TECHNICAL REPORT

American Academy of Pediatrics
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Abusive Head Trauma in Infants and Children: Technical Report

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INTRODUCTION

Abusive head trauma (AHT) in infants and children is a complex and challenging clinical diagnosis. Because of its clinical, social, and legal implications, few pediatric diagnoses evoke as much cognitive difficulty and emotional distress as AHT. Over the past several decades, considerable literature has been published on various aspects of the AHT diagnosis including epidemiology, historical features, clinical findings, biomechanics, differential diagnoses, outcomes, and prevention. The volume and critical analysis of the published literature can be daunting to even the most motivated pediatric provider.

To date, the American Academy of Pediatrics (AAP) has not promulgated, in the form of a technical report, a comprehensive review of the evidence-based literature surrounding AHT. Although the AAP has addressed the subject matter in other treatises, a scientific review in the form of a technical report offers the benefit of openly available, readily accessible, and more frequently updatable scientific information.[1,2] Additionally, as the diagnosis has engendered some media and legal controversy, which then spills into a clinical venue, a technical report can assist the pediatric provider in communicating the scientific information to interdisciplinary colleagues such as child welfare agencies and courts.

With respect to the methodology used for this technical report, the authors recognize that properly conducted formal systematic review methods provide a standardized, relatively objective compilation of strength-graded evidence on a specific focused topic of inquiry. However, the broad topic of AHT, in which evidence comes from a wide variety of sources, fields, and disciplines using many different methods to approach a range of related areas of focus, is not a topic that easily lends itself to a formal systematic review process. Instead, in each area of this technical report, authors or groups of authors performed a wide review

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of peer-reviewed publications within each subtopic (generally limited to articles published in English), emphasizing those sources with the highest quality of evidence but not eliminating sources for which descriptive methods nonetheless provide useful and relevant information. The overall manuscript was reviewed several times by multiple participants with source documents reviewed for content when appropriate. References are provided for statements and conclusions so that the reader can access the sources directly.

Additionally, there are various medical terms utilized in this technical report that are commonly encountered in the AHT diagnosis (ie, acute subdural hematoma/hemorrhage, chronic subdural hematoma/hemorrhage, short distance fall, subdural hygroma, etc) but may have some variance in definition throughout the medical literature. For transparency and to promote a common understanding of these terms, there is an Appendix of definitions at the conclusion of this report. Many of the definitions for the pathoanatomic entities were from the National Institutes of Health Common Data Elements for Traumatic Brain Injury.[3] Some other definitions, such as those within the biomechanics section, are described/defined in more detail in the relevant sections.

DEFINITION

AHT has been known by multiple terms in the past and is referenced by other terms currently in some other countries.[4] Aside from the challenges and confusion that multiple terminologies represent, more challenging has been the lack of consistent case definition amongst clinical and research sectors.

In 2008, the Centers for Disease Control and Prevention (CDC) convened an expert panel group of "pediatricians, child maltreatment experts, abusive head trauma experts, injury surveillance experts, ICD coding experts, and experienced state health department personnel" with the aim of identifying a common set of codes (ICD-9-CM and ICD-10) "that could be uniformly implemented to define abusive head trauma among children under 5 years of age."[5] Through an iterative process, the expert panel group arrived at the following definition of AHT:

> "an injury to the skull or intracranial contents of an infant or young child (<5 years of age) due to inflicted blunt impact and/or violent shaking."

Although this definition was engineered to facilitate consistency in "public health surveillance and research," the breadth of the definition also provides a practical framework for clinical application. It is noteworthy that this definition is intentionally broad (ie, not confined to exclusively intracranial findings or findings indicative of particular mechanisms of injury). The breadth of the definition offers the benefit of reducing heuristic application of certain variables to the AHT diagnosis, thereby minimizing concerns for circularity in research.

EPIDEMIOLOGY

Incidence

AHT is the third leading cause of head injury in children younger than 5 years, only surpassed by motor vehicle collisions and falls.[5] It is also the leading cause of severe head injury in infancy.[5] The mortality rate of AHT ranges from 10% to 20%.[6–9] The current incidence of AHT in the United States is estimated at 25 to 35 children per 100 000 annually younger than 1 year of age.[6,8,10] Some researchers have found an incidence as high as 40 children per 100 000 annually using more rigorous research methods as defined by the CDC.[5,11] Most studies find a peak in incidence at around 2 months of age with a median of 4 months of age; however, some additional studies have noted a second, smaller peak that occurs at 8 months of age.[6,9] After infancy, the incidence drops steeply with an incidence as low as 3.8 per 100 000 for children between 1 and 2 years of age and few cases noted after 5 years of age.[12]

There is geographic variation in the incidence of AHT. A number of researchers have noted that the incidence of AHT varies based on US region: the highest incidences are reported in the Midwest and the lowest in the Northeast.[7,11] At this time, there has been no clear reason found for this regional variation; however, there does appear to be an increased risk of AHT in rural populations, which are more prevalent in the Midwest.[13] The United States appears to have one of the highest reported national incidences of AHT, with Scotland reporting 24.6 per 100 000 infants annually and New Zealand reporting 14.7 to 19.6 per 100 000 infants.[14,15]

TAKEAWAY POINTS

1. AHT is the third leading cause of head trauma in children younger than 5 years.
2. Mortality rate ranges from 10% to 20%.
3. The current incidence of AHT in the United States is estimated at 25 to 35 children per 100 000 annually younger than 1 year of age.
4. Peak incidence is around 2 months of age; the median is at 4 months of age, with decreasing incidence after infancy.

DISPARITIES IN AHT DIAGNOSIS

Children who have experienced AHT are have been reported disproportionately as male, Black, or American Indian/Alaska Native.[6,10,11,15,16] This broad statement impugns race per se as an associated risk factor for AHT in children. Importantly, this overarching statement does not reflect rigorous statistical adjustment for social drivers

of health (SDOHs), which are similarly associated with the incidence of AHT. This is heralded by the findings of Wood et al (2016) demonstrating an increase in the incidence of AHT during the 2007–2009 US recession. Taken together, the data to date suggest considerable confounding affects any assertion of race per se as a risk factor for AHT and instead elevates the probability that AHT may reflect yet another racial disparity (defined as a difference in outcomes not attributable to biology but rather due to social circumstances or structural or implicit bias).[17]

Implicit bias may affect one's ability to appropriately diagnose AHT and likely affects certain groups disproportionately—that is, in overreporting).[18–20] In the converse, Jenny et al (1999) identified an underreporting or lesser likelihood to diagnose AHT in white and intact families. This is juxtaposed with the findings of Lane et al (2002) indicating that Black, Hispanic, and Indigenous children were more likely to be evaluated for abuse.[21,22] Luken et al (2021) used exploratory mapping in the National Child Abuse and Neglect Data System (NCANDS) data to find that there was a 15 times greater rate of reporting abuse in Black communities than in white communities.[23] Regional variations in reporting highlight policy and practice concerns that further identify the importance of place and social/policy circumstances affecting the diagnosis of AHT in minorities.[23] Finally, other risk factors such as poverty and financial stress consistent with known SDOHs are more prevalent in communities of color, present challenges in quantitative assessment, and likely serve as candidate root causes for the observed disparities.[15]

TAKEAWAY POINTS

1. There is an overrepresentation of Black, Hispanic, and American Indian/Alaska Native children having experienced AHT, with the associations potentially and largely influenced by the confounders of social circumstances and bias of reporters. Future study of AHT requires more rigorous assessment of socioeconomic status, SDOHs, and other drivers of AHT.
2. Bias in reporting can also adversely affect nonminoritized communities through underreporting (positive implicit bias), in addition to the previously acknowledged concerns for overreporting in minoritized communities (negative implicit bias).

RISK FACTORS

Risk factors are identified when one group (with the risk factor) is more likely than another (without the risk factor) to experience abuse. Risk factors can be effective in prevention strategies (and public policies) as they can help target interventions[24] but should not be used for individual diagnostic decision making.[25,26]

The reported perpetrators are most commonly male caregivers (father, stepfather, or mother's boyfriend), followed by female babysitters.[27,28] Mothers are the perpetrator in 13% to 16% of cases.[27,28] Other risk factors for AHT include young age of the mother, late prenatal care, low birth weight, and multiple births.[6,12] Kelly and colleagues (2017) found that prematurity was a stronger risk factor than low birth weight and multiple births.[29]

Crying is frequently reported as a trigger for an abusive event.[30] Barr and colleagues (2006) have noted that the normal crying curve is paralleled by the incidence in AHT in infancy, with peak incidence of the normal crying curve occurring at 5 to 6 weeks of age and the peak incidence of AHT hospitalization rates occurring approximately 4 weeks later.[31] The absence of the aforementioned risk factors does not preclude the diagnosis. In fact, AHT has been diagnosed many times in the absence of these risk factors.

TAKEAWAY POINTS

1. The full assortment of risk factors for AHT remains under investigation but includes poverty, maternal factors (eg, young maternal age), low birth weight, multiparity, and a male caregiver.
2. Known risk factors are insufficient to serve as a screening tool for children at risk for AHT, as AHT can occur in the absence of known risk factors.
3. Addressing known risk factors may be effective in developing prevention strategies, including public policy objectives.

CLINICAL PRESENTATION

History

Introduction

A key component in the evaluation of suspected AHT is whether the reported history is consistent with the child's clinical presentation.[32,33] This inconsistency with the history is a cornerstone of the maltreatment diagnosis in general and no different with AHT. Therefore, gathering a detailed history is highly important in the evaluation of suspected abuse, as it is in all of medicine. Detailed components of the trauma history include what and when symptoms were noted by the caregiver, when the child was last observed by the caregiver to have been acting "normally" in the caregiver's estimation (and for what period that normalcy existed), a detailed account of any traumatic events, the child's behavior and symptoms after the traumatic event, and the caregiver's response for medical assistance, if any.[33] When attaining history from a caregiver, interviewing each caregiver independently, if feasible, and asking questions in a nonleading, inquisitive, and nonjudgmental manner assists in attaining the most reliable historical information.[34]

Historical "red flags" have long been taught as cornerstone markers for suspicion of AHT.[35] Over the past couple of decades, several historical features have been studied

more systematically to investigate their association with the AHT diagnosis.

An Absent Trauma History

An absent trauma history, in light of traumatic findings, has been considered a key associative feature of abuse/AHT.[32,36] In 2003, Hettler and Greenes performed a retrospective review of 163 children, aged 0 to 3 years, including patients admitted from 1993–2000 with acute traumatic intracranial injury.[37] The authors classified cases as either "definite abuse" or "not definite abuse" on the basis of radiologic, ophthalmologic, and physical examination findings, without regard to the presenting history. Forty-nine of the cases (30%) were classified as "definite abuse" and 114 (70%) were classified as "not definite abuse." These authors found that an absent history of trauma, in patients with a clear traumatic injury, had a 97% specificity and 92% positive predictive value (PPV) for AHT.[37]

In 2020, Hymel et al published a study that sought to replicate Hettler and Greenes' retrospective analysis on a cross-sectional dataset containing prospective data.[38] Similar to Hettler and Greenes, cases were categorized a priori as "definite" or "not definite" AHT solely on the basis of patients' clinical and radiologic findings unrelated to history. Hymel et al found that a caregiver's specific denial of any trauma had a 90% specificity (95% confidence interval [CI], 0.84–0.94), 81% PPV (95% CI, 0.71–0.88), and a positive likelihood ratio of 4.83 (95% CI, 3.07–7.61) for definite AHT.[38]

An Evolving or Changing Trauma History

The longstanding clinical adage is that "accidental histories don't change."[33] It is uncommon for the history of an accidental injury scenario to vary substantively from provider to provider. It can, however, be difficult to determine what constitutes a "substantive" variance in the trauma history.

There are primarily 2 studies that provide reliable data on this historical variable: Hettler and Greenes (2003) and Hymel et al (2020). In Hettler and Greenes' cohort, only 6% of all cases had an evolving or changing history.[37] *All* of those were in the "definite abuse" cohort (9/49 [18%]). There were no cases (0/114) in the "not definite abuse" cohort that had evolving or changing histories. However, Hettler and Greenes did not operationalize a definition for what constituted a "change" to or "evolution" of the initial history and instead relied on the judgment of the providers as documented in the records.

The Pediatric Brain Injury Network (PediBIRN) data set used by Hymel et al also did not define what constituted a "substantive" change but instead relied on the investigators to determine whether they thought the history had "changed substantively with repetition over time."[38] In their prospective dataset of 346 patients, 248 patients provided an accidental trauma history—81 in the "definite abuse" cohort, and 167 in the "not definite abuse" cohort. Hymel et al found 41 cases in which the history "changed substantively with repetition over time." Of the "definite abuse" cases, 31 of 81 (38%) had "changed" histories. Additionally, of the "not definite abuse" cases, 10 of 167 (6%) had "changed" histories. Hymel et al found a "changed" history to have 94% specificity (95% CI, 0.89–0.97), a 76% PPV (95% CI, 0.59–0.87), and a 6.39 positive likelihood ratio (95% CI, 3.30–12.38) for the "definite abuse" cohort.

Thus, although there is currently no explicit operational definition in the evidence-based literature on what constitutes a "substantive" change in caregiver trauma history, there is some evidence that indicates a substantive change to be much more strongly associated with abuse than accident.

A History That Is Inconsistent With the Child's Developmental Capabilities

There are multiple potential scenarios in which a child's developmental abilities could be inconsistent with a given trauma history. One commonly cited example among AHT scenarios is the history of a 1- or 2-month-old infant "rolling" off an elevated surface (such as a couch or a bed), before the developmental capability of rolling from back-to-front or front-to-back is achieved. But, similar to the earlier discussion on changing histories, there are a limited number of studies that have empirically assessed the association of an inconsistency with the reported developmental capabilities of a child and the abuse diagnosis.[38,39]

In the PediBIRN data set of 346 patients used by Hymel et al, an accidental trauma history was provided in 248 cases (72%).[38] Of those, 22 of 248 cases (9%) had a caregiver accident history that was deemed inconsistent with the child's known or expected gross motor skills. In the a priori "definite AHT" and "not definite AHT" groups, developmental inconsistency with the trauma history had a specificity of 95% (95% CI, 0.90–0.98), a 64% PPV (95% CI, 0.41–0.82), and a 3.61 positive likelihood ratio (95% CI, 1.58–8.25) for the definite abuse cohort.

Although a limited evidence base supports the assertion that incongruence of reported developmental capabilities and abuse are linked, other authors have cautioned providers about overreliance on the normative ranges of developmental capabilities in infants and toddlers in suspected abuse cases.[34]

A Delay in Seeking Medical Care

There are several studies that have addressed the historical variable of delay in seeking medical care in suspected AHT. Vinchon et al offer one of the few studies to provide data surrounding the time range of what constitutes a "delay" in abusive and nonabusive head trauma.[40] In their cohort of 45 confessed abuse cases and 39 witnessed accidents,

the authors found that the mean and median times from injury to referral for care were shorter in the accidental group than the abusive group (21 vs 57 hours mean and 3.5 vs 12 hours median, respectively), but these findings did not reach statistical significance. Similarly, Hettler and Greenes did not find a statistically significant difference of delay in seeking care between their definite and not definite abuse groups. [37]

In distinction to the studies by Vinchon et al and Hettler and Greenes, Kennedy et al did find a statistically significant difference between AHT and non-AHT (nAHT) groups with regard to delay in seeking care.[41] Kennedy et al retrospectively reviewed charts of children younger than 6 years who had acute head injury and were admitted to the pediatric intensive care unit at a pediatric hospital from 2013 to 2017. The authors defined a "delay in seeking care" as waiting approximately 30 minutes or more from severe symptom onset (which was defined as when a caregiver identified that the child was clearly ill). Patients who had AHT were significantly more likely to have a delayed presentation to care than those who had accidental head trauma (61.8% vs 20.0%; $P = .0017$). In a secondary analysis of the AHT group (n = 34), a delay in presentation to care was not significantly associated with age, sex, race, Medicaid insurance status, length of stay, or discharge disposition.

Vadivelu et al sought to evaluate "delay in seeking care" from another perspective—the effect on overall patient neurologic outcome at discharge.[42] In their 10-year retrospective review at a single institution, the authors included only those AHT cases in which a perpetrator was identified by data in the New York State Central Register and predefined "delay" criteria were applied (ie, 0–6 hours = "no delay"; 6–12 hours = "moderate delay"; and >12 hours = "severe delay") based on parallel definition in the stroke literature. The authors identified 28 children who met inclusion criteria and reviewed those charts to assess the association of delay, if any, with neurologic outcomes. They found the majority of patients with AHT (61%) presented with either moderate or severe delay; the majority (61%) had an admission Glasgow coma score (GCS) of less than 7; additionally, the majority of those with a GCS of less than 7 presented with either no or moderate delay.

Vadivelu et al also found that patients presenting to medical care 6 to 12 hours after AHT (moderate delay) appeared to have worse outcomes than those presenting with no delay (P <.0426). Interestingly, however, the authors found that those patients presenting with severe delay did not have worse outcomes than those presenting with no delay or moderate delay. The authors attributed this aberrance to be secondary to higher GCSs in the children with severe delay.[42]

TAKEAWAY POINTS

1. An absent trauma history, in the presence of traumatic findings, has high specificity and positive predictive values for AHT.
2. A "substantive" change in a caregiver's trauma history also holds high specificity and positive predictive values for AHT, but what constitutes a "substantive" change is an area needing further research.
3. There is some literature support to indicate that inconsistency between the caregiver's history and the child's developmental capabilities is more strongly associated with AHT than nAHT; this is also an area needing further research.
4. The current evidence base is unclear on whether there is a strong association between "delay" in seeking medical care and AHT. Additionally, further research is needed on what constitutes a "delay" in seeking medical care in cases of suspected AHT.

Symptoms

Introduction

The neurologic presentation of the infant and toddler with AHT varies widely, ranging from the neurologically asymptomatic patient commonly presenting with macrocephaly to the patient in acute cardiopulmonary arrest with profound neurologic impairment.[43] Frequently, symptoms in the infant are nonspecific; symptoms including vomiting, irritability, and lethargy overlap with medical diagnoses such as viral illness and undernutrition/growth impairment, leading to challenges in injury detection.[15] Jenny and colleagues conducted a 5-year retrospective single center review of children younger than 3 years seen by an institutional child advocacy and protection team.[21] Of the 173 children in whom AHT was eventually diagnosed, 54 (31%) were initially unrecognized with regard to the cause of their symptoms. Children presenting with coma, respiratory compromise, or seizures were more likely to have a diagnosis of head injury, while those presenting with irritability or vomiting were less likely to be identified.

Additionally, it can be challenging for the provider performing the evaluation to describe the degree of neurologic impairment. Pediatric variants of the GCS remain the most commonly used scales for describing the neurologic status of the injured child, despite their reliance on motor and verbal skills (which may be more difficult to determine in young children). [23] There is a paucity of data available on the predictive abilities of the GCS in pediatric neurologic injury.[10] One interesting observation regarding the limited value of neurologic symptoms informing the clinician regarding the diagnosis of AHT is the lack of reliance on neurologic symptoms in AHT clinical decision rules (Pittsburgh Infant Brain Injury Score [PIBIS] and PediBIRN).[44–46]

Factors including the mechanism of injury and magnitude of force applied influence both patient presentation and outcome.[47] On the basis of parallels with accidental trauma, children with high-force inertial injuries can be expected to be more neurologically impaired. As described by Oluigbo and colleagues in a single institutional retrospective study of 37 children undergoing decompressive craniectomy, 14 (38%) children had experienced AHT and presented with acute encephalopathy and a mean GCS of 4.5.[48] Less severe injuries presented with less profound acute symptoms or, in some cases, presented with a delay in time from the initial traumatic event. In contrast to acutely severe presentations, Klimo et al performed a single institutional retrospective study of 26 children presenting for treatment of chronic subdural collections. Twenty-four (92%) children in this cohort who had experienced AHT presented with "global findings" to include macrocephaly, seizure, sun-setting eyes (persistent downward deviation of gaze), lethargy, and irritability. Sixty-two percent of patients presented with occipitofrontal circumference exceeding the 90th percentile, suggesting the collections temporally evolve from an acute hemorrhage or tear in the arachnoid mater and that symptoms likely were not severe at the time of injury but only evolved as the chronic collection enlarged gradually. Mortality in this series was only 4.2%.[49]

Apnea and Seizures

Piteau et al performed a systematic review of clinical characteristics associated with AHT over nAHT in a hospitalized population of children 6 years or younger.[50] Although the authors were challenged by a lack of consistent reporting of variables in the included studies, their meta-analysis determined odds ratios (ORs) for 11 radiographic and 8 clinical variables. Among neurologic signs and symptoms, seizure (OR, 7.25; 95% CI, 3.04–17.27; P <.001) and apnea (OR, 5.31; 95% CI, 2.34–12.05; P <.001) achieved ORs >1.0, suggesting a higher likelihood of a diagnosis of AHT. Similarly, Maguire and colleagues reviewed data from 6 studies in children younger than 3 years, pooling data on 1053 children, among whom 348 (33%) had experienced AHT. Multilevel logistic regression analysis revealed seizure (OR, 5.075) and apnea (OR, 6.948) as neurologic symptoms more highly associated with AHT in this study.[51]

Irritability, Vomiting, or Developmental Delay

Although symptoms such as irritability, vomiting, and developmental delay are commonly present in children with AHT, these symptoms are of limited utility in discriminating AHT from other common childhood disease.[52] Feldman et al conducted a 5-year retrospective multicenter study of children with AHT and subdural hemorrhage.[53] Although the majority (80%) of patients in this study were symptomatic, 40.7% presented with nonspecific neurologic and somatic symptoms including lethargy and vomiting. Ten percent presented with no neurologic symptoms, and 3% presented with asymptomatic macrocephaly alone.

