

Rachel P. Berger and Michael J. Bell

Abstract

Abusive head trauma (AHT) (e.g. shaken baby syndrome) is the leading cause of death from child abuse. Proper diagnosis of AHT is critical; if AHT is not identified, children can be inadvertently returned to a violent environment where they can be re-injured or killed. The intensivist plays a critical role in the identification, evaluation, and treatment of AHT. This chapter will focus on the clinical presentation of AHT, the medical evaluation for cranial and non-cranial injuries in cases of suspected AHT as well as the management and treatment of AHT with a focus on the differences between management of children with AHT vs. non-abusive TBI. Current data related to the mechanism of injury and pathophysiology of AHT will also be discussed. Finally, issues related to mandated reporting and legal proceedings related to AHT cases will be discussed as will the role of the intensivist in all of the above.

Keywords

Child abuse • Abusive head trauma • Retinal hemorrhage

Introduction

Abusive head trauma (AHT), defined as traumatic brain injury which is the result of child abuse, is the leading cause of morbidity and mortality from traumatic brain injury in infants and young children [1–3]. AHT is also the leading cause of death from child abuse. The rate of severe or fatal AHT is approximately 1 in 3,300 infants <1 year of age [2], although unpublished data from a multi-center AHT study suggests that severe or fatal cases may comprise only half of

the total number of AHT cases (Rachel Berger, unpublished data). Even at a rate of 1 in 3,300, AHT is far more prevalent than other diseases of childhood which we often consider to be “common.” For example, the incidence of acute leukemia, the most common childhood cancer, is approximately 1 in 28,000, almost 10 times lower than the rate of AHT in infants [4].

While severe and fatal cases of AHT are the ones more frequently treated by the pediatric intensivist, it is critical to recognize and understand the full spectrum of injury severity in AHT in order to better understand issues of biomechanics and pathophysiology, as well as the spectrum of clinical presentation, intracranial injuries, ophthalmologic findings, and orthopedic injuries. Because of the intense social and legal ramifications of making a diagnosis of AHT, the overall care of a child with possible AHT can be more complex than the care of children with non-abusive TBI. Specifically, standard medical care for children with suspected AHT includes photographic documentation, screening for non-cranial injuries, evaluation and testing for alternative diagnoses, and reporting to Child Protective Services (CPS) – as well as all

R.P. Berger, MD, MPH (✉)
Department of Pediatrics,
Children’s Hospital of Pittsburgh of UPMC,
4401 Penn Avenue, Lawrenceville Medical Building,
Pittsburgh, PA 15224, USA
e-mail: rachel.berger@chp.edu

M.J. Bell, MD, MPH
Critical Care Medicine, University of Pittsburgh,
3434 Fifth Avenue, Pittsburgh, PA 15260, USA
e-mail: bellmj4@upmc.edu

of the care required to take care of the multiply traumatized child outlined in other chapters of this text. The goal of this chapter is, therefore, to provide the pediatric intensivist with an understanding of medical, social and legal issues related to AHT.

Terminology

In 2009, the American Academy of Pediatrics published a policy statement changing the official name from ‘shaken baby syndrome’ to abusive head trauma (AHT) [5]. While the previous term implied a single injury mechanism – shaking – the new term takes into account improvements in our understanding of the injury in AHT which may include a combination of shaking, blunt impact, spinal cord injury, and hypoxic ischemic injury. The term AHT is focused on the etiology of the injury – abuse – rather than the injury mechanism. While the previous definition only included children who had been shaken, the term AHT also includes children, for example, who sustain TBI during a domestic dispute and children with an isolated impact injury (e.g. child hit over the head with a baseball bat by an angry adult). Shaken baby syndrome is, therefore, a subtype of AHT.

Epidemiology

AHT has traditionally been thought of as a disease of infants. And while infants are at greatest risk, AHT can also occur in toddlers and even older children [6, 7]. For example, in a recent multi-center study of more than 400 children with AHT, 24 % were older than 1 year of age [8]. Interestingly, while children greater than 1 year made up only 24 % of the study population, they accounted for 41 % of the deaths in this multi-center study; the higher mortality rate in older children was also reported among AHT cases in Pennsylvania [9]. Recognizing that AHT can occur in children up to 5 or even 6 year of age is important for the pediatric intensivist; AHT should be part of the differential diagnosis whenever caring for a child whose injuries seem out of proportion to the history provided or when an infant or young children’s symptoms cannot be well-explained. While there are demographic, social, child, and parental risk factors for AHT, these risk factors clearly cannot be used to diagnose AHT, [10–13] cases of AHT clearly occur in children with no recognized risk factors. Therefore, while the presence of one or more risk factors should alert the treating physician that an infant or young child may be at increased risk of AHT, the lack of these risks factors cannot be used to eliminate AHT from the list of differential diagnoses.

Clinical Presentation

The clinical presentation of children with AHT can be quite varied, ranging from non-specific symptoms such as irritability or vomiting to extreme cardiorespiratory instability and acute herniation syndromes. In contrast to other disorders that present as critical illnesses, the reliability of the history provided by the caregiver in cases of AHT is always suspect, making the formation of an appropriate diagnosis more difficult. Specifically, in the majority of cases, the caretaker who is providing the medical history does not give the physician any history of trauma [14, 15]. While caretakers may purposely be evasive or lie, it is perhaps more frequent that the caretaker providing the medical history is not the perpetrator and may not know that the child has been abused.

The physical examination, including the neurologic components, can be normal in children with AHT. While bruising can be seen in a subset of children with AHT, children whose primary injury is an acceleration-deceleration injury (e.g. shaking) without an impact would not be expected to have external signs of injury. Even in cases in which there is impact, the impact may be against a soft surface (e.g., a couch) or may not be significant enough to cause a contact injury. Autopsy studies have demonstrated that some infants without bruising on physical examination have signs of impact injury which is only visible when the scalp is retracted [16, 17].

