Child Abuse

Pocket Atlas Series

Volume Three

Head Injuries

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A portion of our profits is contributed to nonprofit organizations dedicated to the prevention of child abuse and the care of victims of abuse and other children and family charities.

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Preface

As more communities work to develop effective methods for recognizing and treating victims of abusive head trauma, investigating cases, protecting victims from further harm, prosecuting offenders, and pursuing education and prevention efforts, there has been a growing interest in educating and training the professionals involved in all phases of response to this problem. The time has come to synthesize what we know, what questions remain, and what scientific studies still need to be done. It is time to share information in an organized manner among professionals working in the field in order to provide improved recognition, treatment, investigation, prosecution, education, and prevention of this deadly form of abuse.

This text is designed to serve as a reference for medical, investigative, legal, social service, and prevention professionals. All of these disciplines are affected by AHT in children and all have made notable progress in handling the results of child maltreatment in general. Prevention efforts have also been cultivated, focusing specifically on avoiding the development of patterns of child abuse within the family. The goal of educating all professionals is to help children and families with the corollary of improving society's concern and care for the most helpless of its citizens.

We have sought to offer a balanced approach to the problem of AHT while exploring current efforts and recommendations to address the concerns of professionals. It is hoped that this publication will become a reliable reference for professionals in the medical, investigative, legal, social service, and prevention areas.

Randell Alexander, MD, PhD
Robert N. Parrish, JD

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Contents in Detail

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Chapter 1

UNINTENTIONAL HEAD INJURIES

Todd C. Grey, MD

The patterns of injury seen in accidental lethal head trauma are striking. The typical findings in a case of immediately or rapidly fatal accidental head injury, in which the child is pronounced dead at the scene or within a short time of arriving at the hospital, have an array of cutaneous, skeletal, and intracranial findings. While the extent of injury in the various structural layers of the head may at times be discrepant, there is always something in the pattern and extent of injury that is indicative of a significant amount of force being delivered to the head. What is even more striking is the clear correlation between the severity of injury and the mechanism of injury provided in the history. The injuries present in the patient are reasonable given the explanation provided for these injuries, which is in sharp contrast to the often trivial mechanisms offered as an explanation for a child's injuries in cases of abusive trauma. The cases in this chapter are graphic in their presentation but serve to emphasize the dramatic and distinct nature of the injuries. It is also notable that tremendous forces are involved when accidental fatal head trauma occurs in the case of motor vehicle collisions, a horse falling on a child, or an adult falling down stairs and landing on a child.

Chapter 2

ABUSIVE HEAD TRAUMA

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Abusive head injury (AHT) has several synonyms including non-accidental head trauma or inflicted traumatic brain injury. Terms such as shaken baby syndrome and shake impact syndrome are often used as well, but they are not as inclusive as the terms aforementioned. The American Academy of Pediatrics noted in their policy statement that the intention of leaning away from terms such as shaken baby syndrome, "is not to detract from shaking as a mechanism of abusive head trauma but to broaden the terminology to account for the multitude of primary and secondary injuries that result from abusive head trauma."[1,2] Regardless of the label, abusive head trauma frequently results in serious and permanent brain damage. The forces to which the infants' brains are subjected tend to be severe. The prevalence of abusive head trauma is highest in children younger than 2 years of age, probably because the size of an older infant makes it difficult to create the extreme forces necessary to inflict such severe injury to the brain and its coverings. The incidence of abusive head trauma is estimated at approximately 14-30 per 100 000 children within the first year of life; the mean age of accidental injuries is 2.5 years whereas the abused are on average 0.7 years (8 months) old.[3-5] Abusive head trauma in infants is more common than all childhood cancers and type 1 diabetes.[1]

When evaluating abusive head trauma, it is best to consider each injury individually since it involves the internal layers of tissue as well as those surrounding the brain. While this is a logical approach to describing the injuries, it is important to recognize that multiple anatomical areas of injury are the rule, not the exception.

External to the brain, the scalp is often the site of a subgaleal hemorrhage after impact (**Figure 2-1**). Hemorrhage within the scalp creates the proverbial “egg” on the head. The subgaleal space is a large potential space; therefore, the blood often flows into a dependent region. This explains why the palpable or visible bump is not always in the region of the trauma. Unless the child has a bleeding disorder or some other abnormality, the presence of a subgaleal hematoma always suggests that there was an impact injury. There is another, less common variant of scalp injury: the cephalhematoma, which is a hemorrhage in the subperiosteal space, external to the bone but localized anatomically to the bone since it is confined by the periosteal layer of each bone of the skull. Cephalhematomas are rarely seen in child abuse and always remain local to the area of hemorrhage or impact.

