ELSEVIER

Legal Medicine 9 (2007) 83–87

LEGAL MEDICINE

www.elsevier.com/locate/legalmed

Abusive head injuries in infants and young children

Mary E. Case

St. Louis University Medical Center, Forensic Pathology, 3556 Caroline, St. Louis, MO 63104, USA

Abstract

Abusive head injuries are among the most common causes of serious and lethal injuries in children. These injuries may result from impact or shaking or a combination of these mechanisms. These mechanisms cause the child's head to undergo acceleration–deceleration movements which may create inertial movement of the brain within the cranial compartment. Differential movement between the brain and skull may result in subdural and subarachnoid hemorrhages and traumatic diffuse axonal injury. This paper will discuss the unique anatomical and developmental features of the immature brain, skull, and neck which render young children particularly vulnerable to shearing injuries, the pathology of those injuries, and the mechanisms of these injuries.

Keywords: Abusive head injury; Subdural hemorrhage; Retinal hemorrhages

1. Introduction

Abusive head injury describes the traumatic brain injury inflicted on an infant or young child. In the recent past such descriptions as "shaken baby syndrome" and "shaken-impact" have been used to describe the assumed mechanisms of injury. Abusive head injury will be used in this chapter to encompass all mechanisms of inflicted traumatic brain injury. Investigation into whether an injury is intentional or unintentional is complicated by the false histories or lack of any history that may be provided by a caregiver to explain a child's injury. The pathological findings may assist in determination of how the injury was caused.

Physical abuse is the leading cause of death in children [26]. Ten percent of all injuries of children under age 2 years are abusive injuries. Forty to 50% of all abusive injuries are head injuries and as many as 80% of fatal abusive injuries are head injuries. Of children under 2 years old, 80% of head injury deaths are abusive [6]. Abusive head injuries are seen in children up to age 4 or 5 years although most occur in infants under 1 year old. Of children with inflicted head injury, up to 30% die, 30–50% have severe neurological deficits, and 30% have a chance of full recovery [5,9,24].

2. Unique features of the immature brain and skull

Head injuries in young children are unique because the trauma affects an organ that is in the process of maturing and developing. The mechanisms, thresholds, and types of injuries which affect the young child's head differ from those occurring later in childhood and adult ages.

The young brain is enclosed in a thin and pliable skull. The bone is not ossified, the sutures are unfused, and the fontanelles are open. The brain attains 75% of its full weight by age 2 years and almost its entire adult weight by age 5–6 years making the head proportionately much larger and heavier than later in life. The weight of the newborn child's head is about 10–15% of its body weight compared to 2–3% for the adult head [14,4]. The large brain of the young child has a relatively large subarachnoid space which is also very shallow in depth with the brain closely positioned next to the skull [44]. The young child's neck musculature is immature and unable to fully support the weight of the head.

At birth, myelination is just beginning in the cerebral hemispheres although it is fairly well developed in the

E-mail address: MCase@stlouisco.com

1344-6223/$ - see front matter
doi:10.1016/j.legalmed.2006.11.017

spinal cord and brain stem. Myelination in the cerebral hemispheres progresses over the first 2 years of life and the early myelin sheath is thin and immature. Glial cells, synaptic connections, and dendritic connections are also minimal at birth and develop progressively over the first years of life. At birth, the water content of the brain is much greater than it is at any later time resulting in a soft, easily deformed brain.

3. Mechanisms of injuries

Biomechanical studies of traumatic brain injury have demonstrated that forces that result in translational movement of the brain in a straight line in relation to its center of gravity have minimal effect on the brain except for those resulting from focal contact. Contact forces may result in scalp bruises, skull fractures, epidural hemorrhage, and focal subdural hemorrhage. In rare incidences of trivial home falls from short distances, contact forces may produce a fatal head injury in a young child by producing a space-occupying lesion from an epidural hemorrhage or subdural hemorrhage. In these rare cases, the child is not immediately concussed but gradually becomes symptomatic of increasing intracranial pressure. Surgical or medical intervention may prevent a fatal outcome by removal of the hematoma but some cases do have a fatal outcome [23,35].

