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SPECIAL ISSUE: ABUSIVE HEAD TRAUMA

Distinguishing accidental from inflicted head trauma at autopsy

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Abstract This article will discuss accidental and inflicted head injuries in infants and young children and how forensic pathologists distinguish between these types of injuries. The article begins with a consideration of the special and unique features of the anatomy and development of the child's head and neck and then relates these features to the mechanisms of traumatic brain injury and how these unique features influence the mechanisms of injury. The article very specifically notes that accidental head injuries in young children that occur in and around the home are focal head injuries in distinction to inflicted head injuries, which are diffuse brain injuries. The article discusses the mechanisms by which traumatic brain injury causes loss of consciousness and relates those mechanisms to the differences in the clinical features that occur in both accidental and inflicted head injury. The article discusses and illustrates the pathological findings in accidental head injuries consisting of the crushing head injuries and the head injuries sustained in short falls including epidural hemorrhage and focal subdural hemorrhage. The article discusses and illustrates the pathological findings that occur in inflicted head trauma, including subdural and subarachnoid hemorrhages and retinal and optic nerve sheath hemorrhages.

Keywords Non-accidental injury · Head injury · Postmortem · Mechanism of injury · Child

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Introduction

This article will discuss how forensic pathologists distinguish accidental from inflicted head trauma at autopsy. It is imperative that the autopsy findings are considered in conjunction with the medical history, beginning with the birth of the child and coming forward to the time of the injury, the family's medical and social services history, the circumstances of the injury event, and all of the investigative information derived from reports from law enforcement and other agencies as well as information from genetic screening and toxicology evaluations. This article will consider the mechanisms of traumatic head injury and how these mechanisms create various types of pathological findings seen in either accidental or inflicted head injury. This article will further discuss the mechanisms by which consciousness is affected by neurotrauma and what the differences are between the manner by which focal and diffuse head injuries cause a loss of consciousness. It is important that the process for arriving at the decision of whether an injury is the result of an accident or is a homicide is thoughtful, deliberate and well delineated. This article aims to provide an understanding of the deliberative process utilized by the forensic pathologist in distinguishing accidental from inflicted head trauma.

Anatomical and developmental characteristics of the child's head and neck

Understanding head injuries that occur from either accidental or inflicted trauma in children requires an appreciation of the very unique features of the brain, skull and neck. These structures are in the process of maturing in young children and injuries during this period will differ from those injuries that occur later in life. The differences in these structures remain until mid-childhood but are most marked in the very

young. In early life, the skull is very thin and pliable so that the infant head can pass through the birth canal. Such a skull provides little protection against impact and force can pass readily through the unossified bones. The human brain is a large organ at birth and grows very rapidly in infancy and early childhood, yet it is not mature for many years. The young child's head is very heavy, 15% to 20% of the body's weight, compared to the adult whose head is 2% to 3% of the body's weight. The consistency of the young human brain is very soft due to a high water content, the immaturity of the glial cells, the lack and immaturity of the myelination of the axons, and the small size of the axons. These latter features make the brain in the young child more easily damaged by strains and shearing forces [1]. The subarachnoid space in a young child is thinner than later in life [2]. The thickness of the subarachnoid space determines how much buttressing effect the subarachnoid space will have, which is obviously much less in the young brain. Lastly, the strength of the neck muscles of the young child is much less than that at older ages. Acceleration-deceleration of the head is the most common mechanism by which head injuries occur and these injuries are facilitated by weak neck muscles in support of the heavy head. Neck muscle strength and the ability to prevent head motion on impact or impulse are critical factors in preventing head injury [3].

