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MINI-SYMPOSIUM: Forensic Pathology of Brain Trauma in Children

Inflicted Traumatic Brain Injury in Infants and Young Children

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Abstract

This article will discuss the subject of inflicted or abusive head injury in infants and young children. Inflicted neurotrauma is a very common injury and a frequent problem in attempting to distinguish between inflicted and accidental injury. Inflicted head injury occurs usually in the home in the presence of the individual who has inflicted the injury outside the view of unbiased witnesses. Distinguishing between inflicted and accidental injury may be dependent upon the pathological findings and consideration of the circumstances surrounding the injury.

The most common finding in an inflicted head injury is the presence of subdural hemorrhage. Subdural hemorrhage may occur in a variety of distributions and appearances. The natural history of subdural bleeding and the anatomy of the "subdural" will be considered. The anatomy of the dura and its attachment to the skull and to the arachnoid determines how subdural bleeding evolves into the cleaved dural border cell layer and as well as how bridging veins are torn and anatomically where bleeding will occur. Different biomechanical mechanisms result in different distributions of subdural blood and these differences will be discussed.

INTRODUCTION

Differentiation between inflicted and accidental traumatic head injury is a problem that arises frequently in the pediatric population. Young children fall frequently and most traumatic head injury is caused by falls, although the great majority of short-distance falls are trivial in terms of the injuries caused by those falls. Unfortunately, inflicted head injury is also very common in infants and young children and often has far worse consequences than the minor falls. The most common cause of severe traumatic brain injury is abuse, which accounts for most cases of head injury in children under 1 year old and 10% of all traumatic injuries of those under 5 years old (66). Billmire and Myers noted that 64% of head injuries serious enough to warrant admission to the hospital (excluding uncomplicated skull fractures) and 95% of serious intracranial injuries are the result of child abuse (6). It is estimated that some 2000 children die yearly in the United States from abuse and neglect (65). Head injury is by far the leading cause of death from child abuse (68). As many as 80% of fatal abusive injuries are caused by head injuries (19). The younger the child who suffers from inflicted injury, the more likely that injury will be a head injury (17). Although the majority of children with inflicted head trauma are under 2 years old, older children may have identical pathological findings (16, 75).

This article will describe the neuropathological features of inflicted traumatic brain injury in infants and young children. Although these injuries predominate in infants under 1 year old, the same injuries may be found in children as old as 4 or 5 years (28). Of children with inflicted head injury about 30% die. Some 30% to 50% have significant neurological deficits including varying degrees of mental retardation or slowness, learning disabilities, seizures, blindness or irritability. Some 30% of the injured children appear to have full recovery (5, 8, 74).

In the Introduction section of this symposium, the mechanisms of head trauma were discussed and will not be described further. Infants and young children can be injured by the same mechanisms that create head trauma at other ages. As noted in the discussion of the unique anatomical and developmental characteristics of young children in the Introduction, these unique characteristics probably cause differences in the thresholds and injuries in the youngest of the human species but there remains uncertainty as to what exact extent those differences exist. In 1972, Caffey described abusive shaking of infants and introduced the idea that young children could be injured by this inertial mechanism (11). Since then, others have described infants who were thought to have been injured by a whiplash-shaking mechanism. Whiplash shaking is considered by some to be a source of acceleration deceleration inertial brain injury. In 1987, Duhaime *et al* questioned whether shaking, as an isolated mechanism, could create the rotational acceleration and velocity necessary to cause concussion, subdural hemorrhage or axonal injury (26). Using a doll model, those authors found that shaking alone did not reach the rotational acceleration necessary for significant injury. Since that time, there has been a continuing debate as to whether shaking can significantly injure the brain of a young child. There is obviously no debate over whether impact can

cause head injury. This article will not further consider the debate as to whether shaking can create significant inertial traumatic brain injury. In those cases that have been described as examples of shaken infants, the pathology of the injury is the same as in cases that have an impact. The absence of an impact site is sometimes cited as evidence that no impact occurred, but there is some disagreement over whether even broad, soft surfaces used as an impacting object will not leave an impact. The aim of this article is to discuss the pathology of the inflicted neurotrauma.

PATHOLOGY OF INFLICTED TRAUMATIC HEAD INJURY

Subdural hemorrhage

The most common finding in abusive head injury is subdural hemorrhage. Knowledge about the nature of the "subdural space" has changed over the past 30 years and it is now recognized that a true subdural space does not exist (42, 43). The dura is composed of fibroblasts and large amounts of extracellular collagen. The innermost layer of the dura is made up of the dural border cell layer and this layer is continuous with the arachnoid outer barrier cell layer. Although a patent subdural space does not exist, a subdural space may be created by blood entering the dura–arachnoid junction and dissecting open the dural border cell layer. The dural border cell layer is the weakest plane and will dissect open as a result of trauma. The dura is attached to the inner table of the skull and the arachnoid is attached to the pia on the surface of the brain. When the skull is removed at surgery or autopsy, the skull is lifted away cleaving the dural border cell layer from the arachnoid so that there appears to be a naturally occurring space beneath the dura. In fact, the space created by the cleaving of the dura border cell layer is intradural. The bridging veins pass through the meninges as they travel from the cortical surface of the brain to the venous sinuses in the dura. The walls of the bridging veins are attached to the dural border cell layer and to the arachnoid cells. The attachment of the bridging veins to the arachnoid is relatively strong whereas the attachment at the dural border cell layer is relatively weak. When either impulse loading or impact causes cranial acceleration sufficient to create differential motion between the brain skull interface, the dura moves with the skull and the arachnoid moves with the brain, placing strain on the bridging veins. When the strain becomes great enough to tear the bridging veins, blood enters the intradural layer of the dural border cells and it is the blood classically noted to be "subdural." Because it is easier to discuss bleeding at various sites within the intracranial compartment by using the terminology "subdural," this article will continue to use that phraseology but does recognize that it is anatomically a potential space.