Timing of Clinical Symptoms

Forensically, a key issue that arises in AHT cases is the timing of onset of clinical symptoms. Because the manifesting symptoms in AHT are variable and often subtle to the lay caregiver, it is important to gather medical and forensic information relating to the last time the child was noted to be "normal" (ie, symptom-free) in the eyes of caregivers.

One forensic issue that arises in the setting of fatal AHT is the topic of a "lucid interval" prior to neurologic decline. The term "lucid interval" classically has been described in the setting of a contact injury to the skull with subsequent expanding extra-axial hemorrhage (epidural hemorrhage) in verbal pediatric or adult patients.[54] De Leeuw reported the presence of lucid interval following confessed AHT only in the setting of contact injuries to the skull.[55]

Arbogast and coauthors illustrated challenges with the study of lucid intervals in patients with AHT in their analysis of fatal head injuries within the Pennsylvania Trauma System Outcome database.[56] Of the 37% of injuries classified as AHT, 6.8% were reported as having a GCS >7 at presentation. Patients younger than 24 months with AHT were 10 times more likely to present with moderate GCSs than those in motor vehicle crashes. The authors acknowledged the methodologic problems inherent in the retrospective nature of their study: "Whether [our results are] because of differences in pathologic injury, neurologic responses unique to the infant brain, or limitations of the bedside methods used to assess neurologic function in young children cannot be determined by this study."

Most cases of fatal AHT are a result of inertial brain injury with acute subdural hemorrhage and brain swelling.[57] Among patients analyzed in their series, Duhaime et al observed "…there is no evidence of a prolonged interval of lucidity between the injury and the onset of symptoms in children with acute subdural hemorrhage and brain swelling."[58] However, in the infant or preverbal child, even the definition of lucidity is problematic, particularly in the setting of AHT, in which the report of the caregiver may be unreliable. "…It is not clear what is meant by a lucid state in an infant, and who should verify whether this is present or not."[59]

Studies of fatal nAHT in this age group represent one means of reducing concerns for bias present in the study of AHT patients. Willman et al examined 95 fatal head injuries in pediatric patients with nAHT and identified only 2 patients with reliable reports of a lucid interval prior to death. Both occurred in the setting of epidural hemorrhage.[60]

TAKEAWAY POINTS

1. Symptoms of AHT are frequently nonspecific and include vomiting, irritability, and lethargy.
2. Seizure and apnea are neurologic signs highly, but not exclusively, associated with AHT.
3. Determination of "lucidity" in the infant is challenging; reliance on this aspect of patient history for the timing of injuries can be problematic.
4. In cases of severe head injury (cerebral edema with acute subdural bleeding), it is unlikely that there is a prolonged period of wellness between the injury and symptom onset.

Physical Findings

Introduction

Much like symptomology, physical examination findings indicative of AHT can be variable and subtle and are often missed.[21,61] Research has identified bruising as a key sentinel injury in infants (ie, younger than 12 months), and its recognition is vital to the prevention of more severe abuse such as AHT. Sheets et al identified sentinel injuries in over 28% of subsequently identified "definite" abuse cases and bruising as the sentinel finding that was missed or underappreciated in 80% of those cases.[62] The physical findings discussed immediately below are those in the clinical literature of nonfatal AHT cases. Epidemiologic and clinical features of fatal AHT cases are discussed in the "Pathology" section of this technical report.

a) Macrocephaly

Macrocephaly is defined operationally as an occipitofrontal circumference greater than 2 standard deviations above mean or above the 97th percentile as measured from the glabella to the most prominent portion of the occiput. Macrocephaly can also be relative, when the head is large out of proportion to the length and weight of the child. The differential diagnosis for macrocephaly is broad and includes conditions resulting in enlargement of the extra-axial spaces, the ventricular system, and brain parenchyma itself.[63] The majority of infants with macrocephaly do not harbor pathologic conditions that warrant diagnostic imaging or evaluations by a neurosurgeon.[64] Haws et al examined 466 patients with benign macrocephaly diagnosed by ultrasonography, none of which required surgical intervention.[65] The relative frequency of macrocephaly led editors reviewing the work of Haws et al to advocate for close observation with a tape measure in the child with isolated macrocephaly and no neurologic findings.[66]

Despite the benign implications of macrocephaly in most infants, AHT is often associated with fluid collections in the extra-axial compartment, more specifically in the subdural space, leading to macrocephaly in some patients.

Repetitive episodes of AHT are well described in the literature, and either repetitive or single injuries with venous and/or arachnoid tears may lead to the formation of subdural collections and macrocephaly.[30] Increasing head circumference is a useful sign that a hemorrhagic subdural collection may be related to a previous subdural hemorrhage and, therefore, to a previous episode of trauma.[67] Hobbs and colleagues reported on 186 children with a diagnosis of subdural hemorrhage collected from a national registry in Great Britain from 1998–1999.[68] Thirty-eight children (20%) were noted to have macrocephaly at presentation.

b) Soft Tissue Injuries

Soft tissue findings in AHT can include scalp swelling, scalp contusions/hematomas, subgaleal hematomas, intra-oral injuries (including frenular contusions/tears, intra-oral mucosal contusions, tooth injuries, and palatal/pharyngeal injuries), subconjunctival hemorrhage, and bruises to the face, ear, neck, or torso. Although many cases of AHT will have no visible soft tissue manifestations about the head, bruising to the head and neck is reported in 29% to 54% of cases of AHT and is the most common site of bruising in children who have been abused.[69,70] However, bruising to the head is also common in nAHT.

Meta-analysis has shown that bruising about the head, *in general*, in a child younger than 3 years with intracranial injury is *not* a discriminating factor between AHT and nAHT (PPV for AHT was 37%; OR, 0.8; $P > .2$).[71,72] However, clinical distinctions between nAHT and AHT bruising can emerge depending on a child's age and the distribution of bruising on certain areas of the head.

Pierce et al found bruising in any region of the body in children 4 months and younger as suggestive of abusive versus accidental bruising.[73,74] Larger systematic reviews have focused more on a child's mobility (rather than a specific age) in correlation to susceptibility to abusive or accidental bruising.[69,75] Accidental bruising is rare (0–1.3%) in premobile infants (ie, those who do not yet crawl, cruise, or ambulate).[76–80] Thus, any bruising in premobile infants will raise concerns for physical abuse/AHT, and thus, an appropriate evaluation is warranted.

With regard to distribution of bruising, large meta-analyses have identified certain body regions that are more characteristic of accidental bruising.[69,75] In mobile children, these include the shins, knees, and bony prominences. When the face is involved, accidental bruising characteristically follows a "T" distribution (ie, the forehead, nose, upper lip, and chin). Even in mobile children, accidental bruising to certain body regions—the soft tissue of the cheek, ears, neck, abdomen, buttocks, and genitalia—is extremely uncommon.[75]

However, abusive bruising tends to appear in a different body distribution and a more nuanced distribution about the head.[69,73,74,77,81–83] In a prospective, multicenter study, Pierce et al validated a clinical decision rule that was designed to distinguish abusive from nonabusive bruising.

They found that bruising in the "TEN-4-FACESp" regions (described as any bruising in an infant 4 months or younger; bruising to the torso, ear, or neck in a child younger than 4 years; and bruising to the frenulum, angle of jaw, cheeks, eyelids, subconjunctiva, or patterned bruising) had a 96% sensitivity and 87% specificity for identifying abusive injury.[73] Two other comparative studies of cohorts of children who were abused and not abused found similar results—that bruising to the ear, face, neck, torso, and buttocks was significantly more common in children who were abused (P <.001).[82,83] Thus, although in general, bruising about the head is not, in itself, a discriminating feature, certain regions of the head (ears, fleshy cheek, angle of jaw, and neck) assist in discriminating between the likelihood of AHT and nAHT.

The number of bruises may also assist in distinguishing AHT from nAHT.[73,74,76,77,79] Although the number of bruises children sustain through normal activity increases as they get older and their level of independent mobility increases, children experiencing accidents have a mean of 1 to 3 and a median of 1 to 5 bruises per child, with a range of 1 to 16.[73,77] Children who have been abused have a median of 3 to 11 bruises per child, with larger medians at each developmental stage.[73]

It is noteworthy that the current clinical practice of assessing a suspected bruise is based on both optics (eg, color perception) and pattern matching. Clinical detection and assessment of bruising in children with darker skin tones can be particularly challenging because of the difficulty in visualizing these injuries in some cases.[84,85] Visualization of bruising may be aided with appropriate lighting. It is important to note that some cultural and nontraditional medical practices, such as cupping and coining, can be misdiagnosed as abusive injury, so it is prudent for providers to be aware of how these appear.[86]

Finally, as bleeding disorders are often a diagnostic consideration in children with suspected AHT, a limited number of studies have examined the location, pattern, and number of bruises in children with known bleeding disorders.[87,88] One study looked specifically at the characteristics of bruising in preschool children with inherited bleeding disorders.[88] In this study, there were 5613 bruises recorded from children with inherited bleeding disorders and 3523 bruises from children without a bleeding disorder. Children with severe bleeding disorders had more and larger bruises than children without bleeding disorder at all developmental stages. However, even children with bleeding disorders rarely had bruises on the ears, neck, cheeks, eyes, or genitalia.

Takeaway Points

1. Sentinel injury research has identified bruising as a key sentinel finding in infants, occurring in upwards of 28% of "definite" abuse cases.
2. Although not specific for AHT, and typically benign in many infants, macrocephaly is present in a majority of patients with AHT.
3. Bruising that is more likely to occur in AHT versus nAHT can be distinguished on the basis of a child's age, developmental abilities, and distribution of bruising.
4. Bruising in nonmobile children has a strong association with abuse over accident.
5. Bruising in the TEN-4-FACESp distribution has high sensitivity and specificity for abusive injury. Thus, in general, bruising in such locations, absent a clear and consistent accidental history, warrants an evaluation for possible abuse.
6. Children with severe, inherited bleeding disorders have more and larger bruises than children without a bleeding disorder; however, even children with inherited bleeding disorders rarely bruise on the ears, neck, fleshy cheeks, eyes, or genitalia.

c) Intracranial Hemorrhage/Hematoma

Subdural hemorrhage has been reported to be the most common intracranial manifestation of AHT, occurring in more than 80% in some series.[89–92] In comparative studies, including a 2011 systematic review by Kemp et al, subdural hemorrhage more commonly occurs following abusive rather than accidental head injury.[50,93–101] Duhaime et al found 13 of 16 cases (81%) of subdural hemorrhage resulted from abusive injury in 100 consecutively hospitalized infants and young children younger than 2 years.[96] Reece et al found subdural hemorrhage in 46% (25/54) of definite AHT cases compared with 10% (23/233) in infants and young children with accidental causes.[98] Feldman et al found 76% of subdural hemorrhage that occurred without a readily identifiable cause occurred as a result of abuse. In contrast, epidural hemorrhage more commonly occurs from nAHT.[94–96,99,100] Subarachnoid hemorrhage, which is common in many types of trauma, is reported as not being a distinguishing feature of AHT versus nAHT in several series.[94–96,100,102]

In AHT, subdural hemorrhage is often multifocal, may be both supratentorial and infratentorial in location, but is frequently located over the cerebral convexities or vertex location, and posteriorly along the tentorium and parieto-occipital locations.[92,95,97,103–106] An association between interhemispheric location and abusive head trauma has been shown in multiple studies.[95] The subdural hemorrhage found in AHT may be small in volume, often with a more severe parenchymal insult, although, it also can be larger in volume, requiring emergent surgical decompression.[107]

Traumatic subdural hemorrhage most often results from bridging vein injury, although nontraumatic etiologies also exist.[108–110] Small, localized vertex clots on imaging, typically centered along parasagittal bridging veins, are thought

to represent focal vein injury and were reported in over 80% (45/55) of abusive head trauma patients compared with only 36.4% (8/22) with accidental causes.[111–113] In one series, concurrent findings of retinal and subdural hemorrhages with parasagittal vertex clots significantly increased the odds of a patient having abusive injuries.[111] However, no vertex clots were present in 88 controls who presented with accidental trauma and had no subdural hemorrhage on computed tomography (CT).

d) Edema, Hypoxic Injury, and Cortical Injury

Patterns of cytotoxic edema and hypoxic-ischemic injury are observed much more frequently following abusive head injuries and strongly contribute to the significant morbidity and mortality seen in this population.[95,107,114–116] In a comparative study, patterns of restricted diffusion indicating cytotoxic edema and/or hypoxic-ischemic injury occurred in 37% (11/30) of abusive cases versus 9% (2/22) of those with accidental trauma undergoing brain magnetic resonance imaging (MRI).[107] Of those with abuse, a pattern of diffuse, bilateral hypoxic-ischemic injury predominated, a pattern also described in several other series.[114,115,117,118] However, asymmetric cortical involvement and unilateral hemispheric involvement also may be observed.[116,119] This diffuse pattern of injury/insult is not in a typical vascular distribution and likely has multifactorial causes, including apnea, hypoperfusion, craniocervical injury, and/or excitatory or metabolic cascades resulting in permanent neuronal injury. [120–123] Although parenchymal findings may be subtle on initial CT, changes occurring as early as 72 minutes following the traumatic insult have been described, becoming more conspicuous over the initial 24-hour period.[90]

Parenchymal contusions, lacerations, and traumatic axonal injury also may be seen following abusive head injury, but these traumatic lesions are infrequent.[107,114,124] Parenchymal contusions, when present, often coexist with other traumatic lesions such as skull fractures or adjacent/overlying extra-axial hemorrhage.[107,114,116,117] Slit-like tears or clefts near the cortical-white matter junction, predominantly in the anterior temporal and frontal lobes, occur in young infants and appear as small fluid-like clefts with layering hemorrhage.[109,125] They may occur from inflicted head injuries, but also are described in the perinatal period following instrument-assisted deliveries.[125,126]

e) Spinal Injury

Spinal injuries occur in abusive and accidental trauma and may involve the vertebral column, ligamentous structures, and intraspinal contents.[121,127–131] Cervical ligamentous injury is more common in abusive (78% or 52/67 cases) compared with cases of accidental trauma (46% or 21/46 cases) undergoing spinal MRI.[121] Abused patients with a diffuse pattern of hypoxic-ischemic brain injury increasingly have been shown to have a significant association with MRI findings of cervical spine injuries, occurring in just over 80% of cases in one series.[129] However, spinal injuries are missed on initial clinical evaluation in over 50% of cases, as they may be clinically silent and/or difficult to detect because of coexistent, devastating brain injuries.[128,131]

Spinal subdural hemorrhage was observed in 60% of children with AHT undergoing spinal MRI but was infrequently observed in cases of accidental injury.[128] It may result from tracking of intracranial hemorrhage into the spine rather than from direct spinal trauma.[109]

Unlike ligamentous injuries in abuse, which more commonly occur in the cervical or craniocervical junction locations, spinal fractures from abuse are more commonly located in older children in the thoracic and lumbar spine, with 22 of 25 being spinal thoracic and lumbar compression deformities in a series by Kleinman et al.[127,130–133] Overall, vertebral fractures are uncommon relative to other skeletal injuries in child physical abuse, accounting for between 0.4% and 2.7% of fractures on skeletal surveys in several series. [127,130,131,134–136] However, spinal fractures may be the only additional finding on a skeletal survey to suggest abuse.[136]

f) Ocular Findings

1. Anatomy. The retina is a multilayered structure that forms the inner lining of the eye. It extends from the posterior pole of the eye anteriorly to the front third of the eye (ie, toward the iris). The retina is composed of 10 layers. The innermost layer, the internal limiting membrane (ILM), is the structural interface between the retina and the vitreous, which in young children is a gelatinous structure filling the posterior segment of the eye, and the retinal pigment epithelium is the outermost layer adjacent to the choroid, which is the layer of tissue between the sclera and the retina. The retinal vasculature is a complex network of vessels and capillaries that permeate the inner retina. Retinal hemorrhages most commonly occur within the retinal layers.[137] The posterior pole encompasses the optic nerve and the macula. The region around the optic nerve is called the peripapillary region. The macula is defined clinically as the region between the major temporal vascular arcades. Anteriorly, the ora serrata is the peripheral termination of the retina at its junction with the ciliary body (the part of the eye that connects the iris to the choroid). The retina between the macula posteriorly and the ciliary body anteriorly is referred to as the peripheral retina, which may be subdivided into the near, mid, and far peripheries, where the near periphery is adjacent to the macula and the far periphery is adjacent to the ciliary body.

Blood that has entered the potential space between the vitreous and the retina is referred to as preretinal or subhyaloid hemorrhage, the hyaloid surface being the outer

surface of the vitreous. Such hemorrhages may obscure the underlying retinal structures and appear denser on examination. Hemorrhages found under the retina between the retina and choroid are called subretinal hemorrhages; they commonly appear slightly fainter and partially obscured by overlying structures, such as the retinal vasculature. Mechanical injury to the retina from inertial forces causing traction between the vitreous and the retina results in extravascular leakage of blood that may be trapped in and between the different retinal layers (intraretinal hemorrhage), under the retina (subretinal), or above the retina but under the vitreous (preretinal).[138,139]

The clinical appearance of intraretinal hemorrhage varies depending on the location of the hemorrhage within the retinal layers. Superficial blood within the nerve fiber layer is flame or splinter shaped because of the orientation of fibers, and hemorrhages in the deeper layers of the retina have a dot or blot configuration because of the configuration of cells in those layers. When there is retinoschisis, or splitting of the retinal layers, hemorrhage may collect in the potential space of the retinoschisis cavity and appear as a blister-like cavity. Traumatic retinoschisis in AHT is often between the ILM and the other layers of the retina but may occur between other layers as well.

2. Findings. Although also seen in nAHT, retinal hemorrhages are the most frequent and most sensitive acute sign of AHT. In a systematic review of 20 observational studies (level II-III evidence) that included eye examinations of 973 children who had experienced AHT, retinal hemorrhages were seen in about 80% of children, with a mean sensitivity of 75% for AHT.[137] The extent, distribution, and pattern of retinal hemorrhages and other associated ophthalmic findings determine their specificity, which can be as high as 94% for abuse.[137]

In addition to retinal and vitreous hemorrhages, the ocular findings of AHT may include retinal folds, retinoschisis, papilledema, and optic nerve sheath and orbital fat hemorrhage on autopsy. Vitreoretinal findings of trauma not visible with indirect ophthalmoscopy, such as vitreoretinal traction, vitreous separation, and subclinical retinoschisis, may require specialized retinal imaging (ie, optical coherence tomography) to identify and may be seen on autopsy in fatal injuries.[140–142] Poor vision on presentation, decreased pupillary response, papilledema, and severe retinal hemorrhages and retinal folds are ophthalmologic findings of AHT associated with increased mortality.[143–146] Longer-term sequelae associated with AHT include optic nerve atrophy and retinal scarring.

Retinal folds appear as arcuate or circular hypopigmented ridges or lines, usually around the macula and less commonly in the more peripheral retina. They may be found at the edges of an area of traumatic retinoschisis but may also occur in the absence of a clinically evident schisis cavity.[147–149] Retinal folds may be unilateral or bilateral. They occur in about 3% of clinical cases of AHT, but the incidence increases to 23% to 42% in severe or fatal cases of AHT.[137,147]

Retinal folds or lines around the schisis cavity may help differentiate it from subhyaloid blood. A "hemorrhagic macular cyst" is a dense macular schisis hemorrhage, sometimes with a meniscal interface between red blood cells and serum.

Papilledema, a term referring to optic disc swelling specifically attributable to raised intracranial pressure, is found in less than 10% of patients with AHT.[150] It usually takes a minimum of 12 to 24 hours to develop in patients with increased intracranial pressure following head injury; therefore, if noted on initial presentation, it may suggest delay in seeking care after the injury occurred. Papilledema is an ominous finding associated with high mortality.[143,151]

3. Mechanism. Retinal hemorrhages arising from head trauma may be caused by one or multiple mechanisms. Vitreoretinal traction from head and ocular acceleration/deceleration injury, as would be caused by shaking and/or head impact, is the leading mechanism, supported by clinical, imaging, and animal study evidence.[140,141] The vitreous is attached to the retina throughout the interface between the 2 structures, but there are areas of particularly strong attachment, which are directly perceptible by surgeons at the time of vitreoretinal surgery. These areas include the posterior pole, especially around the optic nerve head and along the major vascular arcades, and anteriorly at the vitreous base, which straddles the ora serrata and attaches to both the far peripheral retina and the adjacent ciliary body. Retinal hemorrhages in abusive head trauma often appear predominantly in these areas, sometimes producing a pattern in which hemorrhages are in the posterior pole and in the far retinal periphery but relatively sparce in the mid-peripheral retina.[152]

The mechanism of traumatic retinoschisis in AHT is vitreoretinal traction generated by firm adhesions between the vitreous and the posterior retina as the child's head is submitted to acceleration-deceleration forces. This phenomenon results in in splitting of the retinal layers.[140] Most commonly, this split is between the ILM and underlying retinal layers. Damage to blood vessels traversing the split layers may result in blood filling the schisis cavity. Perimacular retinal folds likewise occur in an area of strong vitreoretinal attachment. The resulting retinoschisis results from traction, and optical coherence tomography provides direct visualization and evidence of vitreoretinal traction.[140–142] Animal studies of inertial injury also demonstrate ocular hemorrhages in areas of strong vitreoretinal attachment in the animals, posteriorly and anteriorly at the vitreous base.[153]

Birth-related retinal hemorrhages occur in patterns often resembling those of AHT or nAHT.[154–156] The mechanism of these hemorrhages is not established. However, vitreoretinal traction may be one cause if direct eye compression causes distortion and lengthening along a single axis. Other proposed mechanisms include ocular compression with prolonged highly raised intraocular pressure and subsequent decompression retinopathy, which is known to cause retinal hemorrhage in patients with glaucoma with acutely lowered intraocular pressure following glaucoma surgery, and release of leukotrienes or other vasoactive factors resulting in increased retinal vessel permeability.