In contrast to translational events, much more commonly serious or fatal head injury in young children is related to events that cause rotational movement of the brain about its center of gravity. Rotation of the brain's center of gravity is caused by acceleration–deceleration movement of the head which may be initiated by either impact or nonimpact mechanisms. The majority of cases of abusive head trauma are related to direct impact to the head causing forceful angular acceleration–deceleration of the head. Shaking is a nonimpact inertial mechanism which may also produce angular acceleration–deceleration of the head. Acceleration–deceleration movement of the head is damaging when it is forceful enough to cause rotational movement of the brain within the cranial cavity causing the brain to turn abruptly on its axis. Rotational movement of the brain within the confines of the skull creates shearing of the vascular adnexae on the surface of the brain as well as the axonal processes within the brain [11,22,33,34,39,43,22].

The unique features of the young child's brain and skull make young children particularly vulnerable to diffuse or shearing injuries of the brain. Impact to the immature brain is more likely to produce shearing injury rather than brain contusions. The lack of myelination and the small axonal size predisposes the young brain to shearing injury. The soft consistency of the brain and the pliability of the bone facilitate brain deformation on head impact. When deformation exceeds the elastic threshold of the brain parenchyma, shearing injury occurs. The large heavy head supported on the weak neck allows greater movements of the head and brain in response to acceleration–deceleration forces [13,14,28].

In 1972, Caffey described whiplash shaking as a mechanism of injury in young children causing a syndrome consisting of subdural and subarachnoid hemorrhages, retinal hemorrhages, and metaphyseal fractures [7,8]. Within the forensic pathology community in the United States, there is disagreement on the theory of whether shaking generates enough force to seriously or fatally injure a young child through rotational acceleration–deceleration. In 1987, Duhaime et al. published a study in which they measured the angular acceleration of the head of a doll model resulting from either shaking or impact. Shaking alone did not reach the thresholds for concussion, subdural hemorrhage, or diffuse axonal injury [13]. The thresholds for concussion, subdural hemorrhage, and diffuse axonal injury, however, had been derived from studies on adult primates which do not necessarily truly resemble the immature human infant skull and brain [16]. This study demonstrated that impacts even against soft-padded surfaces markedly increase the angular acceleration and exceed the thresholds for concussion, subdural hemorrhage, and diffuse axonal injury. On impact, it is the sudden angular deceleration experienced by the brain and vascular adnexae, not the specific contact forces applied to the surface of the head, that resulted in the intracranial injury [12].

A current model for the study of inertial brain injuries utilizes a miniature pig to model the effects of rotational acceleration on a human infant. The brain of a young pig closely resembles a 3- to 4-month-old human infant brain. These studies have demonstrated that the young pig's brain is more vulnerable to rotational acceleration than the adult whereas the data from the primate model had suggested the infant would be less vulnerable [36,37]. These newer data thus indicate that the thresholds for concussion, subdural hemorrhage, and DAI used in the 1987 Duhaime study were not appropriate for human infants.

Absence of a bruise on the scalp or subgaleal hemorrhage does not necessarily indicate there has not been an impact. Many impacts are not reflected on the scalp because a young child's scalp is very elastic and may stretch on impact. It is not necessary to distinguish abusive head injuries as to either impact or shaking injuries because the brain pathology is identical in both [13]. If there are focal injuries such as skull fractures, scalp bruises, or subgaleal hemorrhage, certainly impact can be assumed. In the absence of such marks of impact, however, shaking alone should not be presumed as there may well have been impact that cannot be discerned [18].