Mechanisms of traumatic brain injury

Fully appreciating distinctions between accidental and inflicted head injury in young children requires a consideration of the mechanisms by which head injury occurs in all age groups as well as a consideration of the special anatomical and developmental characteristics of the young human head and neck. Traumatic head injury can be divided into static and dynamic injuries based upon the rate at which force is loaded onto the head. Static injuries occur over longer time periods, greater than 200 ms and eventuate in crushing head injury. Such injuries occur when the stationary head is compressed by a heavy weight and the calvarium, basilar skull and facial skeleton sustain comminuted fractures resulting in the brain sustaining fracture lacerations and fracture contusions. Static head injuries are not very common but occur occasionally in children in certain circumstances that will be further discussed in the following sections. Dynamic head injuries are the most common type of injury at all ages and occur when force is rapidly loaded to the head in less than 200 ms. Dynamic injuries result when the head is caused to move (impulsive loading) either by direct impact to the head, which is free to move, or by an action to the body that causes the head to move such as a collision with the body or by shaking the body. Forceful dynamic loading can create inertial movement of the brain within the cranial cavity. Inertial brain motion occurs due to the difference in rigidities between the brain and the skull. When acceleration-deceleration of the head causes differential motion between the brain and the skull, the dura moves with the skull causing the bridging veins that arise from the surface of the cerebrum and ascend into the dura to be stretched and torn resulting in bleeding into the subdural space. Rotational inertial motion of the brain results in damage to axonal processes, traumatic axonal injury.

A further classification of head injury is into focal and diffuse brain injury categories. Impact to the head causes focal contact injuries, which include scalp contusion and laceration, skull fracture, brain contusions, epidural hemorrhage and focal subdural hemorrhage. Focal injuries can be seen by the naked eye of the pathologist on gross examination of the brain and by the radiologist on imaging with CT or MR. Impact, if forceful enough, can also create inertial brain movement. Diffuse brain injury is the result of inertial brain motion and includes interhemispheric subdural hemorrhage and traumatic diffuse axonal injury. Traumatic diffuse brain injury is not visible to either the pathologist on gross inspection at autopsy or to the radiologist on CT or MR [4–8].

Mechanisms of loss of consciousness in traumatic brain injury

The separation of traumatic brain injury into focal and diffuse brain injuries is helpful in considering how these types of injury result in clinical features, particularly in regards to levels of consciousness. Focal brain injury results in symptoms due to either destruction of brain tissue, which can happen in a brain contusion, or by creating increased intracranial pressure through an enlarging hematoma or brain swelling. In an injury in which increasing intracranial pressure leads to loss of consciousness, there is a progression over time of descending levels of consciousness. This is primarily the progression due to tentorial herniation and takes place in hours or in some cases less time but never abruptly or instantaneously. In cases of diffuse brain injury, the mechanism of loss of consciousness is traumatic axonal injury. In these cases, the change in level of consciousness is abrupt and immediate at the time of the injury. This type of unconsciousness is designated as "traumatic unconsciousness" [7].

Pathology of accidental head trauma

To illustrate the numbers of cases of accidental vs. inflicted injury occurring in and around the home in children examined in a large medical examiner's office, the author gathered data from the St. Louis metropolitan area with a population of 2 million during the time periods 1975 to 1985 and 1986 to 1999. This study excluded children who died from gunshot wounds, drowning out of the home, house fires and vehicular

accidents. From 1975 to 1985, there were 160 deaths studied of which 63 (39%) were accidents, 70 (43%) were homicides and 27 (17%) were undetermined. Of the 63 accidental deaths, 39% were under 1 year old, 28% were between 1 and 2 years old, 8% were 2 to 3 years old and 22% were older than 3 years. Thirty-seven (58%) of the deaths were asphyxial, 10 (16%) were head injuries, 7 (11%) were intoxications, 3 (4%) were in home drownings, 2 (3%) were burns, 3 (4%) were electrocutions, and 1 died from a lightning strike. Of the accidental head injuries, there were 4 children struck by an object (1 each by a metal spear, tornado damage, rotary lawn mower and a heavy pole) and 6 children who fell (3 from the second story, 1 from the ninth story, 1 down 13 basement stairs in a baby walker, and 1 child holding onto a toy cart down 10 basement stairs). The head injuries sustained by these children included 2 children with skull fractures and acute subdural hemorrhage, 2 children with penetrating wounds of the head and 6 children (70%) with massive crushing injuries with skull fractures and brain lacerations. The data from 1986 to 1999 produce rather similar types and numbers of head injuries [10].