Subdural hemorrhage is present in approximately 90% to 95% of fatal cases of inflicted head trauma, and is able to be imaged in 40% to 55% of living patients with inflicted traumatic head injury (15, 26, 37, 46). In many of the cases, the hemorrhage is a thin layer of subdural blood diffusely distributed over both cerebral convexities (Figure 1). In some cases, the bleeding may be unilateral rather than bilateral but will be diffusely distributed over the right or left convexity, in contrast to a more focal area of subdural bleeding (Figure 2). The biomechanical mechanism of subdural bleeding in

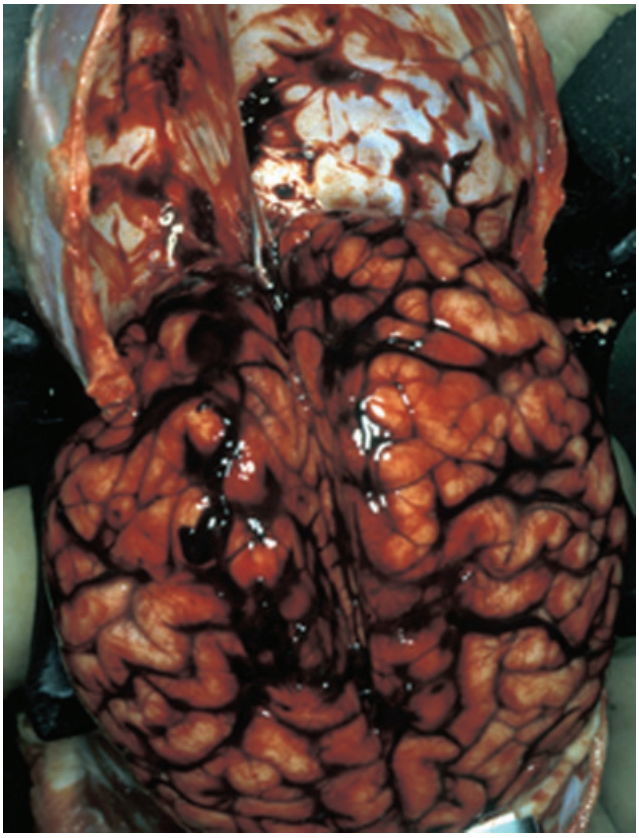

Figure 1. A 7-month-old male with inflicted head trauma with thin layer of subdural blood over cerebral convexities.

inflicted head trauma is inertial brain motion separating the cortical surface from the overlying skull and attached dura, causing tearing of the bridging veins (35, 88). Inertial motion of the brain begins earliest in the posterior hemispheric fissure so that the earliest bleeding develops in that area and then spreads anterior and outward (36, 82). A study that looked at the types of hemorrhage in 293 children under 3 years of age found that the most common type of subdural hemorrhage was in the interhemispheric region in 143 children (49%). Seventy-three percent of these children suffered from inflicted injuries. Accidental trauma caused 15% of the interhemispheric subdural hemorrhages and all these accidental injuries were associated with relatively high force—three falls from heights of more than 2 m, 12 motor vehicular collisions, four falls in an infant walker and two blows to the head. This study found that subdural hemorrhage over the convexities was almost as common as interhemispheric subdural hemorrhage and was found in 47%. Convexity and interhemispheric subdural hemorrhages occurred together in 52% of the patients who had subdural hemorrhages. Inflicted trauma accounted for 72% of the convexity subdural hemorrhages. Of the 10% convexity subdural hemorrhages following accidental trauma, most were from severe injuries such as motor vehicular collisions, falls from heights greater than 2 meters, or infant walker–stair falls. There were three subjects with convexity subdural hemorrhages who fell from less than 2 m (84).

In cases where there is an associated skull fracture, the subdural bleeding is not always immediately adjacent to the fracture.

Figure 2. A 5-month-old female with inflicted neurotrauma with subdural blood over both cerebral convexities greater on the left than right.

(Figure 3A,B) The amount of subdural blood may be very minimal and in some cases less than 5 mL or 10 mL. Such a small amount of subdural blood can be visualized at autopsy by a very close inspection of the cranial cavity as the calvarium is being removed. Once the venous sinuses have been disrupted so that the calvarium can be released, however, blood draining from the venous sinuses may interfere with appreciation of very small amounts of subdural blood. Computed tomography (CT) scans may not be able to demonstrate the very small amounts of subdural blood that is present over the cerebral convexities in some cases but CT scans can visualize subdural blood within the interhemispheric fissure, an area that is not readily examined at autopsy until the brain has been removed and artifactual hemorrhage may be difficult to eliminate (20, 52). Magnetic resonance imaging is better in demonstrating very small amounts of subdural blood over the cerebral convexities than CT scans (31). Following the removal of the brain, subdural hemorrhage is commonly seen in one or more of the cranial fossae in addition to the subdural blood seen over the convexity. Many cases of inflicted head trauma will also demonstrate the presence of blood in the spinal subdural space, and this blood probably descends by gravity from the intracranial compartment.

Subdural hemorrhage may occur in association with inflicted trauma, accidental trauma, medical or surgical manipulation, pre- and perinatal conditions, birth trauma, metabolic diseases, tumorous conditions, genetic disorders, autoimmune disorders, clotting disorders, infectious diseases, long-term shunting for hydrocephalus and other miscellaneous conditions. In suspected cases of inflicted trauma, other sources of subdural bleeding must be considered and can most often be excluded by history, physical examination, radiological imaging and laboratory studies. Subdural hemorrhage isolated over the ventral surface of the brain or in a focal area of the brain requires very close examination for another source of bleeding as such findings are not consistent with an inertial mechanism of bridging vein hemorrhage (71, 83). Contact injuries of the head may also create focal areas of subdural bleeding and these injuries will be considered in the accompanying article on accidental head injuries in children.

In general, acute subdural hemorrhage at all ages is either a mass lesion—in which case it must be surgically removed if the hemorrhage develops significant mass or else the patient will suffer consequences of increased intracranial pressure—or an epiphenomenon of diffuse axonal injury. If the subdural hemorrhage is an epiphenomenon of diffuse axonal injury, it is the typical thin diffuse hemorrhage as described previously, and this blood eventually resolves. Most unoperated acute subdural hemorrhages resolve into liquefied clot in 4 to 6 weeks and will be absorbed (18).

Chronic subdural hemorrhage

In the past, it was presumed that chronic subdural hematomas developed from acute subdural blood. Currently, there is appreciation that few cases of acute subdural hemorrhage evolve into chronic subdural hematomas (24, 55, 69). Experimental models are not able to reproduce a chronic subdural hematoma from acute subdural blood (39). It is now apparent that the great majority of chronic post-traumatic subdural hematomas develop from subdural hygromas (56). A subdural hygroma arises from a traumatically induced tear of the arachnoid, allowing effusion of cerebrospinal fluid into the subdural space, as an acute subdural hemorrhage is resolving (10, 67). Most acute subdural hemorrhages resolve rapidly because of the high levels of tissue thromoplastin in the brain and cerebrospinal fluid (4). Any pathologic process that induces cleavage of the dural border zone tissues can induce proliferation of dural border cells with production of neomembrane. In certain circumstances, when an acute subdural hemorrhage resolves, the cleaved intradural membrane may remain as a persistent space. Persistence of an intradural space may be facilitated by circumstances that lower intracranial pressure such as areas of cerebral atrophy, prolonged drainage of cerebrospinal fluid, or prolonged use of osmotic agents (21, 64). Effusion of cerebrospinal fluid or remnants of the liquid portions of the acute subdural blood can then pass into the cleaved space and create the hygroma. What happens to a hygroma is determined by whether the lowered intracranial pressure persists. If there is return to normal pressure, the hygroma resolves. If the lowered pressure continues, the hygroma may expand and further develop neomembranes from the proliferating dural border cells. As neovascularization creates the ingrowth of new vessels, some of these fragile vessels may tear and result in

