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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*American Journal of Forensic Medicine and Pathology.* They believed that while it was worthy of publication, it should not be published as a position paper because of the controversial nature of the subject.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.

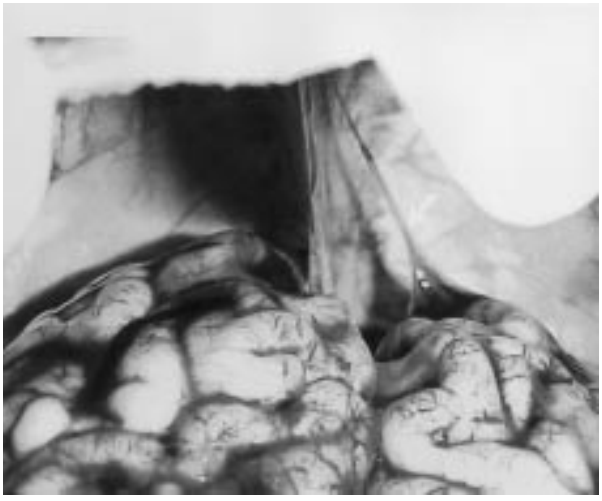

FIG. 1. Thirteen-week-old infant with normal brain and intracranial spaces demonstrating bridging vein (arrow) arising from left cerebral convexity (right).

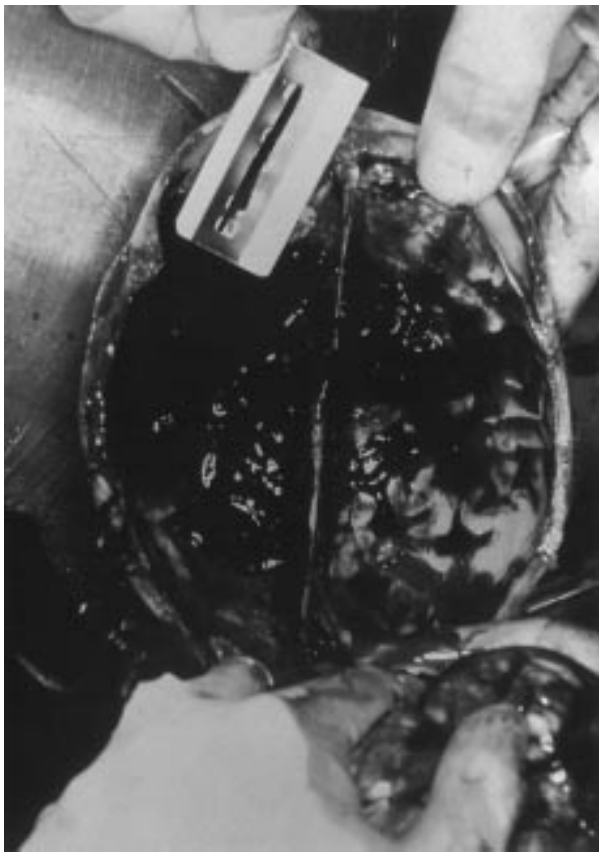

FIG. 2. Nine-month-old infant with fracture of right parietal calvarium showing bilateral acute subdural hemorrhages over the cerebral convexities.

SUBARACHNOID HEMORRHAGE

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

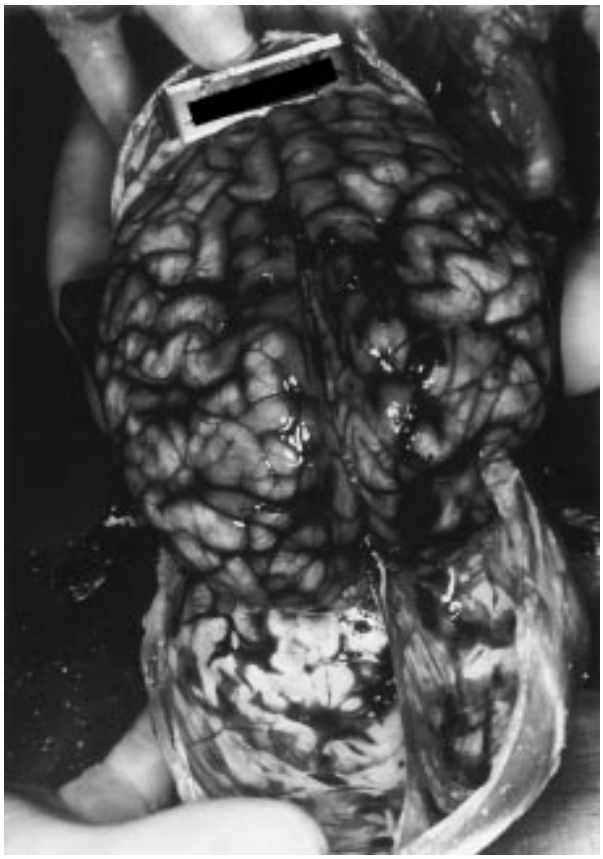

FIG. 3. Eleven-week-old infant with small amounts of acute subdural hemorrhage over both cerebral convexities.

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

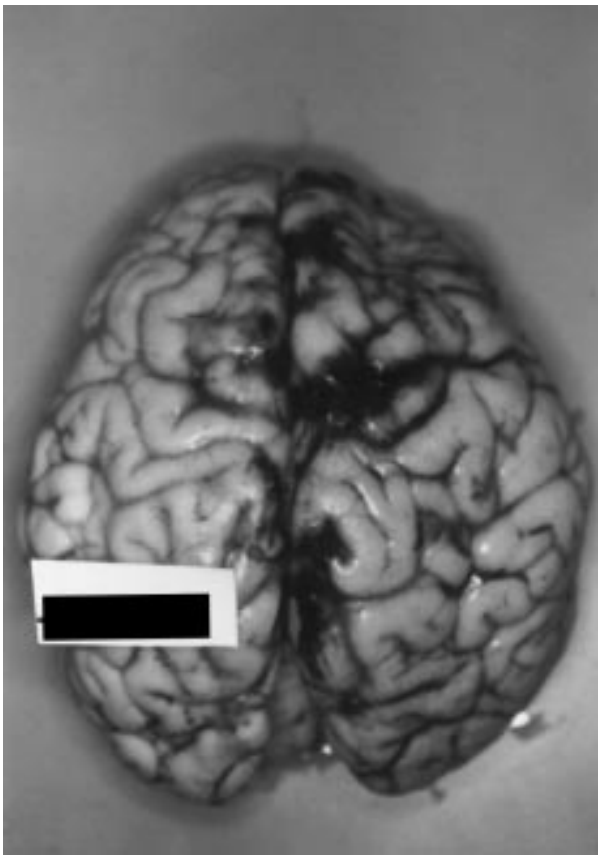

FIG. 4. Seventeen-week-old infant with large (70–80 ml) acute subdural hemorrhage over right cerebral convexity demonstrating patches of subarachnoid hemorrhage over both parasagittal regions, greater on the right than on the left.