In the time period from 2000 to 2013, the author has seen again similar types and numbers of head injuries with the addition of a small number of children with short falls sustaining skull fractures and epidural hemorrhages. These injuries will be further described in a subsequent section.

These data are similar to those of other studies that have looked at such deaths and that show that most accidental child deaths in and around the home, after excluding fires, gunshot wounds, house fires and vehicular accidents, are the result of asphyxial events. The types of deaths excluded in the study are readily recognizable as being of accidental origin. The difficulty that frequently arises with children dying from head injury in and around the home is that the injuries are often not witnessed by anyone other than the care providers who may or may not provide accurate accounts of what happened.

Crushing head injury

Crushing head injuries are static injuries as described in the above section on "Mechanisms of traumatic head injury" as occurring when the stationary head is compressed by a heavy object. While these are not the most common types of head injury mechanism when head injuries at all ages are considered, these injuries are not extremely rare in young children. Examples of such injuries occur when a vehicle wheel runs over the head of a pedestrian or when a heavy object falls onto the child's head. These injuries consist of comminuted fractures of the calvarium, basilar skull and facial skeleton with the fractured bone edges lacerating and contusing the brain. These are interesting injuries because as static injuries the crushing occurs slowly (in terms of head injury rates) and the brain injuries are focal injuries rather than diffuse injury. In nonfatal examples of such injuries, the patients do not have loss of consciousness or amnesia for the event. In these injuries, there is no rotational inertial brain motion and consciousness is maintained until the brain is markedly distorted and the deep central gray structures are damaged [7, 11]. The maintenance of consciousness despite extreme crushing head injury is illustrated by studying individuals who have survived such injuries and observing that the outcome in some of these cases is relatively good. Both Duhaime et al. [12] and Prasad [13] reported cases of young children who sustained crushing head injuries from circumstances such as a vehicular wheel running over the head or having a heavy weight such as a television fall onto the head. These crushing injuries are of great interest because they provide evidence that dynamic brain injury resulting from impulse forces, which are most often accompanied by immediate loss of consciousness, are much more damaging to the brain than is the actual physical distortion of the brain by crushing.

Short falls

For professionals who interact with children with head injuries, the question often arises as to whether a given head injury in a child is due to a short fall or some other mechanism such as inflicted injury. This is true because inflicted head trauma most often occurs outside of public areas where unbiased witnesses can verify what preceded the injury and because a common history provided to explain an inflicted head injury is that of a short fall. Young children fall frequently as a result of their learning to mobilize and in the development of their muscle strength and balance. These falls frequently result in head injuries, which are trivial and of no significance other than having sustained a "bump" to the head. Inflicted head trauma is very common and the consequence of these head injuries is usually much more significant and serious than head injuries sustained in short falls. McClelland and coauthors [14] found that the most common cause of severe traumatic brain injury is abuse, which accounts for the majority of cases in children younger than 1 year of age and 10% of all traumatic injuries in children younger than 5 years of age. Abundant literature on short falls in young children has been gathered for the purpose of providing any distinguishing features of those injuries [15–23]. What these studies overwhelmingly find is that short falls that occur in circumstances in and around the home are rarely fatal and that they cause primarily focal contact injuries including scalp contusion and laceration or no injury at all. However, in 1% to 3% of short falls, a skull fracture occurs that is usually a simple linear fracture without intracranial injury. In a very small number of those short falls that are associated with a fracture, there is an intracranial injury and these injuries may be lethal. The intracranial injuries associated with fractures from short falls are more often epidural hemorrhage and less often focal subdural hemorrhage [10, 24]. In either the epidural hemorrhage or the

focal subdural hemorrhage, the hemorrhage is located at the fracture site, which may or may not be at the site of impact. In short falls, even when associated with a skull fracture, there is little deformation of the brain distal to the point of impact and therefore there is no diffuse brain injury, although at the point of impact there may be fracture contusion [25, 26]. Ommaya et al. [27] propose a divergent viewpoint that contact injury may distribute injury diffusely through the brain of a young child. This view is not supported by the literature noted above, which looked at hundreds of cases of short falls with very few producing any significant head injury.

