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Position Paper on Fatal Abusive Head Injuries in Infants and Young Children

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This article represents the work of the National Association of Medical Examiners Ad Hoc Committee on shaken baby syndrome. Abusive head injuries include injuries caused by shaking as well as impact to the head, either by directly striking the head or by causing the head to strike another object or surface. Because of anatomic and developmental differences in the brain and skull of the young child, the mechanisms and types of injuries that affect the head differ from those that affect the older child or adult. The mechanism of injury produced by inflicted head injuries in these children is most often rotational movement of the brain within the cranial cavity. Rotational movement of the brain damages the nervous system by creating shearing forces, which cause diffuse axonal injury with disruption of axons and tearing of bridging veins, which causes subdural and subarachnoid hemorrhages, and is very commonly associated with retinal schisis and hemorrhages. Recognition of this mechanism of injury may be helpful in severe acute rotational brain injuries because it facilitates understanding of such clinical features as the decrease in the level of consciousness and respiratory distress seen in these injured children. The pathologic findings of subdural hemorrhage, subarachnoid hemorrhage, and retinal hemorrhages are offered as "markers" to assist in the recognition of the presence of shearing brain injury in young children.

Key Words: Abusive head injury—Shaken baby syndrome—Head injury—Inflicted injuries.

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Editor's note: The Board of Directors of the National Association of Medical Examiners charged the authors of this article with writing a position paper on the shaken baby syndrome. This article was the result. The manuscript was reviewed by three reviewers on the Board of Editors of the *American Journal of Forensic Medicine and Pathology*. They believed that while it was worthy of publication, it should not be published as a position paper because of the controversial nature of the subject. The Board of Directors responded to this opinion by stating that position papers always deal with controversial subjects.

The original charge to this ad hoc committee was to produce a position paper on shaken baby syndrome. This terminology was taken by the committee to refer generally to the area of abusive head injury in young children. However, because the term *shaken baby syndrome* has taken on such controversy, this article will address the topic of abusive head injury in young children. In several areas of this article, the term *marker* is used when describing the importance of identifying the presence of subdural, subarachnoid, and retinal hemorrhages. The term *marker* indicates a grossly observable sign to signify the possible existence of diffuse axonal injury that is not grossly evident. Use of the term *marker* does not imply that such hemorrhages cannot exist without such an association but is intended to remind us to be alert to the possibility.

Head injuries account for up to 80% of fatal child abuse injuries at the youngest ages (1). Blunt force impact as well as vigorous shaking may play a role in the pathogenesis of these injuries (2). This article describes the state of knowledge concerning the pathogenesis, clinical features, and pathologic changes of fatal abusive head injuries in young children. The intent is to inform the practicing pathologist about the proper recognition, interpretation, and clinical correlation of these injuries.

Caffey's description of whiplash shaking of infants in the early 1970s introduced the concept that serious and even fatal head injury could be inflicted by a caretaker through shaking (3,4). Caffey described injuries characterized by subdural and/or subarachnoid hemorrhages, brain swelling, and reti-

nal hemorrhage without injuries that would indicate impact, such as facial bruises, scalp bruises, or skull fractures. Since that time, experts in many scientific fields have investigated whether such apparently innocent practices as tossing a baby into the air and other playful maneuvers might cause brain damage by a similar shaking mechanism. Currently, it is generally accepted that such playful practices do not result in injuries to the young child's brain. The type of shaking that is thought to result in significant brain injury involves holding the child by the thorax or an extremity and violently shaking the child back and forth, causing the head to forcefully whiplash forward and backward with repeated accelerations and decelerations in each direction.

Abusive head injury commonly occurs in response to prolonged crying and often is inflicted by a caregiver with limited patience or experience in handling a child. Some individuals who admitted to shaking children as a mechanism of injury have stated that shaking would stop the babies from crying. These assailants have actually used this practice to stop babies from crying on previous occasions without any visible adverse result.

When shaking is the mechanism of injury, the arms and legs of the child may also be violently flung about during the shaking, causing injuries to the long bones. Most frequently affected are the tibia, distal femora, and proximal humeri. These lesions were originally described as bucket-handle and corner fractures and were thought to be caused by the indirect forces of the shaking. These injuries are currently recognized as fractures through the most immature portion of the metaphyseal primary spongiosa and appear radiographically as separation of portions (corner) or of the entire disk (bucket handle) of metaphyseal bone, depending on the radiographic plane (5,6). Many babies with fatal abusive head injuries do not demonstrate any external injury, although in about 25% to 50% of cases, such injuries are evident on external examination (7–10). It is important that a careful search is made to identify any injury to the body, such as a bruise or abrasion. Grasping the child by the arms or thorax may result in bruises in these locations, but children may be grasped in this manner without leaving bruises. Likewise, ribs may be fractured while grasping the child around the thorax. At autopsy, close attention should be directed to the rib cage. Fractured ribs should be removed, decalcified, and examined microscopically. Posterior rib fractures are highly specific for abuse. Ribs are the most common bones fractured in association with other abusive injuries of children who die of fatal child abuse (11,12). Infants may sustain abusive head injury of less than fatal outcome and may sustain injuries to the brain that will later be reflected in degrees of mental retardation or slowness, learning disorders, seizures, blindness, or irritability. Of infants who receive abusive head injuries, approximately 7% to 30% die; 30% to 50% have significant cognitive or neurologic deficits; and 30% have a chance of full recovery (13–15). Lethal abusive head injury is not confined to infants. Children as old as 4 or 5 years can be fatally head injured by abuse, although the great majority are under 2 years of age, and most are under 12 months of age (16). Adults may also sustain head injuries by shaking, with findings identical to those found in shaken infants. Pounder described a slightly built, short, 30-year-old prisoner who was grasped by the shoulders and violently shaken. When he died 3 days later, his autopsy demonstrated subdural, subarachnoid, and retinal hemorrhages with diffuse axonal injury (17).