A patient presenting with skull fracture (**Figure 2-2**), shows evidence of significant traumatic injury; however, injuries following uncomplicated normal vaginal delivery have (rarely) included skull fractures. A tender soft tissue swelling associated with such an injury points to a recent impact, but often injuries such as cephalohematoma will take time to become evident and resolve over the course of several weeks.[6]

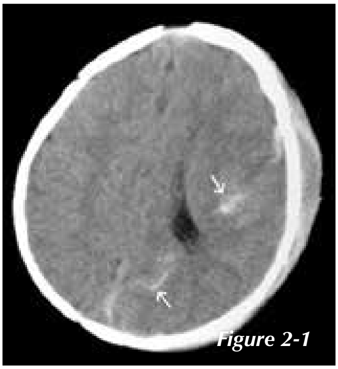
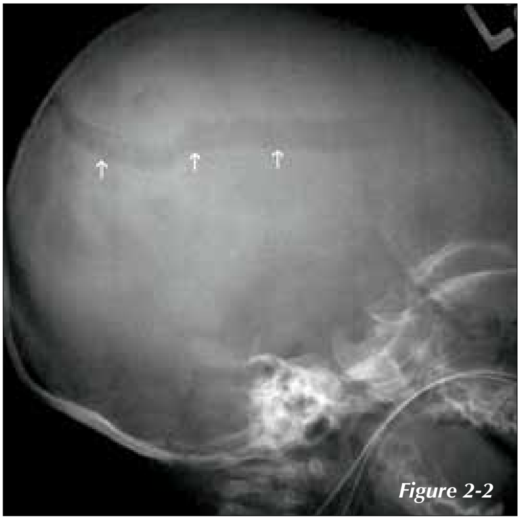


Figure 2-1. *Multifocal contusions involving the left frontal and parietal lobe with evidence of subaracnoid hemorrhage and subgaleal hematoma (white arrow).*

Figure 2-2. *Lateral view of the skull demonstrates linear diastatic parietal fracture.*



As a whole, young patients who present to a health care provider with traumatic injuries often have common events in their history, which often include a recount of a "short fall" (>90cm), falling off a couch, etc. Height from fall, however, is often an inaccurate estimate on the part of parents and the most reliable estimate of short falls are in-hospital falls.[7] Analysis of short falls by Helfer et al, compared the results of short fall events in the hospital versus at home. In the home group (n=176) there were 2 skull fractures whereas in the hospital (n=57) there was only 1 such fracture. None of these fractures was diastatic or defined as greater than 1 mm in width (**Figure 2-2**). No children suffered neurological complications as a result of this head injury. The best current estimate of mortality for short falls affecting infants and children is near zero.[7,8]

Certain fractures are found to occur significantly more often in AHT, these include: multiple or complex fractures, depressed or wide diastatic fractures, those with involvement of more than one bone and those involving other than the parietal bone.[6,9] Skull fractures typically associated with abusive head trauma are similar to those due to high velocity impact. These fractures are long (longer than 5 cm), stellate (many limbs from one point of impact), or diastatic (the edges of the fracture are widely spread). It is possible to have skull fracture from a short fall and in rare cases, some overlap of features between high impact and short fall injuries may occur; however, the presence of long, stellate, or diastatic fractures should lead to enhanced suspicion if they are ascribed to a short fall.

The epidural hematoma is an unusual injury in child abuse (**Figure 2-3**). This type of hematoma occurs because of bleeding, usually arterial, into the epidural space between the inner table of the skull and the dura mater. This lesion is classically associated with a lucid interval and skull fracture. The theory of the lucid interval is that the initial impact causes the fracture and concussion, rendering the victim unconscious. The subsequent bleeding from ruptured branches of the middle meningeal artery then causes the epideral hematoma, which grows rapidly, owing to arterial (as opposed to venous) bleeding, and causes further deterioration of mental status after the patient stabilizes from the concussion.