Epidural hemorrhage

Epidural hemorrhage (EDH) is a contact injury and occurs as a result of impact to the head. About 85% are associated with skull fractures [28]. EDHs that occur without fracture are most likely to be in young children whose heads may deform on impact without fracture but the deformity may strip off the dura to cause an EDH. Bleeding in an EDH is usually from a torn branch of a middle meningeal artery but may also result from tears of a dural sinus or diploic vein. Whether the bleeding is venous or arterial determines the rate at which the EDH occurs. EDH in young children may result from a short fall and is much less likely to result from an inflicted blow to the head, which is more likely to result in acceleration of the head and cause diffuse brain injury. The author has seen a case in which an infant's head was held stationary on a coffee table and beaten with a wooden object, which resulted in skull fracture and epidural hemorrhage (done by a psychotic mother to release evil spirits from the child) (Fig. 1). EDH occurs most often over the parietal and temporal portions of the cerebral convexities but may occur elsewhere. EDH tends to be limited by the dural attachments at the sagittal suture and creates a lens-shaped hematoma that markedly flattens the cerebral surface. In EDHs due to fractures, the cerebral cortex under the fracture will demonstrate fracture contusions. EDH greater than 100 ml in adults will produce a mass lesion but smaller amounts of epidural blood can cause increased intracranial pressure in young children [28]. EDH does not necessarily continue to bleed but may stop and then be resorbed so that not all EDHs represent neurosurgical emergencies although those that produce increased intracranial pressure do. The clinical significance of an EDH depends upon its size and rate of bleeding and its location in the head. Those EDHs that are not associated with other brain injuries and that are recognized early and treated appropriately should have little mortality or morbidity [29]. Unfortunately, EDHs from short falls in young children still cause deaths when they are not recognized and brought to medical care [10] (Table 1). EDHs should be easily distinguished from inflicted head injury by appreciating that they may result from a short fall and with a consistent history should be considered as accidental injuries.

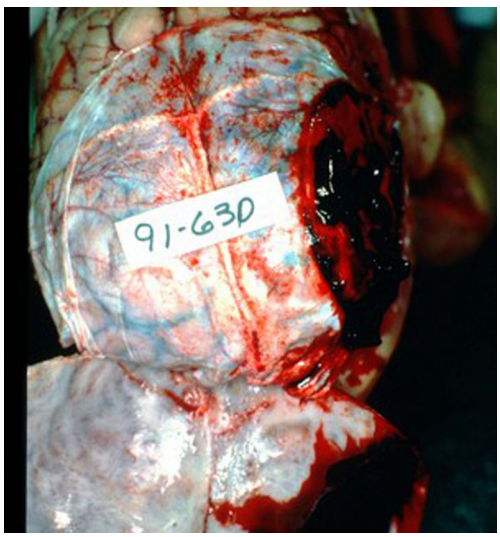

Fig. 1 A 2-year-old child hit on the head with a wooden object with fracture of right parietal bone and 80 ml epidural hemorrhage over right temporoparietal convexity

Pathology of inflicted head trauma

Head injury is the leading cause of death in children who die from inflicted injury [30]. From 75% to 80% of children who die from abuse die from abusive head trauma [31]. The head injuries seen in these children with inflicted trauma occur predominantly in younger children younger than 1 year but the same injuries are seen in children as old as 4 or 5 years of age [32, 33]. In children hospitalized for inflicted head injury, the mortality rate is about 20% [34].

Subdural hemorrhage

The most common finding at autopsy of inflicted head trauma is the presence of subdural hemorrhage (SDH), which is found in about 90% to 98% of such cases [35, 36] (Fig. 2). The number of cases with SDH at autopsy is greater than the number of living victims of inflicted head trauma in whom SDH is detectable on CT imaging because very thin layers of subdural blood over the cerebral convexities may not be visible by CT yet can be observed at autopsy. On the other hand, a very small amount of SD blood that is in the interhemispheric location is detectable by CT imaging but not visible at autopsy [37, 38]. The most typical form of SDH at autopsy in an inflicted head injury is a thin layer of SD blood over the cerebral convexities, one or both and more commonly both, and the amount of blood may be less than 10 ml (Fig. 3). There most often is also SDH in one or more of the cranial fossae. This type of SDH is the result of inertial brain