Central retinal venous occlusion, which is a common cause of retinal hemorrhage in adults, is another possible contributing cause in AHT. However, central retinal vein occlusion produces a characteristic radiating pattern of retinal hemorrhages that is usually absent in head trauma and is accompanied by retinal venous tortuosity and optic disc swelling, which also are not commonly present in head trauma.[151] Branch retinal venous occlusion produces a sectoral pattern not reported in head trauma. Increased intracranial pressure, whether acute or subacute, does not produce retinal hemorrhage in patterns seen in AHT and is discussed below.

4. Incidence. As stated earlier, retinal hemorrhages are seen in about 80% (45%–100%) of children with AHT.[137] The prevalence of retinal hemorrhages in healthy children (ie, without trauma or other medical cause) is estimated at less than 0.1%.[157] A study of 7758 retinal examination of children between 1 and 24 months of age suggests that retinal hemorrhage does not spontaneously occur in healthy children after the postnatal period; even a single hemorrhage should be considered of medical significance and a plausible etiology sought.

Retinal hemorrhages in AHT occur along a spectrum of severity, ranging from mild to severe. Hemorrhages are usually bilateral, although they can be asymmetric or completely unilateral in up to 30% of cases.[137] In approximately two-thirds of cases of AHT, the hemorrhages are too numerous to count, are multilayered, and extend into the retinal periphery, often to the ora serrata.[143] Patients may have vitreous hemorrhage that can obscure the underlying retinal hemorrhages. However, retinal hemorrhages associated with AHT can also be few in number, exclusively intraretinal, and confined to the posterior pole.[58,150–152,158–160]

Retinal hemorrhages are rare in young children with normal neuroimaging and no evidence of head trauma on physical examination.[159,161,162] In one study of 190 children evaluated for AHT, all 15 children with retinal hemorrhages had positive neuroimaging, and none of 85 children with negative neuroimaging had retinal hemorrhages.[162] In a larger study of 353 children without traumatic brain injury (TBI) on neuroimaging, retinal hemorrhages were found in only 2 children (0.6%; 95% CI, 0.1–2.0).[163] Similarly, in one study of 78 infants with isolated long-bone fractures and dilated fundus examinations, none had retinal hemorrhages.[164] This evidence supports the conclusion that dedicated retinal examinations are not needed in children without evidence of traumatic head injury.

5. Signs and symptoms. Children with AHT commonly show no externally visible ocular sign or symptom.[143,165] Signs of external eye trauma, such as periorbital ecchymosis, eyelid edema, or subconjunctival hemorrhage, usually indicate direct trauma to the face or eye, as opposed to inertial or impact injury to the head.[166,167] Corneal injuries, hyphema (blood in the anterior chamber), secondary glaucoma, lens subluxation, traumatic cataract, and pupillary abnormalities have all been reported as a result of child physical abuse. Anterior segment injuries, when present, are rarely isolated and tend to be associated with severe trauma and a poor prognosis.

6. Description. The term "multilayered retinal hemorrhages" refers to hemorrhages that are present at multiple locations (intra, preretinal, and/or subretinal).[151] Hemorrhage within the vitreous (vitreous hemorrhage) may appear as more indistinct localized globules of hemorrhage or sometimes appears as diffuse dispersed hemorrhage. Retinal hemorrhages are detected and characterized with examination of the retina and vitreous best performed through a dilated pupil using an indirect ophthalmoscope, which provides a binocular, 3-dimensional view of the posterior segment of the eye and permits appreciation of the physical locations and morphologic characterization of these different types of hemorrhages. Vitreous hemorrhage, when present, may partially or fully obscure a view of retinal hemorrhages or the retina itself. Specification of these various configurations and locations within the posterior pole is important, because it helps to describe the geographic distribution and patterns of retinal hemorrhage associated with different causes. Of note, some patients may have "white-centered" retinal hemorrhages.[168] Such hemorrhages are not diagnostic of septic emboli or of an infectious origin. White-centered retinal hemorrhages, in fact, are nonspecific and may be caused by central clearing of the blood within any intraretinal hemorrhage from any cause, or artifactual light reflection from an ophthalmoscope or retinal camera. Thus, describing such findings as "Roth spots," a historical eponym associated with endocarditis and septic emboli, can be misleading.

7. Differential diagnosis of RHs. i) Trauma. Although many systemic and ocular conditions may result in retinal hemorrhages in children, the most common cause of retinal hemorrhage in infants and young children is trauma,

birth-related in the first month of life; AHT after the first month; and less commonly, nAHT.[137,155,156,169–171]

The pattern and distribution of the hemorrhages, presence of other retinal findings, clinical history, nonocular physical examination, and laboratory tests help to distinguish traumatic from nontraumatic causes of retinal hemorrhage. Vitreous hemorrhage may also be present in AHT but is less commonly seen than retinal hemorrhage. Retinoschisis and retinal folds are also less common but are highly specific for severe trauma, including AHT.[137] Apart from their diagnostic significance, retinoschisis and retinal folds are indictors of poor prognosis and associated with severe neurologic injury and mortality.[143,144] Retinal detachment is another less common sign and may signify a late presentation.[172]

Generally, increased severity of retinal hemorrhages correlates with increased severity of head trauma.[150,173] Extensive hemorrhages that are "too numerous to count," found at multiple layers, and extend to the peripheral ora serrata are highly specific for severe head trauma, including AHT and unambiguous severe nAHT.[40,174,175] However, the features of multilayered or peripheral RHs in isolation do not define a level of severity and, in and of themselves, are not diagnostic of AHT; the number and geographic distribution must also be considered.[158] Retinal hemorrhages are less common in AHT characterized only by blunt impact, similar to the low incidence observed in accidental falls.[137,151] Retinal hemorrhages occasionally occur from witnessed accidental "household" falls (ie, typically representing low-height or short-distance falls), but in this setting typically are sparse and not in the pattern more specific to AHT.[93,96,175] Retinal hemorrhages tend to occur more frequently in children who have died versus unimpaired survivors.[143,150,151,176,177]

A systematic review of 20 observational studies comprised ocular findings in 1948 children, including 242 children with nAHT and 973 children with AHT as determined by perpetrator confession, third-party witness, legal decision, autopsy findings, or multidisciplinary assessment.[137] This review found that intraocular hemorrhage was present in 44% to 100% of children with AHT but in less than 10% of children with nonabusive head injury. It should be noted that the children with accidental trauma in most comparative series typically have different types of intracranial injuries than do children with AHT, only infrequently having subdural hemorrhages. However, with this caveat in mind, the sensitivity of intraocular hemorrhages for AHT was 75%; the specificity for AHT was 94%. The specificity for AHT was highest for intraocular hemorrhage that was bilateral, multilayered (preretinal in addition to intraretinal), extended beyond the posterior pole, and of moderate to severe number/extent.

In addition to the multilayered, numerous in number, and extending to the periphery retinal hemorrhages, retinal folds and retinoschisis are also highly specific for severe head trauma, most commonly AHT.[178–180] They also have been reported after unequivocal severe accidental head injury, such as fatal motor vehicle crashes, head crush injury, and high-elevation (eg, 10 m) falls, and more rarely in association with certain established medical diagnoses, including a ruptured intracranial aneurysm and leukemia.[152,181] Thus, when macular retinoschisis or perimacular retinal folds are present in a child without a history of major accidental head trauma, the likelihood of AHT is very high. The presence of retinal folds typically implies severe injury that often results in death or neurologic sequelae, although some surviving patients may experience good visual recovery.[143,144]

ii) Increased intracranial pressure. Raised intracranial pressure causes specific patterns of retinal hemorrhages that are different from head trauma. Raised intracranial pressure is associated with superficial intraretinal hemorrhages on or adjacent to a clearly swollen optic nerve head and, therefore, are in a peripapillary distribution.[182] Current evidence and extensive clinical experience with patients with raised intracranial pressure unrelated to trauma does not support raised intracranial pressure as a cause of widespread or multilayered retinal hemorrhages.[183,184] A very rapid elevation of intracranial pressure resulting from intracranial hemorrhage can cause intraocular hemorrhage in a condition referred to as Terson syndrome.[185–190] The pattern of hemorrhages in Terson syndrome is mainly of vitreous and preretinal hemorrhage adjacent to the optic nerve head or in the posterior pole with no or few intraretinal hemorrhages. Terson syndrome is uncommon in infants and children and does not refer to a specific cause, such as a ruptured arteriovenous malformation.[151,191,192]

iii) Other systemic illness. Patients with severe coagulopathies may rarely be found to have retinal hemorrhages, which are usually few in number and confined to the posterior pole; although they may be more widespread in children with leukemia.[193] It is not clear to what degree coagulopathy might worsen the severity of retinal hemorrhage attributable to head trauma, but even coagulopathy is not known to cause spontaneous extensive retinal hemorrhages.[194] Sepsis, meningitis, and endocarditis may be associated with hemorrhages that are typically intraretinal and confined to the posterior pole but can also be more extensive.[195–197] In a prospective case series of 159 critically ill children without abusive head injury receiving ophthalmologic evaluation within 48 to 72 hours of admission, the prevalence of retinal hemorrhages was 15% (95% CI, 9.5%–21%).[193] Most of these children had fewer than 5 single-layered retinal hemorrhages, but 6 children (3.7%) were found to have more numerous or multilayered hemorrhages and had diagnoses of fatal nonabusive traumatic

brain injury, severe coagulopathy, and/or sepsis associated with leukemia.[193] In retinitis attributable to cytomegalovirus, retinopathy of prematurity, and hypertensive retinopathy, retinal hemorrhages are accompanied by characteristic retinal lesions such as cotton wool spots, white retinal patches, lipid exudates, and neovascular tissue.[151] Generally, in medical causes of retinal hemorrhages, accompanying diagnostic clinical history, examination, and laboratory findings are present.

iv) Cardiopulmonary resuscitation. Cardiopulmonary resuscitation (CPR) uncommonly causes retinal hemorrhages in children, with a reported prevalence of approximately 3%.[193,198–201] In these cases, the retinal hemorrhages are few in number, single-layered, and confined to the posterior pole. In most instances there is an associated coagulopathy. Severe and abrupt increases in intrathoracic pressure may cause Purtscher retinopathy, which is characterized by white retinal patches and retinal edema most commonly around the optic disc, with retinal hemorrhages as a minor feature, and not the pattern strongly associated with AHT.[202]

v) Seizures, coughing, vomiting, and others. In multiple studies, severe coughing, forceful vomiting, and seizures have not been found to cause retinal hemorrhages.[203–207] Vaccinations also have not been found to cause retinal hemorrhages.[157] Diabetic retinopathy, a common cause of intraretinal hemorrhages in adults, does not occur in children at this age.[208] Glutaric acidemia type 1 (GA1) is an organic acidemia caused by biallelic pathogenic variants in the GCDH gene that is sometimes associated with retinal hemorrhages.[161] Case reports describing retinal hemorrhages in this disease entity mention few pre- or intraretinal hemorrhage confined to the posterior pole.[161] Neurosurgical procedures that might result in sudden changes in intracranial or intravascular pressure have not been found to be associated with retinal hemorrhages and are unlikely to confound the interpretation of retinal findings in children being evaluated for AHT who recently underwent neurosurgery.[209]

8. Dating retinal hemorrhages. Retinal hemorrhages cannot be accurately dated to determine the precise timing of the traumatic event. However, some conclusions can be drawn on the basis of the known rates of resolution of hemorrhages, depending on the type, size, location, and number of hemorrhages. Resolution timing ranges from less than 24 hours for some intraretinal hemorrhages to several months for nonclearing vitreous hemorrhage.[145] The resolution of retinal hemorrhages has been studied both in newborn infants and in children with nAHT or AHT, with identical findings in both settings with regard to the rate of resolution. General characteristics are as follows:

- Intraretinal hemorrhages resolve very quickly, starting within 24 to 48 hours, with numerous hemorrhages clearing within just days, significantly fewer hemorrhages remaining at 1 week, and nearly all hemorrhages clearing within 2 weeks. Isolated (single) dense intraretinal hemorrhages can persist longer, up to 2 months. On the basis of these findings, the presence of too-numerous-to-count intraretinal hemorrhages suggests an injury that has occurred recently, typically in the previous few days.[156,210]
- Preretinal hemorrhages can take considerably longer to clear, on the order of weeks or months.
- Intraretinal hemorrhages are uniformly present to some degree at presentation when retinal hemorrhages are present. Therefore, initially, preretinal hemorrhages in AHT are always accompanied by intraretinal hemorrhages. However, because they take longer to clear, the presence of preretinal hemorrhages with few or no intraretinal hemorrhages suggests a more chronic injury.[210]
- Because of variability in rate of resolution, the presence of bright red hemorrhages along with fading hemorrhages does not necessarily imply separate traumatic events.
- Blood in a schisis cavity and subretinal hemorrhage resolves more slowly; vitreous hemorrhage is the slowest to resolve.
- It takes time for retinal scars and optic atrophy to develop, so their presence suggests a distant previous injury.

TAKEAWAY POINTS

1. Retinal hemorrhages do not spontaneously occur in healthy children after the postnatal period.
2. Retinal hemorrhages are the most frequent and most sensitive acute ocular sign of AHT; they can be bilaterally symmetric, bilaterally asymmetric, or unilateral.
3. Retinal hemorrhages that are numerous, present in multiple layers of the retina, and extend peripherally beyond the posterior pole are highly specific (94%) for severe head trauma and particularly AHT.
4. Retinal hemorrhages cannot be accurately dated to determine the precise timing of the traumatic event, but the rate of resolution of different types of retinal hemorrhage can sometimes provide general guidance as to injury timing.
5. Retinal hemorrhages are rare in the absence of neuroimaging findings or other evidence of head trauma; thus, dedicated retinal examinations are not needed in children evaluated for physical abuse in whom there is an absence of neuroimaging findings or other evidence of head trauma.
6. Other ocular clinical findings may include vitreous hemorrhage, retinal folds, retinoschisis, and papilledema. Autopsy findings may also include optic nerve sheath

hemorrhage and orbital fat hemorrhage. With specialized retinal imaging (OCT), vitreoretinal traction, vitreous separation, and subclinical retinoschisis can also be identified.
7. Papilledema occurs infrequently in AHT (less than 10%).
8. Nontraumatic causes of retinal hemorrhages usually occur in patterns different from head trauma.

g) Skeletal findings. 1. Skull fractures. Like bruising, skull fractures are commonly seen in both AHT and nAHT. Skull fractures are identified in 25% to 40% of AHT cases.[90,94,211] In nAHT cases, skull fractures are most commonly linear and involve the parietal bone.[212] Isolated skull fractures, in general, while indicative of trauma, are not differentiating features between AHT and nAHT.[12,93,213–217]

Skull fractures may not be associated with major signs or symptoms at the time of the traumatic event, and although scalp swelling is common, not all skull fractures are associated with scalp swelling. Approximately 10% of children with skull fractures have no clinically appreciable or radiographic evidence of scalp or facial soft tissue swelling at the time of presentation.[218] It should be noted, however, that because scalp swelling itself can be an indication for imaging, the true incidence of skull fractures without associated scalp swelling remains unknown.[219–221]

Linear skull fractures may be associated with delayed onset of apparent scalp "swelling" as scalp hematomas liquefy and become more protuberant. This can occur days to several weeks after fracture with a median of 3 days, and is sometimes the only sign associated with a recent fracture. Absent scalp swelling does not necessarily imply a delay in seeking care, especially in active toddlers who may fall frequently with minimal or transient symptoms.[222–224]

It is unclear whether skull fractures in conjunction with intracranial hemorrhage are differentiating features of accidental versus AHT etiologies. Piteau et al found skull fractures plus intracranial injury to have an OR for abuse of 7.76 (95% CI, 1.06–57.08) in children 6 years or younger.[50] Yet, Maguire et al found that skull fracture and intracranial injury in children younger than 3 years had a PPV for AHT of 44% (95% CI, 22%–68%) and an OR of 0.8 (95% CI, 0.3–2.3).[71] Large comparative studies seem to indicate that it is not the fracture itself that is discriminatory but the presence and severity of intracranial injury that was associated with AHT (OR, 2.38; 95% CI, 2.09–2.70).[225]

It is well known that short-distance falls can rarely result in linear, parietal skull fractures with a small underlying extra-axial (most commonly epidural) hemorrhage or focal parenchymal contusion. However, nonfocal (ie, not underlying the skull fracture site) hemorrhage and/or more severe brain injury is an uncommon finding in short-distance falls and other minor accidental trauma.[226,227]

Finally, some older studies suggested that certain features of skull fractures—multiple skull fractures, depressed skull fractures, or skull fractures crossing the suture lines—are more strongly associated with or specific for AHT.[68,228] However, other studies have not confirmed these findings as differentiating features.[212]

2. Rib fractures. The incidence of rib fractures in AHT cases is less well-studied, but can occur in up to 28% of cases.[12,229] Multiple studies have found a strong association of rib fractures with AHT over nAHT.[12,50,51,68,71,217,230,231] Two separate meta-analyses concluded that in a child with intracranial injury and rib fracture, the ORs for AHT were in the range of 3 to 10.[50,71] Another meta-analysis on distinguishing abusive from accidental fractures in all children younger than 48 months (not just confined to AHT cohorts) concluded that, "in those aged less than 18 months with rib fractures the odds ratio (OR) for abuse was 23.7 (95% CI, 9.5, 59.2)" and "the positive predictive value for rib fractures in relation to suspected or confirmed abuse is 66% (95% CI, 42.5–89.7)."[232] Limitations to the larger meta-analyses include small numbers of rib fractures recorded in some studies and use of an imputation strategy to account for the fact that not all cases of nAHT had skeletal surveys.

Rib fractures associated with AHT can occur along the entire anatomic rib arc—anteriorly, anterolaterally, posterolaterally, and posteriorly.[233] The mechanism for rib fractures is predominantly anteroposterior compression of the thoracic cavity.[234] Although not exclusively specific for AHT, posterior fractures at the costovertebral junction have a high specificity for abuse and are thought to result from a squeezing injury leveraging the posteromedial rib over the spinal transverse process.[235] In a review of 1463 fractures in 141 infants who were abused, costovertebral junction rib fractures were the single most common site for a rib fracture; however, the majority of rib fractures were in a location other than the costovertebral junction.[236] Costovertebral junction fractures are much less common in nonabuse cases. When rib fractures occur unilaterally and are located either anteriorly or laterally, then blunt trauma to the chest can also be considered. Finite element modeling studies have further supported the anteroposterior compression mechanism because of their demonstration of stress profiles consistent with fracture locations seen in AHT cases.[212]

Birth trauma and CPR are potential differential diagnostic considerations in suspected AHT cases. In both circumstances, rib fractures are an extremely uncommon occurrence.[237–240] In CPR, rib fractures are almost always anterolateral. Although some evidence argues that 2-handed techniques of CPR may increase the risk of posterior rib fractures, the occurrence of multiple posterior fractures remains rare.[236,240–243]

3. Long bone fractures. Long bone fractures occur in approximately 17% to 36% of AHT cases.[12,120,217] A large meta-analysis aiming to distinguish abusive from nonabusive fractures in children younger than 15 years (and not specifically in AHT cohorts) found that age and fracture locations can be discriminatory features between patients who have been abused and those who have not.[232] Fractures were much more common in children younger than 18 months who were abused than in those who were not.

In children younger than 18 months, tibia/fibular fractures had an OR of 12.8 (95% CI, 5.1, 32.6) for abuse.[232] And radial/ulnar fractures had an OR of 5.8 (95% CI, 2.4, 14.3) for abuse in those younger than 4 years. Abusive femoral fractures are more likely to occur in children who are not yet walking, and spiral or oblique humeral fractures in children younger than 5 years more commonly occur in abused children. However, supracondylar humeral fractures in children are more associated with accidental injury. Finally, metaphyseal fractures, or classic metaphyseal lesions, are more commonly described in abuse than in accidents.[232]

The aggregate of these data demonstrates that in a child with intracranial injury and long bone fracture, the positive predictive value for AHT was 59% (95% CI, 48%–69%) and OR 1.7 (95% CI, 0.8–3.6).[72] However, as with rib fractures, these larger meta-analyses are limited by small numbers of long bone fractures recorded in some studies and the imputation strategy used to account for the fact that not all cases of nAHT had skeletal surveys.