4. Pathology of abusive head injuries

The pathology of abusive head injuries consists of subdural hemorrhage, subarachnoid hemorrhage, retinal hemorrhages, and diffuse axonal injury. Subdural hemorrhage results from tearing of the bridging veins which pass from the cortical surface to the dural venous sinuses. Bridging

veins tear as they are overstretched as the brain moves within the cranial cavity [15,40,43]. Subdural hemorrhage is evident in up to 90% of cases of abusive head injury in children at autopsy [13,19]. The subdural hemorrhage is most commonly a thin film of blood over the cerebral convexities. It may be unilateral or bilateral although there is a tendency to be bilateral (see Fig. 1). The subdural blood tends to extend into the posterior interhemispheric fissure and is usually located on both sides of the falx [45]. On CT scan, small amounts of interhemispheric subdural blood can be detected which may be missed at autopsy because of its location but CT is less sensitive than autopsy in detecting small convexity subdural hemorrhages [27]. MRI may detect subdural hemorrhage along the falx or tentorium which may not visible on CT. The amount of subdural hemorrhage in some cases is as much as 100 ml or more but most frequently is much less. Subdural blood is a marker of shearing injury. Because the dura is firmly attached to the skull and the arachnoid is firmly attached to the cerebral cortex, most brain motion occurs across the potential subdural space and the bridging veins are vulnerable to tearing with sudden head motion. Subdural hemorrhage is not necessarily significant as a space-occupying mass lesion producing increased intracranial pressure. The presence of even a small amount of subdural blood indicates that brain rotation has been produced and such rotation may have caused some amount of diffuse axonal injury.

Subarachnoid hemorrhage usually accompanies subdural hemorrhage and occurs in patches especially over the parasagittal cerebral convexities although it may be very sparse and difficult to observe because the subarachnoid blood may be present on the mesial surfaces between the cerebral hemispheres (see Fig. 2). Bridging veins are enwrapped by an arachnoid sheath so that a tear of the vein also causes subarachnoid hemorrhage.

Retinal hemorrhages are found in 70–85% of abusive head injuries. The mechanism which causes these hemorrhages is not precisely understood but their presence correlates highly with acceleration–deceleration head injury and they are greatly overrepresented in cases of abusive head trauma in young children. Mechanisms which may 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 the retinal hemorrhages which frequently occur in full-term neonates after vaginal delivery [4,31]. Ophthalmological findings in children with abusive head injury include numerous hemorrhages throughout all layers of the retina which extend far into the periphery of the retina [29,30] (see Fig. 3). There may be additional internal eye injuries in these same children consisting of vitreous bleeding and retinal folds. In children with very severe accidental traumatic head injuries, an occasional instance of retinal hemorrhage is found. Nontraumatic causes of retinal hemorrhages include bleeding disorders, sepsis, meningitis, vasculopathies, increased intracranial pressure, and very rarely if ever cardiopulmonary resuscitation [17,21,25,41,42]. Nontraumatic causes of retinal hemorrhage do not result in the multiplicity and peripheral distribution of the hemorrhages associated with abusive head injury. Retinal hemorrhages are not seen on CT or MRI unless they are very severe [14]. Retinal

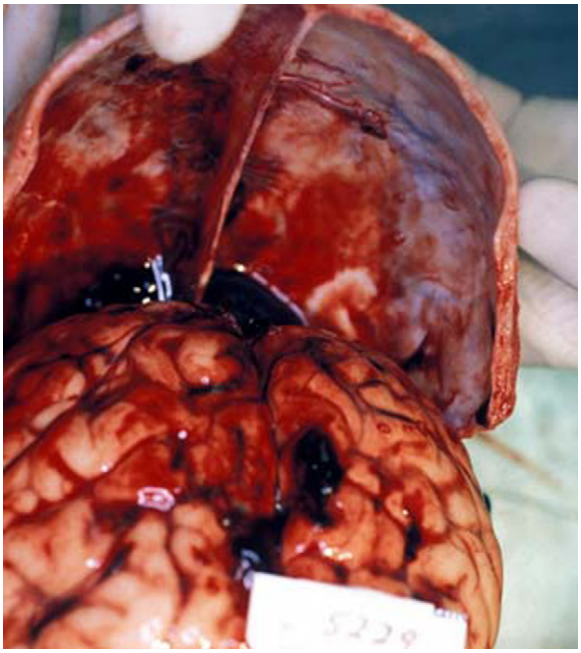

Fig. 1. Thin film of subdural blood over cerebral convexities.