Table 1 Epidural hemorrhages in accidental falls in young children

Age	Sex	How injury occurred	History	Autopsy findings
4 months	Male	Fell out of bed, struck head on bedside table	Taken to children's hospital; CT of head negative; slept and ate poorly next 2 days; died on way to doctor's office	Right forehead contusion, fracture of right frontal bone/orbital plate, epidural hemorrhage right anterior cranial fossa
7 years	Female	Fell 3 feet off tree stump, hit back of head	Complained of headache and vomited numerous times over next 12 h; dead in morning	Left frontal subgaleal hemorrhage, fracture of left frontal bone, epidural hemorrhage over left frontal bone, contusions of left superior and middle temporal gyri
21 months	male	Fell out of bed	Cried following fall; able to be consoled; vomited and put back to bed; dead in morning	Left temporal subgaleal hemorrhage, left temporal fracture, large left epidural hemorrhage over left lateral temporoparietal convexity
10 years	Male	Fell off bike, not wearing helmet	Found on ground by bike, combative; became somnolent in ER; CT showed fracture of right temporal bone; became unresponsive; neurosurgery consulted, recommended observation; died 2 days later	Right temporal scalp contusion, fracture of right temporal bone, epidural hemorrhage over right parietal, contrecoup contusions of left temporal lobe

rotational motion within the cranial cavity in which bridging veins are torn and bleeding occurs into the cleaved subdural space [9, 39, 40]. This type of SDH is considered a diffuse brain injury as it is the result of inertial brain motion.

There are numerous causes of SDH including those from trauma of accidental, inflicted or birth-related origins, metabolic disease, such as glutaric aciduria type 1 [41], Menkes disease,[42], nutritional deficiencies [43], coaglulopathies,[44], tumors such as lymphoblastic leukemia and neuroblastotoma, and infections such as HSV meningoencephalitis and bacterial meningitis. To fully evaluate the cause of SD bleeding requires consideration of the autopsy findings in conjunction with the medical and family histories and all of the investigative information. Other reasons than trauma for SDH must be excluded before that diagnosis can be made. Vascular malformations and aneurysms are rare in young children but do occur and must be recognized when they are present [45, 46]. The distribution of the SD bleeding in the case of an aneurysm is usually basilar over the Circle of Willis rather than the convexity SDH of inflicted head injury. Vascular malformations also have different distributions of SDH related to the type and location of malformation and have the additional pathological features of such malformations.

Traumatic SDH may also occur that is not the result of diffuse inertial injury, but the result of focal head injury and these injuries may occur in short falls. In such cases, the child typically sustains a short fall, does not have an immediate onset of unconsciousness, and may have contact injuries

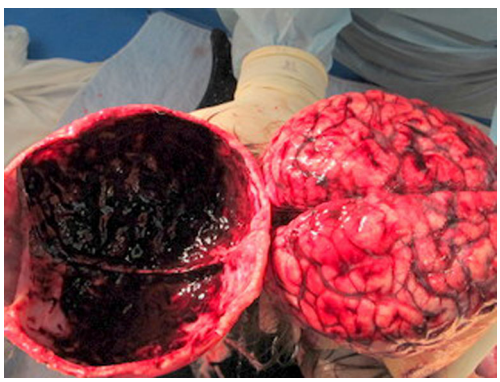

Fig. 2 A 7-month-old infant with inflicted head trauma with subdural blood over both cerebral convexities