TAKEAWAY POINTS

1. Isolated skull fractures, in general, while indicative of trauma, are not differentiating features between AHT and nAHT.
2. No skull fracture type is pathognomonic for AHT, and short-distance household falls frequently present with linear skull fractures, with parietal location most common.
3. Two separate meta-analyses have concluded that, in a child with intracranial injury and rib fracture, there are significant ORs for AHT (in the range of 3 to 10).
4. The primary mechanism for rib fractures is anteroposterior chest compression, and rib fractures associated with AHT can occur along the entire anatomic rib arc.
5. Although a diagnostic consideration, rib fractures related to birth and CPR are an extremely uncommon occurrence.
6. In a child younger than 18 months, femoral, humeral, radius/ulna, and tibia/fibula fractures are much more common in children who have been abused than those who have not.

BIOMECHANICS

Introduction

Determining whether injuries are attributable to accident or abuse in children requires a multidisciplinary team; *if available*, engineers with appropriate expertise can play an important role on this team. Although engineers with injury biomechanics expertise can aid in determining whether a child's head injuries could have been caused by the caregiver-provided history, they are uncommonly involved with daily clinical pediatrics. When available, these engineers should have expertise in pediatric injury biomechanics that includes experience with children who have had injuries attributable to accidental causes, as well as experience with children who have been injured from abuse. It is important for engineers to be knowledgeable of the types of injuries that can result from accidental causes (eg, household falls) in children to be able to effectively differentiate them from abusive injuries.

The Importance of Biomechanical Compatibility of Injury With History

An engineer's role is to determine *biomechanical compatibility* between the caregiver-stated cause and the *constellation of injuries*.[244,245] In the presence of a short-distance fall history, determining biomechanical compatibility rests on answering whether the single fall described by the caregiver biomechanically accounts for *all* of the child's injuries. A comprehensive evaluation of biomechanical compatibility will account for *all injuries* and not just the severe or fatal head injuries.

To determine biomechanical compatibility between the stated cause of injury and the constellation of injuries, a comprehensive biomechanical assessment that includes evaluating biodynamic compatibility and a quantitative biomechanical assessment is needed.[244] For example, if a short-distance fall history is provided, the biodynamic compatibility evaluation must determine whether the fall dynamics (how a child's body falls from their initial pre-fall position to their final post-fall resting position) can account for all of the child's injuries (including all bruises, lacerations excepting those associated with medical intervention) and the evidence of impact to the body. Soft tissue injuries and fractures (in most cases) are indicative of impact and provide a roadmap of the child's exposure to forces. Unless a child impacts an object during a fall descent, evidence of impact (eg, bruise, fracture) typically is limited to 1 or 2 adjoining planes of the body. [246–248] For example, if an infant fell from a bed directly onto a hardwood floor landing in a prone position, evidence of impact would be expected to be limited to the anterior plane of the body. If the child was found to have bruising on 3 or 4 planes

of the body, the bed fall history would be biomechanically incompatible with these injuries.

When a quantitative biomechanical assessment is warranted, engineers attempt to reconstruct the fall to estimate levels of acceleration, velocity, and forces applied to the body and head using physics-based calculations, anthropomorphic test device (ATD; test dummy) experiments, and/or computer simulations. These estimates of biomechanical measures can then be compared to pediatric head injury thresholds and corroborated or video-recorded events with documented head acceleration/velocity/forces and known injuries to estimate the likelihood of injury. However, it is important to remember various techniques used to estimate biomechanical measures have advantages/disadvantages, and limitations must be considered.[244]

Lastly, a critical review of the evidenced-based injury biomechanics and clinical scientific literature will assist in determining whether the assessment of likelihood of injury is consistent. Combining findings from their biodynamic compatibility and quantitative biomechanical assessments, along with review of the evidenced-based scientific literature, will enable engineers to formulate an opinion as to the *biomechanical compatibility* of the constellation of injuries and caregiver history. This opinion is best framed as either *biomechanically compatible, likely biomechanically compatible, indeterminate biomechanical compatibility, unlikely biomechanical compatibility, or biomechanically incompatible.*

Types of Injury Biomechanics Studies Used to Investigate AHT

Various types of injury biomechanics studies are used to investigate injury risk, to evaluate the effectiveness of safety devices, and to study how injuries may occur, how the human body responds to accelerations/forces, and which factors may influence injury risk. These studies can be categorized as in-depth event investigation and/or reconstruction, human surrogate experiments, computational models and simulations, animal injury models, and postmortem human subject (PMHS) experiments.[249–251] It is important to remember, like all scientific studies, biomechanical studies have limitations, each of which must be carefully considered when drawing conclusions regarding likelihood of injury.

In-Depth Event Investigations and/or Dynamic Reconstructions

In-depth event investigations and/or dynamic reconstructions can provide an improved understanding of injuries resulting from a specific event and injury causation. Identification of environmental or human factors related to injury causation are often the goal of in-depth investigations. Reconstructions of event dynamics (eg, fall dynamics) can be accomplished using physics-based software or manual physics-based calculations to estimate key biomechanical measures (eg, head acceleration) associated with the event.[226]

Human Surrogate Experiments

A brief summary of human surrogate studies is provided in Table 1. Human surrogate experiments utilize ATDs or surrogates to represent a specific population (eg, age, mass percentile). Surrogates are a mechanical analogue of a human and provide opportunities to measure mechanical quantities such forces, strains, and accelerations during exposure to impact, accelerations, or other potentially injurious conditions using onboard instrumentation (eg, accelerometers, force sensors). ATDs are designed to represent the anthropometrics (geometry), mass, and mass distribution of the target human. It is extremely important they be as *biofidelic* (human-like) as possible in their response. The reliability and accuracy of biomechanical measures obtained from surrogate experiments are highly dependent on their biofidelity.

Although human surrogate studies have advanced our understanding of pediatric head biomechanics in shaking, forceful impact, and falls involving infants and children, some limitations exist and can influence interpretation of findings. It is important to note that differences in surrogate design can yield substantial differences in outcomes.

Biofidelity of the human surrogate is critical to the accurate prediction of injury probability, regardless of the mechanism (eg, falls, shaking, etc) being investigated.[252] Biomechanical properties of the neck and spine (ie, stiffness or flexibility) are particularly important when investigating head injury risk. A flexible and multisegmented neck and spine are key to achieving infant and child-like kinematics (motion of the body). Slight changes in surrogate design (eg, neck properties) can result in different biomechanical outcomes and thus, differing predictions of head injury risk.[253,254] Limitations related to surrogate biofidelity can influence findings from surrogate experiments.[252,255] Although considerable progress has been made to account for more of the details of these properties in infants of different ages, the exact biomechanical properties of the cervical spine of infants during maturation and with different characteristics remain incompletely understood. For this reason, although models can "bracket" the likely range of possibilities, it is not yet known with complete certainty how to create an exact biofidelic model for children of a specific age or to model a specific injury scenario.

The level of surrogate biofidelity attainable is dependent on the availability of biomechanical properties (ie, how the body responds to dynamic loading) of infants and young children, along with our ability to design mechanical analogues matching those properties.[255]

Surrogate studies cannot predict the injurious effects of repeated or ongoing cumulative trauma that may occur in AHT. Nor can they predict the independent injurious effects

TABLE 1. Human Surrogate Studies

1st Author, Date	Title	Age	Surrogate Design	Mechanism Investigated	Findings	Limitations
Duhaime, 1987	The shaken baby syndrome. A clinical, pathological, and biomechanical study.	1 mo	Just Born Doll with weighted thorax, modified head, neck and skull. Head was filled with cotton and water to achieve desired mass. 3 different neck designs (flexible rubber, stiff rubber, hinged) and thermoplastic skull vs no skull were evaluated.	Shaking, forceful impacts	Accelerations due to impact were approximately 50 times greater than those associated with shaking. SBS, at least in its most severe form, is not usually caused by shaking alone. Although shaking may be a part of the process, it is more likely that such infants experience blunt impact.	Unknown surrogate mass distribution, anthropometrics, neck properties, thorax properties, and joint properties. Extremities had negligible mass. Unclear whether chin-to-chest or occiput-to-back contact occurred.
Cory, 2003	Can shaking alone cause fatal brain injury? A biomechanical assessment of the Duhaime shaken baby syndrome model.	1 mo	Just Born Doll (same as used by Duhaime, 1987) with adjustable design parameters. Investigated varying head-neck joint location (ie, occipital condyle location), neck construction (rubber neck vs hinged neck), torso padding (cotton wool vs silicone), and location of the surrogate center of gravity (CG).	Shaking	Altering certain surrogate design parameters led to increased head accelerations that exceeded those measured by Duhaime et al (1987). Chin-to-chest and occiput-to-back contacts exceeded impact thresholds. Cannot categorically be stated 'pure shaking' cannot cause fatal head injuries in infant. "There must now be sufficient doubt in the reliability of the Duhaime et al (1987) study to warrant the exclusion of such testimony in cases of suspected shaken baby syndrome."	Unknown surrogate mass distribution, anthropometrics, neck properties, thorax properties, and joint properties.
Prange, 2003	Anthropomorphic simulations of falls, shakes, and inflicted impacts in infants.	1.5 mo	Custom-designed surrogate included torso, hinged neck, and head. Head from a toy doll (La Baby) with polypropylene skull and rubber scalp. Extremities were not represented. Mass of arms and legs incorporated into the torso. Torso was constructed of single wooden block.	Shaking, forceful impact, falls	Angular accelerations and velocities associated with shaking were not sufficient to cause trauma to an infant. Inflicted impacts against hard surfaces are more likely to be associated with inertial brain injuries than falls from a height of <1.5 m or from shaking. There are no data demonstrating that the angular acceleration and angular velocity experienced during shaking and impact against a padded surface … cause SDH or TAI in infants. Vigorous shakes produced rotational responses similar to those resulting from minor falls, but inflicted impacts produced responses that were significantly higher.	Surrogate torso was rigid and lacked any spine flexibility, which led to unrealistic kinematics. Extremities were not included in surrogate. Neck motion was limited to the sagittal plane.

(Continued on next page)

TABLE 1. Human Surrogate Studies (Continued)

1st Author, Date	Title	Age	Surrogate Design	Mechanism Investigated	Findings	Limitations
Lloyd, 2011	Biomechanical evaluation of head kinematics during infant shaking versus pediatric activities of daily living.	12 mo	CRABI-12 and National Center for SBS doll 7-month-old child	Shaking, activities of play	Aggressive shaking and resuscitative shaking are not likely to be the primary cause of DAI, primary RH, or SDH in a healthy infant. Rotational accelerations during shaking of ATD were indistinguishable from the accelerations endured by a 7-month-old boy at play.	CRABI-12 ATD designed for evaluating injury risk in seated posture in high-energy events (motor vehicle crashes). CRABI-12 ATD neck is overly stiff compared with an infant's neck. CRABI-12 ATD does not have a segmented spine and is overly stiff. Overall kinematics of shaking CRABI-12 ATD differ greatly from those expected in a child due to overly stiff neck and spine.
Miyazaki, 2015	The mechanism of shaken baby syndrome based on the visualization of intracranial brain motion.	12 mo	CRABI-12 with modified head, including transparent skull, deformable brain, CSF, falx, tentorium - reconstructed from CT images.	Shaking, falls	Relative displacement between skull and cerebrum were measured. Displacement of brain with respect to skull exceeded bridging vein (BV) rupture thresholds during shaking. Potential mechanism for brain injury is reverse rotation of brain relative to skull following chin-to-chest or occiput-to-torso contact. Peak BV stretch occurred just following maximum extension of head/neck when skull reverses direction of rotation. Relative brain motion associated with falls (0.4 m to 1.2 m height) generated BV stretch below thresholds.	CRABI-12 ATD neck is overly stiff compared with an infant's neck. CRABI-12 ATD does not have a segmented spine and is overly stiff. Overly stiff neck and spine led to unrealistic kinematics. Uncertainty regarding mechanical properties of the brain and its subregions.
Jenny, 2017	Biomechanical response of the infant head to shaking: An experimental investigation.	5th percentile Japanese newborn	Aprica 2.5 - custom designed 12 segment surrogate, including flexible 2-segment spine (thoracic and lumbar regions). Mechanical properties of surrogate documented using standardized ATD testing procedures - documented elsewhere.	Shaking	Angular head accelerations and velocities exceeded those measured by Duhaime et al and Prange et al. Maximum angular head accelerations and velocities occurred during chin-to-chest contact and suggest higher potential for injury in shaking than previously reported.	Mechanical properties of thorax not reported but available in other related publications. Mechanical properties of mandible not reported.

ATD indicates anthropomorphic test device; CSF, cerebrospinal fluid; CT, computed tomography; DAI, diffuse axonal injury; SBS, shaken baby syndrome; SDH, subdural hemorrhage; TAI, traumatic axonal injury.

of a combination of mechanisms (ie, shaking combined with forceful impacts or throwing a child). Cumulative deleterious effects associated with multiple mechanisms cannot currently be assessed using human surrogates.

To predict likelihood of brain injury from surrogate experiments, biomechanical outcomes such as head angular and translational accelerations are compared with available injury thresholds.[255,256] Unfortunately, published pediatric brain injury thresholds vary widely, depending on the injury model (eg, adult primates or immature swine or other animals), the techniques used to scale from adult brain size to child brain size (eg, mass, strain, geometry),

the test conditions used to arrive at thresholds (eg, impact, indirect loading), and the injury type (eg, subdural hemorrhage, cerebral concussion).[257] Scaling from adult injury models may fail to capture anatomical differences of the immature skull (eg, open fontanelle) and brain (eg, composition, extent of myelination).[258,259] Additionally, the physiological response of an immature brain to dynamic loading has also been shown to differ from that of an adult brain, further undermining injury predictions based on scaled adult thresholds.[260]

Computational Models and Simulations

Computer models incorporating 3-dimensional (3D) digital humans or body segments (eg, head) in a virtual environment (eg, fall environment, motor vehicle crash) can be used to simulate potentially injurious events such as falls.[261–264] Anatomic accuracy and biofidelic response are key in the development of 3D digital human models. Similar to surrogates, 3D human models may be equipped with virtual sensors to predict biomechanical measures. The motion and response of human models are governed by user-defined body characteristics and the laws of physics; they are not animations.

Animal Injury Models

An overview of studies using animal models to investigate AHT is provided in Table 1. Animal models have been used to gain further understanding of injury mechanisms and injury thresholds since this information is otherwise not attainable using human subjects. Ensuring translatable outcomes to humans and animal welfare are key to animal injury models. The animal or animal specimen must represent the equivalent human structure and function with appropriate scaling for differences in mass, maturation, or material properties, and the animal's biologic response should parallel that of the human population as much as possible.

Animal models of inflicted injury have been used in a number of contexts, with most studies focusing on 2 main research areas. The first category employs animal models to gain insights into possible mechanisms of injury. Such studies reflect the premise that by subjecting an animal to similar mechanical phenomena (eg, forces, accelerations) that an infant or child might experience during an abusive event, the resultant injuries potentially reflect injuries that may happen in humans and, thus, enable investigators to better understand injury mechanisms. Such models might also advance our understanding of predictors of outcome or response to interventions.

The second main use of animal models is to facilitate the study of factors influencing pathophysiology and evolution of injury. Such factors might include maturational stage, specific types of insults, or stressors that might exacerbate damage. This approach applies a specific insult or combination of insults thought to be part of abusive head trauma and evaluates the effects of relevant controlled variables to assess the comparative evolution of injury and, in some instances, to investigate prognostic variables or response to treatment.

Postmortem Human Subject Experiments

PMHS experiments simulating potentially injurious events can provide insight into injury mechanisms, injury types resulting from exposure to mechanical phenomena such as acceleration or force, and injury thresholds. PMHS studies have the advantage of accurate anatomic representation and biomechanical response of the targeted human population. However, the lack of PMHS vascular perfusion and respiratory inflation, the presence of rigor mortis, and the use of preservation techniques could alter biomechanical response compared with living humans.

What Can We Learn From Biomechanical Studies Focused on AHT?

As discussed earlier, different types of biomechanical studies have been used to address questions related to pediatric head injury. These studies have investigated pediatric head injury risk related to forceful impact and shaking mechanisms.[250,251] It is important to note that this is not an exhaustive review of the biomechanics scientific literature related to pediatric head trauma.

Diffuse brain injuries and/or focal brain injuries can occur in AHT. These distinct types of injuries are caused by exposure to different physical phenomena. When a child is subjected to violence, the forces and accelerations they are exposed to are likely complex in nature and may include a combination of direct and indirect forces, along with angular and linear accelerations.

Biomechanical studies have demonstrated that direct or indirect forces applied to the body leading to rotation of the head and angular head acceleration can cause diffuse brain injuries, such as diffuse axonal injury, concussion, or a subdural/subarachnoid hemorrhage that often is in a more widespread distribution rather than being very focal.[265–268] Conversely, forces applied to the body that generate linear motion of the head and translational head acceleration can cause focal brain injuries, such as brain contusions or focal subdural hemorrhage. Impact forces applied directly to the head can also lead scalp contusions, subgaleal hemorrhage, and/or skull fractures as well as intracranial contusions and focal intracranial hemorrhages.

Forceful impacts onto hard surfaces are likely to cause skull fractures and focal brain injuries (eg, brain contusion, epidural hemorrhages, focal subarachnoid or subdural hemorrhage), but sudden deceleration against even a softer surface may also generate rotational accelerations at levels capable of producing diffuse brain injuries (eg, concussion, subdural hemorrhage, diffuse axonal injury).[269,270]

With the exception of one study, surrogate experiments focused on head injury risk typically measure global head accelerations and velocities.[271] Although early animal studies have shown that brain injury risk correlates with angular and translational head accelerations, the prediction of some brain injuries may be better correlated to other measures paralleling their specific anatomic mechanism/etiology of injury. For example, many subdural hemorrhages occur through tearing of bridging veins that accompanies rotational motion of the brain relative to the skull. Thus, measuring bridging vein strain (ie, stretch) and/or brain motion relative to the skull could provide an improved prediction of subdural hemorrhages risk related to rotation. Future surrogates incorporating anatomical detail with high biofidelity that enables investigation of physiologically relevant brain injury mechanisms are needed.[271]

a) Can shaking alone cause severe brain injury or fatality in infants?. Findings from biomechanical studies investigating shaking are mixed as to whether shaking alone can cause severe brain injury or fatality in infants. Studies have used animal models, surrogate models, postmortem tissue studies, and wearable instrumentation from human subjects to approach this question.[253,254,268–283] From a biomechanics perspective, a challenge to answering this question is the threshold at which specific brain injuries occur in infants and young children, as most pediatric brain injury thresholds are derived from adult humans or immature animals and may not account for differences in the infant brain composition or the infant physiological response to trauma.[284]

1. Surrogate studies. Surrogate studies investigating shaking alone as a mechanism of brain injury have mixed findings. Some studies have concluded that severe brain injury is unlikely to be caused by shaking alone, while others concluded that shaking is potentially injurious.[253,254,269–271,278,283] The latter studies identified chin-to-chest and/or occiput-to-back contact/impact as a potential mechanism of injury in the surrogate scenario, generating the highest levels of head acceleration during a shaking cycle, thus combining shaking and impact mechanisms.

It has been shown using several surrogate models that shaking generates lower angular head velocity/angular head acceleration than certain types of impact, but other surrogate models have yielded much higher levels of angular, tangential or centripetal acceleration associated with shaking with some attributable to chin-to-chest and/or occiput-to-back impact.[253,254,269,270,278,283] These latter studies reported head accelerations that exceeded published pediatric brain injury thresholds, but uncertainty remains as to the risk of fatal injury or whether the head-to-torso contact predicted in surrogate shaking is representative of a "shaking" event in infants.

Differences in findings across surrogate studies are primarily attributable to differences in surrogate design and their level of biofidelity. Surrogates that incorporate a flexible spine and neck better approximate the expected motion of an infant during shaking but may include elements of chin-to-chest and/or occiput-to-back contact. Models that capture such events also may not be fully "biofidelic" in that they may not incorporate the compressibility of these structures in living human children, which likely would slow deceleration. Another surrogate model, developed to investigate the mechanism of subdural hemorrhages, generated displacements of the brain relative to the skull during shaking that were consistent with tearing of bridging veins associated with subdural hemorrhages.[271] Despite these findings, the roles of cervical injury, apnea, physiologic and/or electrophysiologic responses, and other factors remain incompletely understood in AHT and more generally, in infant brain injury.[100,285–287]

2. Animal studies. To test the theory that children may be injured by violent shaking, a number of animal models have investigated shaking animals in various ways and then studying the resulting injuries. Although what constitutes "shaking" varies across models, most include a repetitive cyclic motion of some type. Smith et al subjected 6-day old rats suspended upside down in a 2-inch diameter tube to repetitive back and forth movement of the tube and found scattered subarachnoid hemorrhage and cortical atrophy that was not blocked by administration of an aminosteroid antioxidant.[288] Bonnier and colleagues used a similar unrestrained head device and found no subdural hemorrhages but delayed scattered microscopic injury that was decreased by pretreatment with a glutamate antagonist (MK801).[289] Wang and colleagues used an elastic-and-spring device to deliver repeated head flexion-extension forces to young mice. They found a reduction in blood flow and subarachnoid hemorrhage but no mortality.[290] Kawamata and colleagues subjected rat pups to axially oriented shaking (head to tail cyclic motion on a customized laboratory shaker device.[291] Mice behaved normally and did not develop subdural hemorrhages but had very small microhemorrhages visible on microscopy and MRI.