MECHANISMS OF INJURY

Appreciation of the unique characteristics of young children's head injuries requires an understanding of the developmental differences in the skull, brain, and neck before the age of about 4 years. Injuries to the young child's brain are unique in that the trauma occurs to an organ that is in the process of maturing; the mechanisms, the thresholds of injury, and the types of injuries differ from those that affect the older child or adult. The primary features unique to the young child include the thinness and pliability of the skull; the rapid growth of the brain and skull, resulting in a large heavy head; the softness of the brain, which is composed primarily of neurons without dendritic connections; the paucity of the myelin sheath of axons; the relative flatness of the skull base; the undeveloped neck muscles; and the subarachnoid space, which is large in its extent but shallow in depth (18,19). Because of the unique characteristics of the developing skull and brain, children under the age of 4 or 5 years are particularly vulnerable to a type of brain injury that is best described as shearing injury. Shearing injury implies a distortion of the brain shape that elongates it in an anterior–posterior dimension with resulting shifting apart of adjoining brain structures. Impact to the immature brain is more likely to produce shearing injury rather than the typical brain contusions that might occur in older children and adults (19). Impact force is more effectively transferred through the thin pliant skull and across

the large and shallow subarachnoid space of a young child's head. The paucity of myelination, the large number of neurons without glial or dendritic connections, and the small axonal size predispose the young brain to shearing injury by creating a soft consistency. The large heavy head mounted on the weak neck of the young child produces instability of the head, which allows greater movement of the head and brain when acted on by acceleration–deceleration forces. Last, the shallow skull base allows the young child's brain to rotate more readily in response to head acceleration or deceleration than occurs after the skull base has developed more prominent bony ridges and concavities (7,19,20). The mechanical forces that are operative in head injury are primarily translational and rotational (angular). Translational forces produce linear movement of the brain, a type of movement that is quite benign (21,22). The trivial falls that children sustain in falling from furniture and even down stairs primarily involve translational forces. Although such falls may occasionally result in a skull fracture, these incidents are generally very benign and do not result in loss of consciousness, neurologic deficit, or death (23–26). Rotational forces are generated by either impact or nonimpact inertial mechanisms, such as whiplash shaking, which produce sudden acceleration or deceleration of the head. Rotational forces applied to the head cause the brain to turn abruptly on its central axis or its attachment at the brainstem–cerebral junction. Extensive clinical and experimental data have demonstrated that such rotational movements of the brain result in a type of injury referred to as *shearing injury* or *diffuse axonal injury* (23–29). To keep pace with the accelerating skull responding to rotational forces, the brain moves within the cranial cavity, and because of the nonuniformity and different consistencies of the brain structures, strains occur at the junctures between cortex and subcortical white matter, white matter and deep gray matter, and lateral extensions across the midline of the brain (corpus callosum and superior and middle cerebellar peduncles). Disruptions of the axonal processes occur at these junctures as the peripheral parts of the brain rotate farther or more rapidly than the inner, deeper, more relatively fixed parts of the brain. Lesser forces disrupt the most external junctures, whereas greater forces disrupt deeper junctures down to the deep gray matter of the basal ganglia, thalamus, and finally the rostral brainstem. In young children, either an impact or a shaking mechanism may result in diffuse axonal injury, when significant, because both impart rotation to the brain (7,19,20). After the age of 4 or 5 years, the most common cause of diffuse axonal injury is the motor vehicle crash.

Some experimental evidence suggests that shaking alone may not be sufficient to produce the angular acceleration necessary to create fatal shear injury (7). The particular model used in the latter study to determine how much force could be transmitted by shaking to an infant's head utilized as a model a doll with a rubber neck, and the force considered necessary to produce shear injury was derived from studies of adult primates. Neither of these experimental circumstances necessarily truly resembles the immature human infant skull and brain (29). In favor of shaking as a possible mechanism, many forensic pathologists have experience based on confessions by perpetrators or witnesses of how these injuries were inflicted, as well as on autopsies in which no impact site is found on the scalp or skull. However, this experience must be received with some caution. Perpetrators may not remember, or later may not be willing, to fully describe their actions. Children may be violently shaken, then forcefully thrown rather than just placed down gently. Impacts may not be reflected on the scalp if the striking surface is padded or if it is broad and firm. A child's scalp is very elastic and stretches on impact. Not all impacts are registered as hemorrhage in the galea. In the vast majority of cases, it is not possible to definitely characterize children's head injuries as being caused by either pure impact or pure shaking because the pathologic changes in the brain are identical in cases in which either of these two mechanisms has been suggested (7). If there are focal injuries, such as skull fractures, scalp bruises, or subgaleal hemorrhage, an impact can be assumed, but coexistent shaking cannot be excluded. In the absence of signs of an impact, however, shaking alone should not be presumed because there may well have been an impact that cannot be identified (30). Subarachnoid and subdural hemorrhages should be appreciated as markers of brain displacement by angular force and the possibility of accompanying diffuse axonal injury (24,31). In young children, both impact and shaking produce these pathologic findings, which should be appreciated as markers for the underlying problem in the brain: the diffuse axonal injury (32).