The importance of a complete dermatologic examination in children with suspected AHT cannot be overemphasized. While bruises (particularly of the ears and face), petechiae, and abrasions have little clinical significance and do not require treatment, they can be important for diagnosis. Completing and documenting the result of a comprehensive physical examination is critical so that it is clear which injuries were present upon arrival to the hospital (e.g. did not occur as part of medical care); this is particularly important when children are taken to the operating room for a neurosurgical intervention. Even in hospitals with a Child Protection Team (CPT), the child abuse physician often does not examine the child for several hours after admission. The initial examination by the CPT physician is therefore often after resuscitation and neurosurgical intervention. Injuries such as forehead bruising, for example, often cannot be seen after operative procedures; early and accurate documentation is therefore important. Bruising to the forehead, for example provides evidence of impact which can be critical for diagnosis and possibly for subsequent legal proceedings.

While the lack of a history of trauma and/or a lack of dermatologic findings can be barriers to proper diagnosis of AHT, perhaps the greatest barrier to diagnosis of AHT is the fact that infants and young children with subdural hemorrhages and/or other intracranial injuries can be neurologically

normal or have only non-specific neurologic signs such as irritability. In a classic study by Greenes and Schutzman [18], 19 % of children less than 2 years of age who had a subdural hemorrhage, cerebral edema or cerebral contusions due to abusive or non-abusive injury had a GCS score of 15. Therefore, the challenge for the pediatric intensivist is to recognize AHT when presented with a history which is inherently unreliable and a physical examination that is normal or nonspecific. In order minimize missing cases of AHT (which may prove fatal to the child), it is prudent to consider AHT in the differential diagnosis of any infant or young children (1) with an abnormal head CT where it is unclear whether the mechanism provided by the caretaker explains all of the child's injuries and (2) who has not had a head CT performed and in whom the etiology of the symptoms is not yet clear (e.g. an infant with apnea or a young child with altered mental status).

Mechanism of Injury and Pathophysiology

The mechanism of injury in AHT is among the most controversial issues in all of pediatrics. In his seminal paper, Caffey described the unexplained occurrence of 23 long bone fractures in six children who also demonstrated subdural hematomas. In these cases, a nursemaid admitted to shaking the victims while holding them by the arms and trunk [19, 20]. Since that time, there have been multiple studies which support the hypothesis that shaking is an important mechanism of injury in many cases of AHT [7, 21, 22]. In a recently published international population-based study by Runyan and colleagues, more than 20 % of parents admitted to shaking a child <2 year of age [23], suggesting that shaking may be a more common practice than previously thought. The prevalence of extensive multi-later retinal hemorrhages in many cases of suspected AHT also supports a shaking-type mechanism; retinal hemorrhages rarely occur in even severe non-abusive TBI and when present, do not have the same characteristics as the retinal hemorrhages in AHT [24, 25].

While shaking is likely an important mechanism of injury, in a significant proportion of cases, there is also evidence of an impact to the head based on (i) physical examination, (ii) radiologic evaluation and/or (iii) autopsy. The relative contribution of impact and shaking to the clinical symptoms in AHT has been an area of intense discussion. There are also data which support the importance of hypoxemia in AHT and several studies have suggested that the hypoxic-type injury in AHT may be more fundamental to outcome than direct trauma to the brain/skull [26–29].

The importance of cervical spine injury in the pathophysiology of AHT has also been the subject of debate. Early

studies by Shannon and colleagues and others suggest that injury to the cervical spine is common in fatal cases of AHT [30, 31]. More recent studies suggest that spine injury in non-fatal cases of AHT may be far more common than previously thought [32–35]; in a study by Choudhary and colleagues [00], spinal canal subdural hemorrhage was present in more than 60 % of children with AHT compared with only 1 % of children with accidental TBI. Even prior to the Choudary study, a review article by Kemp and colleagues suggest that consideration be given to performing a spine MRI in all cases of AHT [36].

Overall, our understanding of the pathophysiology of AHT has been developed from many years of clinical observations [7, 37–39], histopathologic data from children with fatal injuries [17, 30, 31, 40], confessions of perpetrators [21, 22, 41, 42], and more recently, cases of AHT accidentally caught on 'Nanny-cams'. There has also been a significant amount of progress over the past 10 years in the ability to model the injuries in AHT using a combination of animal models, human and animal tissue models, finite element analysis, anthropomorphic dummies, and computer simulation [43–52]. Although an in-depth discussion of these studies is beyond the scope of this chapter, the interested reader is referred to two excellent articles [53, 54].

The pathophysiology of AHT is complex and multifaceted and likely different in each patient. While improving our understanding of the pathophysiology of AHT is important and has improved significantly over the past 10 years, the focus for the clinical intensivist should not be on specific mechanism of injury in each case. Rather, the focus should be on the fact that the child's injuries were caused by an abusive act which was perpetrated by an adult.

Evaluation of Suspected AHT

In many pediatric hospitals in which children with suspected AHT are treated, there is a CPT, a multi-disciplinary team which evaluates cases of suspected child maltreatment and which often includes a board-certified child abuse physician or another physician with expertise in child abuse. The role of the CPT physician is often to provide recommendations related to evaluation for abuse, provide information to CPS about the child's injuries and the likelihood of abuse, and to be in charge of communication between the family, medical personnel, CPS and police as it relates to the abuse-specific issues. In hospitals without a CPT, the pediatric intensivist is often the physician who speaks with both families and CPS about abuse; in these situations, the intensivist must be able to do a comprehensive evaluation for abuse and relay the appropriate level of concern to others. It is important to remember

that CPS is ultimately responsible for the protection of a child who has been abused. If the level of concern about the likelihood of abuse is not properly relayed to CPS, a child may be placed back into a violent environment. We recommend that the evaluation of AHT be thought of as three evaluations: the evaluation of the cranial injuries, the evaluation for non-cranial injuries and the evaluation by CPS which is done based on the information from the first two evaluations.