There have been several challenges to this approach. First, the type and magnitude of mechanical force to which human infants and children are subjected during nonaccidental trauma remain incompletely understood. The range of injuries and histories, when available, including from confessions reflect the fact that injuries and their mechanisms vary among patients.[30,50,89,292] Secondly, concepts of what constitutes a specific mechanism may vary among individuals. For instance, what research subjects demonstrate or are instructed when asked to perform "vigorous shaking" of a child surrogate is variable.[254,270,278] Thus, reproducing "shaking" comparable to what human children

may experience can be challenging. More importantly, the specific tissue strains resulting from angular acceleration/deceleration are heavily dependent on the direction of motion, center of rotation, and, most notably, the mass of the brain.[293,294] Therefore, creating sufficient angular acceleration in the very small brain of rodents to mimic what the large human brain experiences is especially challenging.

The biomechanical challenges of scaling angular acceleration and inertial forces are more easily met with large animal models, but even here the magnitudes needed to create similar forces are significant because of the much smaller brain size. For instance, 1-month-old human infants have brain weights of approximately 500 g, while infant Yorkshire piglets and lambs have brains approximately $1/10^{th}$ that weight.[269,295,296]

Manual shaking of anesthetized lambs by humans has been used to assess the role of shaking on the immature brain. Variations in shaking kinematics were found, in part related to subject body weight and variable motion of the unrestrained head.[297] At 6 hours postinjury, small focal subdural or subarachnoid hemorrhages were seen in a subset of subjects, and more widespread amyloid precursor protein reactivity and albumin extravasation was found, but the larger subdural hemorrhages and diffuse brain swelling seen in severe cases of human injury were not produced by this mechanism. Some of the smaller animals died, but the exact cause of death was not clearly defined.[275,298]

Immature swine at 5 days and 1 month of age have been subjected to single rapid angular rotations in the horizontal plane at different magnitudes, with physiologic and neuropathologic response noted. Variable degrees of subarachnoid hemorrhage, beta-amyloid precursor protein (APP) and neurofilament-68 staining were found, which was related both to the age of the subjects and the magnitude of angular acceleration. The data also suggested that the younger subjects showed increased susceptibility to injury even accounting for traditional brain weight scaling.[273] To investigate the effects of repetitive rather than single-event rotational forces, Coats et al. scaled the forces created by manual shaking of anthropomorphic models to the infant piglet. Multiple cyclic events separated over time showed only modest injury detected by beta-APP, and animals did not sustain significant subdural hemorrhages, brain swelling, or demonstrate major acute neurologic symptoms.[272]

Large animal models have been used in an attempt to recreate some of the pathologic and clinical findings seen in severe inflicted injuries. By adding various insults, including mechanical trauma, subdural hemorrhage, apnea, and seizures, the combination of insults required to create the widespread hemispheric damage seen in children can be studied. This approach has shown that neither mechanical trauma nor subdural hemorrhage alone can create the widespread injury seen, that younger subjects tend to have more bilateral damage, and that older subjects with a combination of synergistic insults have more widespread injury preferentially on the side of the subdural hemorrhage.[295,299,300] Moreover, the degree of damage correlates with the duration of seizures, similar to human children.[301] Although these animal models may provide some insights, as yet there is no animal model that can reproduce all the feature seen in human abusive head trauma.

Together, these experiments demonstrate that animals can be injured by a variety of mechanisms, but the attempt to "match" the mechanics of human infants and young children to what is experienced by animal surrogates remains challenging. For this reason, firm conclusions about required mechanisms of injury to cause the clinical picture seen in human patients remain elusive.

The role of repetition also remains unclear; does each successive repetition lower the threshold for brain injury? A series of studies using an infant animal model found that cyclic repetitions increase the risk of brain injury compared to a single head rotation, but that multiple repetitions across different intervals still did not cause severe injury.[272] However, AHT may involve multiple exposures including to both shaking and impact; shaking is a common part of the history in many but not all case series, and, on that basis, that repetitive cyclic events seem likely to play a role in some injury consequences. Additionally, there is no evidence (biomechanical or clinical) to support the notion that rocking in an infant swing, normal play, or typical care activities cause fatal subdural hemorrhages in healthy children. Thus, in most cases, whether or not shaking can cause fatal injury is less relevant to the determination of inflicted versus accidental etiology than is the pattern of injuries in the context of a specific history.

b) Can short-distance falls lead to severe head injury or fatality in children?. Numerous studies have focused on describing injuries associated with short-distance falls involving children. To gain an objective understanding of injuries that can result from short-distance falls involving children, one must consider the level of evidence and study design. Short-distance fall studies can be categorized as epidemiologic, clinical-retrospective medical record or autopsy review, clinical with in-depth fall investigation, clinical-corroborated falls, and biomechanical.[219,226,270,302–327]

Biomechanical studies investigating head injury risk associated with short-distance falls can be further divided into those using human surrogates, PMHS studies, computational simulations, and in situ video-recorded falls with wearable sensors.[261,262,264,270,279–282,302–309] Findings from PMHS studies utilizing drop testing have found that linear skull fractures occurred in infants from a fall height of 82 cm (32 in) onto stone, carpet, and linoleum.[279,280] Studies including video-recorded falls, falls corroborated

by noncaregivers, and in-hospital falls provide a high level of evidence and typically offer greater fall detail and reduced concern that abuse has been erroneously included in the sample (Table 2). These studies can be summarized as follows:

- A total of 4060 short-distance falls are included across 7 studies providing the highest level of evidence.[302,324–330] These falls were either video recorded, occurred in-hospital, or were corroborated by a second noncaregiver. Studies including corroborated falls included a rigorous method to rule out abuse.
- The maximum fall height was approximately 1.5 m.
- 1 fatality occurred when a 16-month-old child was struck by another child who was pushed into her. The child fell rearward onto cobblestone pavement impacting the occipital region of her head.
- Serious or fatal head injuries occurred in 0.12% (n = 5) of falls. Amongst studies providing a high level of evidence, 3 cases with subdural hemorrhages (including 1 fatality) were associated with occipital impacts from rearward falls where children were unable to brace themselves[328]; 2 of these falls involved the child being pushed with impact onto concrete and cobblestone. (All falls [n = 3] were witnessed by noncaregivers.)
- Skull fractures occurred in 10 falls (0.24%). When skull fractures occurred as a result of short-distance falls, they typically were nondisplaced, narrow, linear, or curvilinear fractures. Minimally depressed skull fractures occurred in falls with head impact onto edged or cornered surfaces.

TAKEAWAY POINTS

1. Biomechanical compatibility between provided history and presenting injuries is an important factor when determining whether a child's injuries are the result of abuse or an accident.
2. Various types of biomechanical studies have been conducted to investigate the potential for head injury when children are exposed to short-distance falls, inflicted impact to the head, and shaking. These studies include in-depth event investigation and/or reconstruction, human surrogate experiments, computational models and simulations, animal injury models, and PMHS experiments.
3. Biomechanical studies have demonstrated that angular head acceleration/deceleration of sufficient magnitude can cause diffuse brain injuries, such as concussion, subdural hemorrhage, and/or diffuse axonal injury. Forces applied to the body that generate linear or rotational motion of the head and translational or angular head acceleration can cause focal brain injuries, such as brain contusions or focal subdural hemorrhage, as well as more diffuse injury patterns.
4. Findings from biomechanical studies investigating shaking are mixed as to whether shaking alone can cause severe brain injury or fatality in infants.
5. Head impacts onto surfaces causing forces of sufficient magnitude may cause skull fractures and/or focal brain injuries but may also generate rotational head accelerations at levels capable of producing diffuse brain injuries or subdural and subarachnoid hemorrhage, depending on the magnitude of acceleration/deceleration and other factors. Many head injuries in the clinical setting are the result of both direct impact and inertial (brain motion) forces and acceleration.
6. Short-distance falls rarely result in fatality in children. There is a low incidence of serious head injuries in short-distance falls, but they most frequently occur when there is momentum from concomitant activities (eg, play equipment, a child is pushed), in rearward falls onto hard surfaces when children are unable to brace themselves, or from expanding intracranial hemorrhages and/or contusional swelling from contact events. It is important to note that "short-distance" fall heights are measured as head-to-ground height rather than feet-to-ground height or height of support surface, which can lead to underestimating the forces/accelerations imparted to the head.

EVALUATION

Introduction

Children with abusive head trauma may present with clear signs/symptoms of either abuse (unexplained bruising or fractures) or head trauma (seizures, loss of consciousness). However, many children also present with nonspecific symptoms such as vomiting and excessive fussiness.[21] A thorough physical examination including head circumference, the entire skin, oral cavity, and ears can assist in determining which patients would benefit from a more detailed evaluation for abuse.[73]

When children present with clear concerns for trauma, the American College of Surgeons developed widely accepted methodology for the initial evaluation and stabilization of the trauma patient, to include children with AHT. The Advanced Trauma Life Support (ATLS) Program was introduced in 1980 and currently is in its 10th edition.[331] By ATLS methodology, all trauma patients undergo an initial primary survey including assessment of airway, breathing, circulation, neurologic disability, and exposure with immobilization and stabilization of the cervical spine.[331] The neurologic assessment includes pupillary response, GCS, and notation of any lateralization or pattern of neurologic disability such as hemiplegia or paraplegia. Relevant to the patient with suspected or proven AHT, a more detailed secondary survey includes inspection and palpation of the

entire head and face, repeat assessment of pupillary response and GCS, and examination of the cervical spine. Specific guidance regarding the evaluation and management of the pediatric trauma patient to include age-based vital sign ranges, modifications of the GCS verbal score, and appropriate modifications of neutral positioning of the cervical spine are also presented. For offices or facilities lacking appropriate clinical and diagnostic resources, rapid transfer to an appropriate facility is suggested. Institutions with a dedicated trauma team and neurosurgical support prioritize the initial diagnostic evaluation based on the clinical needs of the patient. Although a comprehensive child abuse evaluation[33] might involve a variety of radiologic examinations to include a skeletal survey, the risk of transporting the patient outside of the emergency department or ICU setting needs to be considered.[332]

Laboratory

Occult, nonobvious injuries can occur in the setting of physical abuse, especially in young children who are nonverbal or children who cannot localize pain for various reasons. Given the co-occurrence of head and abdominal trauma in young infants, laboratory evaluation to assess for occult abdominal trauma, which includes hepatic transaminases, is reasonable.[333] If these transaminases are significantly elevated (greater than 80 IU/L), then further abdominal imaging may be required.[333] Renal injuries can manifest with abnormalities in creatinine and/or blood urea nitrogen levels, or they may be evidenced by the presence of blood or other abnormalities on a urinalysis.

The evidence base for bleeding disorder evaluation in the context of suspected AHT has been addressed previously in the AAP clinical report by Anderst et al.[334] It stated that an initial evaluation for bleeding disorders in the context of intracranial hemorrhage should include: a complete blood cell count (CBC), prothrombin time (PT), and activated partial thromboplastin time (aPTT).[334] Mild, transient elevations in PT and aPTT may be seen in the setting of parenchymal injury, but these do not lead to severe, life-threatening bleeding.[105,335,336] A panel for disseminated intravascular coagulation (DIC) may be needed for clinical care if the child is severely ill. Further testing for mild factor deficiencies such as factor VIII or factor IX may be needed if there is a history of trauma causing the intracranial hemorrhage. However, in the setting of other abusive injuries, such as fractures or burns, there may be no need to do the extensive factor testing.[334] Von Willebrand disease, although relatively common, is rarely associated with intracranial bleeding, and testing is only necessary in the setting of a child with intracranial bleeding who returns to neurologic baseline or if there is a clear family or medical history suggestive of such a condition.[334]

Although some rare metabolic conditions can mimic AHT, these diagnoses may be considered on the differential when other clinical indicia of the metabolic condition are present. A review of the newborn screen can assess for many metabolic conditions, and one should consider evaluating for the presence of glutaric aciduria type 1 (GA1) with serum amino acid levels and urine organic acid levels if there are clinical indications such as preexisting developmental delay or microcephaly.[33]

Berger and colleagues have evaluated the ability of serum biomarkers to identify pediatrics traumatic brain injury. They have derived and validated a multivariable mode (using three serum biomarkers and serum hemoglobin) to identify intracranial hemorrhage in children with suspected AHT. To date, this biomarker research has not yet been able to differentiate abusive from nAHT.[337]

Imaging

Neuroimaging

Currently, CT is the imaging modality of choice in acute pediatric head trauma[338] and is indicated in the infant or young child presenting with neurologic findings and suspicions for abusive injury.[339,340] The American College of Radiology Appropriateness Criteria for imaging the child with suspected physical abuse provides additional guidance and stratification of imaging studies for different ages and clinical scenarios in this population.[340] Routine use of multiplanar and 3D reconstructions is helpful and can be generated without additional imaging.[338] These 3D reconstructions provide increased ability to identify convexity and posterior fossa hemorrhages as well as more detailed evaluation of fractures and developmental skull anomalies.[341–343]

Frequently requiring sedation, MRI is often used in the nonacute setting to further evaluate brain parenchymal abnormalities, hemorrhages, and coexistent spinal injuries in the child presenting with injuries suspicious for child physical abuse and concurrent neurologic symptoms.[339,340] Diffusion-weighted imaging is invaluable for the detection of cytotoxic edema and hypoxic-ischemic brain injury and should be included in all suspected abusive head injury magnetic resonance (MR) protocols.[339,340] Gradient echo or susceptibility-weighted sequences, to evaluate for blood products, are also routinely utilized, in addition to standard T1- and T2-weighted sequences, which provide excellent anatomic detail.[340]

Rapid MRI protocols may provide a feasible alternative to CT imaging in the initial assessment of patients with macrocephaly and those at high risk for abuse, potentially reducing exposure to unnecessary radiation and sedation.[344–349] Flom et al proposed a screening MR protocol, using a reduced number of typical MR sequences, to potentially replace CT in the initial screening of infants and found it was highly sensitive in the detection of intracranial hemorrhage in both the study and control cases.[347] More recently, this protocol was tweaked and included the addition of a

Table 2. Short Distance Falls in Children: Studies with High Level of Evidence

1st Author, Date	Title	Study Design	Age	Sample Size	Findings/Injuries
Bertocci, 2020	Injuries and biomechanics of falls involving young children in a child care setting.	Prospective, observational; video recorded falls in a child care center with wearable biometric devices.	12–36 months	3255	- no fatalities, no serious head injuries - no children had multiple injuries - 4 minor (AIS 1) injuries to soft tissue (3 lacerations; 1 nosebleed) - 0.12% of falls resulted in minor injury - max fall height 1.2 m (measured to head COM)
Williams, 1991	Injuries in infants and small children resulting from witnessed and corroborated free falls.	Retrospective; witnessed by 2nd person other than caregiver.	< 3 years	106	- 3 serious (non-life threatening) injuries in falls between 4–5 ft (1.2–1.5 m); small depressed skull fractures without loss of consciousness from falling against edged surfaces - 1 fatality from 70 ft (21.3 m) - not short distance fall - fall heights were estimated
Helfer, 1977	Injuries resulting when small children fall out of bed.	Retrospective; review of hospital incident reports; in hospital falls.	< 6 years	85	- 57 falls: no injury - 37 falls: minor injury - 1 fall: skull fracture w no sequelae (fall from ED bed) - approx. 0.9 m fall height
Nimityongskul, 1987	The likelihood of injuries when children fall out of bed.	Retrospective; review of hospital safety records; in hospital falls.	< 16 years (75% 1–5 years)	76	- 1 nondisplaced skull fracture in 1-year-old child w no ICH – no treatment required - 1 tibia fracture in patient with OI - 74 children had minor or no injuries; 4 required sutures - approx 1–3 ft (0.3–0.9 m) fall height
Levene, 1991	Accidents in hospital wards.	Retrospective; in-hospital incidents.	< 16 years	328	- 42% of incidents involved falls from heights - 2 limb fractures (3 m fall and unspecified fall) - 2 skull fractures (fall from bed and fall from chair); no treatment required
Lyons, 1993	Falling out of bed: a relatively benign occurrence.	Retrospective; review of hospital incident reports; in-hospital falls.	< 7 years	207	- 31 injuries: 29 "trivial" injuries (no sutures), 1 simple linear skull fracture with no neurologic signs (10-month-old, fall from crib), 1 clavicle fracture (21-month-old) - no children had multiple injuries - approx 25–54 in (0.6–1.3 m) fall height
Atkinson, 2018	Childhood falls with occipital impacts.	Retrospective; survey of child abuse physicians requesting occipital impacts from falls w SDH ± RH; child abuse team ruled out abuse.	7–16 months	3 directly witnessed - noncaregiver/ nonrelative witnesses	- Fall 1: pushed by 3-year-old and fell rearward onto concrete from standing: unilateral small to moderate SDH w mass effect; bilateral RHs (multiple) - Fall 2: standing on couch, fell rearward impacting head on wooden table and then floor: unilateral SDH w midline shift required surgical evacuation; bilateral RH (numerous) - Fall 3: fatality (16-month-old) – one child pushed by another child who fell into child who died; child who died fell rearward from standing impacting head on cobblestone pavement (multiple witnesses): unilateral SDH overlying frontal, parietal and temporal lobes; diffuse edema; SAH; unilateral few RH + optic nerve sheath hemorrhage - no skull fracture or other fractures - all developed immediate symptoms - median 4.5-day hospital stay; 2 survivors returned to baseline on discharge
			TOTAL	**4060**	

AIS indicates Abbreviated Injury Scale; COM, center of mass; ED, emergency department; ICH, intracranial hemorrhage; OI, osteogenesis imperfecta (a metabolic bone disorder); RH, retinal hemorrhage; SDH, subdural hemorrhage; SAH, subarachnoid hemorrhage.

more rapid T2-acquisition, a single-shot T2 fast spin echo sequence, and diffusion-weighted sequences.[346] The authors concluded this protocol had the potential to significantly reduce head CT imaging in high-risk infants. Limited MR studies, either using limited numbers of sequences or more rapid sequences, may be used for follow-up to assess parenchymal volume loss, ventricular sizes, and subdural hemorrhages or collections in pediatric trauma patients. Limitations, however, include limited availability for 24-hour MR coverage in many institutions, more motion artifact, more complex to perform than CT in critically injured patients, and less sensitivity in the detection of skull fractures and small hemorrhages.[346,347,350]

Radiographic images of the spine for fracture detection are an important part of the routine skeletal survey in cases of suspected abuse and include both dedicated anterior/posterior and lateral views.[340] Cervical spine MRI is valuable in cases with high clinical concerns of spinal injury

or in cases of spinal injury identified on other imaging modalities and has been shown to be superior to CT assessing injury to the pediatric cervical spine.[340,351] On the basis of the increased detection of spinal injuries described earlier in this report, it is prudent to strongly consider spinal MRI in cases of abusive head injury in which spinal injury would be difficult to exclude clinically or by plain radiography.[131] MRI is especially important in infants and young children with diffuse bilateral hypoxic-ischemic brain injury, which has been shown to have a high association with cervical ligamentous injuries.[129] MRI of the entire spine also may be valuable to evaluate for additional sites of spinal injury or spinal subdural hemorrhage.[128,340]

Spine MR protocols include multiplanar T1- and T2-weighted sequences to provide anatomic detail, as well as either STIR (short tau inversion recovery) or fat-saturated T2-weighted sequences, which optimize evaluation of soft tissue and marrow edema and, thus, aid in assessing for ligamentous or vertebral injures.[340,352]

a) Dating intracranial hemorrhage. Aging or dating subdural hemorrhage strictly on the imaging appearance may be challenging, as the imaging appearance depends on the stage and composition of the blood products and potential admixture of cerebrospinal fluid in the subdural compartment.[43,53,90,353–360] Dating intracranial hemorrhage based on neuroimaging is estimative. In children with suspected abuse, serial CT and/or MRI may provide an increased ability to estimate the acuity or chronicity of imaging findings and intracranial hemorrhage, especially when correlated with a multidisciplinary child protection team discussion.[90,91]

High-attenuation subdural hemorrhage on CT typically represents an "acute" hemorrhage, indicating that the hemorrhage occurred within approximately 7 days of the date of the CT.[358,361] "Subacute" hemorrhage gradually becomes lower in attenuation on CT over a period of several days up to 2 to 3 weeks from the date of the CT.[358,361] A "chronic" subdural hemorrhage contains aging blood products, may contain neomembranes and be compartmentalized, is low in attenuation on CT, is generally present beyond 2 to 3 weeks of the date of the CT, and often is well demonstrated on MRI.[354,355,358] However, a subdural hygroma is a term occasionally used to refer to a low-attenuation subdural containing fluid attenuation on CT or signal on MRI without the presence of membranes. Hematohygroma is a subdural collection containing both blood and fluid (either cerebrospinal fluid or serum) and may be used to describe a mixed-attenuation subdural hemorrhage.[353] Both a subdural hygroma and hematohygroma may present acutely after trauma, again emphasizing that dating solely on the basis of the appearance of the subdural may be challenging.[353]

In AHT, up to 67% of cases of subdural hemorrhage may demonstrate mixed attenuation on the initial CT study,[102] whereas the majority of nAHT cases presented with more uniformly high-attenuation subdural hemorrhage.[90,95]

Mixed-attenuation subdural hemorrhages may exist in several scenarios.[53,90,354–356,362] It may be the result of differing stages or components of hemorrhage, such as clotted and unclotted blood or serum in an acute and hyperacute subdural hemorrhage or clotted subdural hemorrhage mixing with serum in an acute subdural hemorrhage.[354,355] It may also represent acute hemorrhage mixing with cerebrospinal fluid resulting from traumatic injury to the arachnoid.[353] Finally, it may represent acute bleeding or rebleeding into a preexisting chronic subdural hemorrhage.[53,90,354,355,362]

1. Skeletal surveys. Skeletal surveys are recommended in children younger than 2 years when there are concerns for physical abuse.[33,340] Duffy and colleagues found that skeletal surveys found almost 11% of children with suspected abuse had a positive skeletal survey.[363] Skeletal surveys should be performed by an institution with experience in caring for and imaging children, following the American College of Radiology (ACR)-Society of Pediatric Radiology (SPR) "Practice Parameter for the Performance and Interpretation of Skeletal Surveys in Children."[364] This document, as well as the ACR Appropriateness Criteria, provide guidance regarding the skeletal images that are recommended, relative radiation levels for the imaging modalities, and additional guidance for radiation safety and imaging techniques as this skeletal imaging uses ionizing radiation.[340,364] Skeletal surveys may consist of over 22 images detailing each area of the body with some imaged in multiple planes. Despite the number of images, the radiation risk of a skeletal survey has been estimated to be low, ~0.2 mSv, and the risk of not performing a skeletal survey and missing a case of child physical abuse must be weighed against the potential low radiation risk associated with skeletal imaging.[365]

ACR Appropriateness Criteria indicates that, in children younger than 24 months of age with a high clinical suspicion for abuse and a negative initial skeletal survey, a repeat follow-up skeletal survey performed 2 to 3 weeks after the initial examination may add diagnostic information.[340] Abusive fractures may not be visible on initial skeletal surveys and prospective studies have shown that repeat skeletal imaging increases the number of skeletal fractures diagnosed in more than 25% of children who were abused. According to a meta-analysis by Maguire et al, new fractures were found by repeating the skeletal survey in 8.4% to 37.6% of cases and repeat skeletal surveys added new information that influenced child protection procedures.[366] The addition of further injury findings on repeat skeletal

surveys can improve diagnostic specificity and sensitivity for abuse.[33,367,368]

2. Other studies (literature on abdominal CT, etc). There is limited literature specific to evaluation of abdominal trauma in the setting of suspected or confirmed AHT.[369] However, a child with suspected abuse and concern for thoracic or abdominal injuries should have a skeletal survey.[340] The investigation of suspected abdominal and thoracic injuries in suspected physical abuse should be the same imaging used for accidental trauma with body CT as the imaging modality of choice.[370]

3. Postmortem imaging. Postmortem skeletal surveys can provide important information regarding the presence and chronicity of extremity fracures that may not be documented at autopsy. McGraw and colleagues reported postmortem skeletal findings in 14 children who were abused including 11 with blunt force injuries to the cranium.[371] In 6 (43%) of these 14 patients, radiography detected 26 extremity fractures not detected at autopsy. All fractures carried a high index of suspicion for abuse.