SUBDURAL HEMORRHAGE

The grossly and microscopically identifiable pathologic changes in young children's rotation or acceleration–deceleration head injuries include subdural hemorrhage, subarachnoid hemorrhage,

and retinal lesions, including hemorrhages and schisis. Subdural hemorrhage results from tearing of bridging veins, which extend from the cortical surface to the dural venous sinuses (Fig. 1). These rather transparent veins tear when they are stretched as the brain moves within the subdural space of the cranial cavity (23,33–35). Subdural hemorrhages occur most frequently over the convex cerebral surfaces, especially posteriorly within the interhemispheric sulcus, and may be either unilateral or bilateral, although more commonly they are bilateral (Fig. 2). If the subdural hemorrhage is associated with a skull fracture, it need not be on the same side as the fracture. Subdural hemorrhage is probably uniformly present in cases of shearing injury but is evident at autopsy in about 90% to 98% of cases (7,36). Small amounts of interhemispheric blood that can be detected by computed tomography (CT) may not be seen at autopsy (5,37). Very thin layers of subdural blood over the cerebral convexities may not be visible on CT but can be found at autopsy. Magnetic resonance imaging (MRI) is able to detect a much greater number of subdural hemorrhages than can CT; however, many critically ill young children are not able to undergo MRI (19). As a result, studies that report data on nonfatal cases of abusive head injury find that about 80% to 85% of patients have subdural hemorrhage (38). At autopsy, the subdural hemorrhage may consist of only 2 to 3 ml of blood and may not be observed if the prosector does not personally inspect the subdural space as the calvarium is being removed (Fig. 3). Extreme caution should be taken to not misinterpret as premortem subdural hemorrhage the blood draining from the dural sinuses when these are incised at autopsy. The importance of subdural hemorrhage is typically not that of a space-occupying mass lesion producing increased intracranial pressure and the consequences of tentorial herniation, although some hemorrhages are large enough to bring about these complications. Rather, it is important as a marker of brain movement within the cranial cavity and may accompany shearing injury. Subdural bleeding may continue and accumulate to some extent if the child experiences postinjury survival. At autopsy, large subdural hemorrhages resulting in part from postinjury accumulation have been observed. Even a small amount of subdural hemorrhage indicates that brain displacement has been produced, which may have caused some shearing brain injury.

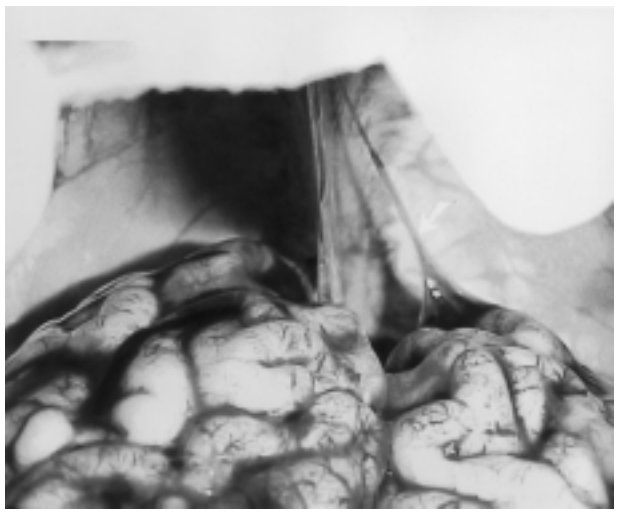

FIG. 1. Thirteen-week-old infant with normal brain and intracranial spaces demonstrating bridging vein (arrow) arising from left cerebral convexity (right).