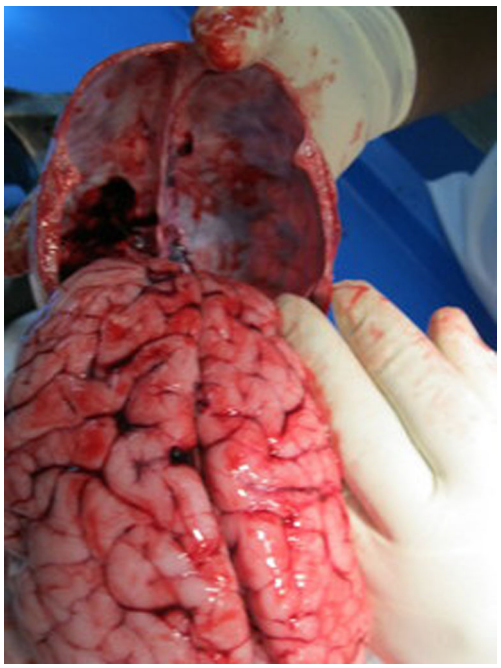

Fig. 3 A 3-month-old infant with inflicted head trauma with small amounts of subdural blood over cerebral convexities, greater on the right side

resulting from the impact such as scalp contusions and rarely fractures. These SDHs may increase in size over time and may become a mass lesion with the complications resulting from that problem such as is seen with an epidural hemorrhage. Hymel et al. [47] described 54 children under 36 months of age hospitalized with acute head injuries resulting from inflicted trauma (11), noninflicted trauma (30) or undetermined etiology (13). In the noninflicted trauma group, 26 were caused by a fall. In this group, 7 of the 26 had SDH on CT and in 3 of the 7, the SDH extended or originated from the interhemispheric region. These 3 children fell from 3 to 6 feet and had contact injuries but only superficial focal cortical brain injuries and did not have any loss of consciousness. These cases illustrate that there are differences between contact and noncontact SDHs and the latter are much more correlated with concussion.

Four children with "disappearing SDHs" were described by Duhaime and colleagues [48]. These 4 children were 10 months, 9 months, 4 years and 9 years of age and had unilateral convexity SDHs on CT from household falls in 3 cases and in the fourth case a fall from the second story. The clinical symptoms in the 3 children with household falls were quite minor and consistent with the forces involved. In these 4 children, the SDHs resolved very rapidly over 1 to 2 days. These authors noted that the blood, which was thought to be SD on CT, may have actually been in the subarachnoid space, which would explain why it would resolve so rapidly and that finding blood in the SD compartment by CT is not always accurate as these two compartments may be indistinguishable.

Subarachnoid hemorrhage

Subarachnoid hemorrhage (SAH) generally accompanies SDH caused by rotational inertial brain injury in which tearing of bridging veins occurs. This is true because the bridging veins pass through the arachnoid membrane to reach the dural sinuses and when a bridging vein tears, some amount of blood may enter the subarachnoid space. In cases of inflicted neurotrauma, the typical appearance is small patches of SAH along the cerebral parasagittal convexities (Fig. 4). SAH can result from other causes of blood entering the subarachnoid space whether natural causes such as a ruptured aneurysm or vascular malformation or from non-inertial traumatic causes produced from contact force such as contusions and lacerations.

Retinal hemorrhages

At the autopsy of any child in which there is a possibility or suspicion of physical abuse, the eyes should be removed. Removal of the eyes is best carried out by removing the orbital roof so that the eyes can be dissected out of the surrounding fat and then fixed in formaldehyde for examination after fixation [49]. The eyes should be removed to allow as much of the optic nerve as possible to be retrieved with the eye. The surrounding fat is left attached as this may contain hemorrhage. Following fixation, the eyes are examined grossly and then sectioned into segments that allow visualization of the interior of the eye. All findings are documented and photographed. Sections are taken for microscopic examination and findings should be described and documented as to the presence of optic nerve sheath hemorrhage, retinal hemorrhages and the presence of any schisis of the retina or hemorrhage in the vitreous. Descriptions of any retinal hemorrhages should include notation of the locations of the hemorrhages (posterior, periphery or reaching the ora serrata), the number of hemorrhages present (few, many or too numerous to count) and which of the retinal layers are affected. The optic nerve sheath is examined closely to determine whether there is hemorrhage within the sheath or the nerve.