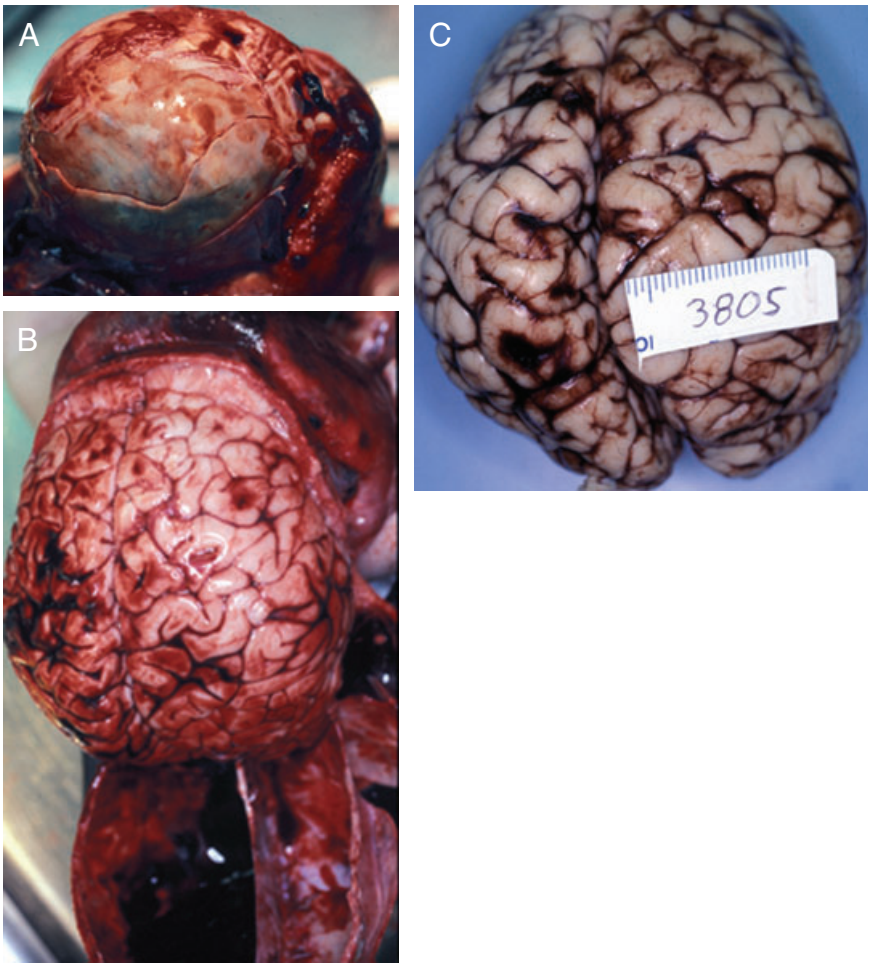

Figure 3. A. A 3-month-old male with inflicted head injury with fracture of right parieto-occipital skull. **B**. Subdural blood over left cerebral convexity. **C**. Subarachnoid hemorrhages over left cerebral convexity.

microhemorrhages with little or no trauma. It is the repeated microhemorrhages and repeated resolution of these hemorrhages that lead to an enlarging subdural hygroma and transformation to the chronic enlarging subdural hematoma (53, 56, 85). The differences between the subdural hygroma and the subdural hematoma depend upon the degree of evolution of the neovascularization, microhemorrhaging and resolving of those microhemorrhages. Hygromas tend to be less than 3 weeks old, static or decreasing, contain clear fluid, lack membranes and do not or rarely produce a mass effect. A chronic subdural hematoma tends to be older than 3 weeks, enlarging, contains dark brown fluid (crank case), has membranes and may cause a mass effect. Both appear similar on a CT scan (56). The histopathology of subdural membranes in young children is similar to those described in adults. A chronic subdural hematoma is a liquefied hematoma with a membrane. The membrane consists of fibroblastic proliferation and neovascularization (69). An acute subdural hemorrhage is a clot and never has a membrane. The fluid within a subdural hygroma is frequently a mixture of blood and cerebrospinal fluid. The chronic subdural hematoma shows excessive activation of both clotting and fibrinolytic systems (50). Because blood within the cleaved border cell layer will be resolved through coagulation and fibrinolysis, a disordered hemeostatic mechanism is probably necessary for the origin and development of a chronic subdural hematoma.

In infants and younger children it may be difficult to distinguish between subdural hygroma and chronic subdural hematoma. Because the infant's skull is deformable, posttraumatic brain atrophy may allow significant enlargement of a chronic subdural hematoma. Later, as the sutures fuse and the skull becomes rigid, an enlarging chronic subdural hematoma may cause a mass effect.

An acute subdural hemorrhage will usually image on CT scan as a uniform high density. Hyperacute subdural hemorrhage may imagine on CT as high and low density, low attenuation, and may be mistaken for an older chronic subdural hemtoma or rebleeding in a chronic subdural hematoma (76). The low-density component may represent clot retraction or serum extrusion from portions of the hemorrhage. Interpretation of CT imaging evidence of a subdural collection for purposes of establishing age of the collection or to evaluate for rebleeding should be approached cautiously because of the heterogeneity within the collection of subdural blood.

Subarachnoid hemorrhage

Subarachnoid and subdural hemorrhages can both result from disruptions of bridging veins, as these veins pass from the cortical surface through the arachnoid membranes. Subarachnoid hemorrhage occurs in about the same number of cases as subdural hemor-

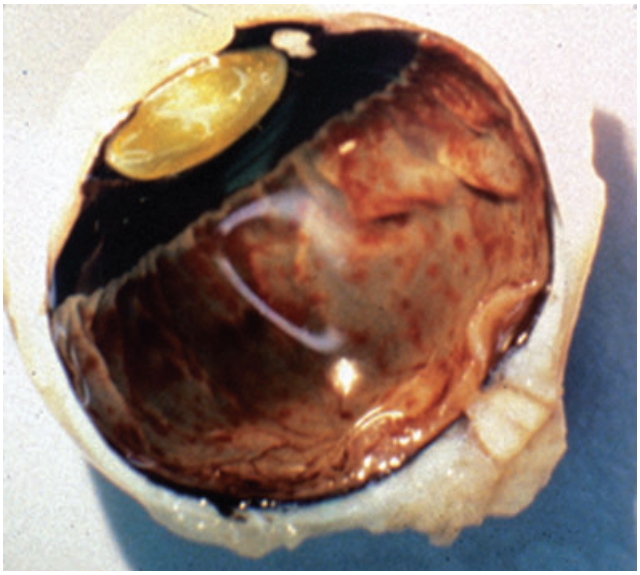

Figure 4. Eye transected through pupil-optic nerve plane of a 2-month-old male with inflicted head injury showing multiple retinal hemorrhages extending far into the periphery to the ora serrata.

rhage but appears in patches over the cerebral convexities and to some extent on the interhemispheric surfaces where it is difficult to see (23). Subarachnoid hemorrhage is usually contiguous to or lies under areas of acute subdural hemorrhage (Figure 3C). On a CT scan, patches of subarachnoid hemorrhage are more difficult to appreciate than the accompanying subdural hemorrhage. Experiments on primates have demonstrated that the parasagittal bridging veins tear from high-magnitude angular deceleration with inertial brain displacement (35, 87). Subarachnoid hemorrhage in cases of inflicted head injury appears to be related to inertial brain displacement that also causes the subdural hemorrhages seen in these cases.