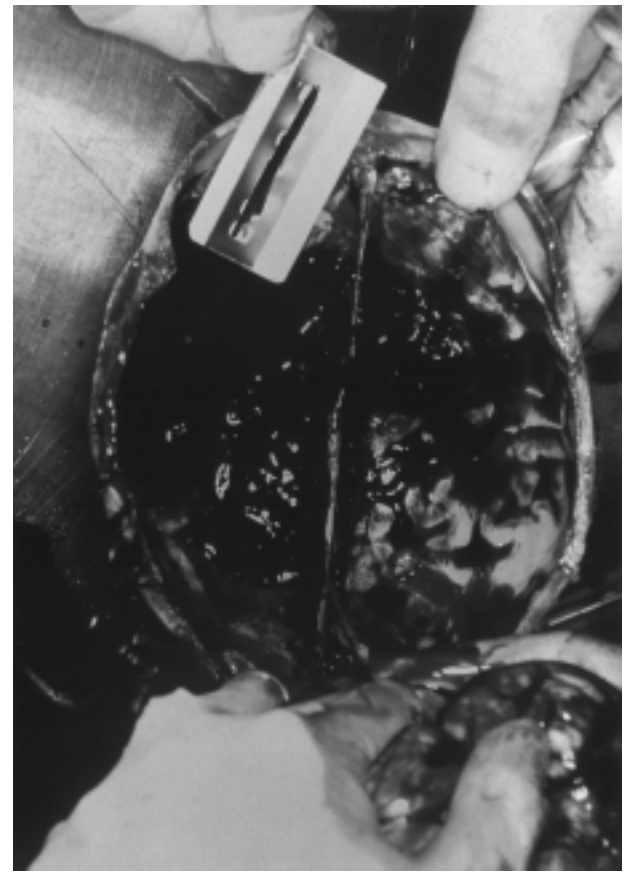

FIG. 2. Nine-month-old infant with fracture of right parietal calvarium showing bilateral acute subdural hemorrhages over the cerebral convexities.

SUBARACHNOID HEMORRHAGE

Subarachnoid hemorrhage occurs in patches over the cerebrum, especially over the parasagittal cerebral convexities (Fig. 4). It is present in virtually all

FIG. 3. Eleven-week-old infant with small amounts of acute subdural hemorrhage over both cerebral convexities.

fatal cases, although it may be very small and difficult to identify, especially on the interhemispheric surfaces (Fig. 5). Subarachnoid hemorrhage arises from tearing of arachnoid vessels at the same time bridging veins are torn, because the bridging veins are surrounded by an arachnoid sheath as they cross the subdural space to enter the inner dural layer and finally the dural sinuses. Tearing of bridging veins usually produces both subdural and subarachnoid hemorrhages.

RETINAL HEMORRHAGES

Retinal lesions are observed in 70% to 85% of young children with severe rotational brain injuries (32,39). Currently, their pathogenesis is not precisely understood. Their presence highly correlates with rotational head injury, and they are greatly overrepresented among cases of nonaccidental trauma in young children. Possible mechanisms to account for retinal hemorrhages include increased pressure transmitted to the central retinal vein from increased intrathoracic or intracranial pressure, direct trauma to the retina from being struck by the vitreous moving within the eye, and traction on the retina by the movement of the vitreous pulling away from the retina. The retinal hemorrhages seen in abusive head injuries are similar to those that are frequently observed in full-term neonates after vaginal delivery. In neonates, the hemorrhages appear to be consequent to increased intrathoracic or intracranial pressure from squeezing of the thorax during the passage through the birth canal. Most of the neonatal retinal hemorrhages completely resolve by 5 or 6 days, although a few persist longer (40,41). In children older than 30 days who have retinal hemorrhages, the great majority have abusive head injuries.

Ophthalmologic findings in abused children include peripheral retinal hemorrhages associated with retinal detachments, retinal tears, and large numbers of retinal hemorrhages (39). There may be other internal eye injuries in these children, consisting of vitreous bleeding and retinal folds. There is

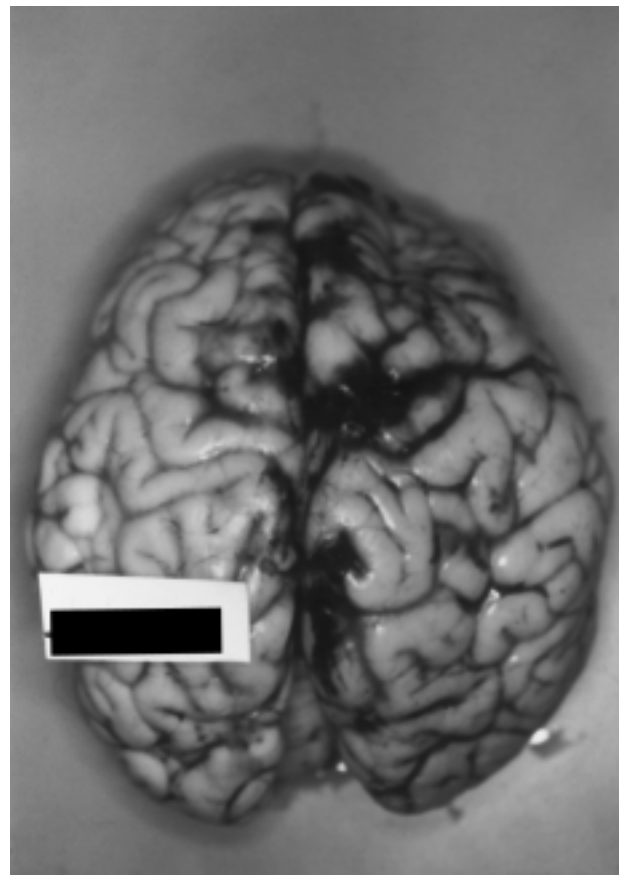

FIG. 4. Seventeen-week-old infant with large (70–80 ml) acute subdural hemorrhage over right cerebral convexity demonstrating patches of subarachnoid hemorrhage over both parasagittal regions, greater on the right than on the left.