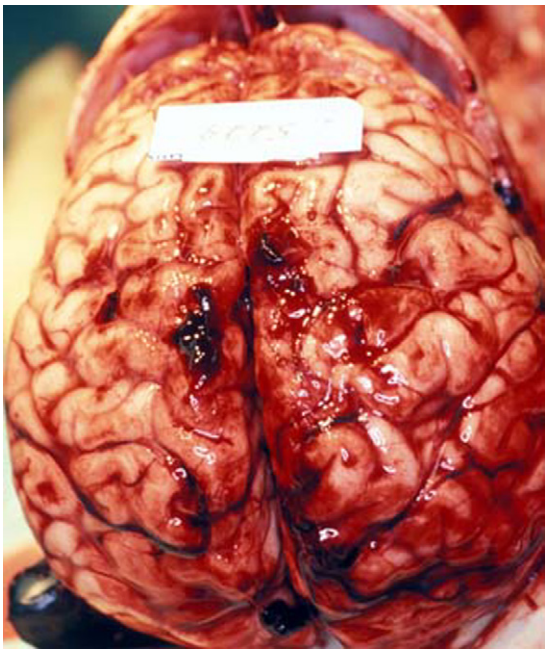

Fig. 2. Patches of subarachnoid hemorrhage over cerebral convexities.

Fig. 3. Multiple retinal hemorrhages throughout retina.

hemorrhages caused by increased intracranial pressure consist of small numbers of hemorrhages at the posterior pole of the retina around the optic nerve head.

Diffuse axonal injury consists of tears of axonal processes and small blood vessels and rarely actual tissue tears in certain areas of the brain [2,3]. The areas of predilection for axonal injury are the corpus callosum, subcortical white matter, periventricular areas, and the dorsolateral quadrants of the rostral brainstem. Axonal tears are very difficult to see in young children because of the small size of the axonal processes. Immunohistochemical staining for β-amyloid precursor protein has enabled demonstration of axonal damage in some cases of abusive head injury as early as 2 h after injury [20,38].

The blood vessel tears of diffuse axonal injury usually appear as streak or punctate hemorrhages which vary from less than 1 mm up to many centimeters in patients who survive for several days. Blood vessels in young children are very elastic and do not tear readily even when adjacent axonal processes are torn so that streak and punctate hemorrhages are very seldom found as gross evidence of diffuse axonal injury in young children.

Tissue tears are rare lesions found on gross examination of the brain, usually in children under the age of 5 months. Lindenberg described these lesions as contusion tears [32]. They appear as small tears which occur at the cortex–white matter junction or within the layers of the cortex and are due to the differential movement of the brain as some portions of the brain shear or slip apart as the brain rotates. Contusion tears are difficult to see and in those cases in which they are found, they are accompanied by the usual findings of subarachnoid and subdural hemorrhage and do not constitute an isolated lesion by which shearing injury can be identified without the other features.

It is not always possible to 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 all these findings are demonstrated in some cases [40]. Appreciation of subdural/subarachnoid and retinal hemorrhages as "markers" of possible rotational acceleration–deceleration forces is helpful in trying to discern the mechanism of injury in abusive injuries [10].

Abusive head injuries are frequently accompanied by brain swelling of various degrees. Initial CT scans may demonstrate 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 [27].