Intracranial Injuries

Our understanding of intracranial injuries after TBI has improved with the increased availability and sophistication of MRI imaging. As is described within other chapters of this text, newer MRI techniques are now capable of identifying subtle white matter injuries that have previously gone unrecognized. This may be particularly relevant in AHT as diffuse injuries have not been well-recognized in previous studies. In addition to obtaining information regarding white matter injuries, MRI is also helpful in evaluating whether extra-axial collections contain blood products (e.g. chronic SDH) or not.

Within the last decades, it has been recognized that the end-stage of some cases of AHT can be severe loss of cortical and subcortical matter – the so-called “Big Black Brain” or multicystic encephalomalacia (MCE). MCE is a well-recognized phenomenon in the neonatal period and neonatal MCE is thought to be the result of a hypoxic-ischemic event. This has led others to hypothesize that unrecognized hypoxia may be an important contributor in cases of AHT which result in MCE. Animal models [55, 56] as well as clinical experience also suggest that a period of maturational vulnerability at the time of injury may also be important. [57, 58]

Extra-Cranial Injuries

The diagnosis of AHT is rarely based solely on the brain injury itself. With a few exceptions, the brain injuries discussed above are not specific to AHT. The combination of the brain injury and the non-cranial injuries in a patient without a history which adequately explains them is what defines AHT. As a result, identifying the non-cranial injuries can be critical in making the diagnosis of abuse.

Fractures

Up to 50 % of children with AHT will have either an acute or healing non-cranial fracture [59, 60]. In the vast majority of cases, these fractures cannot be diagnosed by physical examination alone. A complete skeletal survey ideally including oblique rib films [61, 62] should be performed whenever AHT is part of the differential diagnosis. In addition, a follow-up skeletal survey [63, 64] should be performed

10–14 days after the initial skeletal survey to assess for fractures which can be difficult to visualize in the acute setting.

Retinal Hemorrhages

A dilated ophthalmologic examination should be performed as soon as possible by an experienced pediatric ophthalmologist. While retinal hemorrhages are not specific for AHT and occur in about 10 % of cases of non-abusive TBI, certain patterns of retinal hemorrhages are highly-specific for AHT [65, 66]. Retinal hemorrhages which are multi-layered and/or extend beyond the periphery are almost unique to AHT. In the absence of significant direct trauma to the eye (e.g. crush injury), retinoschisis is virtually pathognomonic of AHT [24, 67]. In children with severe AHT, a dilated ophthalmologic examination is often not possible in the acute setting because of concern that the mydriatic will interfere with assessment of the pupillary response. The simplest solution to this problem is to request that the ophthalmologist perform an initial, non-dilated exam. Although the view of the retina will be limited to the posterior pole, a non-dilated exam can provide preliminary information about the presence or absence of retinal hemorrhages and a sense of how extensive they are. This can be particularly helpful in cases in which the possibility of AHT is raised, but the concern may not be high enough to make a report to authorities (e.g. there is a history of a fall, but the brain injury seems out of proportion to the history). If there are other young children in the home, timely reporting to CPS is especially important; though the child in the PICU is safe from abuse, other children in the home may still be with the perpetrator. An alternative to an undilated examination is serial dilation of the pupils.

Abdominal Injury

Although it is rarer than AHT, abdominal injuries are the second leading cause of death from abuse [68]. All children being evaluated for AHT should have liver function tests, amylase and lipase. A recent multi-center study evaluating the use of these screening labs suggested a low threshold for obtaining an abdominal CT [69].

Evaluation for Bleeding Disorders

The concern about whether a bleeding disorder could account for the intracranial hemorrhage children with AHT needs to be a consideration in certain cases. Recent clinical and technical reports from the American Academy of Pediatrics provide recommendations for which children with suspected AHT should undergo testing for bleeding disorder and what that testing should be [70, 71]. Briefly, the recommendation is that a CBC with platelets, PT/PTT, Factor VIII, Factor IX, d-dimer and fibrinogen be measured when a bleeding disorder is being considered and that a hematologist become involved if any testing is abnormal. Testing may not be needed when there are other medical findings consistent with abuse (e.g. fractures). The details of these reports are beyond

the scope of this chapter; the reports should be considered required reading for any physicians who evaluates children with suspected AHT.

Evaluation for Disorders Which Can Mimic AHT

The diagnosis of AHT is only occasionally obvious from the outset and AHT is often part of the differential diagnosis for a many infants and young children with TBI. As with every disease, identifying the correct diagnosis is paramount importance to patient care – in cases of AHT, there are additional social and legal implications of the making a diagnosis. There are very few other diagnoses which can result in children being removed from their parents home and/or people going to jail. There are also very few other diagnoses which, if missed, can result in a child being killed. Both sensitivity and specificity are therefore critical; while one does not want a child to be removed from a non-abusive home, one also does not want to return a child to an abusive one.

The most common differential diagnosis is non-abusive TBI. In about 50 % of cases of AHT, the caretaker provides a history of trauma as the explanation for the child's injuries; the issue is whether this history can account for the child's symptoms as well as the constellation of cranial and non-cranial injuries. It is incumbent upon the physician who is evaluating the child to be cognizant of the extensive literature related to injuries in short falls and stair falls, a common history provided by caretakers of children in whom AHT is part of the differential diagnosis [72–75]. In addition to the knowledge of the literature, one should not underestimate the importance of clinical experience. The pediatric intensivist is in the unique position of also evaluating children with non-abusive TBI; assessment of children with non-abusive TBI can provide important information which can be used when assessing children with possible abuse. It can be very instructive, for example, to listen carefully to the histories provided in cases of non-abusive TBI, specifically, the level of detail which the caretaker provides and the consistency with which he/she provides it. It can also be helpful to look at the non-cranial injuries sustained by children with non-abusive TBI – the number and location of bruises (particularly in premobile infants), the prevalence of acute or healing non-cranial fractures or a chronic SDH, and the number and type of retinal hemorrhages. Using this type of evaluation in cases of non-abusive TBI allows the intensivist to better assess the likelihood of abuse in cases which may be due to AHT.