Retinal hemorrhages

Retinal hemorrhages are common in inflicted traumatic brain injuries and occur in as many as 85% of cases (51, 58, 60, 61, 81). The characteristic pattern of retinal hemorrhages that is highly associated with inflicted head injury is the numerous multilayered hemorrhages extending far into the periphery of the retina and often reaching the ora serrata (17, 59) (Figures 4 and 5). They are more often bilateral but may be unilateral. The pathogenesis of these retinal hemorrhages is not fully understood but is thought related to the same acceleration deceleration forces which initiate the other pathological features of inflicted brain injury. Retinoschisis is the splitting of the layers of the retina and is believed to be caused by the shearing forces generated through acceleration–deceleration causing the strong attachment of the vitreous to the retinal surface to be pulled upon and to shear the retinal surface (59, 40). Retinoschisis may be difficult to identify at autopsy when the eyes are removed and processed in the usual manner because artifactual disruption of the retina may be created that is difficult to distinguish from actual retinoschisis (75).

Retinal hemorrhages may also occur, although in many fewer cases, in very severe accidental head trauma particularly vehicular collisions (27, 47). Retinal hemorrhages are not found in cases of mild accidental trauma. Because of the very common association of retinal hemorrhages with inflicted head trauma, it is necessary to examine the eyes in all cases in which there is suspicion of abuse. For forensic purposes, the best technique for removal of the eyes is to remove the orbital plates of the anterior cranial fossae, then dissect the fat and muscle away from the eyes and finally lift the eyes out of the fat pad at the same time keeping as much optic nerve attached as possible.

Retinal hemorrhages may have a variety of pathological features and pathogeneses. Retinal hemorrhages occur in a great number of disorders including bleeding disorders, sepsis, meningitis, vasculopathies, some newborns, certain genetic conditions and many others (25, 29, 30, 86). Retinal hemorrhages occur rarely in association with chest compressions with cardiopulmonary resuscitation (49, 54, 57). When retinal hemorrhages occur in these conditions that are unrelated to inflicted neurotrauma, the hemorrhages tend to be few in number and confined to the posterior pole of the retina.

A finding commonly accompanying the retinal hemorrhage associated with inflicted head trauma is optic-nerve sheath hemorrhage. Hemorrhage within the perineural tissue is seen in many

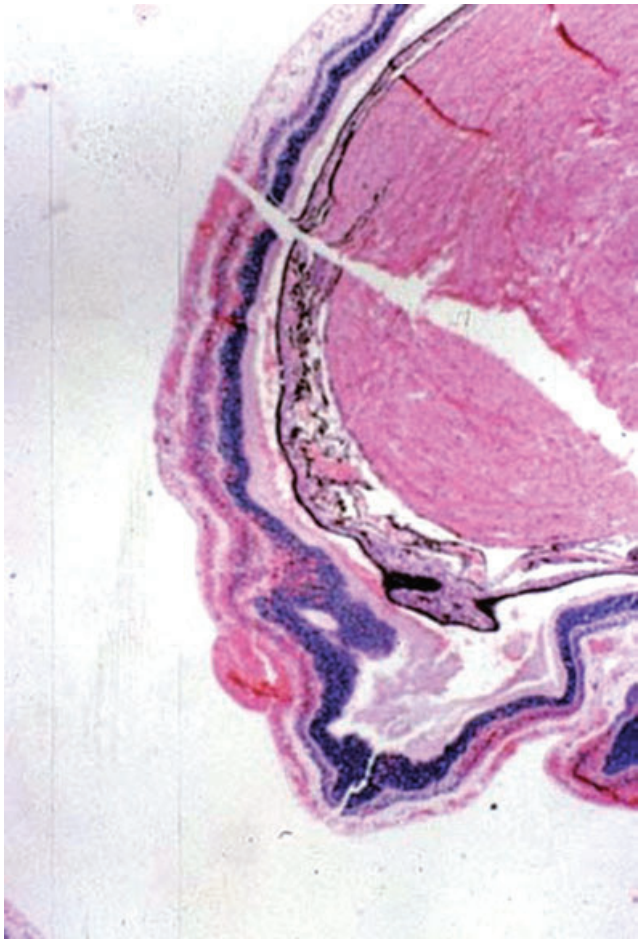

Figure 5. Eye of a 2-month-old male with inflicted head trauma showing multiple hemorrhages throughout all layers of retina (40× magnification).

cases of retinal hemorrhage associated with inflicted neurotrauma. These hemorrhages are seen in other causes of subdural hemorrhage and are not specific to abusive injury. Optic nerve sheath hemorrhages can also be seen in association with some cases of increased intracranial pressure even those unrelated to cranial trauma (40).

Traumatic Diffuse Axonal Injury

Severe rotational acceleration deceleration injuries create diffuse shearing injury of the brain resulting in widely distributed damage to axons, small blood vessels and even on rare occasions gross tissue tears. The inertial brain movement that accompanies acceleration deceleration of the head creates differential movement of the brain beginning at the cerebral periphery and extending deeper into the center of the brain as greater force is transmitted to the brain. In descending through the cortex, through white matter, through deeper gray matter and into the brainstem, tissues of different consistencies and structures are encountered and these differences facilitate shearing at those points (1, 3).

Traumatic diffuse axonal injury can be classified into three grades based upon the system used by Adams *et al* (3):

- Grade 1: microscopic damage to axons without gross hemorrhage.
- Grade 2: microscopic damage to axons with hemorrhage in the corpus callosum.
- Grade 3: microscopic damage to axons with hemorrhage in the dorsal aspect of the brainstem.

The hemorrhages described in association with the axonal damage are in areas adjacent to the axonal damage and appear grossly as streak or punctuate hemorrhages as small as 1 mm and ranging up to several centimeters when bleeding has continued over several days (2). In young children, hemorrhages are seldom found in association with the axonal damage because the blood vessels in young children are quite elastic and tend not to tear as readily as adjacent axons on tissue deformation. The grading scheme of Adams is not useful in young children as hemorrhages are so infrequent.