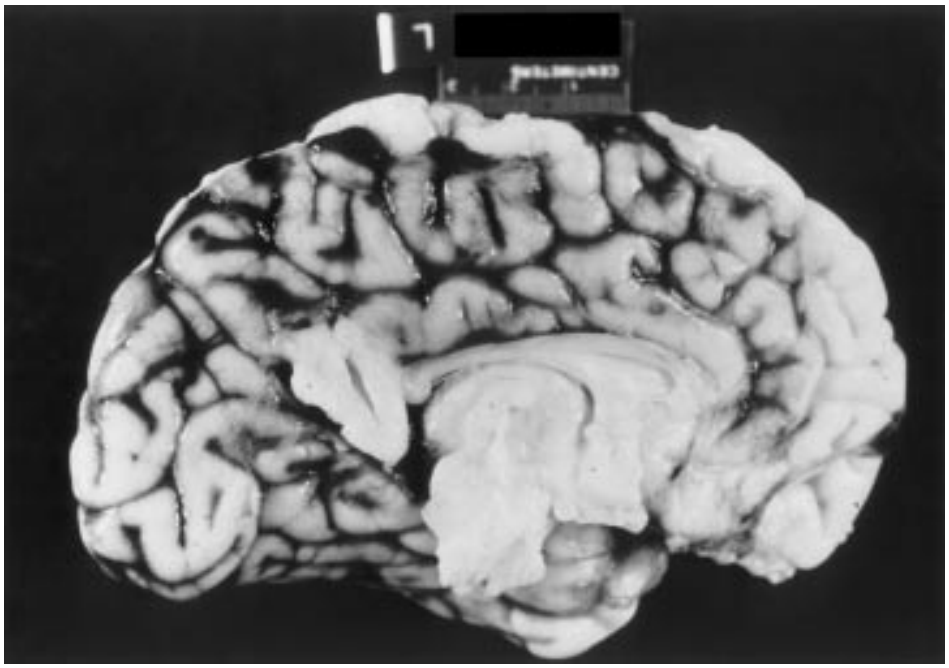

FIG. 5. Five-month-old infant with large bilateral acute subdural hemorrhages over cerebral convexities showing patches of subarachnoid hemorrhage on the mesial surface of the right cerebral hemisphere.

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

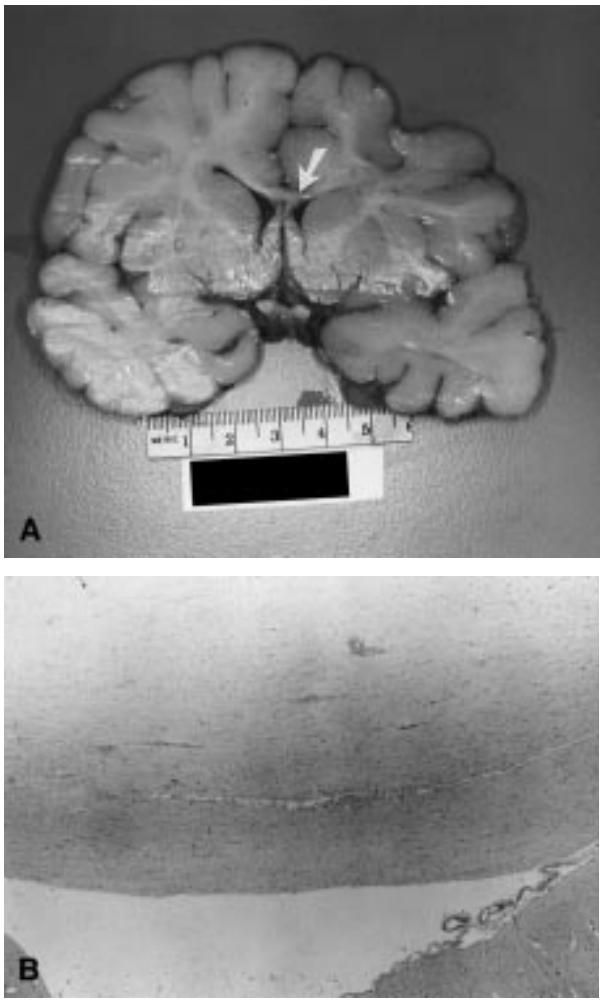

FIG. 6. Seventeen-week-old infant (same infant as in Fig. 4). (**A**) Right side of corpus callosum has a 6-mm linear tear (arrow). (**B**) Photomicrograph of linear tear in corpus callosum with split in the tissue and fresh hemorrhage.

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

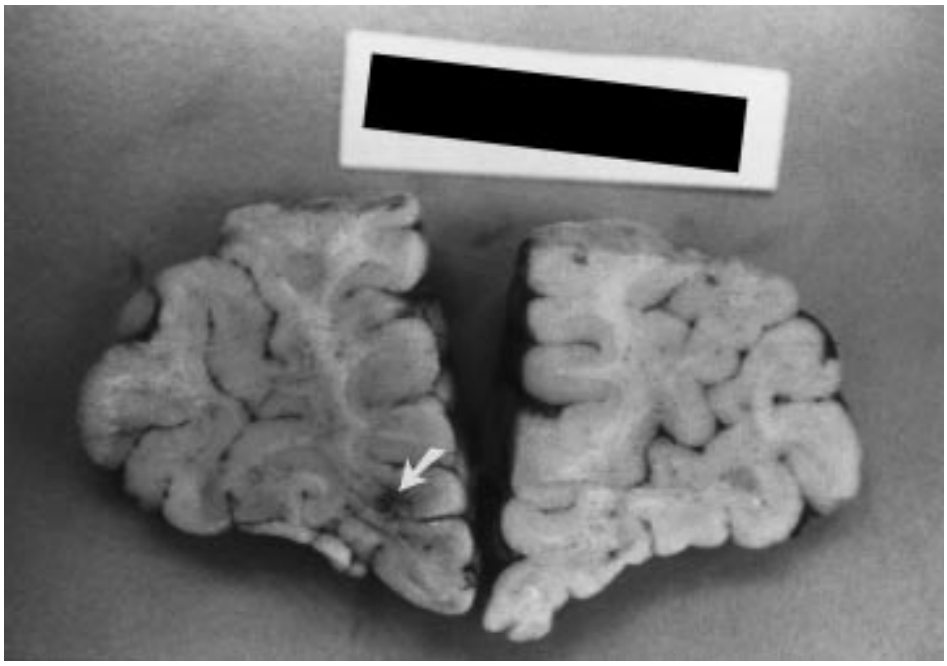

FIG. 7. Seven-month-old infant with depressed skull fracture of left parietooccipital calvarium with small acute subdural hemorrhage over the posterior aspects of both cerebral convexities demonstrating a 3- to 4-mm contusion tear (arrow) in left orbital subcortical white matter.

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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Symptoms Following Head Injury

To the Editor:

A recent proposed position paper regarding pediatric nonaccidental abuse head injury would allege that we may depend on symptoms to appear immediately upon injury (1). I have a concern about that claim, which I will explain with the following case. CASE REPORT

A 13-month-old Hispanic girl was brought to the University of Wisconsin Hospital on the morning of September 18, 1999. The complaint was of vomiting that had lasted for 24 hours. She was described as irritable, sleepy, and vomiting. In our emergency room she was noted to have extensive bruises on the cheeks, chest, back, and arms; the mother attributed these to bites by a 3-year-old housemate. She was admitted and given intravenous fluids. She was sedated with pentothal followed by head computed tomography, which was negative. She was then admitted to the pediatric ward. The resident who saw her described her in the chart and in discussion as being fussy and clingy, but interactive and responsive. Because of the numerous bruises, the police were notified and took pictures.