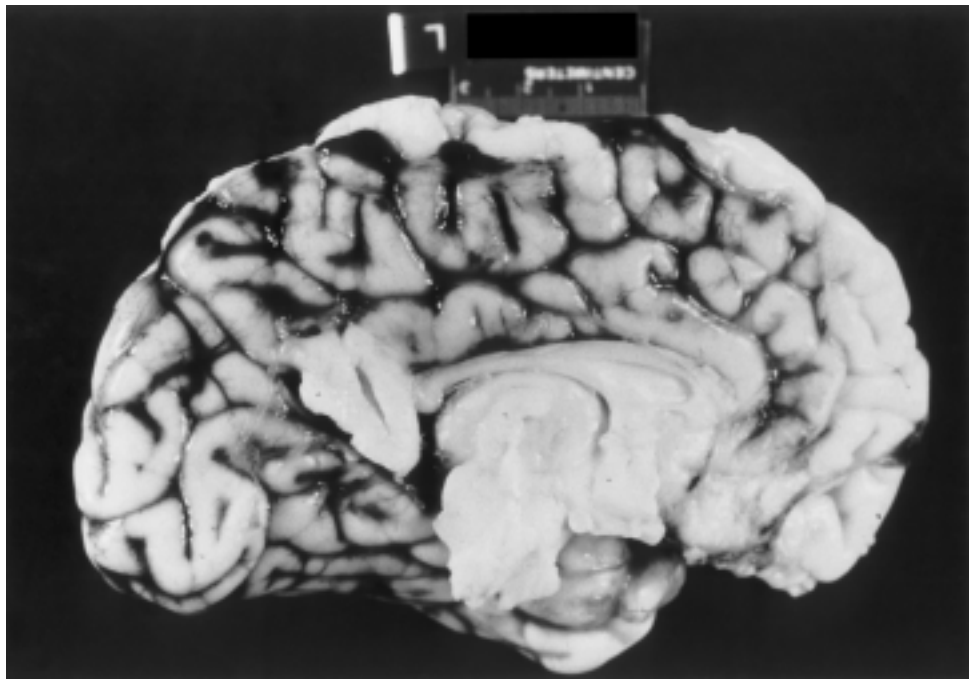

FIG. 5. Five-month-old infant with large bilateral acute subdural hemorrhages over cerebral convexities showing patches of subarachnoid hemorrhage on the mesial surface of the right cerebral hemisphere.

evidence that increasing severity of trauma to the head directly correlates to severe eye damage, beginning with subhyloid and intraretinal hemorrhages and progressing to retinal detachment and finally choroidal and vitreous hemorrhage (42). In children with very severe accidental head injury, (e.g., from a car accident), retinal hemorrhage is occasionally found (26). The retinal hemorrhages associated with nonaccidental head injuries tend to be bilateral, although they may be unilateral, multiple, and extensive and reach far into the periphery of the retina (39). Nontraumatic causes of retinal hemorrhages include bleeding disorders, sepsis, meningitis, vasculopathies, increased intracranial pressure, and, very rarely if ever, cardiopulmonary resuscitation (43–47). Retinal hemorrhages that occur in association with increased intracranial pressure are found at the posterior pole of the retina around the optic disc and are accompanied by papilledema (39).

Optic nerve hemorrhage is observed in association with inflicted head injuries in children but is not specific for those injuries. Optic nerve hemorrhage is hemorrhage in the perineural area. These hemorrhages are seen commonly whenever subdural hemorrhage is found in the cranial cavity, although there is not necessarily a direct connection between the subdural compartment of the orbital sheath and the subdural compartment of the intracranial cavity (42). Optic nerve hemorrhage can be seen in some cases of increased intracranial pressure that are not related to any form of trauma (48).

DIFFUSE BRAIN INJURY

Diffuse brain injury consists of tears of axonal processes and small blood vessels and, rarely, more extensive tissue tears (49,50). The areas of predilection are the corpus callosum; the subcortical white matter, especially of the superior frontal gyri; the periventricular areas; and the dorsolateral quadrants of the rostral brainstem. The axonal disruptions result in microscopic lesions that may be visible by light microscopy after 18 to 24 hours as retraction bulbs or varicosities. Retraction bulbs are accumulations of axoplasm, which appear on hematoxylin and eosin staining as pink bulbs. They are observed as the axoplasm of the disrupted axons accumulates at the damaged end and creates a bulbous enlargement. These axonal lesions are very difficult to see in young children because of the small size of the axonal processes. Immunohistochemical stains for β-amyloid precursor protein may allow demonstration of axonal injury as early after survival as 2 hours (51,52). The blood vessel tears of diffuse brain injury may be visible grossly as linear streaks or punctate hemorrhages, which vary from less than 1 mm up to many centimeters if bleeding continues for several days (31). However, these hemorrhages are very seldom seen in young children with diffuse brain injury because the blood vessels in young children are very elastic and do not readily tear even when adjacent axonal processes are torn.