Damaged axons may be visible on light microscopy after 18 to 24 h using hematoxylin–eosin (HE) staining. These damaged axons appear as retraction bulbs where axoplasm has accumulated adjacent to a damaged axonal process. On HE staining, these bulbs are pink. Retraction bulbs are not easily seen in young children on HE stains because the axons are very small and the bulbs are even smaller. Beta-amyloid precursor protein (B-APP) immunohistochemical stains will demonstrate axonal damage in less than 2 h (78). A recent report noted that axonal injury can be detected within 35 minutes in adults using B-APP (45). The axonal damage detected is damage of any type and not specific to traumatic injury.

In relatively rare cases of diffuse axonal injury in infants usually under 5 months of age, tissue tears may be evident grossly in the brain. These tears were described by Lindenberg as contusion tears (62). These tears appear as splits or slits at the cortex–white matter junction or within lamina of the cortex (Figure 6). The same inertial deformation of the brain that causes the axonal damage causes these tears in very young infants (20). Handling of the brain may create similar defects particularly at the cortex-white matter junction.

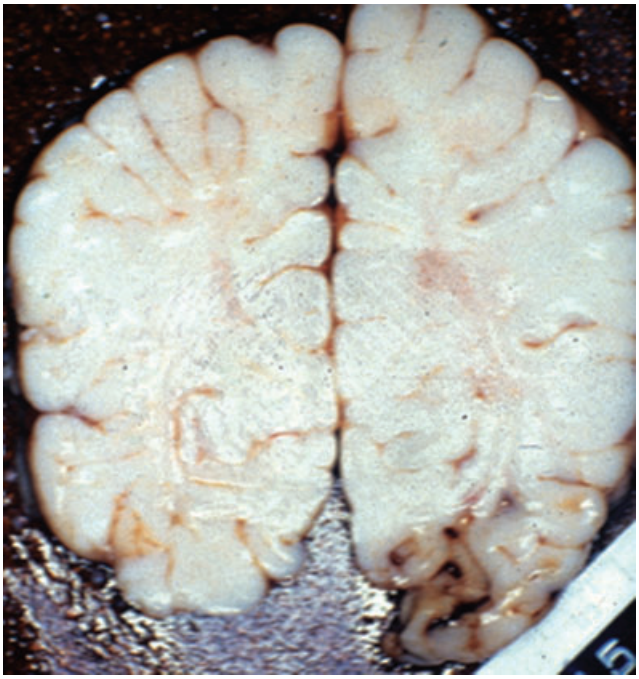

Figure 6. Coronal section of brain of a 6-month-old female with inflicted head trauma showing contusion tear of right inferior parietal lobe.

Studies of young animals, which hope to elucidate patterns of inertial injury of the brain, demonstrate that differences exist among the various models examined. Some animal models react to inertial injury by developing axonal damage whereas some show neuronal damage and hypoxia that may be an additional factor that determines which patterns develops. Bonnier *et al* studied brains of a mouse pup model of shaken baby syndrome using 8-day-old pups shaken for 15 s on a rotating shaker and then sacrificed at 13, 15, 19 and 31 days (9). These animals did not appear to have hypoxia associated with their injuries so they represent a pure shaking model. The study demonstrated that shaking these young pups caused mortality, multifocal white matter lesions in two-thirds of the survivors and significant white-matter thinning. The brain damage in these pups was axonal. The pups were also examined for retinal hemorrhages and were found to have shaking-induced retinal hemorrhages. Bittigau *et al* studied a newborn-rat model using focal impact-induced brain trauma and found that the damage was extensive neuronal cell death in several gray structures (7). Smith *et al* studied rat pups subjected to shaking without impact and found that the resulting damage was neuronal degeneration (79). The differences between these studies are: Bittigau's rat pups were shaken on three separate occasions whereas Bonnier's mouse pups were shaken once; and Smith's rat pups were hypoxic during the injury. Smith *et al's* rat pups also demonstrated cortical hemorrhaging that was not found in the mouse pups. Smith *et al* also described evidence of retinal hemorrhages in their shaken rat pups. Smith *et al* noted that although diffuse axonal injury was not found in their study, axonal damage was likely triggered by the primary neuronal injury, and diffuse axonal injury is difficult to demonstrate in the neonatal rat because the brain is undergoing rapid development in terms of myelination and growth of nerve fibers.

NEUROPATHOLOGY OF INFLICTED BRAIN DAMAGE

The neuropathology of the brain in cases of inflicted head injury in young children remains poorly studied or documented. Part of the problem is that many, if not most cases, of children dying from inflicted trauma are autopsied by forensic pathologists under the jurisdiction of a medical examiner or coroner system. Forensic pathologists are not generally familiar with neuropathological processes or techniques. Most cases of inflicted neurotrauma do not have detailed studies of the brain carried out by a neuropathologist in a manner conducive to fully elucidating the neuropathological findings. An additional problem is that many of the past studies that attempted to study the neuropathology contained few cases (12, 38, 41, 77, 82). Recent studies of B-APP staining in cases of inflicted neurotrauma by forensic neuropathologists have discussed the sampling techniques necessary to diagnose diffuse traumatic axonal injury (dTAI) and vascular axonal injury (VAI) as well as the interpretation of these stains (22, 73). These authors note that B-APP expression may be absent in damaged axons even when there is a length of survival, where reactive changes would be expected because cerebral perfusion was halted very rapidly early on in these cases.

In the largest study of the neuropathology of inflicted head trauma to date, Geddes *et al* studied 53 cases of fatal inflicted neurotrauma in children, 37 under 9 months of age and 16 between 13 months and 8 years (33, 34). Skull fractures were found in 36%, acute subdural hemorrhage in 72%, and retinal hemorrhages in 71%. The most common cause of death was raised intracranial pressure. The brains were systematically sampled to include several blocks of hemispheric white matter, corpus callosum, internal capsule, cerebellum, midbrain, pons, medulla and spinal cord. HE and B-APP staining were carried out on all cases. Clinical histories on presentation were: apnea, abnormal breathing or collapse, with the child suddenly turning blue and limp (33 cases); found dead (six cases); child dropped (four cases); child fell (two cases); found unconscious at bottom of stairs (two cases); and thrown, shaken or stabbed (one case each). In three cases, no details of presenting history were available. Fifty-one percent (27 of 53) had additional injuries: 10 had recent or old fractures of ribs or clavicle; four had long-bone fractures; six had serious abdominal injuries; and seven had burns or extensive bruising. Eighty-five percent (45 of 53) had evidence of impact to the head at autopsy with scalp bruises or skull fracture. Of the 19 skull fractures, 18 were either parietal or occipital. In six cases, the fractures were bilateral. None of the fractures had epidural hemorrhages. Subdural hemorrhages were present in 43 and 38 of them were acute. The older hemorrhages consisted of pigmentation and membrane formation. Of the acute subdural hemorrhages, 34 were thin films of blood and 28 were bilateral. Four of the subdural hemorrhages were large enough to produce mass lesions. These four hemorrhages occurred in older children aged 8 months, 9 months, 3 years and 4 years. Brain weight was increased in 82% of cases. Subarachnoid hemorrhage was identified in 25 of 52 and was always seen in association with either subdural bleeding or fracture. Cortical contusions were present in five cases and these children were under 4.5 months old. Of the 38 cases in which the eyes were examined, 71% had retinal hemorrhages and all but one case were bilateral. The distribution of the retinal hemorrhages was not studied. Retinal hemorrhages had a significant association with subdural hemorrhage, all the retinal hemorrhages occurred in cases with subdural bleeding. The most common microscopic finding was global neuronal hypoxia-ischemia or VAI that was identified by the usual neuropathological criteria. VAI was present in 84% of infants and 63% of older children. B-APP staining identified patterns of VAI and dTAI and the authors noted that it is critical to distinguish between these two patterns because many children with these injuries suffer breathing abnormalities and sustain global hypoxia. Geddes described the VAI pattern as broad geographic areas of B-APP expression that was often related to vessels, whereas dTAI demonstrated B-APP expression in scattered small groups of axons in the hemispheric white matter, corpus callosum and internal capsule (Figures 7 and 8). Geddes found evidence of dTAI in only three of the 53 cases, an 8 year old boy and two infants both of