At about 2:00 the following morning, a nurse coming in to care for the child noted that she had decreased respirations. It was then shown that she was unresponsive and had a right dilated unreactive pupil with a sluggish left pupil. She was taken emergently to the pediatric intensive care unit, where she was intubated and given mannitol. A subsequent computed tomography scan showed very poor differentiation of gray/white matter interface. A Codman catheter was placed and then replaced with a ventriculostomy tube after an intracranial pressure of 21 mm Hg was noted.

On the evening of the day after admission, a cerebral blood flow study showed no cerebral blood flow. She was pronounced brain dead.

In the interim, her mother had fled town and has not been found since.

An autopsy was done on September 20, 1999. This showed hemorrhage in the left optic nerve sheath and left retinal hemorrhages as well as marked cerebral edema and thin widespread subdural hemorrhage. Diffuse axon injury was demonstrated with amyloid precursor protein antibody.

My point is that the child did have some symptoms, but clearly the severe intracranial injury symptoms, which were confirmed on repeat computed tomography and autopsy, were delayed for several hours, during which time she was under our view and review in the hospital. Others have noted similar problems (2).

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Madison, Wisconsin, U.S.A.

REFERENCES

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Author's Response

To the Editor:

In response to Dr. Robert Huntington's letter in which he expresses concern about the Position Paper's position on interval from injury to symptoms in young children with abusive head injuries, I would make the following comments. Dr. Huntington describes a child who is noted to be "irritable, sleepy, and vomiting" on admission. The Position Paper states, "Symptoms demonstrated by these severely injured children include an immediate decrease in the level of consciousness (either lethargy or unconsciousness)." Lethargy is defined as a condition of drowsiness or indifference *(Dorland's Illustrated Medical Dictionary)*. The sleepiness in this child is the neurologic symptom that marks the time at which the injury to this child occurred. It indicates a decrease in the level of consciousness. After the child was in the hospital several hours, she showed signs of increased intracranial pressure (right dilated pupil and sluggish left pupil) and went on to brain death. Dr. Huntington remarks that the severe intracranial injury symptoms were delayed for several hours. The symptoms to which he is referring were not the initial symptoms of injury but those related to increased intracranial pressure. The brain injury that precipitated this course of events (diffuse axonal injury) occurred when the child first became neurologically symptomatic. Brain swelling followed the diffuse axonal injury and resulted in increasing intracranial pressure and eventually brain death. Certainly, the child's neurologic symptoms changed with time, reflecting this changing neuropathology. It is the initial neurologic change that marks the time of injury.

The article by Gilliland that Dr. Huntington mentions is not helpful in elucidating the interval from injury to symptoms. The cases reported in that article relied on histories from possibly biased witnesses (caregivers who

might have injured a child) and took at face value the time intervals provided in each case. Dr. Gilliland noted this problem in her article, stating, "It should be noted that in all of the cases where information was supplied by someone other than the perpetrator, the child was not normal during the interval."

The claim that a young child has been fine for hours after a fatal head injury was inflicted and then suddenly developed symptoms is a claim that has no support from legitimate or mainstream medicine. When a child has suffered a serious acceleration injury to the brain that will result in long-term neurologic impairment or cause death, the so-called lucid interval is a fiction. The change from "fine" to "not fine" may be lethargy or it may be unresponsiveness, but it is a neurologic change, and it occurs at the time of injury.

Mary Case, M.D.
St. Louis, Missouri, U.S.A.

To: Leestma, Jan
Cc: Hanzlick, Randy L.; Bandt, Cal; Contostavlos, Dimitri; Davis, Gregory G.; Davis, Joseph; DiMaio, Vincent; Dragovic, Ljubisa; Hutchins, Kenneth; Lindsey, Lindsey; Massello, William; Pestaner, Joe; Rose, Earl F.; Stephens, Peter; Walker, Shirl; Baden, Michael; Bandak, Faris; Barnes, Patrick D.; Carlstrom, Thomas; Clark, Brian J.; Cory, Corrina; Geddes, Jennian; Gilles, Floyd; Goldsmith, Werner; Howard, Matt; Jones, Michael; Justis, David; Nelson, Marvin; Ommaya, Ayub; Stephenson, John; Thibault, Kirk; Uscinski,Ron; Whitwell, Helen
Subject: RE: Recent trial experience

Jan: I am forwarding this to selected NAME members plus Geddes, Barnes, Thibault, Goldsmith, etc.

This makes me sick. Case testified in a trial last Wednesday and the "Position Paper" played a prominent part. How do you make a judge or jury UNDERSTAND that the "collected wisdom" of the "medical profession" is WRONG, provably WRONG? How do you make a judge or jury UNDERSTAND that "widely accepted" does NOT mean "true"? How do you make them understand that the sum of "vast clinical experience" is not science? (I would FORCE every potential Board-certified FP or "Child Abuse" Pediatrician to read and UNDERSTAND all of S. J. Gould's collected essays published over the past ten years in "Natural History" as a prerequisite for Board Certification and testimony.) How do you address the "collected wisdom" of physicians "only concerned about the welfare of the child"? The pious parade of pediatricians and pathologists. How do you explain that the theories of Case et al. violate EVERY principle of classical mechanics and EVERY conclusion from EVERY study done by EVERY University department of Biomechanics for the past half century? How do you address statements AND testimony from Chadwick and Alexander (exact quote) that "application of the principles of mechanics has nothing to offer in the evaluation of infant injury, since kids are different and we already know what causes the injury"?

The disclaimer at the bottom of the first page does NO good. The judge and the jury do not CARE that the "peer-reviewers 'recommended that it not be published as a Position Paper' because of the controversial nature...". It WAS published, and IS the "Official Position" until and unless the Board of Directors rescinds it. This is ultimately going to be more of a scandal than the repressed memory, satanic rituals, day-care sexual assault, etc., scandals COMBINED.

Families, finances, and lives are being destroyed.

John

John Plunkett
Regina Medical Center
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Position Paper, 7-01.pdf

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Plunkett, John

From: dimaio@co.bexar.tx.us[SMTP:dimaio@co.bexar.tx.us]
Sent: Monday, July 23, 2001 9:50 AM
To: Plunkettj@ReginaMedical.org
Subject: RE: Recent trial experience

You have my permission to send it on.

-----Original Message-----
From: Plunkett, John [mailto:Plunkettj@ReginaMedical.org]
Sent: Sunday, July 22, 2001 3:58 PM
To: DiMaio, Vincent
Subject: RE: Recent trial experience

Vince: Would you please forward this to everyone to whom I sent the original message? I think that you have made a very important point. (I can forward it if you want, but only with your permission.) John

John Plunkett
Regina Medical Center
1175 Nininger Road
Hastings MN 55033
651-480-4251

From: dimaio@co.bexar.tx.us[SMTP:dimaio@co.bexar.tx.us]
Sent: Saturday, July 21, 2001 12:39 PM
To: Plunkettj@ReginaMedical.org
Subject: RE: Recent trial experience

The problem is much bigger than this Position Paper. The disclaimer should have caused the Judge to tell the jury that this article was NOT accepted as a peer review article and the content is therefore suspect. The object of the peer review system is to prevent the dissemination as fact of nonscientific contentions. It cannot prevent publication.

That the judge did not comprehend this is part of a much bigger problem of the Courts letting unqualified experts testify. When rulings as to cause and manner of death are made by non-physicians (coroners); when organizations issue dubious credentials; when anyone is an expert and when the media presents as fact fiction, this paper is only a minor injustice.