Aside from non-abusive TBI, it is important to consider whether there might be a non-traumatic cause for a child's medical findings. These non-traumatic etiologies are often referred to as 'mimics'. A mimic is defined by Webster's dictionary as "something which closely resembles something else." The most common mimics of AHT discussed in the literature are glutaric aciduria type I [76], hemophagocytic lymphohistiocytosis [76–78] hemorrhagic disease of the

newborn [79] and arteriovenous malformations [80]. While these diseases can resemble AHT, they rarely, if ever, share all its characteristics. It is important that the pediatric intensivist consider these mimics and in some cases, it is important to test for these mimics. In most cases, however, the clinical presentation and/or injuries are inconsistent with the mimic (e.g. multiple metaphyseal fractures in hemorrhagic disease of the newborn). When a mimic is strongly being considered and there are no other children in the home, then filing a report with CPS can sometimes wait until if the additional data can be obtained within a day or two. If there are other children in the home, however, reporting should not be delayed since the other children in the home need to be evaluated to ensure their safety; the presence or absence of abusive injuries in contact children can provide important information about the probability of abuse in the index child.

Management and Treatment of AHT

The overall management and treatment of children who have suffered AHT is not significantly different from children with non-abusive TBI. However, given the relatively younger age population (and consequently smaller physical size), performance of some of the interventional procedures for the AHT population may be more challenging. EMS should be activated as soon as it is recognized that the child may be injured. Once the child arrives at a trauma center, assessment of airway, breathing and circulation (the "ABCs") is an essential part of the primary survey. A secondary survey, based on Advanced Trauma Life Support Guidelines, should then be performed to assess for systemic conditions and neurological injuries. The "gold standard" for neurological assessment is the Glasgow Coma Scale (GCS) score. Though there are various adaptations of the GCS score to account for developmental age [81], none have been sufficiently validated as measures of disease severity or as prognostic of outcome and thus the GCS remains the gold standard.

Mild and moderate AHT is generally treated expectantly with supportive measures, essentially to avoid secondary insults (e.g. hypoxia, hypotension, seizures, and hyperthermia). For severe AHT, management from a comprehensive team that includes trauma surgeons, neurosurgeons, intensivists and others is essential. In 2003, evidenced-based guidelines for the medical management of severe TBI in children were published [82]. While this document represents a synthesis of the TBI literature, none of the articles within the document is specific to AHT. In addition, the guidelines include children across the entire age spectrum – therefore, most of the literature includes a subset of children with AHT within a much larger population of children with non-abusive TBI. Despite these limitations, this document represents the best current evidence for caring for children with all types of TBI.

As with children with non-abusive TBI, AHT can lead to intracranial hypertension if the compensatory mechanisms to maintain the volume/pressure relationship within the cranium are overcome. Despite the lack of ossification of the skull of young children (and the presence of membranous fontanels for the first 18 months of life), critical intracranial hypertension leading to cerebral herniation can be observed in children of all ages. Therefore, minimizing intracranial pressure (ICP) and maintaining cerebral perfusion pressure (CPP) is a mainstay of neurocritical care for both non-abusive and AHT. Recent data from a study of young children many of whom had AHT suggest that maintenance of CPP may be more important than ICP control in this population [83].

After a rigorous resuscitation and assessment outlined above, prompt evacuation of extra-axial hematomas that are causing disturbances in cerebrohemodynamics and placement of ICP monitors are the next essential steps. This may be accomplished in the operating room with a basic craniotomy for simple evacuation of a minimal collection of blood or may require a larger craniectomy to decompress the brain. Insertion of an ICP monitor may occur at the time of the surgical procedure or after admission to the PICU. Intraparenchymal monitors or externalized ventricular drains are essential to detect periods of intracranial hypertension and decreased CPP. Precise therapeutic thresholds for these parameters – ICP and CPP (Mean arterial pressure – ICP) – have been sought for decades. In general, most studies suggest that an ICP target less than 20 mmHg is associated with the best outcome. Chambers and colleagues found a relationship between age and optimal CPP in children with TBI, with the youngest age group exhibiting slightly lower CPP. Specifically, in the age group of 2–6 years, a CPP threshold of 53 mmHg was observed compared to CPP greater than 60 mmHg for older children [84]. A recent study in children with predominantly AHT (81 % of all subjects) suggests that a threshold of CPP less than 45 mmHg is associated with poor outcome which, if confirmed, may suggest a therapeutic target for a larger study [83].

The high prevalence of seizures in children with AHT has been the subject of a significant amount of literature. In a large series of children with all severities of AHT, 73 % had clinical seizures and an additional 16 % had EEG abnormalities during hospital admission [85]. It has been hypothesized that the high rate of seizures is related to the importance of hypoxemia in the pathophysiology of AHT. In a provocative case study, Hartings and colleagues found that use of electrocorticography could detect multiple depolarizations and seizures in the subcortical region that led to severe tissue hypoxia in an adolescent after severe TBI – implying that if seizures are more frequent after AHT, this mechanism may be even more important in that patient population [86]. Further study is required to understand the secondary insults that may adversely affect outcome after AHT.

Outcomes After AHT

While outcome after AHT is variable between different series, multiple studies demonstrate that mortality and morbidity after AHT is higher than after non-abusive TBI of similar severity [87, 88]. While different studies report on different outcomes (e.g. GOS, disability), the most standard outcome parameter is mortality. In a retrospective review of 11 Canadian trauma centers over a 10-year period, the mortality rate from AHT was 19 % [89]. Scavarda and colleagues found a mortality rate of 28 % and also demonstrated that the Pediatric Risk of Mortality Score II (PRISM II) was associated with mortality in 36 children with AHT. Both of these studies included children with mild, moderate and severe AHT – indicating that overall mortality rates children with AHT are greater than those observed for large studies of children with severe non-abusive TBI. [88, 90]

While mortality is the most commonly used measure of outcome, more detailed neurological outcomes are particularly important after AHT because the GOS score is too gross a measure to use in an age group in which dependency on others for activities of daily living can be age-appropriate rather than a sign of pathology. We recently reported a 50 % rate of unfavorable outcome (defined as GOS = 3–5 [severe disability + vegetative + dead]) of a population of young children who predominantly had AHT [83]. Using more detailed neurological assessments, King and colleagues found that only 22 % of 364 children with AHT demonstrated no neurological sequelae 6 months after injury [89]. Recently, a task force recommended standard outcome assessments for all pediatric TBI studies; this would greatly increase the generalizability of studies that include children with AHT [91]. The reason for the increased mortality and morbidity in AHT compared with non-abusive TBI is not entirely clear. It is likely to be due to a combination of factors including characteristics of the injury itself, a delay by caretakers in seeking medical care, a delay by medical providers in identifying trauma, the effect of prior maltreatment, particularly prior AHT, on the response of the brain to subsequent injury and possibly developmental factors. Additional studies are needed to determine the relative impacts of these various putative mechanisms.