Whole-body post-mortem computed tomography (PMCT) also may detect relevant findings that can explain sudden unexpected death and can be extremely valuable for detecting non-accidental injuries. Proisy and colleauges reported high concordance of PMCT and autopsy in 15 of 18 children with unexplained death.[372]

Ophthalmologic Evaluation

Consultation with an ophthalmologist is extremely important for any child suspected to have sustained AHT.[373] Ideally, this examination takes place within 24 to 36 hours after the patient presents for medical care, because retinal hemorrhage patterns may change quickly. The number of intraretinal hemorrhages can decrease noticeably within even 48 hours, and preretinal hemorrhage can spread into the vitreous and prevent a view of the retina, so whenever possible, the ophthalmologic assessment should be performed within that time window, regardless of whether or not the pupils can be pharmacologically dilated.[183,374]

It is important that an ophthalmologist with experience and tools to identify, describe, and manage retinal hemorrhage in children perform the evaluation.[373,375] An indirect ophthalmoscope (a head-mounted unit used with a handheld lens) provides a 3-dimensional wide-angle view of the retina and is used as the primary technique for the assessment of the retina. Sometimes an eyelid speculum and scleral depression, a technique in which the sclera is indented to bring into view the farther peripheral retina and ora serrata, are necessary. It is important that the presence, number, types, and locations of hemorrhages be documented in a detailed written description for each eye, as well as the presence or absence of retinal folds, retinoschisis, optic disc swelling, or other retinal findings, such as white lesions or lipid exudates.[375] Although nonophthalmologists may identify if retinal hemorrhages are present using a direct ophthalmoscope, there is a high false-negative rate and a direct ophthalmoscope provides a small field of view, inadequate to examine wide areas of retina or to clearly see the details of the pattern of retinal hemorrhage.[376] Similarly, ocular ultrasonography and radiographic studies are inadequate for discerning the details of the pattern of hemorrhage.

Pupillary dilation facilitates the best view of the retina, whether by pharmacologic dilation or pathologic dilation, and the examination is best when conducted through dilated pupils. However, the initial examination can be performed as soon as possible and not delayed because of inability to administer eye drops. In patients whose pupils are being closely monitored for signs of extreme intracranial hypertension, short-acting drops (dilation for 2–3 hours) and/or dilation of 1 eye at a time may be an option. In addition to fundoscopy, a complete bedside ophthalmologic examination includes assessment of visual function, keeping in mind that normal visual behavior does not preclude the presence of intraocular injury, pupillary reactivity, gaze preference and ocular motility, and external examination of the ocular adnexa and ocular anterior segment to assess for signs of direct ocular or orbital trauma. Retinal photography with a digital fundus camera can help to document retinal findings. However, photography is not obligatory.[375] The ophthalmologist's detailed written description is sufficient.

Evaluation of Contacts

Siblings, or other children in the same environment of a child identified as having experienced abuse, are also at risk for being abused. When siblings were evaluated, Alexander and colleagues found abuse or neglect in siblings of 33% of their AHT cases.[377] In a similar vein, Sinal and Ball's case series of children who had experienced AHT revealed 2 suspicious deaths in siblings of children who were abused.[378]

In the multicenter ExSTRA study (Examining Siblings To Recognize Abuse), conducted in 2010–2011, skeletal surveys identified at least 1 abusive fracture in 16 of 134 contacts (11.9%; 95% CI, 7.5–18.5) <24 months of age.[379] The prevalence of a fracture or other abusive injury was greater for the younger children.

Twins have a substantially increased risk of abuse. Groothuis and colleagues found that child maltreatment was more prevalent in families with twins, and Hansen described a case in which the twin of a child with AHT was also found to have indications of being injured.[380,381] The ExSTRA study found that twins were at substantially increased risk of fracture relative to nontwin contacts (OR, 20.1; 95% CI, 5.8–69.9).[379]

In 2023, an international consensus statement addressed the screening of contact children in the setting of suspected child physical abuse, as uniformly established guidelines are lacking.[382] This statement and imaging recommendations were based on expert consensus and suggests that contact children younger than 1 year undergo a dedicated skeketal survey and cross-sectional imaging of the brain, ideally MRI imaging of the brain utilizing both routine anatomic and advanced techniques to detect brain insult and intracranial hemorrhage. Head CT with pediatric dose techniques and multiplanar reconstructions could be considered, if MRI is not available. Screening sagittal spine MRI is also recommended, if the brain imaging is abnormal in the contact child. A skeletal survey without dedicated neuroimaging is recommended in a contact child 1 to 2 years of age, with no imaging specifically recommended in contacts 2 years or older.

TAKE AWAY POINTS

1. The initial work up of children with suspected abuse includes an evaluation for medical causes for the child's condition as well as evaluating for other abusive injuries.
2. Laboratory evaluation may include assessment for bleeding disorders, genetic conditions, and other organ injuries including the liver and kidney.
3. A head CT is the current imaging modality of choice for detection of cranial and intracranial injury in children presenting with acute head injury or neurologic symptoms and concerns for abuse. MRI is playing an increasing role as well.
4. A skeletal survey looking for occult skeletal injuries is performed in a child 2 years or younger with concerns of abuse. In addition, a follow-up skeletal survey at 2 to 3 weeks after presentation is also considered, as this will allow detection of now healing fractures that were occult or subtle on the initial skeletal survey.
5. Postmortem skeletal surveys can provide important additional information regarding the extent and chronicity of extremity fractures that may not be documented at autopsy.
6. Siblings of children with suspected abuse are also at risk; the literature supports that their health and safety be considered during a child abuse evaluation.

DIFFERENTIAL DIAGNOSIS

Accidental Trauma

Introduction

When children present with an apparent traumatic injury in which AHT is considered to be in the differential diagnosis, accidental trauma often is one of the primary diagnostic considerations. To determine whether an injury is likely to have been caused by accidental mechanisms, the clinician needs to analyze the history provided including developmental abilities of the child and the clinical presentation in the context of the physical examination and radiologic findings, to determine whether the pattern fits with known data on accidental injury.[310] The certainty with which a specific patient can be determined to have a nonaccidental versus an accidental etiology varies among patients and injury constellations.[94,383] In this section, the most relevant elements of the history and presentation that the provider can obtain to facilitate comparison to what is known about accidental injury mechanisms are reviewed.

Obtaining an Optimal History to Help Differentiate Accidental Trauma From AHT

There are few quantitative studies regarding the most reliable way to elicit a history from caregivers to obtain accurate data regarding reported mechanism of injury and/or clinical appearance of the child before, during, and after a presumed traumatic event, to attempt to differentiate accidental trauma from AHT. However, some methods, including those adapted from biomechanical reconstruction questionnaires, have been suggested. These include gathering details relevant to known data on accidental and nonaccidental head injury mechanisms provided by both clinical series and biomechanical studies.[33,47,96,226,316] Suggested methods for data-gathering from caregivers include facilitating an initial uninterrupted narrative of the history.[33] Gathering specific details of the history from the last time the child was seen well, through the events surrounding the injury or onset of symptoms, and up until presentation to medical care, has been recommended, including how the child was positioned or held, what anatomic structures sustained impacts against what types of surfaces, and from what height the child fell.[47,384]

Most standard furniture dimensions in the United States include couches and other seating heights about 18 to 20 inches, beds 1 to 3 feet, and changing tables and countertops 2.5 to 3 feet.[37,314,316,327,385] Children held by parents, while in a standing position, at the parent's shoulder level, typically have head-to-ground falls of approximately 4 to 5 feet.[96,386] Biomechanical scene reconstructions have shown that most caregivers overestimate fall height when providing a history.[226] Because different authors define "low height," "short height," or "household" falls differently, more exact measurements of head-to-ground distance are more accurate than using these more general terms; however, most papers define low-height or short-distance falls as under 3 to 4 feet.[37,96,226,310,316]

In addition to the reported mechanism of injury or, alternatively, a description of the events surrounding the presentation when no history of trauma is provided, a number of authors have advocated that providers elicit details in the history including a description of the level of consciousness, degree of eye opening, movements of extremities, behaviors that might represent seizures, and

breathing status, since clinical features such as apnea and seizures have been associated with a higher likelihood of AHT compared with accidental trauma.[14,50,93,387,388] (Additional data on other features of the history in AHT can be found in the History section of this report.)

Besides the history of the event itself, in order to help differentiate accidental injury from AHT, some authors have pointed out the importance of gathering information about the child's past medical history, including typical normal behaviors and capacities, such as whether the child sits, rolls, crawls, or cruises.[33]

a) General characteristics of the history associated with accidental trauma compared with AHT. Most often, in the setting of accidental trauma, caregivers are able to give a clear, consistent description of events surrounding the injury and presentation.[96,389] In contrast, a history describing a possible accidental mechanism in which caregivers are unable to describe events, the description of events changes in major ways over time, or the description is clearly developmentally implausible have been noted as characteristics occurring more often in AHT compared with accidental trauma.[50] Some accidental injuries will happen when children are unsupervised, and guidance has been offered to help differentiate this from neglect or abuse, depending on the overall clinical context.[96,390]

In one study of children younger than 2 years with accidental injuries and AHT serious enough to lead to hospitalization in an intensive care or monitored unit or who died, 75% of the accidental injuries were witnessed by caregivers, while witnesses were reported in 2.5% of children with AHT.[387] For children younger than 3 years with subdural hemorrhages, Feldman et al found that parents are not always present when an accidental injury occurred, especially when children are mobile (crawling, walking) or are supervised by older children or other caregivers.[94] A history of a child who falls, cries, and is readily consoled, with the injury only becoming apparent later, is common in low-height/short-distance falls in accidental trauma, most often in the setting of liquefaction of a scalp hematoma associated with a linear skull fracture, as will be discussed further in the next section.[222–224]

In contrast, in various clinical series, approximately 85% to 95% of children judged to have AHT present with either a) no history of trauma, with presentation because of signs or symptoms (range 43%–72% of children); or b) a description of an accidental traumatic mechanism, usually of a minor nature, such as a fall less than 3 feet (25%–42% of children). The remainder present with various other descriptions of potential traumatic events.[37,50,89,94,387] In a series from New Zealand, about 14% of cases determined to stem from assault had an associated admission (confession) of injury causation, but this was age dependent, with a higher percentage of assault admissions in children older than 2 years. Variable injury mechanisms were described in the younger children with admitted AHT, while all children older than 2 years who had been assaulted had blunt impacts reported.[89]

b) Associations between history, clinical, and radiologic findings in accidental trauma. Data linking the history, physical examination, and imaging findings assists in assessing whether the injury pattern is typically associated with the accidental mechanism described, or, when the event is unwitnessed, is likely in the developmental context of the child. Several lines of research have investigated these associations. As mentioned earlier, "low-height falls" or "short-distance falls" have been defined variably in the literature but generally include free-fall translational forces under about 3 to 4 feet (head to ground), as noted below. Some of the relevant studies and findings are summarized below.

- Low-height/short-distance falls can cause linear skull fractures, especially in infants.[328,386]
- Bilateral skull fractures can occur from a single impact event, typically in the occipital or frontal region.[391] These fractures may be separate and noncontiguous, can cross over the suture, and generally depict contiguous fracture planes.[392]
- Epidural hemorrhages, which can be life-threatening, most often occur from low-height/short-distance falls in young children and most often are associated with skull fractures but are uncommonly associated with abuse.[220,393,394]
- Thin, crescent-shaped hemorrhagic collections underlying skull fractures often represent venous epidural hemorrhages from the fracture itself and can be misread as subdural hemorrhages on CT scan.[47]
- Subdural hemorrhages occur in both AHT and accidental trauma but are much more common in infants and young children in the setting of child abuse.[92]
- Low-height occipital impacts and other contact mechanisms have been reported to be associated with subdural and/or subarachnoid hemorrhage, but these are usually clinically benign.[328,389]
- Falls involving higher head-to-ground heights, additional velocity (such as being pushed, or on moving equipment), or greater angular forces can be associated with more significant injuries, including contusions and subdural and subarachnoid hemorrhages.[219,328] Falls from playground equipment have been associated very rarely with subdural hemorrhage, contusion, brain swelling, and death; however, these have included children from 1 to 13 years of age and in some cases involved head-to-ground heights over 3 to 4 feet and/or additional forces such as falls from moving equipment or while being pushed.[311]
- As noted earlier, free falls with head-to-ground height under about 3 feet are uncommonly associated with

serious injury except for skull fractures and epidural hemorrhage. In contrast, inflicted impacts (throwing the subject down onto a surface) using instrumented anthropomorphic models are associated with significantly greater angular deceleration forces than low-height/short-distance falls, with magnitudes in the range predicted to cause injuries such as concussion and subdural hemorrhage.[270,307] (More on biomechanics studies can be found in the Biomechanics section.)

c) Deaths from short-distance falls. Most head injuries in infants and children younger than 24 months occur as a result of falls.[96] Typical, short-distance falls are not life threatening, often with little to no neurologic symptoms.[96,314,325,327] Other than in the setting of epidural hemorrhage, low-height/short-distance falls rarely, if ever, cause severe injuries or death.[98,219,226,310,316,395]

However, some literature has suggested that fatal or significant intracranial injuries may result from short-distance falls.[311,317] Plunkett described 18 children sustaining fatal head injuries from reported falls off playground equipment from more than 75,000 cases in the United States Consumer Product Safety Commission database.[311] Some limitations of this study are that 6 of 18 were witnessed only by a single caretaker; only 5 were younger than 24 months, and none were infants. Additionally, these numbers do not include all of the children who might have sustained insignificant injuries after falling off playground equipment but who were never reported or taken for medical treatment.

Hall et al reviewed pediatric fall fatalities seen at the Cook County Medical Examiner's office.[317] Nearly 41% (18/44 cases) had isolated intracranial injuries associated with short-distance falls ≤3 feet, several with a delay in seeking care. The authors acknowledged that some of these injuries might have been the result of abuse, because significant injuries from these types of short-distance falls are exceedingly rare and should raise suspicions of child physical abuse.

Numerous case series in the medical literature find very few, if any, significant or fatal head injuries occurring as a result of witnessed and corroborated, short-distance falls or falls occurring in a hospital setting.[314,324,325,327,396] However, presenting histories in children in whom abusive head injuries are diagnosed are not infrequently short-distance falls.[89]

Chadwick et al estimated the potential mortality rate from a short-distance fall for infants and young children to be much less than 1 fatality per 1 million young children per year.[310] This is based on an extensive review of the available literature as well as several public database reviews and supports the conclusion that a fatal head injury resulting from a short-distance fall is exceedingly rare.

TAKEAWAY POINTS

1. Accidental trauma is a primary differential consideration in children suspected of experiencing abuse. Detailed history of the child and reported trauma is essential in attempting to differentiate the cause of the child's injuries.
2. Accidental household falls of low height/short distance occur frequently in this population and may result in skull fractures with small volume adjacent intracranial hemorrhage, often venous epidural blood that may be misinterpreted on imaging as subdural hemorrhage.
3. Epidural hemorrhage is common following accidental trauma but is uncommon in abusive head injury.
4. Deaths or clinically significant intracranial injury from low-height/short-distance, household falls are exceedingly rare.

Bleeding Disorders

Unlike some alternative medical explanations, bleeding disorders are known causes of intracranial hemorrhage (ICH) and retinal hemorrhage (RH) and are a heterogeneous group of conditions that vary in etiology, presenting symptoms, and prevalence. When considering bleeding disorders as the potential etiology for the hemorrhagic findings seen in AHT, the AAP has previously suggested, in addition to detailed symptom and family history gathering, a testing strategy for evaluating a possible bleeding disorder that incorporates the probability of an ICH occurring in a child with a given congenital bleeding disorder.[334,397] For example, the probability of an individual in the general population having an ICH because of factor XIII deficiency can be calculated by using the estimated prevalence of the condition (1 in 2 million people) and the prevalence of intracranial hemorrhage within the population with the condition (33%), or 1 in 6 million [(1 in 2 million) × (1/3)].[397]

Within this testing strategy, it is helpful for the pediatric provider to know where the data for prevalence of ICH within a particular congenital bleeding disorder population is derived. A primary source of data for symptoms in children with bleeding disorders is the Universal Data Collection (UDC) database of the Centers for Disease Control and Prevention. The UDC contains specific information on intracranial hemorrhage and subdural hemorrhage in subjects with congenital bleeding disorders. Thus, on the basis of these data and prevalence of conditions, the AAP clinical report on bleeding disorders provided a table of "Probabilities for Congenital Coagulopathies Causing Intracranial Hemorrhage," which may be helpful to the pediatric provider.[334] The essential takeaway point of this table is that the probability of a congenital coagulopathy to cause ICH ranges from "low" to "1 in 50 million," depending on the coagulopathy.

To further evaluate these probabilities, Anderst and Carpenter reviewed the UDC in subjects <4 years of age to "characterize the prevalence and calculate the probabilities of any intracranial hemorrhage, traumatic intracranial hemorrhage, and nontraumatic intracranial hemorrhage in children with congenital bleeding disorders."[398] The authors found that of 3717 subjects, 255 (6.9%) had any intracranial hemorrhage and 206 (5.5%) had nontraumatic intracranial hemorrhage. The highest prevalence of intracranial hemorrhage was in severe hemophilia A (9.1%) and B (10.7%). Of the 1233 subjects <2 years of age in which the specific location of any ICH was known, 13 (1.1%) had spontaneous subdural hemorrhage (12 with severe hemophilia; 1 with type 1 von Willebrand disease). The authors concluded that "In congenital bleeding disorders, nontraumatic intracranial hemorrhage occurs most commonly in severe hemophilia. In this study, von Willebrand disease is not supported as a 'mimic' of AHT."

With regard to the prevalence and probability of RHs in congenital coagulopathies, a systematic review by Thau et al sought to identify whether coagulopathies have been reported with RH similar to those of AHT.[194] Of 61 articles that met inclusion criteria, the authors found that there were only 32 children within the AHT age range (younger than 5 years) who had RH and concomitant coagulopathy. RH has not been reported in congenital factor II, VII, X, XII, or XIII deficiencies. More extensive RH (with extension to the periphery) has been reported in hemophilia A and B, and mild von Willebrand disease, albeit rare in these entities as well. Of the 32 children, only 5 of those cases could be confused for abuse. Of those 5 cases, none had retinoschisis or retinal folds. The authors concluded that disorders of coagulopathy rarely result in RH in children.