From: Vincent Dimaio[SMTP:dimaio@CO.BEXAR.TX.US]
Reply To: National Association of Medical Examiners
Sent: Thursday, February 07, 2002 2:44 PM
To: NAME-L@LISTSERV.CC.EMORY.EDU
Subject: Position Paper Fatal Abusive head injuries

I have just been given a copy of a Internet posting by The National Center on Shaken Baby Syndrome. In it they cite "Position paper on fatal abusive head injuries in infants and young children". I have been dreading this. I would like to clarify some aspects of this position paper.

As editor of the AJFMP, I had serious misgivings about publishing this paper, not because of its contents but in that it is described as a position paper. Position papers in NAME reflect the opinions of the Board of Directors, not necessarily the membership or even the forensic pathology literature.

The AJFMP is a peer review journal. If one bothers to read the box in the lower left corner of the first page of the article, one will see that the paper was rejected as a position paper by the three reviewers. They felt that it should be published as a regular paper if published. As an aside, the paper in its original form was rejected by 4 of 5 reviewers (the reason for so many reviewers was the fact that this was a position paper). Thus, the "peers" rejected it as a position paper. The paper was published because (1) whether one agrees or not, the authors have a right to their opinion and (2) as a courtesy to the Board of Directors (though with the stipulation that the peer reviewers opinion also be printed).

Shaken baby syndrome is controversial in that a number of individuals doubt its existence - including Gennarelli whose research is cited by many as an explanation for the injuries produced.

16. Consumer Product Safety Review 4:#2:3–7, 1999. Available at: www.cpsc.gov/cpscpub/pubs/cpsr.html
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Sent: Thursday, March 06, 2003 9:13 AM
To: Plunkettj@ReginaMedical.org
Subject: RE: Position paper

The "position paper" was reviewed by peer reviewers and determined not to be a position paper but an ordinary article expressing the opinion of the authors. It was so published. People forget that little box at the front of the paper. The paper doses not meet the criteria of a position paper. To quote Lincoln: If you call a dog's tail a leg then how many legs does a dog have? Four. Calling a tail a leg does not make it one.

-----Original Message-----
From: Plunkett, John [mailto:Plunkettj@ReginaMedical.org]
Sent: Wednesday, March 05, 2003 7:38 PM
To: Albert King (E-mail); Ayub Ommaya (E-mail); Brian J. Clark (E-mail); Chris Milroy (E-mail); David Justis (E-mail); Dimitri Contostavlos (E-mail); Don Jason (E-mail); Douglas Knittel (E-mail); Earl F. Rose (E-mail); Faris Bandak (E-mail); Floyd Gilles (E-mail); Gill Adams (E-mail); Gregory D. Reiber (E-mail); Helen Whitwell (E-mail); Horace Gardner (E-mail); Jan Leestma (E-mail); Janis Amatuzio (E-mail); Jennian Geddes (E-mail); Joe Pestaner (E-mail); John Plunkett (E-mail 2); John Plunkett (E-mail); John Stephenson (E-mail); Joseph Davis (E-mail); Joseph Scheller (E-mail); Julie Niedermier (E-mail); Ken Monson (E-mail); Kenneth Hutchins (E-mail); Kirk Thibault (E-mail); Larry Thibault (E-mail); Lindsey Thomas (E-mail 2); Ljubisa Dragovic (E-mail); Mark Donohoe (E-mail); Mark J. Shuman (E-mail); Marvin Nelson (E-mail); Patrick D. Barnes (E-mail); Patrick Lantz (E-mail); Pauline Alakija (E-mail); Peter Stephens (E-mail); Randy Frost (E-mail); Richard Reimann (E-mail); Roger McLendon (E-mail); Ron Uscinski (E-mail); Shirl Walker (E-mail); Susan Roe (E-mail 2); Thomas Carlstrom (E-mail); Thomas L. Bohan (E-mail 2); Vincent DiMaio (E-mail); Waney Squier (E-mail); Werner Goldsmith (E-mail); William Massello (E-mail 2); William Massello (E-mail)
Subject: Position paper

One of the members of this group called me a few minutes ago and asked how the "Position Paper", which had recently been presented to him by a prosecutor in an SBS case, could be accepted for publication. I told him that it was under duress, and with a disclaimer. Please pay particular attention to Dr. DiMaio's comments in the last few pages, which he gave me permission to forward last year. John

-----Original Message-----
From: ljubisa j. dragovic [mailto:dragoviclj@co.oakland.mi.us]
Sent: Thursday, March 06, 2003 2:06 PM
To: Plunkett, John
Subject: Re: Position paper

John,

For a better understanding how the position paper eventually got published, after being rejected as such several times, one has to comprehend the "democratic" process involved. In functioning of any political body, maintaining the democratic process, i.e., the rule by majority vote is fundamental. The NAME Executive Committee (composed of two authors of the position paper, and several other members fairly oblivious to a scientific process in the first place) put the item on the NAME Board of Directors agenda, to be voted on as a political decision of a political body. The Board decided by a huge majority that the paper must be published as a position paper in the AJFMP, since the AJFMP is essentially the MEDIA ORGAN of NAME. This political decision was made in spite of the fact that more than a half of the Board members at the time were not at all cognizant of the profound problems with that paper, and there are some of those members that have not read the paper to this day and/or never have developed a clue about it. A reminder: Democracy is not applicable to a scientific process, which is essentially a dictate of facts placed in an appropriate logical sequence.

Despite the Editor's vocal objections, he was ordered to publish this type of science using democratically established political instruction. The Editor was then left with no professional choice. To protect the integrity of the Journal, the Editor placed a disclaimer.

When the position paper came out few interested individuals read it carefully. The important thing for many was that the "gospel" had been legitimized for all practical/legal purposes. It really took months that the people became aware of the disclaimer. It took far less for the authors to turn livid and request the Editor's head on a platter.

The first part of the Epilogue (two years) has been spent on efforts to root out the Editor. The Editor has been upset and confronted the protagonists in a democratic fashion-the election in
Shreveport, LA. There, the oblivious majority reaffirmed the status quo, paving a path into quick sands of the world of position papers. In that world there is no need to think. All you need to do is apply the wisdom of position papers and continue to play golf without feeling any responsibility for the determinations you have made using the position papers as a pigeonhole system.
For anything that is not covered by position papers yet, there is a powerful committee for position papers, and given time, quality decrees will become available, to the point that one day there will not be a need to even take your hat off (something that some use as an initiation of thinking as a process!).

Meanwhile, NAME listserv is always there for those that don't like to read, or think, so that they can send their cases (scatchy accounts) through the network and get fast/off the cuff (and free) authoritative consults.

The old skeptic H.L. Mencken wrote: "For every human problem there is a solution that is fast, plausible and wrong." Well, I cannot say I was not there, but it seems it did not make any difference... Regards, LJD.

"Plunkett, John" wrote:

One of the members of this group called me a few minutes ago and asked how the "Position Paper", which had recently been presented to him by a prosecutor in an SBS case, could be accepted for publication. I told him that it was under duress, and with a disclaimer. Please pay particular attention to Dr. DiMaio's comments in the last few pages, which he gave me permission to forward last year. John

John Plunkett
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September 21, 1998

John E. Pless, M.D.
President
National Association of Medical Examiners
635 Barn Hill Road
Indiana University Medical Center
Medical Science Building Room 157
Indianapolis, Indiana 46204

Dear John:

I have finished reviewing the manuscript forwarded to me by Denise with a request to indicate my acceptance or rejection as the official position of NAME in regard to the issue of "Shaken Baby Syndrome".