Reporting and Legal Issues

In the United States, physicians are mandated reporters of child abuse. As part of the mandated reporting laws, physicians are protected from lawsuits related to reporting suspected abuse as long as the report of abuse is made in good faith. In cases of AHT, particularly severe AHT, first responders and/or emergency department physicians often make the initial report to CPS. The pediatric intensivist, however, is sometimes the first physician to consider the

possibility of trauma and specifically, AHT. This is particularly true in young children with cardiac arrest, for example, who can be admitted to an ICU prior to a head CT or in cases in which infants may be thought to have RSV or meningitis, but who then undergo a head CT when the other diagnostic possibilities seem less likely and/or when the infant does not respond as expected to standard treatment. The pediatric intensivist may also be the one to make a report to CPS when a child is admitted with what is initially thought to be non-abusive TBI, but is later assessed as being the result of abuse when additional testing demonstrates additional injuries which are incompatible with the history provided. When the pediatric intensivist is the first physician to recognize the possibility of AHT, it is his/her responsibility to be sure that CPS is notified. While timely reporting is always important, it is particularly important in cases in which there are other young children in the home. Violence is often a pattern and while the child who is in the ICU is safe from further abuse, other young children may still in the home with the perpetrator. Children who are left in the home may be particularly vulnerable to abuse immediately after one child is brought to for medical care since the perpetrator may be angry and/or stressed about whether the injuries in the index child will be identified as abusive.

In cases in which a child is seriously injured, reporting to CPS will almost always trigger a report to local police. Early scene investigation by police can provide critically important information both about the etiology of the injuries and/or the perpetrator. Most referring institutions have dedicated social services staff or other designated personnel who can assist physicians in making a report to CPS. Notifying parents that a report is being made to CPS can be done at the same time that the physician discusses the child's injuries and treatment plan with the family. Including a hospital social worker or other support staff with expertise in the CPS system can be very helpful when child abuse reporting needs to be discussed. Discussions about the possibility of abuse can be brief. Physicians should tell the parents the injuries which are concerning for abuse, while being respectful and non-accusatory. It is helpful to remember that the parents may not be the perpetrators and may feel intense guilt for leaving their child in the care of someone who abused him/her. Some physicians feel that informing parents of the legal obligation to report abuse makes the discussion less stressful and less accusatory. While notification of parents is important, if no parent is available, physicians should not delay reporting.

Reporting possible abuse to CPS can be similarly brief: list the injuries in language which is as simple and non-medical as possible (e.g. use the word 'bruise' instead of 'ecchymosis'), inform CPS about the severity of these injuries (e.g. whether or not they are life-threatening) and provide information about the strength of the diagnosis (e.g. there is

concern for abuse vs. the injuries are diagnostic of abuse). A report to CPS is not a static document and more information including a change in the assessment of the likelihood of abuse can always be added as it becomes available (e.g. after a dilated eye exam or skeletal survey is performed).

Pediatric intensivists are often concerned about the need to testify in legal proceedings related to abuse cases. There are two types of court proceedings: civil and criminal. Civil cases are in family court and revolve around decisions related to safety and placement of children rather than prosecution of perpetrators. Testimony for these cases can often occur by phone and the intensivist is often only needed if there is no CPT physician or if there are specific medical questions which can only be answered by the intensivist. The level of evidence required in civil court is lower than in criminal court in which all statements about the etiology of injury must be "to a reasonable degree of medical certainty." Criminal proceedings relate to crimes such as endangering the welfare of a child or manslaughter that occur in association with AHT. Only a very small percent of cases of even unequivocal AHT go to criminal court; if a physician is needed for testimony, courts are often very flexible and will accommodate schedules, assist with transportation, and reimburse for time and expertise. Because of the difference in the level of evidence required in civil versus criminal court, there are many cases of suspected AHT in which the level of concern about the possibility of abuse is high enough that it is necessary to protect the children – either through a change in caretakers or through placement of services into the home – but not high enough for a criminal prosecution.

Conclusion

In summary, the pediatric intensivist is likely to care for numerous infants and young children in whom the possibility of AHT is being considered in the differential diagnosis. It is incumbent upon pediatric intensivists to be aware of this diagnosis, its epidemiology and its clinical characteristics and to be comfortable obtaining and interpreting the diagnostic testing needed to evaluate a child for AHT. In hospitals without a CPT, it is also imperative that the intensivist be able to relay this information to non-medical personnel (e.g. CPS) so that the proper actions can be taken to protect the child and his/her siblings from further abuse. As with most other diseases, AHT comes in all severities from 'mild' to 'severe' and the strength which one can give a diagnosis ranges from 'possible' to 'definite;' it is important that this information is accurately given to the family, CPS and law enforcement. There are very few clinical scenarios in which failure to make a proper diagnosis carries such a risk of re-injury or death; [92, 93] the pediatric intensivist plays a crucial role in protecting this very vulnerable group of young children.