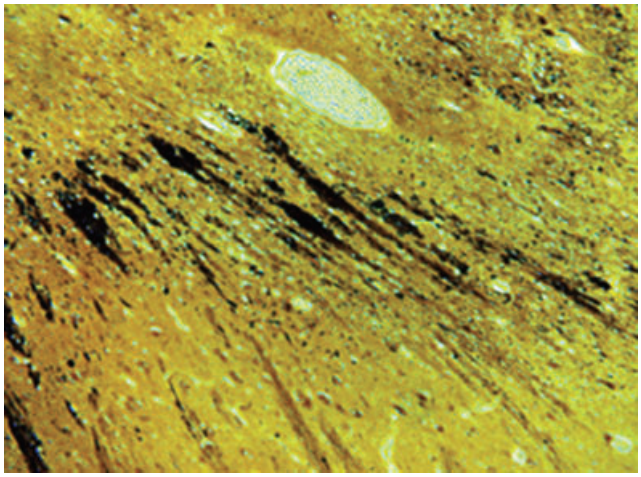

Figure 7. Beta-amyloid precursor protein expression in a vascular axonal injury pattern with large clusters of axons distributed around a vessel (100× magnification).

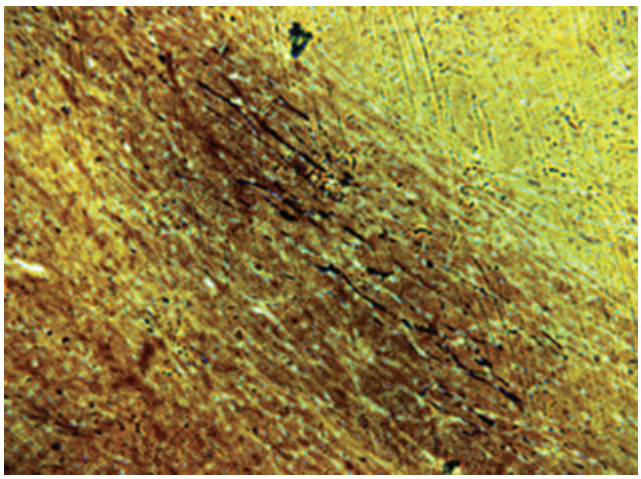

Figure 8. Beta-amyloid precursor protein expression in a diffuse traumatic axonal injury pattern with small numbers of scattered axons in the corpus callosum (100× magnification).

whom had severe head injuries with bilateral skull fractures. Of the 37 infants under 1 year old, Geddes found that five cases showed minimal axonal damage affecting only the corpus callosum or central white matter, and severe traumatic axonal damage in two of the cases. In eight of the cases, Geddes found damaged axonal bulbs in the corticospinal tracts of the medulla and pons. Geddes concluded that focal axonal damage to the craniocervical junction was probably more frequent and significant than dTAI. Geddes suggested that this damage was secondary to stretch injury from cervical hyperextension/flexion. Although the Geddes' studies suggest that severe dTAI is rare in infant inflicted neurotrauma, they do note that none of their control group of non-head injuried infants who also presented with respiratory abnormalities showed changes of VAI as did the group with inflicted head trauma. In some of their cases, Geddes found that both VAI and dTAI patterns existed together. Geddes noted that the interpretation of axonal damage is difficult and requires extensive sampling using large blocks of brain (32).

In a study similar to the Geddes' *et al* 2001 studies, Case examined 34 cases of inflicted neurotrauma in children from 1 month to 8 years (14). Eighteen or 52% were under 1 year old and the remaining cases were 13 months (three cases), 17 months (two cases), 18 months, 2 years (six cases), 3 years, 4 years, and 8 years (two cases). The 19 controls ranged from 5 weeks to 10 years old and 63% were under 1 year. All the cases of inflicted head injury presented unresponsive and apneic at the scene. Survivals ranged from dead at the scene to 12 days. The cause of death in all cases was complications of raised intracranial pressure. Other injuries in the inflicted head injury group included: multiple bruises—50%; burns—5%; rib fractures—20%; laceration of liver—5%; torn frenulum—5%; laceration of mesentery—2%; healing or healed skull fracture—5%; healed clavicle fracture—8%; and long bone fractures—29% (five were injuries in the humerus, one in the radius site, two in the femur, two in the tibia). The inflicted head injury group showed subgaleal contusions in 76% and acute skull fractures in 20%. The children with skull fractures were 4 months (two children), 6 months, 8 months, 17 months and 2 years old. The fractures were: parietal, frontal and occipital; frontal and parietal; occipital and parietal; and occipital. Subdural hemorrhage was found in 91% of the inflicted neurotrauma group. Retinal hemorrhages described as multiple layered and extending into the periphery of the retina were found in 80% of the eyes examined. Using the same sampling techniques, staining techniques and identification of VAI and dTAI patterns as used in the 2001 Geddes studies, the inflicted head injury group showed 27% with VAI pattern of B-APP expression and 73% with dTAI pattern of B-APP expression. In eight of the 11 cases in which specimens of cervical cord were available for study there was B-APP expression in the cervical cord and corticospinal tracts of the medulla. Two children had contusion tears; an 8 month old with a tear of the right inferior frontal gyrus and a 4 month old with a tear of the corpus callosum. The three children with inflicted head trauma without subdural hemorrhage were of interest. One was a 6-month-old with multiple bruises, fracture of the left humerus, scalp bruises, occipital and parietal skull fractures, retinal hemorrhages and microscopic evidence of dTAI. The aunt admitted to shaking and throwing the child. The second was an 8-week-old with retinal hemorrhages and microscopic evidence of dTAI. This child was found unresponsive with no history and later the father admitted to "killing" the child. The third was a 4-month-old with retinal hemorrhages and microscopic evidence of dTAI. This infant was found unresponsive with no further history. The mother later admitted to shaking the infant. In three other cases, confessions of the mechanisms of injury were made by caregivers and included the following: a 23-month-old with multiple bruises, fracture of right radius, scalp bruises, subdural hemorrhage and microscopic evidence of dTAI (eyes were unavailable for study) whom the mother's boyfriend admitted to severely shaking child; a 2-year-old with multiple bruises, scalp bruises, skull fracture, subdural hemorrhage (eyes unavailable for study) and microscopic evidence of dTAI whom the mother's boyfriend confessed to shaking and throwing child; and a 13-month-old with multiple bruises, healing fracture of left clavicle, scalp bruises, subdural hemorrhage, retinal hemorrhages and microscopic evidence of dTAI whom the mother was seen shaking the child. This study demonstrated a much higher incidence of microscopic evidence of dTAI in the children with inflicted head injuries than did the Geddes studies. In some of these cases, both patterns of VAI and dTAI were present together and could be distinguished. It seems very likely that many of these children with inflicted head injury have such global hypoxic damage that it overshadows much of the traumatic axonal pattern and makes it very difficult to recognize the traumatic component of the damage.