First of all, it was an excellent idea to appoint a committee to review this matter and prepare a position paper for further discussion. The national publicity given to the "Nanny" case in Massachusetts along with similar but lower profile cases that occur in most major jurisdictions in this country could not help but move us to action on this important matter in forensic pathology.

I was unable to be at the meeting in San Francisco because of the need for me to present my budget at the Maryland Legislature, so I am unfamiliar with what the Ad Hoc Committee was charged to do. I do not know if there was a discussion of what a position paper should include, how it should be structured and how it should be worded so that it can be presented as an official voice of our Organization.

It is my personal and professional opinion that a position paper should include the following:

1. a distillation of the scientific knowledge available on a certain topic, lucidly and concisely described;

2. a description of the role that the National Association of Medical Examiners assumes in dealing with this particular issue;

3. examples of disputes between findings of medical examiners and forensic pathologists over the scientific matter;

4. a recommendation on the fundamental principles governing the action of medical examiners in dealing with a death of a child which may fit into this particular diagnostic category.

While I commend the members of the committee who authored this paper I do not believe it represents a NAME position paper on this topic. It is certainly a nicely written academic treatise on the topic "Shaken Baby Syndrome". But even overlooking such statements as in the opening paragraph that "most infant deaths are the result of this mechanism", which is certainly disputed, it does not convey the message, which I believe NAME would want other members of the scientific community or the public to hear. One example of a specific case that caused widespread international criticism of forensic pathology practices in this country is the Matthew Eappen case. This case should be analyzed by NAME to determined how the case came to be "mishandled". It should address the legal issues raised by such cases and how these issues can be addressed during immediate investigation and autopsy preventing future problems.

Issues such as a failure to investigate the cause of a healing skull fracture as present in Matthew Eappen, represent a fundamental departure from what NAME should recommend to its members in the investigation of such a death. To minimize the role of the medical examiner in the investigation of these deaths and emphasize the anatomical pathology element does a disservice to what our members would expect such a paper to provide.

I strongly recommend that this paper be discussed at the up-coming meeting in Albuquerque. I don't believe there is any urgency for having this published.

I hope you give these comments your serious consideration and I hope you will call if you have any interest in discussing these points further.

I look forward to seeing you in Albuquerque.

Best regards,

Yours sincerely,

John E. Smialek, M.D.
Chief Medical Examiner

JES/skw

THE AMERICAN JOURNAL OF FORENSIC MEDICINE AND PATHOLOGY

Vincent J.M. DiMaio, M.D., Editor
Bexar County Forensic Science Center
7337 Louis Pasteur
San Antonio, Texas 78229-4565

Please return the paper, photos and this form in the enclosed post paid envelope.

Reviewer **Dr. John E. Smialek** Return By January 14, 2000 MS# **99-136**

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

RATINGS	CIRCLE THE GRADE					
		Most				Least
1. Accept as submitted						
2. Accept, suggested changes (may be noted on manuscript)	Original	1	2	3	4	5
3. Provisional acceptance. Required changes (listed on carbon packets)	Valid	1	2	3	4	5
4. I would like to see paper after revision ✓	Significant	1	2	3	4	5
5. Unacceptable, reconsider with major revision ✓	Comprehensive	1	2	(3)	4	5
6. Unacceptable						

Comments See attached

99-136

This paper, authored by an Ad Hoc Committee of the National Association of Medical Examiners, purports to address "the topic of abusive head injuries in young children. The article does address this topic and reviews a considerable amount of the existing literature on this subject. However, the paper has several serious flaws.

Another major shortcoming, over which the committee had apparent little control, is the stated purpose of this manuscript. The authors refer to their original charge, apparently given to them by either the Board or the President of the National Association of Medical Examiners, "to produce a position paper on Shaken Baby Syndrome (Pg. 5)." The committee states that they interpreted this terminology to refer, generally, to the subject of abusive head injury in young children. A charge to a committee to produce a position paper on a topic, as poorly defined as this, immediately undermines any effort on the part of a committee to produce a meaningful result.

A position paper on Shaken Baby Syndrome should refer to diagnostic criteria, which must be met before the term is used. It should place this entity in a historical and scientific perspective with recommendations for resolving the "controversy" (pg. 5) surrounding this topic. This paper failed to address the nature of the controversy and never refuted literature from their own bibliography, which conflicted with their position.

Early in the introduction (pg. 5), the authors state their "intent" to inform the practicing pathologist about the proper recognition, interpretation and clinical correlation of these injuries." The body of the paper is then divided into these sections: mechanisms of injury, subdural hemorrhage, subarachnoid hemorrhage, retinal hemorrhage, diffuse brain injury, brain swelling,

timing of injuries, chronic subdural hematoma, interpretation of injury and conclusion. The aforementioned intent is finally addressed on page 26 where the reader is informed that "in cases in which subdural and/or subarachnoid hemorrhage is found at autopsy, the brain must be thoroughly examined to exclude the possibility of other causes of bleeding in these spaces."

Nowhere in this paper is a recommendation made to the pathologist to carry out a thorough, objective investigation of the circumstances, under which a child sustained the injury. The lack of such a component in arriving at a determination of whether an injury is abusive or non-abusive, warrants the rejection of this paper, by the Journal, in its present form. Furthermore, it raises a serious question as to the validity of this information to an organization like the National Association of Medical Examiners, especially as a "position paper."

This paper gives the impression that it represents the position of one of the authors who in defense of his/her position, relies on his/her own personal experiences (Case M, unpublished autopsy studies). This author states that 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 to the 90-98% incidence of these hemorrhages associated with abusive head injuries. The authors further state that virtually all injured children, who have not be in a motor vehicle accident or fallen from a building are victims of abuse.

They state "the trivial home accidents that children so frequently sustain are associated with primarily translational forces and not with rotational forces necessary to develop tearing of bridging veins, which would produce subdural hemorrhage or other shearing injury." This statement represents one side of the controversy that presently exists in the forensic science

community regarding these injuries and it ignores the position of a large community of forensic scientists who believe either to the contrary or that the matter is not so clear cut.

The authors cite nine scientific articles that agree with this statement, but in doing so, choose to ignore information produced by authors included in their own bibliography that conflict with this opinion. For example, they cite in reference #53, Lindenberg and Freytag, while ignoring other observations made by the same authors who state, "the degree of acceleration does not depend on the height of the fall. It may be greater and produce more damage in a fall from a sitting position than from a standing position." (Reference 1)

Certain key elements of the paper, such as the mechanism of injury involving rotational forces being responsible for abusive head injuries, imply that rotational forces do not occur with non-abusive head injuries. For example, in accidental falls, (Page 10) "the trivial falls that children sustained in falling from furniture and even downstairs, primarily involve translational forces. Really?? Although such falls may occasional result in a skull fracture, these incidents are generally benign (just a skull fracture!) and do not result in loss of consciousness, neurological deficit, or death." In other words, falls from furniture or downstairs don't involve rotational forces, according to the authors. How can one know this without having familiarized himself with the information gathered in the investigation of the circumstances and scene of the death. On the other hand, the authors firmly believe that "shaking" consistently does produce rotational force of a nature that would cause subdural/subarachnoid hemorrhage, as well as retinal hemorrhages with diffuse axonal injury.