Diffuse brain injuries become symptomatic immediately if there is a moderate to severe degree of diffuse axonal injury as would be expected in cases with significant neurological outcome or death [1,3,34]. The symptoms demonstrated by these severely injured children consist of an immediate decrease in the level of consciousness, either lethargy or unconsciousness followed by respiratory irregularity, difficulty, or apnea, and frequently seizures. A child with such a decrease in the level of consciousness would appear limp, unresponsive, would not eat, and would be recognizably abnormal even to a medically untrained person.

References

[1] Adams JH, Graham DI, Murray LS, Scott G. Diffuse axonal injury due to nonmissile head injury in humans: an analysis of 45 cases. Ann Neurol 1982;12:557–63.
[2] Adams JH, Graham DI, Scott G, Parker LS, Doyle D. Brain damage in fatal nonmissile head injury. J Clin Pathol 1980;33:1131–45.
[3] Adams JH, Doyle D, Ford I, Gennaralli TA, Graham DI, McLellam DL. Diffuse axonal injury in head injury: definition, diagnosis, and grading. Histopathology 1989;15:49–59.
[4] Berger R, Margolis S. Retinal hemorrhages in the newborn. Ann Ophthalmol 1985;8:53–6.
[5] Bonnier C, Nassogne M, Evrard P. Outcome and prognosis of whiplash shaken syndrome; late consequences after a symptom-free interval. Dev Med Child Neurol 1995;37:9–13.
[6] Bruce D, Zimmerman R. Shaken impact syndrome. Pediatr Ann 1989;340:482–94.
[7] Caffey J. On the theory and practice of shaking infants. Am J Dis Child 1972;124:161–9.
[8] Caffey J. The whiplash shaken infant syndrome: manual shaking by the extremities with whiplash induced intracranial and intraocular bleeding, linked with permanent brain damage and mental retardation. Pediatrics 1974;54:396–403.
[9] Carty H, Ratcliff J. The shaken infant syndrome. BMJ 1995;310:344–5.
[10] Case M. Head injury in a child. ASCP Check Sample 1997;39:79–93.
[11] Duhaime AC, Alario AJ, Lewander WJ, Schut L, Sutton LN, Seidl TS, et al. Head injury in very young children: mechanisms, injury types, and ophthalmological findings in 100 hospitalized patients younger than 2 years of age. Pediatrics 1992;90:179–85.
[12] Duahime AC, Christian CW, Rorke LB, Zimmerman RA. Nonaccidental head injury in infants – the "shaken-baby syndrome". N Engl J Med 1998;338:1822–9.