BRAIN SWELLING

Brain swelling is a common finding in inflicted head injury in young children who survive for some period of time. If the child is dead at the scene, the brain may show little swelling. The thin subdural hemorrhages seen in many of these head injuries does not readily explain the progressive brain swelling that is seen in most children who survive for some period. The swelling has been thought by some investigators to be due to hypoxia that follows the apnea, which commonly accompanies these injuries (48). Hypoxia does not fully explain the pathological findings such as the subdural hemorrhages, retinal hemorrhages and the pattern of atrophy that develops in surviving children. As noted in the Geddes' studies, their control group, who also presented with respiratory abnormalities, did not show changes of VAI as did the inflicted head injury group (33, 34).

EPIDURAL HEMORRHAGE OF CERVICAL SPINE

In 1967, Towbin observed epidural hemorrhages in the cervical spine of five children who had died suddenly and unexpectedly (80). He suggested that these findings might be traumatic and might be related to the deaths of these children by producing mechanical injury to the spinal cord. In 1969, Harris and Adelson described 19 infants under 1 year of age who had died suddenly and unexpectedly (44). Five of these infants were found to be dying of natural deaths from diseases noted at autopsy. Fourteen of the deaths were unexplained by findings at autopsy and were considered to be sudden infant death syndrome deaths. Eighteen of the 19 infants had epidural hemorrhages within the epidural spinal region that varied from venous congestion to hemorrhage within the adjacent soft tissues and the hemorrhage was greatest at the lower cervical region. They made three conclusions: (i) these epidural hemorrhages were not traumatic; (ii) the presence of blood was not

damaging to the spinal cord; and (iii) probably hemodynamic forces were responsible for this congestive phenomemon.

In 1989, Hadley *et al* described 13 infants from 1.5 to 14 months old who were diagnosed as shaken infants (41). Eight of these infants died and then six were autopsied. Of the six who were autopsied, 5/6 had epidural hemorrhages in the cervical spine, 4/6 had subdural hemorrhage in the cervical spine, 4/6 had contusions of the high cervical cord and 1/6 had none of these findings. Hadley's findings were of interest for several reasons. Subdural hemorrhage around the cervical spine cord would not be an unexpected finding in infants with abusive head trauma because intracranial subdural blood can descend by gravity into the spinal subdural space. Although Harris and Adelson had considered the question of epidural hemorrhages in the spinal region in infants dying suddenly and unexpectedly and concluded that these epidural hemorrhages were benign, their 1969 study preceded the time when the diagnosis of inflicted traumatic head injuries were being readily recognized (44). No one had specifically looked at infants with abusive head injury to determine whether those children had hemorrhage in the spinal epidural spaces. To elucidate the findings in the spinal area in young children with abusive head trauma, Case conducted a study from 1989 to 1992 of children under 3 years of age who died in a large metropolitan medical examiners' system (13). After examining 50 cases of inflicted head trauma and control cases, it was apparent that both groups frequently had spinal epidural hemorrhage and no relationship could be established between head injury and spinal epidural hemorrhage (Figure 9). Nevertheless, it is recommended that in all children under 3 years of age, the autopsy should include a posterior neck dissection, and examination of the vertebrae and spinal cord in all cases in which there is any type of head injury.

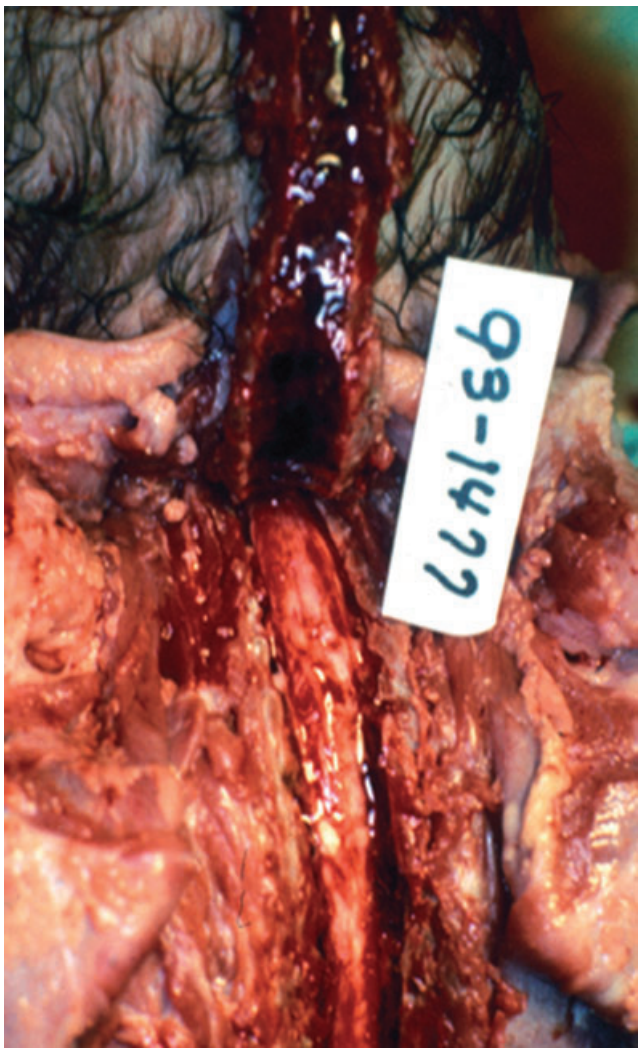

Figure 9. Posterior dissection of the spine to demonstrate extensive blood within the epidural space in a 3-month-old female infant dying from causes unrelated to head trauma.