The authors use of critical terms like "shearing injury" is confusing to the reader. On page 10, reference is made to shearing injury, which "implies the distortion of brain

shape...creating a soft consistency." What does that mean? Several paragraphs later, the authors describe, in more detail, shearing injury which results from strains which "occur at the junctures between cortex and subcortical white matter, white matter and deep gray matter, and lateral extension across the midline of the brain..." (No photos – no references)?

In one of many examples of fuzzy thinking, they refer to motor vehicle accidents and state that after the age of 4 or 5 years, the most common cause of diffuse axonal injury (DAI) is the motor vehicle crash, (no citation) page 12. How is DAI produced? The author's state, on page 2, "rotational movement of the brain damages the nervous system by creating shearing forces, which causes DAI 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." Then on page 16, "in children with very severe accidental head injury, (eg. car accident), an occasional instance of retinal hemorrhage is found." Why only occasional if the MVA is the most common cause of DAI and DAI is very commonly associated with such hemorrhages?

The author's state that "small amounts of interhemispheric blood, which are able to be detected by CT scan, may not be appreciated at autopsy", page 14. Furthermore, at autopsy "the subdural hemorrhage may consist only of 2-3 ml of blood and may not be observed if the prosector does not personally inspect the subdural space as the calvarium is being removed. The writers sound like radiologists lecturing to medical students, rather than forensic pathologists addressing their peers. The reader is referred to figure 3. It is unclear in their poor quality photograph what is being portrayed. I was left with the impression that a good CT scan was better than a forensic autopsy.

The authors spend considerable time on the challenging topic of DAI. However, after being informed that these axonal disruptions may be visible by light microscopy after 18 to 24 hours. I was eagerly anticipating learning how often the members of the committee identified such evidence of diffuse brain injury, associated with subdural hemorrhage and retinal hemorrhages in their experience. No such luck! While they caution that "many of these children died too soon after their injury before these pathological changes have become established," there are certainly numerous examples of infants surviving beyond this time period, where opportunities to conduct studies for diffuse axonal injury could have been conducted. These unsupported and controversial comments are inappropriate in this kind of paper.

I found use of the term "brain swelling" unscientific. Why not use a more appropriate term as cerebral edema, or are we talking about some other condition other than cerebral edema?

One of the most credible statements in this paper was (page 15), the authors' description of retinal hemorrhages as "observed in 70 to 85% of young children with severe rotational brain injuries. Currently, their pathogenesis is not precisely understood." This characterization fits much of the content of this article.

If the authors' felt it necessary to include photographs to support their position, the photographs should add to the paper. The photographs here were consistently poor and of little value.

Summary

I recommend that this manuscript be returned to the committee with a request that they obtain a more clearly, defined set of instructions or expectations from the National Association

of Medical Examiners for what they are attempting to do as well as researching their topic more extensively. This would help ensure that members of their organization obtain a professional and scientifically credible recommendation(s) on which to carry out investigations into these very difficult cases of fatal childhood injuries.

Reference

1. Spitz & Fisher, Medicolegal Investigation of Death, 3rd edition, Charles C. Thomas Publishers, page 616,

M R C O

Minnesota Regional Coroner's Office

REGINA MEDICAL CENTER • HASTINGS, MN 55033 • (651) 480-4253 • FAX (651) 480-4257

COUNTIES OF:
Carver
Chisago
Dakota
Fillmore
Houston
Scott

December 7, 1998

John E. Pless, M.D.
President, National Association of Medical Examiners
635 Barn Hill Road
Indiana University Medical Center
Medical Science Building, Room 157
Indianapolis, IN 46204

Dear John:

I learned that the Board of Directors had accepted a "Position Paper On Shaken Baby" during a trial in Sun Valley, Idaho on November 9, 1998. I called Vince DiMaio and John Smialek and discussed the "Paper" with them, and John sent me a copy of his letter to you dated September 21, 1998. I agree completely with John's concerns and have a few additional comments.

Fundamentally, the paper is little more than a reiteration of standard prosecution testimony in any "shaken baby" case, reinforced at local or national "shaken baby" conferences, or in any number of written documents. The paper has none of the thoughtful introspection or fair analysis I would expect from a group of forensic pathologists, and there is no attempt to present or even acknowledge the existence of legitimate opinion or scientific data which could lead to a conclusion different from that of the authors.

I am particularly concerned about Case et al's conclusions regarding "rebleeding". The first sentence in the paragraph discussing "rebleeding" uses the word "erroneously" twice to characterize any opinion or testimony that rebleeding may be due to "trivial" trauma or no trauma at all. To support this statement, the authors reference Chadwick et al's letter "Shaken Baby Syndrome and the Death of Matthew Eappen", published on the Internet on November 11, 1997, and subsequently published in the Journal Pediatrics. (I was not aware that Pediatric published previously published material). Chadwick et al, and by inference Case et al, dismiss defense expert testimony in the Woodward trial Police as being either ill informed or nefarious. (The defense testimony was given by a Board Certified forensic neuropathologist who wrote the only available textbook on forensic neuropathology, and by a Board Certified forensic pathologist who was formerly Chief Medical Examiner for New York City and is currently the forensic pathologist for the New York State Police.) Further, Case et al cite three references to support their conclusion that "trivial" trauma does not cause a

rebleed. Even cursory examination of these references indicates that none even remotely supports the authors' statements.

I have personally reviewed more than thirty cases of children, living and dead, who have had an acute subdural hemorrhage superimposed upon an old grossly recognizable subdural hematoma and/or subdural hygroma. More than 80% of these cases had no evidence for an acute impact injury, or any other acute injury, and these re-bleed examples are the only rotational deceleration head injury cases I have seen or reviewed (more than 100) in which there was no impact. (Significantly, the autopsy reports and/or medical records in a number of the acute only cases fails to mention scalp/subgaleal trauma, and in some cases specifically states that there is no scalp trauma, but the impact injury is easily identifiable when reviewing autopsy photographs or CT/MRI studies [in children who are alive and did not die]). The caretaker who had custody of the child when he/she became symptomatic was charged with murder or felony assault in each of the re-bleed cases, even though there were no other acute injuries and/or historic data to support "abuse" as the etiology.

To belittle the defense testimony in the Woodward case as "erroneous" is condescending and does little to increase our understanding of head injury in infants and young children. These issues need to be carefully evaluated and discussed by our members before we make any official statement regarding head injury in children.

Best wishes,

John Plunkett, M.D.

JP:kg
12-7-98
cc: Vincent J.M. DiMaio, M.D.
John Smialek, M.D.
Mary Case, M.D.
Michael Graham, M.D.
Tracy Corey-Handy, M.D.
Jeffrey Jentzen, M.D.
James Monteleone, M.D.
Jan Leetsma, M.D.
Michael Baden, M.D.