The subdural hematoma (SDH) is a hallmark of abusive head

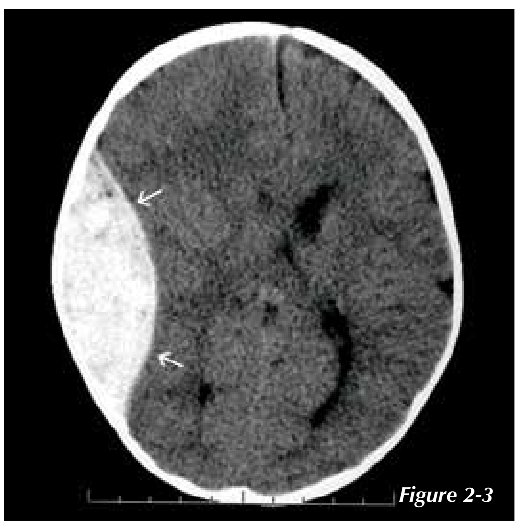
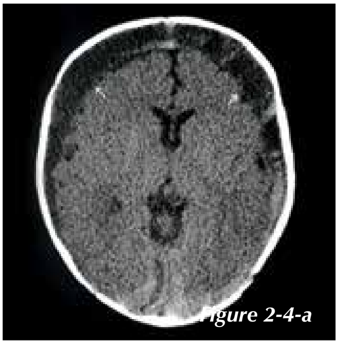


Figure 2-3. *Axial CT of the head demonstrates right frontal epidural hematoma with mass effect and midline shift to the left side.*

injury and is the most frequently diagnosed intracranial injury in child abuse. More specifically, subdural supratentorial convexity and interhemispheric SDH are seen significantly more often in nonaccidental head injury. SDH in accidental injury are uncommon, but when it does occur it appears to be focal and adjacent to the site of impact.[10] Bleeding in the subdural space occurs because of a rupture of the bridging veins that drain blood from the surface of the brain to the dural venous sinuses. The principal route of drainage of surface veins is to the sagittal sinus. As a result, subdural hematomas due to child abuse most often occur over the convexities of the parietal, frontal, and occipital lobes (**Figures 2-4-a** and **b**). Frequently, subdural hematomas can be identified as new or old depending upon the characteristics of the blood degradation products on a computed tomographic (CT) scan or magnetic resonance imaging (MRI) scan (**Figure 2-5**). The relative insensitivity of CT scans for definition of anatomical spaces has led to some confusion in older literature, particularly regarding benign subdural hygromas, most of which are merely enlarged or prominent subarachnoid spaces, and are of no clinical or pathological significance.

Bleeding into the subarachnoid space occurs when the vessels are ruptured between the arachnoid membrane and the pia mater. The subarachnoid space readily communicates with the cerebrospinal fluid (CSF) cisterns and the spinal subarachnoid space. Blood obtained on spinal taps in abused children can be used to indicate subarachnoid hemorrhages (SAH). Subarachnoid hemorrhages can be identified on imaging by bleeding into the cerebrospinal fluid cisterns surrounding the brain or by a serpiginous, gyriform pattern of hemorrhage. Subarachnoid blood also accumulates along the cerebral tentorium or within the thecal sack over the spine. Subarachnoid hemorrhages are very important clinically because there is almost universal agreement among experts that they have distinct symptoms. Adults with subarachnoid hemorrhages, most commonly victims of rupture of an intracranial aneurysm, describe a typical “thunderclap” headache as the worst of their lives. In infants, the symptoms manifest as extreme irritability, discomfort, and pain. An infant with a subarachnoid hemorrhage is highly unlikely to act normally.

Parenchymal injuries to the brain include both bland and hemorrhagic contusions. There are injuries to the surface of the brain from an impact mechanism similar to a contusion elevation in the body. There is a tendency for the brain to suffer contrecoup injuries, an injury opposite the side of impact. The contrecoup injury is usually larger than the direct impact injury. The other characteristic hemorrhagic injury to the brain is the ***diffuse axonal injury*** (DAI), an injury to the axons of the neurons that has a prepovdicean at areas of differing physical density in the brain, such as the watershed areas along the cortex or the deep gray and pericollosal white matter areas. Parenchymal injuries of the brain tend to be rapidly and severely symptomatic (**Figure 2-6-a** to **c**).



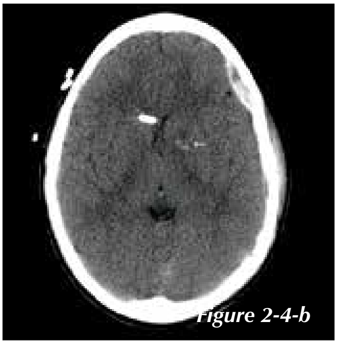
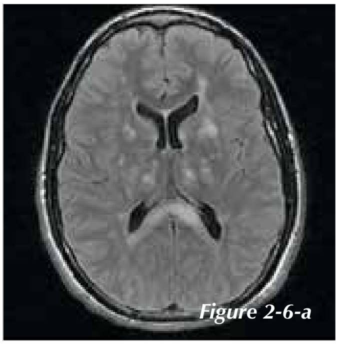
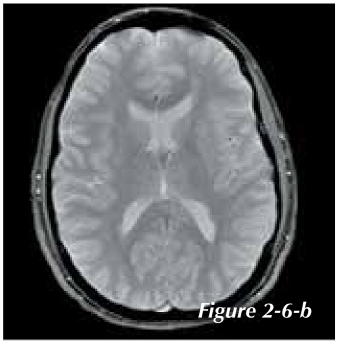