DISTRACTION INJURY OF CERVICAL SPINE

Hadley's 1989 paper described six autopsied infants dying from inflicted head injury and found that 4/6 had contusions of the cervical cord (41). Hadley did not elaborate on the pathology of these contusions so it is difficult to know precisely whether they were injuries similar to those described by Geddes in some of their infants—who showed focal axonal injury in the cervical region—or whether they were truly contusional hemorrhages (33, 34). A very rare abusive injury of the cervical cord is a distraction injury of the cervical spine. In 1995, Priatt described a 15-month-old child who presented to the emergency department with quadriplegia after reportedly falling from a couch (72). The child had linear bruises in front of the right ear and jaw, showers of petechial hemorrhages over the left side of the neck and over both ears, the right side of the jaw and the upper chest. The child had older bruises at the corners of both eyes, on both arms and the right thigh. There was an old fracture of the right clavicle. MRI demonstrated fusiform swelling of the midcervical cord that represented hematomyelia. Two months later, MRI showed spinal cord atrophy and the child's quadriplegia persisted. Following that report, Parrish reported a case of isolated spinal cord injury in a case of child abuse (70). This child was a 2-month-old infant who had contusion of the upper cervical spinal cord and lower medulla. The eyes of the infant were studied at autopsy and found to have anterior chamber bleeding bilaterally, a dislocated left lens and vitreous hemorrhage. These injuries were thought to be the result of direct compressional force. It was theorized that the perpetrator had picked up the child by the head with the perpetrator's thumbs positioned over the child's eyes and violently shook the child's body at the same time grasping the head.

In the years since these reports, the author has seen three cases with findings that suggest a distraction mechanism of injury to the cervical cord and lower medulla (15). Two of the cases were siblings. In the first of those two children, the father described grasping the 3-month-old infant by the head and shaking her body. That child survived with a central cord syndrome. A second sibling was found dead at age 3 months and autopsy demonstrated petechial hemorrhages of the right bulbar conjunctiva and hematomyelia of the upper cervical cord and medulla. It was suspected that the father had performed a similar abusive act by grasping the head and shaking the child thus causing a distraction injury of the cervical cord and medulla. The third child was a 25-day-old infant who was

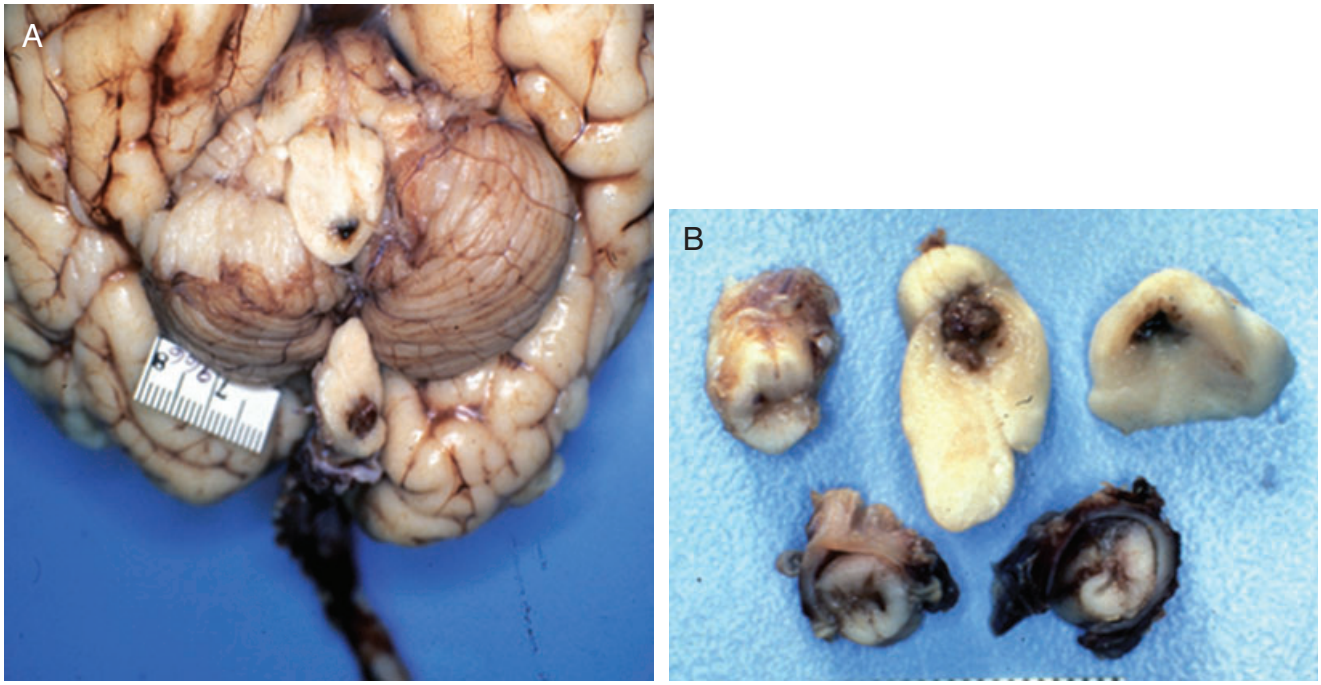

Figure 10. A. A 4-month-old male with distraction injury showing hemorrhagic lesions within the caudal medulla and upper cervical spinal cord. **B.** Sections of brainstem and cervical cord demonstrating the hemorrhagic lesions within the central portions of caudal medulla and upper cervical cord.

reported to have been found unresponsive in the early morning after having cried all night. He was dead on arrival in the emergency department. Autopsy demonstrated contusions of the right mandible, below the left lower lip, right shoulder, left abdomen, posterior left arm, right thigh and both legs. Several of the contusions on the extremities looked like gripping marks where the body had been held. There were petechial hemorrhages of the right preauricular area, left lateral forehead, and bulbar and palpebral conjunctivae. There was a single small retinal hemorrhage. There was fracture of the right clavicle with early callus. There was a separation of the C3–4 intervertebral disc and hematomyelia of the upper cervical cord and lower medulla (Figure 10A,B).

The mechanism of grasping a child by the head and then violently shaking the child's body is suggested by the findings in these rare cases. Grasping a child by the head would create distraction of the upper cervical spinal cord lower medullary region by the weight of the body that is unsupported when the child is suspended. Petechiae are frequently found on portions of an infant's skin that are forcefully grasped or struck. Rows or showers of petechiae are commonly seen in place of contusions in these areas from firm gripping.

There is an artifact that may resemble a cervical cord contusion or a distraction injury of the cord. Patients who are brain dead and not pronounced dead for a day or two after cerebral circulation cease will begin to show fragmentation of the cerebellar tonsils. As the tonsils disintegrate into the subarachnoid space, the material may fall downward into the cervical spinal cord subarachnoid space where it may compress small vessels and result in ischemia to the cervical cord. The resulting lesion is a hemorrhagic softening of the central cervical cord which resembles a contusion. Cerebellar detritus can be found within the subarachnoid space of the cervical cord in these cases. Identical lesions have been seen in experimental animal models of brain death (63).

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