In some (rare) cases of diffuse axonal injury in children under 1 year of age, parenchymal tears are

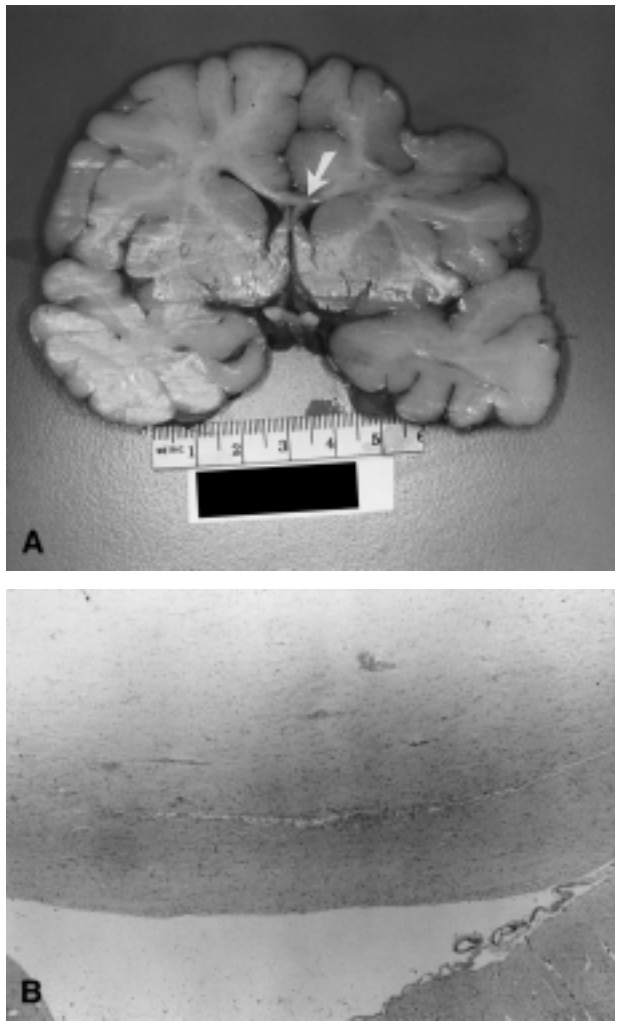

FIG. 6. Seventeen-week-old infant (same infant as in Fig. 4). (**A**) Right side of corpus callosum has a 6-mm linear tear (arrow). (**B**) Photomicrograph of linear tear in corpus callosum with split in the tissue and fresh hemorrhage.

also grossly observable. This is the lesion Lindenberg described as the contusion tear (53). These are slitlike tears that occur at the cortex-white matter junction or within the layers of the cortex and are caused by the differential movements within the brain while some portions of the brain shear or slide apart during differential rotation of the brain tissues (Figs. 6 and 7). Contusion tears are rare, and when seen they are accompanied by the usual markers of diffuse axonal injury, the subarachnoid and subdural hemorrhages in the usual locations (33). Care must be taken not to misinterpret a cross-section through the depth of a sulcus as a contusion tear. It is also prudent to take care to not mistake artifacts created by the process of handling or cutting the brain as true tears. Contusion tears should not be diagnosed on the basis of finding only a microscopic tear without other evidence of diffuse axonal injury.

It is not usually possible to morphologically establish the existence of diffuse axonal injury in young children by demonstrating the classic pathologic changes of retraction bulbs, tissue tears, or intraparenchymal hemorrhages, although these findings may be demonstrated on occasion (33,54). Many of these children die too soon after injury for these pathologic changes to be established. For this reason, it is important to appreciate the markers of shearing injury to identify these cases as diffuse axonal injury.

BRAIN SWELLING

Shearing injuries in young children are accompanied by various degrees of brain swelling. The swelling may not be apparent at autopsy in infants with brief survival intervals. Initially, CT may demonstrate progressive brain swelling and decreased ventricular size without other lesions being visible. The swelling is probably related both to direct injury to the axonal processes, causing localized edema, and to generalized swelling caused by changes in vascular permeability and autoregulation (5). Some investigators have postulated that hypoxia occurring when a child is shaken and becomes apneic accounts for the underlying cerebral insult and brain swelling (55). However, hypoxia does not explain why the injury is sometimes more unilateral than bilateral, the atrophy that develops in the brains in children who survive, or that the appearance of these brains at autopsy is not typical of hypoxic injury.

