details of the accidents were provided. In a carefully detailed study of children less than 2 years of age who were admitted to the hospital for head injury, 10 of 100 patients sustained RH (8). An extraordinary 24% of the injuries were presumed inflicted, and an additional 32% were suspected of being caused by abuse or neglect. Nine of 10 RHs were nonaccidental (9/24 or 38%); the only accidental RH was found in the victim of a fatal motor vehicle accident, who also had an SDH. The results of the study reinforced its authors' opinion that the mechanism of injury of inflicted trauma is sudden deceleration and is associated with impact. In a multicenter cooperative study of 823 head-injured children, RHs were found in 27; 4 hemorrhages were associated with falls, and 1 with a motor vehicle accident (24). The remaining children with hemorrhages were victims of abuse. None of the 70 children in our study who

suffered falls significant enough to cause skull fracture and/or intracranial hemorrhage sustained a RH. Our study extends the range of these observations by studying the incidence of RH in accidental head injuries that are associated with forces sufficient to cause skull fracture and/or intracranial hemorrhage; nonaccidental injuries were specifically excluded. Well-defined criteria were used for inclusion in the study, thorough ophthalmological examinations were performed, and the mechanism of injury was refined.

Lacking a reliable history, the severity of a head injury is sometimes first recognized by radiographic findings and judged nonaccidental if RHs are present (9). The injury is minimalized by the perpetrator whose defense rests on the falsified history of an accidental fall or casual shaking. We reasoned that if falls or casual shaking (Caffey) could cause the syndrome, then RH should be very common in moderately severe accidental head injuries. We found instead that RH was rare (2 of 166 or 1.2%) and occurred only with severe head injuries sustained in side or lateral impact crashes. Lateral impact causes a similar constellation of injuries in the “tin ear syndrome” of child abuse (16). Blunt impact to the ear creates rotational acceleration of the head, which produces severe brain injury, RH, and ear bruising.

Traumatic SDH is probably caused by bleeding from veins bridging the brain and the dural venous sinuses. Because the venous sinuses are adherent to the skull, the bridging veins are torn by the sudden jarring movement of the brain within the skull and dura. Acceleration and deceleration forces are greatest in the parasagittal plane, which is the most common site of SDH in child abuse (45). Retinal, preretinal, and vitreous hemorrhages can be found in physically abused children (13). The vitreous of the young eye is firmly attached by a dense web of collagen fibrils along major retinal vessels. The retinal capillary networks (one in the nerve fiber layer and the other between the inner nuclear and outer plexiform layers) are suspended between the retinal precapillary arterioles and postcapillary venules. A firm attachment also exists between the vitreous and the lens. Shaking and abrupt deceleration impels the lens-vitreous complex back and forth, exerts traction forces on the retina, and tears the vascular attachments. The magnitude of the forces determines the extent of the hemorrhage, but the threshold for hemorrhage is not known. RH is also seen in as many as 40% of full-term infants delivered vaginally (2,28,32,33,34,38) and, rarely, after cardiopulmonary resuscitation (11,19,26,35,37,42) (Purtscher's retinopathy), extracorporeal membrane oxygenation (37), Valsalva maneuver (6,36), chest compression from shoulder restraints (20), and chest compression from being run or rolled over (29). The mechanism involves chest compression, increased intrathoracic pressure, increased pressure in the vertebral venous plexus, and elevated intraocular venous pressure (14). This mechanism may also play a role in the battered child who is strangled or whose chest is crushed or compressed. SAH but not SDH can be seen under these circumstances, and in contrast to child abuse, the history is well documented and indisputable. Venous congestion also accounts for hemorrhages associated with papilledema and intracranial hypertension.

CONCLUSION

RH occurs only rarely in association with accidental trauma, birth, cardiopulmonary resuscitation, and chest compression. Without an adequate history, the origin of RH can be problematic, especially when child abuse is suspected. The magnitude and violence of the

experimental and clinical forces that are necessary to duplicate SBS are, however, extraordinary and cannot be attributed to casual shaking.

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COMMENTS

This is an important article for the general neurosurgical audience. Although those neurosurgeons who deal predominantly with children now realize that retinal hemorrhages rarely if ever occur from minor trauma or resuscitation and are the result of either nonaccidental trauma (child abuse) or severe trauma in the child less than 2 years old, acceptance of these facts may be difficult for the neurosurgeon who sees only a few children a year. If the epidemic of child abuse is to be halted, all physicians who see traumatized children must be aware of the importance of looking for retinal hemorrhage during the initial examination, because overlooking this finding may lead to the neglect of a potentially serious injury and the return of a child to a potentially hazardous environment.

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COMMENTS

In this article, Johnson, Braun, and Friendly describe the retinal findings in 140 children evaluated at the National Childrens Medical Center during 1 year. These 140 patients are those patients evaluated by an ophthalmologist for retinal hemorrhages after presenting to the emergency room with skull fracture and/or intracranial hemorrhage. Children suspected for child abuse and those with gunshot wounds to the head were not included. Significantly only two patients were found to have retinal hemorrhages. Both of these children were restrained in the back seat of a car that was impacted in the side. The authors emphasized that retinal hemorrhages occur only in patients with severe head injuries. If there is no history of severe head injury, then all children with retinal hemorrhages should be considered to be the victims of child abuse until proven otherwise. This report serves to remind neurosurgeons of the importance of retinal hemorrhages. Vigilance on the part of the neurosurgeon seeing a child in the emergency room for a head injury is necessary to identify potential victims of child abuse

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