TAKEAWAY POINTS

1. Most bleeding disorders are rare.
2. The more common bleeding disorders typically are mild.
3. Intracranial hemorrhage resulting from bleeding disorders is a rare complication of the more severe bleeding diatheses.
4. When bleeding disorders are a reasonable clinical consideration (based on parental concern of bleeding/bruising symptoms and/or family history), it is prudent to obtain a hematology subspecialty consultation.

Birth Trauma

Injuries common to both birth trauma and AHT include scalp hematoma, skull fracture, intracranial hemorrhage, and retinal hemorrhage. Although much of birth-related injury is clinically asymptomatic, presentation varies depending on the severity of the injury.

Intracranial Hemorrhage

Acute subdural hemorrhage has been a reported finding following vaginal and cesarean deliveries.[399–403] The prevalence of birth-related subdural hemorrhage ranges from 7% to 50%.[399,400,402,404,405] The precise mechanisms of birth-related ICH remain unknown. Multiple mechanisms have been proposed, including mechanical deformation of the cranium with consequent disruption of the dural border cell layer, mechanical deformation resulting in bridging vein rupture, and increased intracranial pressure.

Birth-related subdural hemorrhages can be either supratentorial or infratentorial.[399,400,404,406] Although more commonly located in the posterior fossa, birth-related subdural hemorrhages can also be located along the frontal, parietal, and interhemispheric regions.[399,400,404,406] Thus, the location of subdural hemorrhage alone is not specific enough to distinguish AHT from birth-related intracranial injury.

Most birth-related subdural hemorrhages are asymptomatic.[399,400,402,407] However, when symptomatic, the severity of presenting symptoms correlates to the presence of underlying parenchymal injury.[406,408,409] The majority of clinical symptoms present within the first 24 hours of life but can present as late as 11 days postnatally.[399,406,410] Delayed symptoms (weeks to months from birth) have not been reported outside the context of birth-related intracranial injury complications (eg, anoxic brain injury, hydrocephalus, etc).

The vast majority (greater than 94%) of birth-related subdural hemorrhages resolve by 4 weeks of age.[399,402] Current literature involving longitudinal radiologic evaluation of birth-related subdural hemorrhage has not documented the persistence of birth-related subdural hemorrhage beyond 3 months of age.[399,400,402] Rebleeding of birth-related subdural hemorrhage (in association with significant neurologic sequelae) is discussed herein below in the "alternative medical theories" section.

The outcomes of asymptomatic infants with birth-related subdural hemorrhage (and no concurrent parenchymal injury) are relatively benign. Rooks et al analyzed 2-year clinical follow-up of 43 of 46 infants with birth-related subdural hemorrhage, and none had gross motor delay; 6 (14%) children were noted to have speech delay.[399] Zamora et al showed similar cognitive and neurodevelopmental outcomes at age 24 months between normal controls and those with asymptomatic birth-related subdural hemorrhage.[411]

Retinal Hemorrhages

RHs are a relatively common finding with parturition. Several well-conducted studies have placed the incidence of birth-related RHs at approximately 20% to 35%.[154,155,412–415] Birth-related hemorrhages occur in 20% to 30% of infants in the first 24 hours of life, decreasing to

10% to 15% when infants are examined in the first 72 hours of life.[151,415,416] They can occur with any type of delivery, including cesarean section (6% incidence rate), but they are more common in spontaneous vaginal (25.6%) or vacuum-assisted deliveries (42.6%). A systematic review of 13 clinical studies representing 1777 infants revealed that birth-related retinal hemorrhages are commonly bilateral (59%), predominantly intraretinal, and localized in the posterior pole but rarely can be numerous and extend to the periphery.[156] Preretinal hemorrhages may also occur, but subretinal hemorrhages are rare.[177] Despite extensive incidence and descriptive studies referenced earlier, to date, retinoschisis has not been a reported feature of parturition.[154,155,412–415]

The rate of disappearance of birth-related retinal hemorrhage depends on the size and type of hemorrhage. The majority of intraretinal hemorrhages (85%) resolve within 14 days.[415] Dense isolated intraretinal hemorrhages may persist past 3 weeks of age, with the longest reported being 58 days, but numerous intraretinal hemorrhages do not.[415,417] Preretinal and vitreous hemorrhage may last significantly longer than intraretinal hemorrhage. This evidence implies that on the basis of the pattern of resolution, the presence of numerous intraretinal hemorrhages after 3 weeks of age, or single or few intra-RHs after 8 weeks of age, cannot be ascribed to birth.

The mechanism of birth-related RHs is incompletely understood. However, vitreoretinal traction may be one cause if direct eye compression causes distortion and lengthening along a single axis.[155,415] Other proposed mechanisms include ocular compression with prolonged highly raised intraocular pressure and subsequent decompression retinopathy, and release of leukotrienes or other vasoactive factors resulting in increased retinal vessel permeability. Finally, some literature indicates that poor visual outcomes are much more common in AHT than in birth-related retinal hemorrhages.[418–421]

TAKEAWAY POINTS

1. Birth-related subdural hemorrhages are common, often asymptomatic, and can occur in similar locations as those in AHT cases.
2. The vast majority of birth-related subdural hemorrhages resolve by 4 weeks of age, with current literature not documenting the persistence of birth-related subdural hemorrhage beyond 3 months of age.
3. The outcomes of asymptomatic infants with birth-related subdural hemorrhage (and no concurrent parenchymal injury) are relatively benign.
4. Birth-related RHs are common and can be bilateral, involving multiple layers; however, unlike AHT, they uncommonly are numerous or extend to the retinal periphery.
5. Additional distinguishing features between birth-related RHs and retinal findings seen in AHT are the lack of retinoschisis documented in parturition, the lack of persistence of birth-related RHs beyond 2 months of age, and a stronger association of severe retinal findings and poor visual outcomes with AHT.

Benign Enlargement of the Subarachnoid Spaces

Infants and young children often have more prominent subarachnoid spaces than older children and young adults.[422,423] Some hypothesize that the presence of these enlarged subarachnoid spaces predispose a young infant to the development of subdural hemorrhages with little or no trauma, thus potentially causing confusion and a misdiagnosis of abusive head injury.[424–431] Most of these are small series or case reports without clear methodology, and it has been demonstrated that subdural hemorrhages are not a common finding following pediatric minor head injury.[96]

Several studies have evaluated infants and young children with enlarged subarachnoid spaces and macrocephaly.[432–435] Each found a small percentage of patients that might have developed subdural collections/hemorrhage with little to no trauma. Importantly, each patient had little to no clinical symptoms, and each article discusses the importance of considering inflicted injury. Greiner et al found no subdural collections in asymptomatic patients with macrocrania and normal subarachnoid spaces.[432] Tucker et al found the prevalence of subdural collections was higher in patients with a larger degree of expansion of the subarachnoid spaces.[434] Holste et al found that in 480 patients with enlargement of the subarachnoid spaces, 8.1% developed spontaneous subdural hemorrhages and in those with any subdural hemorrhage in their study, only 15.5% required surgical intervention.[435]

Hansen et al studied 149 children younger than 2 years who were evaluated by the child protection team and presented with a subdural hemorrhage.[436] Forty-three were either asymptomatic at presentation or had very mild symptoms attributable to a small subdural. These were compared with 106 severely injured patients. Benign enlargement of the subarachnoid spaces was present in 34 of the total cohort (22.8%), half in each category. The presence of enlarged subarachnoid spaces was associated with a lower occurrence of other suspicious injuries. However, over one-third of patients presenting with a subdural and mild associated symptoms in the presence of enlarged subarachnoid spaces also had other concerning abusive injuries.

In conclusion, it has been shown that a small percentage of patients with benign enlargement of the subarachnoid spaces (BESS) may present with small, asymptomatic subdural collections with little to no reported trauma. However, the authors in each of these articles stress the need to consider abuse as a potential etiology in each case

and not simply dismiss the presence of the subdural as a result of the enlarged subarachnoid spaces.

TAKEAWAY POINTS

1. Enlargement of the subarachnoid spaces in common in infants and young children.
2. Infrequently, enlarged subarachnoid spaces may predispose to the development of small, asymptomatic subdural collections with little or no head trauma.
3. Each case of unexpected subdural collection or hemorrhage needs thoughtful consideration as to potential of abuse and not simply dismissed as being attributable to the enlarged subarachnoid spaces.

Alternative Medical Hypotheses Lacking Evidence-Based Research

Hypoxic-Ischemic Injury

Some authors suggest that subdural hemorrhage in infants and young children may result from hypoxic-ischemic injury or hypoxic-ischemic encephalopathy in the perinatal period.[437–439] Several studies, however, have found no such causal association.[440–443] Hurley et al, in analyzing 50 infants with nontraumatic cardiorespiratory collapse, found no extra-axial hemorrhage in the 24 infants with brain imaging and no macroscopic subdural hemorrhage in 39 of 40 with a postmortem examination.[441] One infant had a very small clot adjacent to the dura that was determined to be birth related. Byard et al, evaluating a potential causal relationship of hypoxic ischemic injury and subdural hemorrhage, investigated 82 fetuses, infants, and young children and found no cases of macroscopic subdural hemorrhage on postmortem examinations.[440] Rafaat et al looked at CT findings in 156 young children experiencing near drowning events and found no intra-axial or extra-axial hemorrhage on initial or follow-up imaging in any patients who presented with abnormal initial CT imaging or in those who had normal initial but subsequent abnormal follow-up CT imaging.[444]

Paroxysmal Coughing and Dysphagic Choking

Some authors suggest that rapid, repetitive episodes of elevated intrathoracic pressure, such as paroxysmal coughing or choking on feeds, may result in elevated intracranial venous pressure from venous backflow causing the thin film subdural and retinal hemorrhages that might be seen in cases of suspected AHT.[437,439,445,446]

However, Goldman et al found no retinal hemorrhages in 100 infants and young children presenting with severe coughing episodes requiring hospitalization.[203] Herr et al found no retinal hemorrhages in 100 infants undergoing surgery for hypertrophic pyloric stenosis, 89% being described as having multiple episodes of projectile vomiting.[207] Multiple authors have demonstrated that retinal hemorrhages in the setting of an apparent life-threatening event are exceedingly rare, but when present, should raise concerns for AHT.[447–449]

Hansen et al studied 170 patients younger than 2 years presenting with subdural hemorrhage, 64 presenting with a reported apparent life-threatening event (ALTE) (now described as a brief, resolved, unexplained event [BRUE]) and subdural bleeding.[436] Those with an ALTE-associated subdural hemorrhage were more likely to have a fatal outcome, have permanent neurologic injury, and were 5 times more likely to have at least 1 extracranial injury that was suspicious for inflicted trauma. Ten patients presenting with an ATLE-associated subdural hemorrhage and a dysphagic/choking history witnessed only by 1 caregiver all had evidence of at least 1 other suspicious extracranial injury.

Cerebral Sinovenous Thrombosis

Intracranial or cerebral sinovenous thrombosis (CSVT) historically has a reported incidence between 0.67 and 1.58 cases per 100,000 pediatric patients per year.[450,451] However, the incidence rate in young infants, especially in the neonatal period, is much higher.[450–452] Underlying risk factors for the development of CSVT are present in the majority of pediatric patients and vary with patient age.[450–453] Neonatal CSVT usually occurs from perinatal complications.[450] In the older infant and child, etiologies are often multifactorial and may include dehydration, head and neck infections, underlying malignancies and chronic disease states, prothrombotic factors, and trauma.[451–456]

Some physicians and investigators have suggested that spontaneous or nontraumatic CSVT is a potential source of subdural hemorrhage, thus mimicking the imaging findings of abusive head injury.[437,445,446,457,458]

Outside of the perinatal period, except in rare case reports in the medical literature, subdural hemorrhage is not a frequent finding in nontraumatic cases of pediatric CSVT.[450,455] The Canadian Pediatric Stroke Registry described 9% of patients as having extra-parenchymal hemorrhage on imaging in their report.[450] Subsequently, it has been reported by McLean et al that the registry patients were imaged during the neonatal period, a time when small subdural hemorrhages can be found routinely.[399,400,402,455] This more recent study found no subdural hemorrhage in 36 patients with nontraumatic CSVT, even in the patients with extensive, intracranial thrombosis.[455]

Rebleeding of Intracranial Hemorrhage With Severe Neurologic Sequelae and/or Death

Some authors have suggested that spontaneous rebleeding into preexisting subdural hemorrhage, including birth-related hemorrhage, may lead to the acute compromise of the child.[457,459,460] Several authors have shown that many cases of mixed-attenuation subdural hemorrhages found during an AHT evaluation do not necessarily represent new hemorrhage into "chronic subdural

hemorrhages."[53,90,362,461] Many occur without acute clinical symptoms and suspicious extracranial injuries and represent acute, low-attenuation subdural fluid mixing with different components of blood (hematohygroma), not necessarily different episodes or ages of injuries.[353–356,362]

Datta et al studied 74 children younger than 2 years with either subdural hemorrhage or effusions on neuroimaging.[461] Twenty-six had mixed-attenuation subdural hemorrhages. However, 20 of 26 had extracranial findings of prior abuse and signs of elevated intracranial pressure or acute mental status changes suggesting repeated episodes of abuse, not simple rebleeding. No spontaneous bleeding occurred in postinfectious subdural collections.

Bradford et al found evidence of rebleeding into a preexisting subdural hemorrhage in 16% of 105 cases of abusive head injury, all rebleeds being small and asymptomatic at the time of imaging.[90]

Feldman et al performed a multicenter retrospective review of 383 abused infants and children with suspected acute (291/383) and reported mixed acute/chronic (92/383) subdural hemorrhage.[53] Nearly 80% of each group presented with acute neurologic symptoms and other injuries concerning for abuse, suggesting each likely experienced an acute or repeated inflicted injury, not spontaneous rebleeding.

Wright et al studied 85 children younger than 3 years with a subdural hemorrhage, diagnosis of AHT, and both initial and follow-up imaging.[362] Almost 64% (54/85) had imaging evidence of rebleeding within 1 year. This was significantly associated with parenchymal volume loss, ventricular enlargement, large subdural depth, and macrocephaly, but not new brain injury at follow-up. Most rebleeding was found on routine follow-up imaging and was in the original subdural location in 94.4% (51/54) of cases. All rebleeding was asymptomatic and occurred into preexisting low-attenuation subdural collections. None had acute neurologic compromise or new concerning abusive injuries, and none occurred in a previously resolved subdural hemorrhage.

Feldman et al and Wright et al specifically mention an association of asymptomatic rebleeding in patients with preexisting subdurals and macrocephaly.[53,362] In both of these studies, the rebleeding was isolated to the location of the prior subdural supporting that rebleeding could occur with little or no additional trauma in children with chronic subdurals.[108] However, the neovascularity in the chronic subdural hemorrhage would not result in rebleeding in other distinct locations.

TAKEAWAY POINTS

1. The correlation between hypoxic-ischemic injury and the development of subdural hemorrhages lacks evidence base.
2. Paroxysmal coughing and choking are not associated with the development of RHs and/or subdural hemorrhage.
3. Isolated subdural hemorrhage has not been demonstrated to occur as a result of CSVT. However, in pediatric patients, CSVT often has an underlying risk factor and can occur as a complication of traumatic head injury.
4. Rebleeding into preexisting subdural hemorrhage or collections is common in patients having experienced prior abusive head injury. However, it is almost always asymptomatic and does not result in acute, significant neurologic compromise in the child.

DIAGNOSIS

As discussed earlier, the diagnosis of AHT is made in the context of a complete history, physical examination, and medical evaluation using the same process as in any other medical diagnosis: the differential diagnosis.[52] In addition, many providers have the advantage of a multidisciplinary hospital child protection team.[462] Such a team can help to minimize the risk of individual error that may occur from judgment heuristics or cognitive bias.[463] Throughout the diagnostic differentiation process, the pediatric provider will have to manage various uncertainties until the individual threshold of diagnostic sufficiency is reached.[464]

Prediction Rules and Pooled Analyses

Assisting providers in reaching a diagnosis are recently published prediction rules and pooled analyses. "Clinical decision rules," and "clinical prediction rules" are evidence-based tools that combine clinical features to assist clinicians in probabilistic determinations of clinical diagnosis, prognosis, or response to therapy.[465] Several of these tools—the PredAHT tool, the PEDIBIRN clinical decision rule, and Maguire's pooled analysis—have been discussed earlier.[51,466,467] The value of these tools is that they offer clinicians a meta-analysis of the import of these collective variables and, thereby, minimize inherent risks of individual biases, cognitive errors, and local circumstances.

Communicating the AHT Diagnosis in Court and Other Medicolegal Considerations

With regard to the differential diagnosis methodology, courts have varied in their interpretation and understanding of it. Some courts have interpreted it to be the methodology for arriving at causation by "ruling out" alternative causes.[468] Others have interpreted it to require both a "ruling out" and "ruling in" process for arriving at causation.[469] And others have even created legal concepts, such as "differential etiology," declaring it to be different from "differential diagnosis."

Most courts have found the differential diagnosis methodology to be "a well-recognized and widely used technique in the medical community to identify and isolate causes of

disease and death." [Gunderson, 279 S.W.3d at 107 (citing Globetti v. Sandoz Pharms. Corp., 111 F.Supp.2d 1174, 1177 (N.D. Ala. 2000)); State v. Edwards, 2011 WL 1378927 (Ohio Ct. App. April 13, 2011); State v. Carr, 2010 WL 2473337 (Ohio Ct. App. June 18, 2010)] However, some courts have questioned its reliability.[470]

Tangent to this misunderstanding has been the prevalent issue of referring to the AHT diagnosis as a "triad" (ie, diagnosed if the child has subdural hemorrhages, RHs, and cerebral edema).[471] It is this relegation to heuristic application of the 3 more common findings of the diagnosis that has fostered legal misunderstanding of the aforementioned complex diagnostic process. The pediatric provider will need to be prepared to explain to courts that the evolution of diagnostic understanding of AHT has broadened to try and capture, in definition, the myriad of ways inflicted injury to a child's head can occur and manifest. This understanding, again, may be fostered by current clinical prediction rules and pooled analyses, showing how variable combinations of clinical features can hold extremely high odds for the AHT diagnosis and not just the "triad."[51]

Finally, and probably most importantly, has been the prevalent issue of ethical expert testimony in AHT cases. Some authors have asserted that AHT cases have been a minefield of irresponsible expert testimony.[472,473] In a medicolegal analysis of overturned shaken baby syndrome (SBS)/AHT convictions in the US appellate system over a 10-year period, Narang et al found that: 1) medical evidence-related themes raised on appeal seldom reflected new scientific or clinical discoveries but rather were alternative or differing medical opinions from those offered at the original trial; and 2) that their review of over 1400 appellate cases over a 10-year period "tended to support the concerns of other authors regarding irresponsible communication of medical information in AHT/SBS cases."[473] Thus, the pediatric provider should be prepared to educate the legal system not only about what is sound science in AHT, but also what is junk science. To complete this task, it will be important for the pediatric provider to be versed in the principles of ethical expert testimony espoused by the AAP.[474]

MANAGEMENT

Introduction

Although AHT comprises a significant fraction of head injuries requiring hospitalization in children younger than 5 years, no evidence-based guidelines for the management of the child with AHT currently exist.[98] Guidance regarding the medical and surgical treatment may be abstracted from a combination of existing literature on the management of pediatric and adult head injury as well as a small volume of literature specific to AHT.