From: dimaio@co.bexar.tx.us
Sent: Tuesday, February 15, 2005 3:28 PM
To: plunkettj@frontiernet.net
Subject: RE: Position paper on SBS in AJFMP

The problem with the Position Paper is that it is not a position paper but just an article in which the authors state their opinion. That was why it was peer rejected, something that everyone seems to ignore. The Box in the lower left corner of the first page says it was rejected by peer reviewers. What many do not realize that the outcry was such that no position paper like it will appear again. Look at the position papers since then

-----Original Message-----
From: John Plunkett [mailto:plunkettj@frontiernet.net]
Sent: Tuesday, February 15, 2005 2:03 PM
To: Edwilley@aol.com; JLeestma@aol.com
Subject: RE: Position paper on SBS in AJFMP

Agree. See also Ref #43 and 44 to "Wilbur LS". It should be "Smith WL". Doesn't ANYONE check these references? Feel free to forward to DiMaio. John

John Plunkett
13013 Welch Trail
Welch, MN 55089
507-263-4022 (H)
651-343-4324 (C)

From: Edwilley@aol.com [mailto:Edwilley@aol.com]
Sent: Tuesday, February 15, 2005 1:53 PM
To: JLeestma@aol.com
Cc: plunkettj@frontiernet.net
Subject: Position paper on SBS in AJFMP

Dear Jan:

Yes, I know that Mary's work will never be published. Are you aware references in the position paper do not support her statements on timing of injuries (p118)? How can she misrepresent these articles? Adams et al in Histopathology 1989, 15 p49-59 at p57 indicates 14% of DAI have "lucid intervals". Also, Adams et al in Annals of Neurology 1982, 12 p557-563 at table 4, p560 indicates 44% of cases with non-DAI have lucid intervals. Of those 25 were total and 33 were partial lucid intervals. Ommaya and Gennarelli in Brain 1974, 97 p633-654 described recovery after concussion (p648) and mentioned at (p649) "secondary factors...adds further damage to the primary insult." The only other paper cited, Gilles and Nelson in Pediatric Neurology 1998, 19 p119-128 reports a total of 14 cases, too few to be likely to detect variations in presentation.

It all reminds me of political misrepresentations where reporters cast the matter as a difference of opinion rather than a falsehood. People are simply too lazy or too credulous to check any facts.

In my opinion these misrepresentations warrant judicial notice. Please let me know if you agree with my interpretations. If you wish I can forward the 4 articles referenced, in the Position Paper: #24, 31, 36 & 49.

Misrepresentation of the same subject in the Monteleone textbook never had to pass peer review. There were wrong references, in both editions. At least these from AJFMP are correctly cited and easy to recover.

I wonder what Dr. DiMaio may think of this? Perhaps something so important as a "position paper" should have the data and references checked by someone, preferably someone with no investment in the paper or the subject?

Best regards,

Ed Willey

POSITION PAPER ON FATAL ABUSIVE HEAD INJURIES IN INFANTS AND YOUNG CHILDREN

Revised 2003 – Expires 2008

Mary E. Case, M.D., Michael A. Graham, M.D.,
Tracey Corey Handy, M.D., Jeffrey M. Jentzen, M.D.,
and James A. Monteleone, M.D.
National Association of Medical Examiners Ad Hoc Committee on Shaken Baby Syndrome

From the Department of Pathology, Division of Forensic Pathology, (MEC and MAG) and Department of Pediatrics (JAM), St. Louis University Health Sciences Center, St. Louis, MO; Office of the Chief Medical Examiner of the State of Kentucky (TCH); and Office of the Medical Examiner of Milwaukee County, Milwaukee, WI (JMJ).

ABSTRACT

This paper represents the work of the National Association of Medical Examiners Ad Hoc Committee on Shaken Baby Syndrome to provide a consensus opinion within the forensic pathology profession on the subject of abusive head injuries in infants and young children. Abusive head injuries include injuries caused by shaking as well as impact to the head either by directly striking

the head or causing the head to strike another object or surface. Due to anatomical and developmental differences in the brain and skull of the young child, the mechanisms and types of injuries which affect the head differ from those which 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 pathological 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

INTRODUCTION

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 paper will address the topic of abusive head injury in young children. In several areas of this paper the term “marker” is used when describing the importance of identifying the presence of subdural, subarachnoid, and retinal hemorrhages. The use of the term “marker” indicates a grossly observable sign to signify the possible existence of diffuse axonal injury which is not grossly evident. Use of the term “marker” does not imply that such hemorrhages can not exist without such an association but 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 paper will describe the state of knowledge concerning the pathogenesis, clinical features, and pathology 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 1970's 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 retinal hemorrhage without injuries which would indicate impact such as facial bruises or 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 which causes the head to forcefully whiplash forward and backward with repeated accelerations and decelerations in each direction.

The circumstances in which abusive head injury commonly occurs is frequently in response to prolonged crying and often is inflicted by a care giver 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 due to 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.7,8,9,10 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 from fatal child abuse.11,12

Infants may sustain abusive head injury of less than fatal outcome and may sustain injuries to the brain which will later be reflected in degrees of mental retardation or slowness, learning disorders, seizures, blindness, or irritability. Of infants that are abusively head injured, approximately 7-30% die, 30-50% have significant cognitive or neurological deficits and 30% have a chance of full recovery.13,14,15

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 old, with most being under 12 months old.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 which exist in the skull, brain and neck prior to about the age of 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 which 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 dentritic 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 age 4 or 5 years are particularly vulnerable to a type of brain injury which is best described as shearing injury. Shearing injury implies a distortion of the brain shape which 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 which 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, large number of neurons without glial or dentritic connections, and the small axonal size predisposes 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 upon by acceleration-deceleration forces. Lastly, 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 which are operative in head injury are primarily translational and rotational (angular). Translational forces produce linear movement of the brain, a type of movement which 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, neurological deficit, or death. 23,24,25,26

Rotational forces are generated by either impact or non-impact 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 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.23,24,25,26,27,28,29 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 act to disrupt the most external junctures while 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 which was 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 both on confessions by perpetrators or witnesses of how these injuries were inflicted as well as from 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 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 due to either pure impact or pure shaking because the brain pathology is 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 co-existent shaking cannot be excluded. In the absence of signs of an impact however, shaking alone should not be presumed as there may well have been 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 pathological findings which should be appreciated as markers for the underlying problem in the brain which is the diffuse axonal injury.32

SUBDURAL HEMORRHAGE

The grossly and microscopically identifiable pathology of 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.(Figure 1) These rather transparent veins tear when they are stretched as the brain moves within the subdural space of the cranial cavity.23,33,34,35 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 it is bilateral.(Figure 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% - 98% of cases.7,36 Small amounts of interhemispheric blood which are able to be detected by CT scan may not be appreciated at autopsy.5,37 Very thin layers of subdural blood over the cerebral convexities may not be visible on CT scan yet can be found at autopsy. 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 which report data on nonfatal cases of abusive head injury find that about 80 to 85% of cases have subdural hemorrhage.38 At autopsy, the subdural hemorrhage may consist of only 2-3 ml of blood

and may not be observed if the prosector does not personally inspect the subdural space as the calvarium is being removed.(Figure 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 will be large enough to have 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 post-injury survival. At autopsy, large subdural hemorrhages resulting in part from post-injury accumulation have been observed. The presence of even a small amount of subdural hemorrhage indicates that brain displacement has been produced which may have caused some shearing brain injury.

SUBARACHNOID HEMORRHAGE

Subarachnoid hemorrhage occurs in patches over the cerebrum, especially over the parasagittal cerebral convexities. (Figure 4) It is present in virtually all fatal cases although it may be very small and difficult to identify especially on the interhemispheric surfaces.(Figure 5) Subarachnoid hemorrhage arises from tearing of arachnoid vessels at the same time as 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 70-85% 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 over-represented 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 upon 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 which frequently are 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.