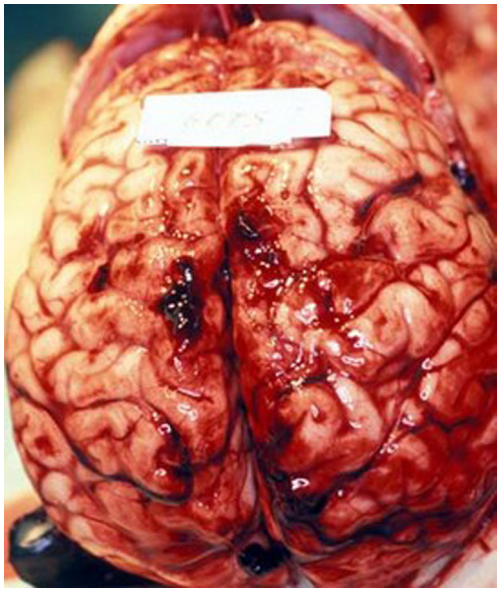

Fig. 4 A 6-month-old infant with inflicted head trauma with patches of subarachnoid hemorrhage in posterior parasagittal regions

In cases of inflicted head injury, retinal hemorrhages (RHs) are common findings occurring in as many as 80% of fatal cases [50–53]. Characteristics of the retinal hemorrhages most commonly associated with inflicted head injury are large numbers of RHs that are usually too numerous to count in multiple layers of the retina and that extend far into the periphery (Fig. 5). In some cases, the numbers are fewer. The RHs may be in one or both eyes although they are most commonly present in both eyes [51]. Other eye findings that have been associated with severe inflicted head trauma include retinal folds and retinoschisis (Fig. 6). Retinoschisis is the splitting apart of the retinal layers to produce a cavity filled with blood. These are often near the macula and the margins appear as perimacular folds. At autopsy, folds may occur artifactually in the retina and these are usually circumferential and linear while the folds associated with schisis cavities are

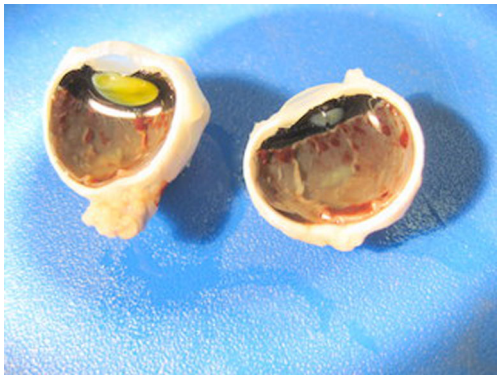

Fig. 5 Eye sectioned from pupil to optic nerve of infant with inflicted head trauma shows retinal hemorrhages extending far into the periphery to the ora serrata

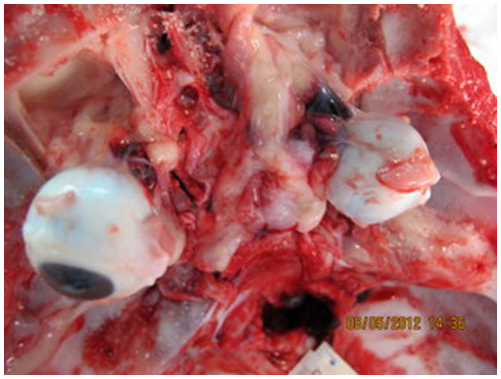

Fig. 7 Eyes being removed from cranial cavity by unroofing roof of orbit; eye on the right shows extensive optic near sheath hemorrhage; infant with inflicted head trauma

along the margins and around the cavity. Schisis cavities are most commonly seen in association with severe degrees of RHs in inflicted neurotrauma but have also been reported in a small number of crushing head injuries in young children [54–56]. RHs may occur in accidental head trauma although they are found in many fewer cases than with inflicted head injury and the type of accidental trauma tends to be very severe such as motor vehicle accidents [57]. Obviously in cases such as vehicular accidents, the circumstances of the injury are quite evident.

Retinal hemorrhages may be found in certain nontraumatic disorders including bleeding disorders, sepsis, meningitis and vasculopathies [58–64]. Retinal hemorrhages in these disorders tend to be rather sparse in number and located in the posterior pole of the retina. In very rare cases, RHs may occur from CPR with chest compressions [65–67]. In those cases where CPR was associated with RHs, Pham et al. [68] noted that the types seen were distinctly different than those seen in association with inflicted neurotrauma and when they do occur with CPR there are coexisting risk factors for the hemorrhages.