[13] Duhaime AC, Gennarelli TA, Thibault L, Bruce D, Mrgulies S, Weser B. The shaken baby syndrome. J Neurosurg 1987;66:409–15.
[14] Gean AD. Pediatric trauma. In: Imaging of head trauma. New York: Raven Press; 1994.
[15] Gennarelli TA, Spielman GH, Langfitt TW, Gildenberg PL, Harrington T, Jane JJ, et al. Influence of the type of intracranial lesion on outcome from severe head injury: a multicenter study using a new classification system. J Neurosurg 1982;56:26–32.
[16] Gennarelli TA, Thibault LE, Adams JH, Graham DI, Thompson CJ, Marcincin RP. Diffuse axonal injury and traumatic coma in the primate. Ann Neural 1982;12:564–74.
[17] Gilliland MGF, Luckenback MW. Are retinal hemorrhages found after resuscitation attempts? Am J Forensic Med Pathol 1993;14:187–92.
[18] Gilliland MGF, Folber R. Shaken babies – some have no impact. J Forensic Sci 1996;41:114–6.
[19] Gilles EE, Nelson MD. Cerebral complications of nonaccidental head injury in childhood. Pediatr Neurol 1998;19:119–28.
[20] Glickman A, Bell B, Evans R, Smith T. Immunohistochemistry: a powerful tool for demonstrating diffuse axonal injury in shaken baby syndrome. Presented at the NAME meeting, Albuquerque, NM; 1998, 11/3/98.
[21] Goetting MG, Sow B. Retinal hemorrhage after cardiopulmonary resuscitation in children: an etiological reevaluation. Pediatrics 1990;85:585–8.
[22] Hanigan WC, Peterson RA, Njus G. Tin ear syndrome: rotational acceleration in pediatric head injuries. Pediatrics 1987;80: 618–22.
[23] Hymel KP, Jenny C, Block RW. Intracranial hemorrhage and rebleeding in suspected victims of abusive head trauma: addressing the forensic issues. Child Maltreat 2002;7:329–48.
[24] Jayawant S, Rawlinson A, Gibbon F, Price J, Schulte J, Sharples P, et al. Subdural hemorrhages in infants: population based study. BMJ 1998;317:1558–61.
[25] Johnson DL, Braun D, Friendly D. Accidental head trauma and retinal hemorrhage. Neurosurgery 1993;33:231–4.
[26] Keenan HT, Runyan DK, Marshall SW, Nocera MA, Merten DF, Sinal SH. A population-based study of inflicted traumatic brain injury in young children. JAMA 2003;290:621626.
[27] Kleinman P. Diagnostic imaging in infant abuse. Am J Radiol 1990;155:703–12.
[28] Kriel RL, Krach LE, Sheehan M. Pediatric closed head injury: outcome following prolonged unconsciousness. Arch Phys Med Rehabil 1988;69:678–81.
[29] Levin A. Ocular manifestations of child abuse. Ophthalmol Clin North Am 1990;3:249–64.
[30] Levin A. Ocular complications of head trauma in children. Pediatr Emerg Care 1991;7:129–30.
[31] Levin S, Janive J, Mintz M, Kreisler C, Romem M, Klutznik A, et al. Diagnostic and prognostic value of retinal hemorrhages in the neonate. Obstet Gynecol 1980;55:309–14.
[32] Lindenberg R, Freytag E. Morphology of brain lesions from blunt trauma in early infancy. Arch Pathol 1969;87:298–305.
[33] Margulies SS, Thibault LE. An analytical model of traumatic diffuse brain injury. J Biochem Eng 1989;111:241–9.
[34] Ommaya AK, Generelli TA. Cerebral concussion and traumatic unconsciousness: correlation of experimental and clinical observations on blunt head injuries. Brain 1974;97:633–54.
[35] Plunkett J. Fatal pediatric head injuries caused by short-distance falls. Am J Forensic Med Pathol 2001;22:1–12.
[36] Raghupathi R, Margulies SS. Traumatic axonal injury after closed head injury in the neonatal pig. J Neurotrauma 2002;19:843–53.
[37] Raghupathi R, Mehr MF, Helfer MA, Marmulies SS. Traumatic axonal injury is exacerbated following repetitive closed head injury in the neonatal pig. J Neurotrauma 2004;21:307–16.
[38] Sheriff FE, Bridges LR. Early detection of axonal injury after human head trauma using immunocytochemistry for beta-amyloid precursor protein. Acta Neuropathol 1994;87:55–62.
[39] Stahlhammer D. Experimental models of head injury. Acta Neurochir Suppl (Wien) 1986;36:33–46.
[40] Vowles I, Scholtz CL, Cameron JM. Diffuse axonal injury in early infancy. Clin Pathol 1987;108:185–9.
[41] Wilbur LS. Abusive head injury. APSAC Advisor 1994;7:16–9.
[42] Wilbur LS. Magnetic resonance imaging evaluation of neonates with retinal hemorrhages. Pediatrics 1992;89:332–3.
[43] Wilkins RH, Rengachary SS, editors. Neurosurgery. New York: McGraw-Hill; 1985. p. 1531–6.
[44] Williams P, editor. Gray's anatomy. New York: Churchill Livingstone; 1995. p. 607–9.
[45] Zimmerman RA, Bilaniuk LT, Gennarelli TA. Computed tomography of cerebral injury in the abused child. Radiology 1979;130:687–90.