Figure 2-4-a. *Axial CT of the head shows acute and chronic bilateral subdural hematoma.*

Figure 2-4-b. *Small left frontal acute subdural hematoma (black arrow) with hemorrhagic shearing injury to left internal capsule (white arrow).*





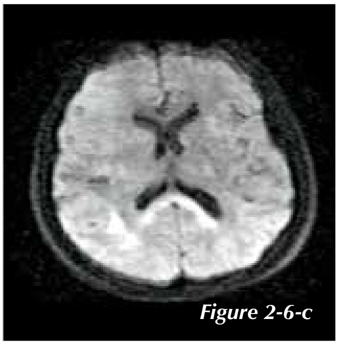


Figure 2-5. *GRE T2W image shows large bilateral chronic subdural hematoma with new acute subdural hemorrhage on the left (black arrow) with blood sediment level.*

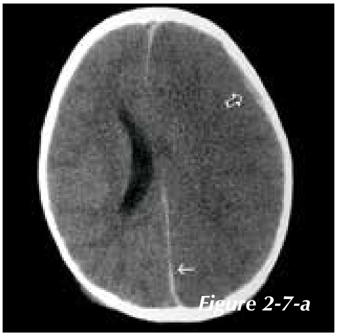
Figure 2-6-a. *Axial flair T2W image shows multiple foci of shearing injury.*

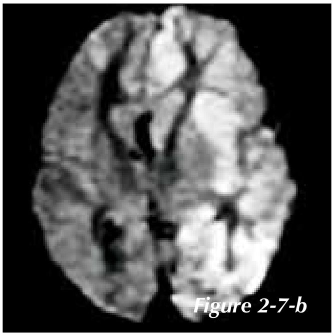
Figure 2-6-b. *Axial GRE T2W images obtained immediately after trauma demonstrates no obvious evidence of hemorrhage.*

Figure 2-6-c. *Axial DWI image of the brain shows diffusion restriction along the corpus callosum (black arrow) (shearing injury) and right posterior parietal subcortical white matter (white arrow) (contusion).*

The final serious injury of the brain ascribable to child abuse is hypoxic ischemic injury. This injury occurs due to a complex interaction of events that leads to either a lack of perfusion of brain tissue or a lack of sufficient oxygenation of the blood perfusing the brain tissue (**Figure 2-7-a** and **b**). As the brain tissue begins to die, a complex event called a neuronal cascade begins, further increasing intracranial pressure and compromising both blood flow and oxygen delivery. The visible result is cerebral edema, which, in its extreme, results in a pattern of injury known as the "bad black brain" or "reversal sign" (**Figure 2-8**). In this imaging picture the structures of the brain are obscured and the ventricles are often compressed due to the increased intracranial pressure. This results in an extremely poor prognosis.

Type of injury, age, and presentation of the patient help to determine the best mode of imaging to perform. Children with skull fractures, clinical abnormalities, and symptoms of intracranial injury should be evaluated with an immediate noncontrast CT scan of the head. If this CT does not indicate a lesion requiring immediate neurosurgical intervention, and the clinical presentation requires further analysis thus an MRI scan of the head should be performed. This MRI series should include T1-weighted and T2-weighted sequences with inversion recovery and gradient echo sequences (**Figure 2-9**). In addition, diffusion-weighted sequences (**Figure 2-10-a**) help to elucidate the presence of acute cerebral injury. Additional MR





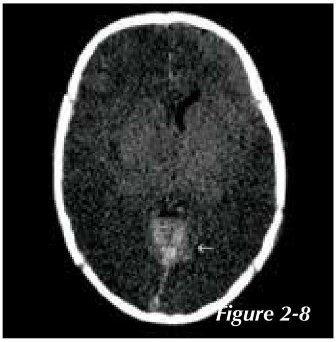


Figure 2-7-a. *Axial CT of head shows small left frontal and interhemispheric acute subdural hematoma with mass effect.*