Optic nerve sheath hemorrhage is a common finding in young children with inflicted neurotrauma [69–71]. Optic nerve sheath hemorrhage appears as blood grossly visible at the portion of the nerve most proximal to the bulb of the eye and may extend outward some distance and even into the surrounding fat (Figs. 7 and 8). The hemorrhage around the sheath of the nerve is usually in the subdural area of the nerve. Optic nerve sheath hemorrhage can be seen in rare nontraumatic cases usually related to increased intracranial pressure from other causes [55].

Fig. 6 Eye sectioned from pupil to optic nerve of infant with inflicted head trauma shows schisis cavity at posterior part of retina

Traumatic diffuse axonal injury

Traumatic diffuse axonal injury (tDAI) is widespread damage to axonal processes resulting from inertial movement of the brain caused by rotational acceleration-deceleration of the head [72, 73]. Deformation of the brain strains and damages the axons and in some cases (rarely in young children) causes tearing of small vessels. In very young infants, usually younger than 5 months old, the deformation also causes tearing of the brain tissue to result in a finding Lindenberg [74] described as contusion tears. Contusion tears appear as slit tears of the cortex-white matter junction or within the lamina of the cortex where differences of tissue architecture exist. Damaged

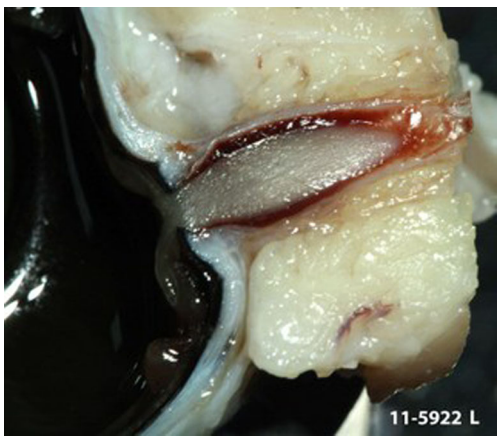

Fig. 8 Eye sectioned from pupil to optic nerve of infant with inflicted head trauma shows hemorrhage surrounding the optic nerve within the sheath

axons are microscopic lesions and may be visible on light microscopy after 18 to 24 h of survival with routine staining techniques (H & E). The retraction bulb is the microscopic appearance of a damaged axon whose axoplasm has accumulated adjacent to the damaged axon. In young children, the bulbs are difficult to observe by H & E staining because the axons are very small. An immunohistochemical process using beta-amyloid precursor protein staining (BAPP) will allow damaged axons to be seen as early as 2 h after damage [75]. The interpretation of the immunohistochemical staining with BAPP requires understanding that a variety of patterns of axonal damage exists including damage from trauma (tDAI), hypoxic/ischemic insult (vDAI), and metabolic insult (mDAI). To properly evaluate the axonal damage requires taking numerous microscopic sections from cerebral deep white matter, deep gray structures, corpus callosum, brainstem and cerebellum. Many cases of inflicted neurotrauma sustain vDAI because they develop respiratory failure following their injuries. Microscopically, BAPP patterns of expression of vDAI appear as broad swaths or zigzagged patterns of staining of damaged axons often in relation to vascular structures. In some cases of inflicted neurotrauma, isolated BAPP stained axons can be found scattered within the brain indicating tDAI. Several studies have evaluated cases of inflicted head injury for the frequency of such patterns and have generally shown that vDAI is the most common pattern seen while the tDAI pattern is seen less often [76–78]. The value of being able to demonstrate BAPP expression in tDAI pattern is that as noted in the section above on mechanisms of traumatic brain injury, the basis of traumatic unconsciousness is tDAI. In those cases in which the neuropathology is tDAI, the mechanism for loss of consciousness is that axonal damage and the loss of consciousness is immediately at the time of the injury.

Conflicts of interest None

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