References

- Barlow KM, Minns RA. Annual incidence of shaken impact syndrome in young children. *Lancet*. 2000;356:1571–2.
- Keenan HT, Runyan DK, Marshall SW, Nocera MA, Merten DF, Sinal SH. A population-based study of inflicted traumatic brain injury in young children. *JAMA*. 2003;290:621–6.
- Ellingson KD, Leventhal JM, Weiss HB. Using hospital discharge data to track inflicted traumatic brain injury. *Am J Prev Med*. 2008;34:S157–62.
- Xie Y, Davies SM, Xiang Y, Robison LL, Ross JA. Trends in leukemia incidence and survival in the United States (1973–1998). *Cancer*. 2003;97:2229–35.
- Christian CW, Block R. Abusive head trauma in infants and children. *Pediatrics*. 2009;123:1409–11.
- Kesler H, Dias MS, Shaffer M, Rottmund C, Cappos K, Thomas NJ. Demographics of abusive head trauma in the Commonwealth of Pennsylvania. *J Neurosurg Pediatr*. 2008;1:351–6.
- Salehi-Had H, Brandt JD, Rosas AJ, Rogers KK. Findings in older children with abusive head injury: does shaken-child syndrome exist? *Pediatrics*. 2006;117:e1039–44.
- Berger RP, Fromkin JB, Stutz H, et al. Abusive head trauma during a time of increased unemployment: a multicenter analysis. *Pediatrics*. 2011;128:637–43.
- Berger R, Fromkin J, Kochanek P, et al. Inflicted traumatic brain injury in children older than 1 year of age: an emerging concern. In: 137th American Public Health Association (APHA) annual meeting and exposition, Philadelphia; 2009.
- Hussey JM, Chang JJ, Kotch JB. Child maltreatment in the United States: prevalence, risk factors, and adolescent health consequences. *Pediatrics*. 2006;118:933–42.
- Gibbs DA, Martin SL, Kupper LL, Johnson RE. Child maltreatment in enlisted soldiers' families during combat-related deployments. *JAMA*. 2007;298:528–35.
- Alexander RC, Smith WL. Investigating abuse in the asymptomatic twin. *Arch Pediatr Adolesc Med*. 1996;150:444–5.
- Becker JC, Liersch R, Tautz C, Schlueter B, Andler W. Shaken baby syndrome: report on four pairs of twins. *Child Abuse Negl*. 1998;22:931–7.
- Ettaro L, Berger RP, Songer T. Abusive head trauma in young children: characteristics and medical charges in a hospitalized population. *Child Abuse Negl*. 2004;28:1099–111.
- Hettler J, Greenes DS. Can the initial history predict whether a child with a head injury has been abused? *Pediatrics*. 2003;111:602–7.
- Duhaime AC, Gennarelli TA, Thibault LE, Bruce DA, Margulies SS, Wiser R. The shaken baby syndrome. A clinical, pathological, and biomechanical study. *J Neurosurg*. 1987;66:409–15.
- Alexander R, Sato Y, Smith W, Bennett T. Incidence of impact trauma with cranial injuries ascribed to shaking. *Am J Dis Child*. 1990;144:724–6.
- Greenes DS, Schutzman SA. Occult intracranial injury in infants. *Ann Emerg Med*. 1998;32:680–6.
- Caffey J. On the theory and practice of shaking infants. Its potential residual effects of permanent brain damage and mental retardation. *Am J Dis Child*. 1972;124:161–9.
- Caffey J. The whiplash shaken infant syndrome: manual shaking by the extremities with whiplash-induced intracranial and intraocular bleedings, linked with residual permanent brain damage and mental retardation. *Pediatrics*. 1974;54:396–403.
- Starling SP, Patel S, Burke BL, Sirotiak AP, Stronks S, Rosquist P. Analysis of perpetrator admissions to inflicted traumatic brain injury in children. *Arch Pediatr Adolesc Med*. 2004;158:454–8.
- Biron D, Shelton D. Perpetrator accounts in infant abusive head trauma brought about by a shaking event. *Child Abuse Negl*. 2005;29:1347–58.
- Runyan DK, Shankar V, Hassan F, et al. International variations in harsh child discipline. *Pediatrics*. 2010;126:e701–11.
- Morad Y, Wygnansky-Jaffe T, Levin AV. Retinal haemorrhage in abusive head trauma. *Clin Experiment Ophthalmol*. 2010;38:514–20.
- Levin AV. Retinal hemorrhage in abusive head trauma. *Pediatrics*. 2010;126:961–70.
- Ewing-Cobbs L, Prasad M, Kramer L, et al. Acute neuroradiologic findings in young children with inflicted or noninflicted traumatic brain injury. *Childs Nerv Syst*. 2000;16:25–33; discussion 4.
- Rao P, Carty H, Pierce A. The acute reversal sign: comparison of medical and non-accidental injury patients. *Clin Radiol*. 1999;54:495–501.
- Ichord RN, Naim M, Pollock AN, Nance ML, Margulies SS, Christian CW. Hypoxic-ischemic injury complicates inflicted and accidental traumatic brain injury in young children: the role of diffusion-weighted imaging. *J Neurotrauma*. 2007;24:106–18.
- Parizel PM, Ceulemans B, Laridon A, Ozsarlak O, Van Goethem JW, Jorens PG. Cortical hypoxic-ischemic brain damage in shaken-baby (shaken impact) syndrome: value of diffusion-weighted MRI. *Pediatr Radiol*. 2003;33:868–71.
- Geddes JF, Hackshaw AK, Vowles GH, Nickols CD, Whitwell HL. Neuropathology of inflicted head injury in children. I. Patterns of brain damage. *Brain*. 2001;124:1290–8.
- Shannon P, Smith CR, Deck J, Ang LC, Ho M, Becker L. Axonal injury and the neuropathology of shaken baby syndrome. *Acta Neuropathol (Berl)*. 1998;95:625–31.
- Katz JS, Oluigbo CO, Wilkinson CC, McNatt S, Handler MH. Prevalence of cervical spine injury in infants with head trauma. *J Neurosurg Pediatr*. 2010;5:470–3.
- Feldman KW, Avellino AM, Sugar NF, Ellenbogen RG. Cervical spinal cord injury in abused children. *Pediatr Emerg Care*. 2008;24:222–7.
- Ghatan S, Ellenbogen RG. Pediatric spine and spinal cord injury after inflicted trauma. *Neurosurg Clin N Am*. 2002;13:227–33.
- Choudhary AK, Bradford RK, Dias MS, Moore GJ, Boal DK. Spinal subdural hemorrhage in abusive head trauma: a retrospective study. *Radiology*. 2012;262:216–23.
- Kemp AM, Joshi AH, Mann M, et al. What are the clinical and radiological characteristics of spinal injuries from physical abuse: a systematic review. *Arch Dis Child*. 2010;95:355–60.
- Johnson DL, Boal D, Baule R. Role of apnea in nonaccidental head injury. *Pediatr Neurosurg*. 1995;23:305–10.
- Pounder DJ. Shaken adult syndrome. *Am J Forensic Med Pathol*. 1997;18:321–4.
- Gilliland MG. Interval duration between injury and severe symptoms in nonaccidental head trauma in infants and young children. *J Forensic Sci*. 1998;43:723–5.
- Case ME, Graham MA, Handy TC, Jentzen JM, Monteleone JA. Position paper on fatal abusive head injuries in infants and young children. *Am J Forensic Med Pathol*. 2001;22:112–22.
- Bell E, Shouldice M, Levin AV. Abusive head trauma: a perpetrator confesses. *Child Abuse Negl*. 2011;35:74–7.
- Adamsbaum C, Grabar S, Mejean N, Rey-Salmon C. Abusive head trauma: judicial admissions highlight violent and repetitive shaking. *Pediatrics*. 2010;126:546–55.
- Eucker SA, Smith C, Ralston J, Friess SH, Margulies SS. Physiological and histopathological responses following closed rotational head injury depend on direction of head motion. *Exp Neurol*. 2011;227:79–88.
- Ibrahim NG, Margulies SS. Biomechanics of the toddler head during low-height falls: an anthropomorphic dummy analysis. *J Neurosurg Pediatr*. 2010;6:57–68.
- Ibrahim NG, Natesh R, Szczesny SE, et al. In situ deformations in the immature brain during rapid rotations. *J Biomech Eng*. 2010;132:044501.