Ophthalmological 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 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,(eg, car accident), an occasional instance of retinal hemorrhage is found.26 The retinal hemorrhages associated with non-accidental head injuries tend to be bilateral although they may be unilateral, multiple, extensive and extend far into the periphery of the retina.39 Nontraumatic causes of retinal hemorrhages include bleeding disorders, sepsis, menigitis, vasculopathies, increased intracranial pressure, and very rarely, if ever, cardiopulmonary resuscitation.43,44,45,46,47 Retinal hemorrhages which 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 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 which 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 18-24 hours as retraction bulbs or varicosities. Retraction bulbs are accumulations of axoplasm which appear on H & E 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 after survival as short as 2 hours.51,52

The blood vessel tears of diffuse brain injury maybe 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 found in young children with diffuse brain injury because 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 old, parenchymal tears are also grossly observable. This is the lesion Lindenberg described as the contusion tear.53 These are slit-like tears which occur at the cortex-white matter junction or within the layers of the cortex and are due to the differential movements within the brain as some portions of the brain shear or slide apart as the brain tissues differentially rotate.(Figure 6 A and B and Figure 7) Contusion tears are rare lesions and in those cases in which they are 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 pathological 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 their injury before these pathological changes have become 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 scans 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 as well as 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 these children 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 neurological outcome or death. Correlations of clinical and experimental observations on 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 and not that the unconsciousness is the result of increasing intracranial pressure or hypoxia.24 Symptoms demonstrated 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 which occurs in these children may be related to damage to lower brainstem (medullary) centers of respiratory control. The timing with which respiratory difficulty develops is not very precise but 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 neurological 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 as there is no currently available method to demonstrate the underlying pathology 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 scan or MRI.38

CHRONIC SUBDURAL HEMATOMA

Rebleeding following trivial injury or spontaneous rebleeding from a pre-existing 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 scanning. 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 which 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.58,59,60 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. Factors which promote such 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 alcoholics, those with hydrocephalus who have been treated by placement of a ventricular shunt, or those with traumatic encephalomalacia.59 Children with glutaric aciduria type 1 develop frontotemporal atrophy and occasionally develop subdural hemorrhage without trauma 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 most determined 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 fibrinolytic systems in the subdural fluid.62,63,64 A young child whose subdural hemorrhage subsequently organizes into a membrane comprised of large vascular channels at risk for rebleeding would have been symptomatic preceding the time of rebleeding since there would have been preexisting brain abnormality. Signs and symptoms which would be expected preceding 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 upon the demonstration of a chronic subdural membrane which 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 hematopoetic 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 are 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 the head injuries demonstrated by children who sustain truly accidental injuries and those who sustain abusive head injuries. 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 to the 90% - 98% incidence of these hemorrhages associated with abusive head injuries.(Case M , 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.26,32,65,66,67,68,69,70,71 In low level falls of less than about 8 feet and witnessed by uninvolved and nonbiased individuals, about 1 to 2% of children will sustain a narrow simple linear skull fracture. A small proportion of the children who sustain these skull fractures will develop an epidural hemorrhage. 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 consciousess due to tentorial herniation.

It is essential that a meticulous autopsy examination be performed in all cases of possible injury to children. In cases in which subdural and/or subarachnoid hemorrhage is found at autopsy, the brain must be thoroughly 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 AVMs is unlikely to resemble that of head injury but these malformations need to be excluded by careful examination of the brain.72,73

CONCLUSION

Anatomical and developmental differences of the brain and skull of children under age 4 or 5 years old make the head injuries and mechanisms of injury which affect these children different in certain respects from those which occur following 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 pathological 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 following their injury.

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FIGURES

1. 13 week old infant with normal brain and intracranial spaces demonstrating bridging vein (arrow) arising from left cerebral convexity(right of photograph.)

2. 9 month old infant with fracture of right parietal calvarium showing bilateral acute subdural hemorrhages over the cerebral convexities.

3. 11 week old infant with small amounts of acute subdural hemorrhage over both cerebral convexities.

4. 17 week old infant with large (70-80 milliliters) acute subdural hemorrhage over right cerebral convexity demonstrating patches of subarachnoid hemorrhage over both parasagittal regions greater on the right than on the left.

5. 5 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.

6. 17 week old infant (same infant as Figure 4) A. demonstrating 6 millimeter linear tear in right side of corpus callosum (arrow); B. photomicrograph demonstrating linear tear in corpus callosum with split in tissue and fresh hemorrhage.

7. 7 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 3-4 millimeter contusion tear (arow) in left orbital subcortical white matter.

From: Gregory G. Davis [gdavis@path.uab.edu]
Sent: Tuesday, October 17, 2006 9:43 AM
To: 'plunkettj@frontiernet.net'; 'rkwrightmd@gmail.com'
Subject: NAME position paper renewal process

Dear John and Ron,

Having returned from the NAME meeting, I am now able to answer your questions, mostly.

Beyond the details that Randy has already related to you, I do not know how the "Shaken Baby" position paper was renewed. No mechanism for renewal of position papers existed at that time. The Board of Directors of NAME has addressed the matter by taking the following two steps.

1. A new addition to the guidelines for position papers was adopted. The ten points listed on the NAME website remain in effect, and to those ten points has been added an eleventh point, which is quoted below.

"11. NAME's endorsement of a position paper ends five years from publication of the position paper. NAME may renew its endorsement of a position paper by following steps 2-10 above, with revision of the original manuscript as appropriate for changes in knowledge or understanding that occur during the five year life of the original position paper."

2. The Board rescinded the renewal of the Shaken Baby position paper, because that paper did not go through the approved renewal process. The Shaken Baby paper may be revised and resubmitted, whereupon it will go through steps 2-10 listed below.

_____

1. Position papers state positions officially endorsed by the National Association of Medical Examiners (NAME) as authorized by the Board of Directors and are intended to assist in the practice of medicolegal death investigation. Position papers discuss subjects in the field of forensic pathology of vital interest to the public and to the membership at large. A position paper will discuss not only the majority opinion about its subject but will also address accepted (mainstream) minority opinions.

2. A position paper is initiated by the President or by direction of the Board of Directors.

3. The President, with the Executive Committee's approval, appoints the lead author.

4. The lead author, with the Executive Committee's approval, selects experts and co-authors to assist in preparing the first draft of the paper.

5. The first draft is submitted to the Board of Directors for review and comment. The Board of Directors votes (simple majority) to accept or reject the paper in concept.

6. The draft is re-edited and posted on the NAME website for 30 days during which time the general membership has the opportunity to review and comment.

7. The authors re-edit the paper based on members' comments and then submit it to the Executive Committee for approval. The Executive Committee votes (simple majority) to accept, accept with additional revisions, or reject the paper.

8. Following Executive Committee approval, the position paper is presented to the Board of Directors no less than 1 month prior to the next Board of Directors' meeting for final approval. Final approval requires a super-majority (3/4) for acceptance.

9. Accepted position papers will be published as approved (with the exception of stylistic changes) in the American Journal of Forensic Medicine and Pathology as a position endorsed by the National Association of Medical Examiners for five years.

10. A paper rejected by the Executive Committee or Board of Directors may be submitted to any medical journal as the product of the authors, but it may not be identified as an endorsed opinion of NAME.

I thank you for bringing this matter to my attention. I thank you also for your patience as I got my bearings.

Sincerely,

Gregory G. Davis
Chairman, NAME Position Paper Committee