Figure 2-7-b. *Diffusion-weighted image demonstrates extensive diffusion restriction secondary to likely combination of shearing injury and hemispheric infarct of left frontal temporal parietal lobes.*

Figure 2-8. *Diffuse low attenuation involving the bilateral frontal temporal parietal gray and white matter secondary to nonaccidental trauma (reversal sign).*

spectroscopy (**Figure 2-10-b**), MRA of the circle of Willis, and MRV of the dural venous sinuses will be helpful to include in the protocol and should be strongly considered. Diffusion tensor imaging is a new and promising application of MRI, which is a form of DWI and help better evaluation of white matter tracts on the bases of intrinsic directionality (anisotropy) of water diffusion in brain. Perfusion CT (**Figure 2-11**) and MRI is also important potential application of advanced neuroimaging in AHT, which allow us to understand underlying vascular injury secondary to AHT.

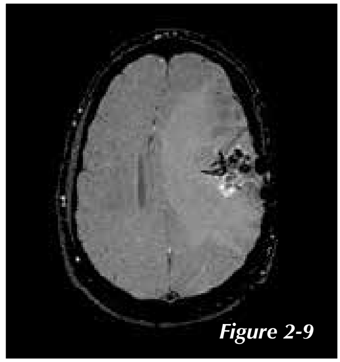


Figure 2-9

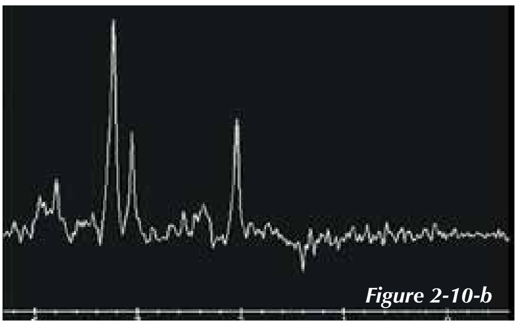


Figure 2-10-b

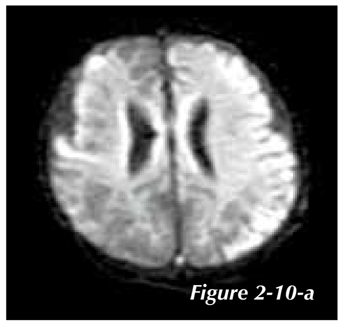


Figure 2-10-a

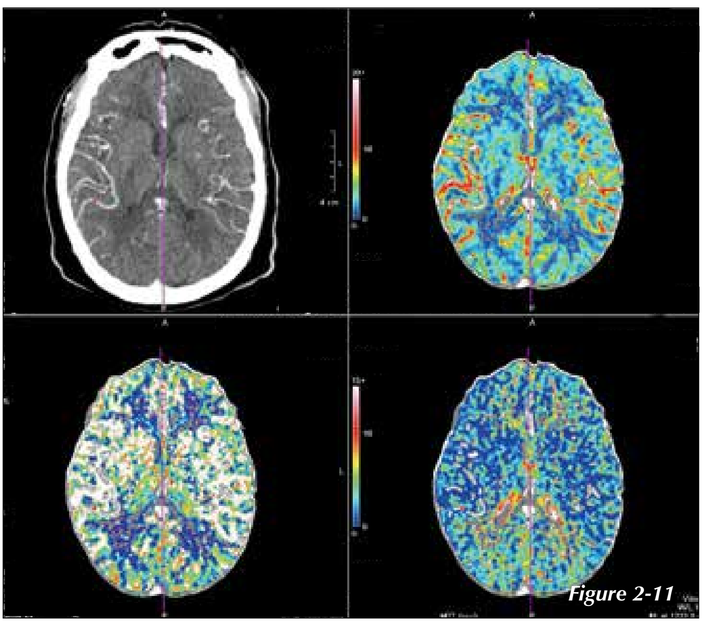


Figure 2-11

Figure 2-11. *CT perfusion image after single I.V. injection of contrast with volumetric scanner shows cerebral blood volume, cerebral blood flow (ml/100g/min) and mean transit time.*

Figure 2-9. *Susceptibility-weighted image shows evidence of blood.*

Figure 2-10-a. *DWI (diffusion-weighted Image) shows citotoxic edema (white) within the left cerebral hemisphere.*

Figure 2-10-b. *MR Spectroscopy obtained from left cerebral hemisphere shows evidence of lactate (arrow) in a patient with non-accidental trauma; this indicates poor prognosis.*

INDEX

F

G

H

I