46. Levchakov A, Linder-Ganz E, Raghupathi R, Margulies SS, Gefen A. Computational studies of strain exposures in neonate and mature rat brains during closed head impact. *J Neurotrauma*. 2006;23:1570–80.
47. Deemer E, Bertocci G, Pierce MC, Aguel F, Janosky J, Vogetley E. Influence of wet surfaces and fall height on pediatric injury risk in feet-first freefalls as predicted using a test dummy. *Med Eng Phys*. 2005;27:31–9.
48. Ibrahim NG, Ralston J, Smith C, Margulies SS. Physiological and pathological responses to head rotations in toddler piglets. *J Neurotrauma*. 2010;27:1021–35.
49. Prange MT, Coats B, Duhaime AC, Margulies SS. Anthropomorphic simulations of falls, shakes, and inflicted impacts in infants. *J Neurosurg*. 2003;99:143–50.
50. Margulies SS, Thibault KL. Infant skull and suture properties: measurements and implications for mechanisms of pediatric brain injury. *J Biomech Eng*. 2000;122:364–71.
51. Bertocci G, Pierce MC, Knight A, Bialczak K, Kaczor K, Deemer BC. Head injury risk associated with free falls from varying heights in children. In: *Pediatric Academic Societies*, San Francisco, May 2006.
52. Cory CZ, Jones BM. Can shaking alone cause fatal brain injury? A biomechanical assessment of the Duhaime shaken baby syndrome model. *Med Sci Law*. 2003;43:317–33.
53. Pierce MC, Bertocci G. Injury biomechanics and child abuse. *Annu Rev Biomed Eng*. 2008;10:85–106.
54. Talbot G. Abusive head trauma in children. In: Rowin M, Spinella PC, editors. *Current concepts in pediatric critical care*. Prospect: Society of Critical Care Medicine; 2010. p. 9–19.
55. Shaver EG, Duhaime AC, Curtis M, Gennarelli LM, Barrett R. Experimental acute subdural hematoma in infant piglets. *Pediatr Neurosurg*. 1996;25:123–9.
56. Durham SR, Duhaime AC. Basic science; maturation-dependent response of the immature brain to experimental subdural hematoma. *J Neurotrauma*. 2007;24:5–14.
57. Duhaime AC, Durham S. Traumatic brain injury in infants: the phenomenon of subdural hemorrhage with hemispheric hypodensity (“Big Black Brain”). *Prog Brain Res*. 2007;161:293–302.
58. Matlung SE, Bilo RA, Kubat B, van Rijn RR. Multicystic encephalomalacia as an end-stage finding in abusive head trauma. *Forensic Sci Med Pathol*. 2011;7:355–63.
59. Reece RM, Sege R. Childhood head injuries: accidental or inflicted? *Arch Pediatr Adolesc Med*. 2000;154:11–5.
60. Ghahreman A, Bhasin V, Chaseling R, Andrews B, Lang EW. Nonaccidental head injuries in children: a Sydney experience. *J Neurosurg*. 2005;103:213–8.
61. Hansen KK, Prince JS, Nixon GW. Oblique chest views as a routine part of skeletal surveys performed for possible physical abuse - Is this practice worthwhile? *Child Abuse Negl*. 2007;32:155–9.
62. American Academy of Pediatrics. Diagnostic imaging of child abuse. *Pediatrics* 2009;123:1430–5.
63. Harlan SR, Nixon GW, Campbell KA, Hansen K, Prince JS. Follow-up skeletal surveys for nonaccidental trauma: can a more limited survey be performed? *Pediatr Radiol*. 2009;39:962–8.
64. Zimmerman S, Makoroff K, Care M, Thomas A, Shapiro R. Utility of follow-up skeletal surveys in suspected child physical abuse evaluations. *Child Abuse Negl*. 2005;29:1075–83.
65. Levin AV, Christian CW. The eye examination in the evaluation of child abuse. *Pediatrics*. 2010;126:376–80.
66. Togioka BM, Arnold MA, Bathurst MA, et al. Retinal hemorrhages and shaken baby syndrome: an evidence-based review. *J Emerg Med*. 2009;37:98–106.
67. Bhardwaj G, Chowdhury V, Jacobs MB, Moran KT, Martin FJ, Coroneo MT. A systematic review of the diagnostic accuracy of ocular signs in pediatric abusive head trauma. *Ophthalmology* 2010;117:983–92 e17.
68. Lane WG, Dubowitz H, Langenberg P. Screening for occult abdominal trauma in children with suspected physical abuse. *Pediatrics*. 2009;124:1595–602.
69. Lindberg D, Makoroff K, Harper N, et al. Utility of hepatic transaminases to recognize abuse in children. *Pediatrics*. 2009;124:509–16.
70. Anderst JD, Carpenter SL, Abshire TC. Evaluation for bleeding disorders in suspected child abuse. *Pediatrics*. 2013;131:e1314–22.