TIMING OF INJURIES

Timing of the head injury is often an important issue because most abusive injuries occur only in the presence of the individual who injured the child and who may not provide an accurate history. Studies in children dying of accidental head injuries indicate that children with diffuse injury show an immediate decrease in the level of consciousness (16,56). Studies in children with nonaccidental head injuries also indicate that they show an immediate decrease in their level of consciousness at injury (36). Individuals sustaining diffuse brain injury of moderate to severe degree become symptomatic immediately (24,31,49). Young children with moderate to severe degrees of diffuse brain injury would certainly include those in whom there is a significant neurologic outcome or death. Correlations of clinical and experimental observations on

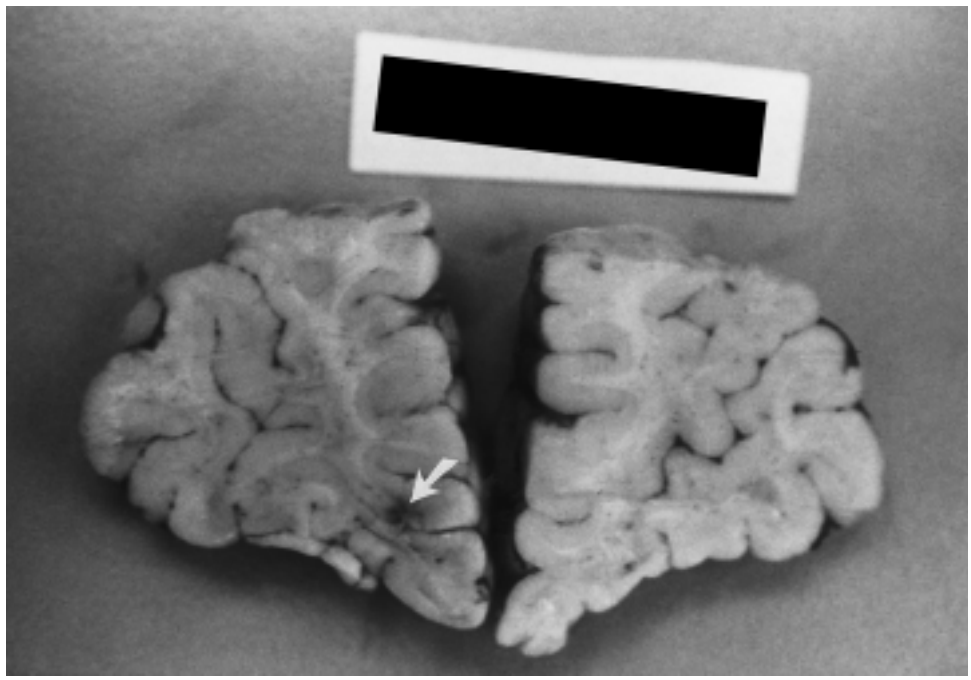

FIG. 7. Seven-month-old infant with depressed skull fracture of left parietooccipital calvarium with small acute subdural hemorrhage over the posterior aspects of both cerebral convexities demonstrating a 3- to 4-mm contusion tear (arrow) in left orbital subcortical white matter.

cerebral concussion and traumatic unconsciousness demonstrate that progressively deeper disconnections of axonal processes affecting the deep gray matter and rostral brainstem are the cause of the unconsciousness in these children, not that the unconsciousness is the result of increasing intracranial pressure or hypoxia (24). Symptoms experienced by these severely injured children include an immediate decrease in the level of consciousness (either lethargy or unconsciousness); respiratory irregularity, difficulty, or apnea; and frequently seizures. The respiratory difficulty in these children may be related to damage to the lower brainstem (medullary) centers of respiratory control. The timing with which respiratory difficulty develops is not very precise, but it is concurrent with or follows the decrease in level of consciousness in these children, who cannot survive for many hours without ventilatory support. It is not currently possible to predict the exact length of time such an injured child may survive.

Children who sustain repetitive episodes of mild diffuse brain injury may gradually accumulate brain damage and acquire neurologic deficiencies (14). The exact timing of such mild additive injuries is not possible. There is no reason to believe that remote shearing lesions would make the brain more susceptible to new shearing injury. Mild injuries unaccompanied by loss of consciousness are not usually brought to medical attention. Some mild shearing injuries are manifested as seizures and clinically present difficult diagnostic problems, because there is no currently available method to demonstrate the underlying pathologic changes of the shear injury until more severe degrees of diffuse axonal damage have been sustained and can be recognized by the markers of subdural or subarachnoid hemorrhage on CT or MRI (38).

CHRONIC SUBDURAL HEMATOMA

Rebleeding after trivial injury or spontaneous rebleeding from a preexisting chronic subdural hematoma should not be offered as an explanation for the presence of acute subdural blood lacking obvious demonstration of such an old subdural membrane (57). The pathogenesis of subdural bleeding has become better delineated after more than 20 years' experience with CT. The classic multilayered chronic subdural hematoma is currently considered a unique type of hemorrhage for several reasons. A chronic subdural hematoma very rarely follows severe head injury in a previously normal person, in whom an acute subdural hemorrhage transforms by aging to become a chronic subdural membrane. Instead, the blood of the acute subdural hemorrhage in these head injuries is readily resolved or rapidly organized (58–60). The resorption of subdural blood tends to be even more rapid and more complete in children than in adults (19). The development of the classic multilayered chronic subdural hematoma results from venous bleeding under low pressure and requires the potential for the subdural space to enlarge without a significant increase in pressure. The