71. Carpenter SL, Abshire TC, Anderst JD. Evaluating for suspected child abuse: conditions that predispose to bleeding. *Pediatrics*. 2013;131:e1357–73.
72. Trenchs V, Curcoy AI, Morales M, Serra A, Navarro R, Pou J. Retinal haemorrhages in-head trauma resulting from falls: differential diagnosis with non-accidental trauma in patients younger than 2 years of age. *Childs Nerv Syst*. 2008;24(7):815–20.
73. Tarantino CA, Dowd MD, Murdock TC. Short vertical falls in infants. *Pediatr Emerg Care*. 1999;15:5–8.
74. Lyons TJ, Oates RK. Falling out of bed: a relatively benign occurrence. *Pediatrics*. 1993;92:125–7.
75. Chadwick DL. A witnessed short fall mimicking presumed shaken baby syndrome (inflicted childhood neurotrauma). *Pediatr Neurosurg*. 2008;44:517.
76. Rooms L, Fitzgerald N, McClain KL. Hemophagocytic lymphohistiocytosis masquerading as child abuse: presentation of three cases and review of central nervous system findings in hemophagocytic lymphohistiocytosis. *Pediatrics*. 2003;111:e636–40.
77. Hansen K, Frikke M. Dual and discrepant case publication in regard to hemophagocytic lymphohistiocytosis and child abuse. *Pediatr Radiol*. 2007;37:846.
78. Fitzgerald NE, MacClain KL. Imaging characteristics of hemophagocytic lymphohistiocytosis. *Pediatr Radiol*. 2003;33:392–401.
79. Ruddy GN, Smith CM, Malia RG. Late-form hemorrhagic disease of the newborn: a fatal case report with illustration of investigations that may assist in avoiding the mistaken diagnosis of child abuse. *Am J Forensic Med Pathol*. 1999;20:48–51.
80. Reddy AR, Clarke M, Long VW. Unilateral retinal hemorrhages with subarachnoid hemorrhage in a 5-week-old infant: is this non-accidental injury? *Eur J Ophthalmol*. 2010;20:799–801.
81. Durham SR, Clancy RR, Leuthardt E, et al. CHOP Infant Coma Scale (“Infant Face Scale”): a novel coma scale for children less than two years of age. *J Neurotrauma*. 2000;17:729–37.
82. Adelson PD, Bratton SL, Carney NA, et al. Guidelines for the acute medical management of severe traumatic brain injury in infants, children, and adolescents. Chapter 3. Prehospital airway management. *Pediatr Crit Care Med*. 2003;4:S9–11.
83. Mehta A, Kochanek PM, Tyler-Kabara E, et al. Relationship of intracranial pressure and cerebral perfusion pressure with outcome in young children after severe traumatic brain injury. *Dev Neurosci*. 2010;32(5–6):413–9.
84. Chambers IR, Stobart L, Jones PA, et al. Age-related differences in intracranial pressure and cerebral perfusion pressure in the first 6 hours of monitoring after children’s head injury: association with outcome. *Childs Nerv Syst*. 2005;21:195–9.
85. Bourgeois M, Di Rocco F, Garnett M, et al. Epilepsy associated with shaken baby syndrome. *Childs Nerv Syst*. 2008;24:169–72; discussion 73.
86. Hartings JA, Gugliotta M, Gilman C, Strong AJ, Tortella FC, Bullock MR. Repetitive cortical spreading depolarizations in a case of severe brain trauma. *Neurol Res*. 2008;30:876–82.
87. Beers SR, Berger RP, Adelson PD. Neurocognitive outcome and serum biomarkers in inflicted versus non-inflicted traumatic brain injury in young children. *J Neurotrauma*. 2007;24:97–105.
88. Adelson PD, Ragheb J, Kanev P, et al. Phase II clinical trial of moderate hypothermia after severe traumatic brain injury in children. *Neurosurgery* 2005;56:740–54; discussion 54.

89. King WJ, MacKay M, Sirnick A. Shaken baby syndrome in Canada: clinical characteristics and outcomes of hospital cases. *CMAJ*. 2003;168:155–9.
90. Hutchison JS, Ward RE, Lacroix J, et al. Hypothermia therapy after traumatic brain injury in children. *N Engl J Med*. 2008;358:2447–56.
91. McCauley SR, Wilde EA, Anderson VA, et al. Recommendations for the use of common outcome measures in pediatric traumatic brain injury research. *J Neurotrauma*. 2012;29(4):678–705.
92. Jenny C, Hymel KP, Ritzen A, Reinert SE, Hay TC. Analysis of missed cases of abusive head trauma. *JAMA*. 1999;281:621–6.
93. Oral R, Yagmur F, Nashelsky M, Turkmen M, Kirby P. Fatal abusive head trauma cases: consequence of medical staff missing milder forms of physical abuse. *Pediatr Emerg Care*. 2008;24:816–21.