factors that promote such a development within a low-pressure intracranial space exist only in specific categories of people, such as those with brain atrophy (i.e., the elderly and those with alcoholism), those with hydrocephalus who have been treated by placement of a ventricular shunt, or those with traumatic encephalomalacia (59). In children with glutaric aciduria type 1, frontotemporal atrophy develops, and occasionally subdural hemorrhage without trauma develops on that basis (61). Minor trauma in these specific categories of patients may result in tearing of bridging veins and small amounts of subdural hemorrhage, which induces an ingrowth of granulation tissue from the dura. This granulation tissue contains fragile capillaries, which may produce microbleeds leading to enlargement of the hematoma. The further evolution of these hematomas is determined mostly by the nature of the vascular neomembrane formed in these patients. The expansion of these hematomas also appears to be related to the excessive activation of both the clotting and the fibrinolytic systems in the subdural fluid (62–64). A young child whose subdural hemorrhage subsequently organizes into a membrane composed of large vascular channels at risk for rebleeding would have been symptomatic before the time of rebleeding, because there would have been a preexisting brain abnormality. The signs and symptoms that would be expected before rebleeding include seizures, macrocephaly, anorexia, lethargy, headache, and apnea (60).

About 20% to 30% of asymptomatic neonates have small amounts of subarachnoid and subdural hemorrhage during delivery. The resolution of this blood may result in the presence of small numbers of dural macrophages containing hemosiderin and, sometimes, small fibrous patches consisting of a few layers of granulation tissue on the dura. These patches of thin membrane or scattered macrophages are not at risk of rebleeding with trivial trauma. Proof of rebleeding of a chronic subdural membrane should be based on the demonstration of a chronic subdural membrane that is grossly evident at autopsy, followed by microscopic confirmation of the vascularized membrane, and should not be based solely on the microscopic finding of fragments of fibrous tissue or a few macrophages containing hemosiderin.

The dura is a tough, fibrous, bilayered membrane overlying the arachnoid. It consists of an inner layer (menigeal) and an outer layer (periosteal). The periosteal layer serves as the periosteum of the inner table of the skull. The dura of young children, particularly along the basilar skull sutures, is a very cellular structure, which contains growing fibrous tissue along with numerous hematopoietic cells, including macrophages, many of which normally contain hemosiderin. The appearance of the normal young dura may be misinterpreted as having a thin chronic subdural membrane by microscopists who are not familiar with looking at these young duras, who may not be able to tell the inner from the outer dural surface, and who may incorrectly believe a chronic membrane to be the cause of acute subdural hemorrhage.

INTERPRETATION OF INJURY

The distinction between nonaccidental and accidental head injury in children is an area of concern for pathologists as well as other medical specialties. Fatal accidental shearing or diffuse brain injuries require such extremes of rotational force that they occur only in obvious incidents such as motor vehicle accidents. Besides vehicular accidents, other fatal accidental childhood head injuries tend to involve crushing or penetrating trauma, which is readily evident. These injuries tend to be the result of falling from considerable heights (greater than 10 feet) or having some object penetrate the head. There are distinctions between head injuries that are truly accidental and those that are abusive. The incidence of isolated subdural/subarachnoid hemorrhage as the only gross finding in fatal accidental head injuries in young children is less than 2%, compared with the 90% to 98% incidence of these hemorrhages associated with abusive head injuries (Case ME, unpublished autopsy studies). The trivial home accidents that children so frequently sustain are associated with primarily translational forces and not with the rotational forces necessary to develop tearing of bridging veins, which would produce subdural hemorrhage or other shearing injury (26,32,65–71). In low falls of less than about 8 feet, witnessed by uninvolved and nonbiased individuals, about 1% to 2% of children sustain a narrow simple linear skull fracture. In a small proportion of the children who sustain these skull fractures, an epidural hemorrhage will develop. These epidural hemorrhages are not accompanied by a decrease in the level of consciousness at the time of injury. If the epidural bleeding continues and produces significant increased intracranial pressure, there may be a subsequent decrease in the level of consciousness attributable to tentorial herniation.

It is essential that a meticulous autopsy examination be performed in all cases of possible injury to children. When subdural and/or subarachnoid hemorrhage is found at autopsy, the brain must be thor-

oughly examined to exclude the possibility of other causes of bleeding in these spaces. Although berry aneurysms are uncommon in young children, they may occur. Vascular malformations may also occur in young children and cause hemorrhage in intracerebral and intracerebellar subarachnoid and subdural areas. The distribution of bleeding in aneurysms and arteriovenous malformations is unlikely to resemble that of head injury, but these malformations need to be excluded by careful examination of the brain (72,73).

CONCLUSION

Anatomic and developmental differences of the brain and skull of children under the age 4 or 5 years make the head injuries and mechanisms of injury that affect these children different in certain respects from those occurring after that age. Inflicted head injuries in these young children usually create shearing injuries of the brain and blood vessels, resulting in diffuse axonal injury and subdural, subarachnoid, and retinal hemorrhages. The pathologic findings of subdural and subarachnoid hemorrhages and very, frequently, retinal hemorrhages are the most common findings by which these rotational head injuries in young children are identified at autopsy. Recognition of the underlying mechanism of the rotational brain or shearing injury is important to an understanding of the clinical course of these children, particularly with respect to the decrease in the level of consciousness and respiratory distress demonstrated after injury.

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