Medical Treatment

Guidelines for the management of pediatric severe traumatic brain injury were initially developed in 2003 by a panel of experts in traumatic brain injury during the fifth Annual Aspen Neurobehavioral Conference.[475,476] The guidelines were updated most recently by the Brain Trauma Foundation in 2019.[477] Current guidelines present evidence-based recommendations on 3 broad categories: monitoring (3 topics), thresholds for treatment (2 topics), and treatment (10 topics). These guidelines are not specific to the infant population or to the child with AHT. In 2 topics covered by the guidelines, additional literature specific to the infant or child with AHT mandates special consideration of this patient population:

- Intracranial pressure monitoring: The 2019 Guidelines for the Management of Pediatric Severe Traumatic Brain Injury suggest the use of intracranial pressure monitoring as an option capable of improving overall neurologic outcomes in pediatric patients. However, the guidelines clarify that "(b)ecause there are no imaging or other biomarkers that indicate a patient with intracranial hypertension, it is recommended that ICP is measured to determine if intracranial hypertension is present" and that "studies support the association of successful ICP monitor–based management of intracranial hypertension with improved survival and neurologic outcome."[477] Furthermore, as suggested treatment thresholds are based on measurements, a lack of intracranial pressure monitoring in the setting of intracranial hypertension limits management.
- Thresholds for Treatment: The 2019 Guidelines for the Management of Pediatric Severe Traumatic Brain Injury suggest the treatment of intracranial pressure (ICP) targeting a threshold of <20 mm Hg as "mortality is often due to refractory sustained increases in ICP." Thus, "preventing intracranial hypertension is central to current neurocritical care of these children."[477] In addition, thee 2019 guideline further suggests treatment to maintain CPP (cerebral perfusion pressure) at a minimum of 40 mm Hg using a range from 40 to 50 mm Hg, "with infants at the lower end and adolescents at or above the upper end of this range."[477]
- Treatment Topics: The 2019 Guidelines for the Management of Pediatrics Severe Traumatic Brain Injury further identified the following recommendations[477]:
  - Bolus hypertonic saline (3%) is recommended in patients with intracranial hypertension. Recommended effective doses for acute use range from 2 to 5 mL/kg over 10 to 20 minutes.
  - Continuous infusion hypertonic saline is suggested in patients with intracranial hypertension. Suggested effective doses as a continuous infusion of 3% saline range from 0.1 to 1.0 mL/kg of body weight per hour,

administered on a sliding scale. The minimum dose needed to maintain ICP <20 mm Hg is suggested.
    - Bolus of 23.4% hypertonic saline is suggested for refractory ICP. The suggested dose is 0.5 mL/kg with a maximum of 30 mL. (Level II Evidence)
    - With use of multiple ICP-related therapies as well as appropriate use of analgesia and sedation in routine intensive care, avoiding bolus administration of midazolam and/or fentanyl during ICP crises is suggested because of risks of cerebral hypoperfusion.
    - Cerebrospinal fluid drainage through an external ventricular drain is suggested to manage increased ICP.
    - Prophylactic severe hyperventilation to a partial pressure of arterial carbon dioxide <30 mm Hg in the initial 48 hours after injury is not suggested.
    - If hyperventilation is used in the management of refractory intracranial hypertension, advanced neuromonitoring for evaluation of cerebral ischemia is suggested.
    - Prophylactic moderate (32°–33°C) hypothermia is not recommended over normothermia to improve overall outcomes.
    - Moderate (32°–33°C) hypothermia is suggested for ICP control.
    - High-dose barbiturate therapy is suggested in hemodynamically stable patients with refractory intracranial hypertension despite maximal medical and surgical management.
    - Decompressive craniectomy is suggested to treat neurologic deterioration, herniation, or intracranial hypertension refractory to medical management.
    - Use of an immune-modulating diet is not recommended.
    - Initiation of early enteral nutritional support (within 72 hours from injury) is suggested to decrease mortality and improve outcomes.
    - The use of corticosteroids is not suggested to improve outcome or reduce ICP.
- Treatment of the patient with severe traumatic brain injury patient is complex, and the recommendations are often difficult to translate into bedside management. Kochanek et al in 2019 established an algorithm for first- and second-tier therapies, in part utilizing the recommendations from the 2019 Guidelines for the Management of Pediatric Severe Traumatic Brain Injury and supplementing with consensus when evidence was insufficient to fully formulate an evidence-based approach.[478] Although the first-tier pathway distinguishes a linear ICP, CPP, brain tissue oxygen (if monitoring), and herniation pathways, the paper specifically noted that treatments may warrant a rapid and nonlinear approach.[478]
- Evaluation and management of post-traumatic seizures: The 2019 Guidelines for the Management of Pediatric Severe Traumatic Brain Injury suggest the use of prophylactic treatment for the first 7 days post-TBI to reduce the occurrence of early post-traumatic seizures in pediatric patients. References cited in the guidelines include authors citing rates of 2.4% and 17% of early post-traumatic seizures in the pediatric population studied.[479,480] However, the incidence of clinical and nonconvulsive seizures is significantly higher in the patients with AHT with a seizure incidence ranging from 51% to 77%, providing additional evidence for the use of anticonvulsant use in this pediatric subgroup.[480–482] Hasbani et al performed a retrospective study reviewing use of continuous encephalogram (EEG) monitoring in AHT patients at their intensive care unit.[388] Of 32 children admitted during the periods studied, 21 (66%) were monitored with seizures noted in 12 (37.5%) and status epilepticus in 8 (25%). Two-thirds of children never had clinical signs associated with their electrographic seizures; thus, EEG monitoring is required to diagnose and guide acute seizure management. In a larger study performed by O'Neill et al, age less than 2.4 years and AHT mechanism was strongly associated with the presence of seizures (OR, 8.7 and 6.0, respectively).[483] In a prospective study of patients with consecutive moderate to severe TBI admitted to the intensive care unit at 2 institutions, AHT mechanism was a significant risk factor for seizures, with 17 of 22 (77.3%) experiencing seizures.[482] AHT was also a significant risk factor for electrographic-only seizures.

Surgical Management

A significant number of children presenting with AHT require surgical intervention. In a 5-year review of children presenting to Le Bonheur Children's Hospital, 58 of 213 children with AHT required surgical intervention. Unfortunately, no evidence-based guidelines exist to guide the selection of surgical strategies for the management of the patient with AHT. Case series and pathology-specific literature can guide providers in the care of these patients.[9]

- Skull fractures and epidural hemorrhage: Literature-based guidance regarding the management of depressed skull fractures and epidural hemorrhage is limited. The Brain Trauma Foundation sponsored a "rigorous literature-based" review to provide recommendations for the surgical management of patients with post-traumatic intracranial mass lesions.[462,463] The document provided indications for elevation of open and closed fractures as well as evacuation of epidural hemorrhage on the basis of size and neurologic status of the patient.[484,485]
- Subdural hemorrhage: Fluid collections are common in the subdural space of children who have experienced AHT. Subdural hemorrhagic collections have variable physical properties depending on factors to include the age of blood in the fluid present and include jelly-like

collections of clotted blood, pink-tinged mixtures of spinal fluid and blood, and clear yellow or thick motor oil consistency fluid. In addition to influencing the selection of surgical technique used for management, the heterogeneity of these collections can lead to inconsistent use of terminology and resultant confusion among those reading diagnostic reports.[354]

At the time of this publication, no established guidelines for the management of subdural fluid collections in AHT have been established. Options to include simple transfontanelle tap, irrigation/closed drainage, irrigation/open drainage, minicraniotomy, and subdural peritoneal shunt placement have been described on the basis of anecdotal experience and provider preference.[49,486,487] For subdural hemorrhages with mass effect, standard craniotomy and sometimes decompression is undertaken. In children requiring surgery for solid clots with mass effect or those failing maximal medical management, decompressive craniectomy has been advocated, although the mortality and complication rates of this procedure are significant in the infant and toddler. Oluigbo et al described their experience with this population, with a reported mortality rate of 35.7%.[48]

- Cerebral infarction: The most severe injuries sustained by children with AHT result in regional ischemia/infarction; the mechanisms of these injuries are incompletely understood, but they affect up to 30% of patients with AHT with an estimated mortality of 25%.[118]

Team Approach to Management of the Child With AHT

Several organizations have cited the value of a multidisciplinary team approach to the evaluation and treatment of the child with AHT.[31,466] Essential team members include the trauma surgeon, neurosurgeon, pediatrician (ideally, a child abuse pediatrician), and a social worker.[488] The expertise of additional medical and surgical subspecialists to include orthopedics, ophthalmology, critical care, neurology, hematology, radiology, and others may be required in individual cases. Additional resources to include nursing, behavioral health, physical and occupational therapy, child life, child protective services, and law enforcement. All parties strive to ensure the timely collection of clinical information affecting both medical decision-making and forensic analysis of the patient's presentation. Excellent communication between neurosurgeons, intensivists, and ophthalmologists can allow for safe diagnostic eye evaluations without effect on patient monitoring.[373] Additional radiographic evaluations and interviews between child abuse pediatricians, social workers, and families can provide critical information regarding timing and mechanism of injury.

Community Management

Individual clinicians are responsible for the assessment of their own capabilities and those of their place of practice. There is currently no literature that establishes which patients can be safely evaluated and/or cared for by whom and in what setting. Likewise, no literature exists that provides guidance regarding which patient requires transfer for specialized care and by what means. However, materials are available relevant to the consideration of this topic. The consequences of missed injuries are well established; in a review of 54 children seen by physicians for symptoms related to AHT, Jenny et al reported a reinjury rate of 27.8% and a mortality of 7.4%.[21] The American College of Surgeons (ACS) established "Best Practice Guidelines for Trauma Center Recognition" that establish the challenges and extensive resource requirements for certification by their organization regarding the diagnosis and treatment of abusive head trauma.[488] This same organization provides instruction on the initial evaluation of the trauma patient that establishes the referring health care provider as responsible for selecting and initiating the appropriate mode of transfer for the patient.[331] Finally, the potential for conflict of interest influencing family decision making regarding self-transport has been recognized in prior research examining the selection of transport means for pediatric patients seen at a level 1 trauma center.[489] Cost of transport was a recognized driver of decision making, suggesting that factors other than patient well-being may influence family decisions. The referring provider may strongly consider the patient's safety in their selection of a mode of transport for a patient with known or suspected AHT.

TAKEAWAY POINTS

1. Medical guidelines for the management of pediatric severe closed head injury are not specific to AHT.
2. Existing literature points to low utilization of ICP monitoring and high rates of subclinical status epilepticus in patients with AHT.
3. Treatment options for chronic/liquid subdural hemorrhage include observation, transfontanelle tap, irrigation with open or closed drainage, craniotomy, and subdural peritoneal shunt placement; management of acute hemorrhage with mass effect follows similar principles for older children including consideration of decompression.
4. AHT with cerebral infarction caries significant mortality.
5. Children with AHT have the potential for multisystem medical disease and require evaluation and treatment that is optimally provided through multidisciplinary collaborative care.
6. Physicians may strongly consider the patient's safety when referring/transporting a patient from either an

outpatient facility or small hospital with limited resources.

PATHOLOGY

Introduction

The National Association of Medical Examiners (NAME) and the AAP endorse universal forensic autopsies for all unexplained infant deaths.[490] All unexpected pediatric deaths are investigated by a medicolegal death investigation system consisting of a mixture of physician medical examiners and lay coroners spread over approximately 2000 autonomous jurisdictions; autopsies are usually performed by forensic pathologists certified by the American Board of Pathology.[491] Forensic pathologists routinely serve as panel members on regional child death/fatality review committees.[492–494]

The medicolegal autopsy aims to understand the cause, manner, and mechanism of death, each of which has a specific connotation when used in this context.[495–497] The *cause* of death refers to the injury or disease process that initiated the lethal sequence of events (eg, traumatic head injury) while the *manner* considers the circumstances.[495] The *mechanism* of death refers to the terminal pathophysiologic derangement that ended in death (eg, seizure, hypoxic-ischemic encephalopathy, cardiac arrythmia, etc) and is usually not etiologically specific.[498]

The potential causes of sudden unexpected death in early childhood are numerous and span a spectrum from rare natural diseases to inflicted injuries. To accurately catalog the cause of pediatric demise, death investigation is a dynamic and comprehensive process that integrates pathoanatomic findings at autopsy with available history, imaging data, circumstances of the death, and the results of ancillary studies that include but are not limited to histology, toxicology, microbiology, metabolic, and molecular studies, among others.[499,500]

As mentioned earlier in this report, the literature on physical findings was derived primarily from the clinical literature of non-fatal AHT cases. Epidemiologic and clinical features of fatal AHT cases are discussed in the section below.

Autopsy in Suspected AHT

Although as many as one-third of AHT cases have a lethal outcome and require an autopsy, determination of the cause of death may be challenging, because a history of trauma is often not easily forthcoming and external signs of injury may be subtle or absent.[500,501] Published reports and guidelines from professional societies assist forensic pathologists in the investigation and certification of sudden pediatric death.[490,502–506] The National Association of Medical Examiners currently has no formal recommendations for suspected head trauma in infants and young children, the 2014 position paper having sunsetted per the society's routine 5-year limitation policy; however, this resource is still informally referenced by many pathologists for guidance on autopsy procedures, interpretation, and documentation of findings.[500,501,507]

An autopsy in suspected AHT is, thus, a specific and large independent dataset with core data elements that include digital photography, radiologic skeletal survey comprising full-body plain radiography, with comprehensive external and internal examination, and ancillary studies to document injury and exclude significant natural disease.[500] These findings are interpreted in the context of history from medical records and other agencies including law enforcement.[500,508] A forum has addressed some of the challenges and controversies faced by forensic pathologists in the autopsy investigation and analysis of childhood death.[509–516]

Limitations of Autopsy Data

Autopsy data are an endpoint with published descriptions of AHT findings generally consisting of small descriptive series. These data are often limited by small sample sizes; incomplete and variable data; selection bias; small numbers of adequate controls; varied survival intervals; confounding variables, such as secondary changes from life-saving interventions instituted during life or artifacts of maintenance on artificial ventilation before death in children with hypoxic-ischemic brain injury; inconsistent inclusion of histopathologic descriptions; and noncontemporaneous methodology to assess axonal pathology in older series limiting comparisons across datasets.[122,133,269,286,517–528]

Even though specialized autopsy techniques for examination of the central nervous system and related structures are described, some of these procedures require technical expertise and are variably implemented by forensic pathologists.[529–535] In addition, AHT fatalities are typically investigated as discrete individual events, such that robust study designs including blinding are almost never feasible. The situation is further strained by an underresourced, understaffed, and nonstandardized death investigation system.[536,537] Despite these limitations, autopsy observations continue to inform the pathobiology and classification of pathologic mechanisms of AHT and represent the bedrock of forensic investigation in sudden and unexplained childhood death.

The most common neuropathologic findings reported across AHT autopsy series include subdural hemorrhage (90%–100%), subarachnoid hemorrhage (60%–100%), RH (20%–100%), cerebral edema (28%–100%), global hypoxic-ischemic injury (37%–78%), traumatic axonal injury (less than 6%), and contusions (7%–67%).[109,122,133,269,501,517–520,522,523,527,538–542] It should be noted that these broad ranges are derived primarily from small qualitative studies, many of which had low numbers

(less than 20 cases), missing datapoints, or nonspecified diagnostic criteria. In isolation, none of these findings is specific to inflicted trauma. However, the constellation of these findings in a pattern not explained by the available history, or combined with unexplained extracranial injuries, alerts the pathologist to the potential of inflicted trauma.[543] These findings are, in turn, modulated by developmental host responses to injury, which are subject to age-dependent maturational differences of the developing brain.[544–546] Importantly, neuropathologic examination serves also to exclude other potential nAHT pathologic processes, such as infection or a ruptured vascular malformation that could account for unexpected death.

FINDINGS AT AUTOPSY

External Findings

The proportion of AHT cases with external contact injuries of the head or facial region varies markedly across autopsy series, with contact injury found in 16% to 100% of cases, although many series are again limited by low case numbers.[133,269,517,519,522,523,527,528,540,547] However, not all fatally injured children have an impact site despite careful examination.[133,521,522,548] This absence of external contact findings could support the inference that injury in these children relates to violent shaking or head impact with deceleration against a padded surface.[133,269,501,522,540] Case selection criteria influence reported skull fracture frequency across autopsy series with reported proportions ranging from 17% to 64% and a propensity for the posterior parietal or occipital bone.[269,517,518,523,540,549] Nondisplaced skull fractures may be difficult to detect at autopsy, and microscopic confirmation of a suspected fracture site stage of healing might be necessary.[500]

Acute Subdural Hemorrhage

Acute subdural hemorrhage (SDH) is observed in more than 90% of AHT autopsies, typically as a thin film of hemorrhage, (<5–10 mL), along the posterior interhemispheric fissure or convexity surfaces of the posterior cerebrum.[90,106,109,501,517,539,550] Although considered a marker for the amount of force transmitted through the brain and underlying brain injury, it is rarely a space-occupying lesion in infants.[109,539] The pathogenesis includes laceration of bridging veins extending from the cerebral surface to the dural sinuses as a consequence of acceleration-deceleration of the head, and less frequently laceration of a dural venous sinus from displaced skull bone fragments.[68,92,109,110,112,551–555] Nontraumatic causes including coagulation disorders, neurosurgical intervention, ruptured vascular malformations, neoplastic and metabolic disease, among others, are rare and can be distinguished with assistance of a complete autopsy and ancillary studies as indicated.[556]

Chronic SDH

The pathogenesis of chronic SDH is complex and poorly understood but includes extravasation of blood and cerebrospinal fluid into the subdural space following border cell layer disruption resulting in fibroproliferative and chronic inflammatory responses with neomembrane formation, angiogenesis, and localized coagulopathy.[557,558] Estimating the timeframe of these pathologic processes by microscopic examination is imprecise and potentially error prone.[559] Gradual enlargement of a chronic subdural hemorrhage may result from microhemorrhage associated with neovascularization.[109] Prospective imaging studies have demonstrated that acute rebleeding within chronic SDH is uncommon and usually minor and asymptomatic when it occurs.[90,362]

Subarachnoid Hemorrhage

Acute subarachnoid hemorrhage (SAH) is common in traumatic brain injury, with a propensity for parasagittal surfaces of the cerebrum, often in a patchy distribution. Although not a distinguishing feature between AHT and nAHT in clinical studies of live children, SAHs are reported in 60% to 100% of fatal AHT cases.[109,269,501,517,519,520,523,540,547] Nontraumatic causes of SAHs, such as ruptured vascular malformation or venous sinus thrombosis, are usually readily distinguished from traumatic causes on examination of the brain at autopsy.

Epidural Hemorrhages

Intracranial epidural hemorrhage (EDH) of arterial or venous origin is uncommon in AHT autopsy series and is rare without associated skull fracture.[517,523,560–563] The dura in infants is more tightly adherent to the skull than later in life, which may be one reason why EDHs are less common in children. Arterial EDHs typically result from laceration of the middle meningeal artery; venous EDHs are associated with skull fractures and lacerated diploic veins, particularly in the posterior fossa.[564] EDHs are relatively infrequently described in autopsy series, reflecting amenability to surgical intervention and an overall favorable prognosis, although some may spontaneously resolve.[565,566]

Cerebral Edema

Rapidly progressive cerebral edema leading to diffuse brain swelling is a prominent autopsy finding in children with fatal head injury.[567,568] Although limited data exist for AHT, several nAHT series report a high prevalence of cerebral edema associated with ischemia and traumatic axonal injury following head injury in young children.[569–573] The pathogenesis of rapid cerebral edema following brain trauma remains poorly understood and is likely multifactorial with cellular and vascular mechanisms implicated.[571,574,575]

Diffuse Axonal Injury

Diffuse traumatic axonal injury (dTAI) refers to a pattern of white matter axonal injury following rapid acceleration-deceleration, characterized pathologically by the accumulation of APP in axonal swellings and bulbs.[576,577] This test is the gold standard for detection of axonal injury in fatal injuries, with injured axons potentially detected within 35 to 45 minutes following head trauma.[576–589]

Although APP is a sensitive marker for axonal injury, it is not specific for mechanical injury, and accumulation occurs in other disease states including hypoxia, infection, and inflammatory disorders, underscoring the necessity for clinicopathologic correlation in all cases.[581,590–592] The inability to reproducibly distinguish mechanical from hypoxic causes of axonal injury using APP staining undermines direct evidence for dTAI since hypoxic-ischemic injury is common in AHT and more specific diagnostic makers are not available. This is further compounded by limited, nonstandardized autopsy data with low case numbers and lack of uniform diagnostic criteria for dTAI.[122,500,517,519,520,542,571,593] Further, older autopsy series predating APP immunohistochemistry lack the requisite sensitivity to detect axonal injury, resulting in inconsistencies when AHT autopsy datasets are reassessed.[122,518,593] Despite the aforementioned limitations of APP staining, the presence of cortical contusions and subcortical white matter lacerations in some cases suggests the etiology of axonal injury might not be adequately explained by hypoxia-ischemia alone in all cases.[519] Several published AHT autopsy series report macroscopic brain descriptions only, and although typical macroscopic features of dTAI such as dorsolateral quadrant brainstem hemorrhages are usually not described, more comprehensive microscopic descriptions of axonal pathology are lacking in several series.[133,269,593]

Localized Axonal Injury

Hyperextension neck injury and cervical spine distraction during shaking or impact are potential mechanisms of spinal cord stretch injury characterized by localized accumulation of APP in axons of white matter brainstem tracts in the cervico-medullary region and pons, a finding described in infants with AHT.[122,133,500,517,519,520,542,571,593] These localized axonal changes are potentially survivable but represent a heightened potential for progression to more severe hypoxic-ischemic injury and cerebral edema, which in turn leads to death.[122] However, apnea complicating cervico-medullary injury does not fully explain the unilateral pattern of cerebral hemispheric injury following AHT seen in some cases, suggesting nonglobal mechanisms of injury are also at play, at least in some instances.[594,595] Localized traumatic axonal injury associated with AHT has been described in brainstem corticospinal tracts and high cervical cord and spinal nerve roots and is associated with cervical epidural hemorrhage in some cases, although complete brainstem data are not reported for all cases.[122,133,286,517,519,596] Isolated epidural or meningeal hemorrhage, reported in some series, is of unclear significance and should be interpreted cautiously without additional supporting evidence of inflicted trauma.[535,597]

Cervical Spinal Cord Injury

Cervical spinal cord injuries are common, although not universal in fatal AHT series, with spinal cord injury reported in up to 80% of cases in some series that document spinal findings.[122,286,519,523] Reported injuries include primary spinal cord trauma, cord edema, spinal nerve root injury, dorsal root ganglion, and extramedullary compartment hemorrhages.[120–123,128,132,133,286,519,598]

Autopsy techniques to assist pathologists in the evaluation of these structures are described.[529,530,535] Localized stretch injury of the spinal cord is postulated to result from hyperextension or distraction movement of the neck, which is thought to predispose to mechanisms of death that include trauma-induced apnea, hypoxic-ischemic injury, and cerebral edema.[120,122,123,599] Suggested diaphragmatic denervation attributable to cervical spinal injury does not fully explain similar changes in AHT cases without cervical cord damage or account for the nonglobal patterns of hypoxic-ischemic injury described in some infants.[122,286,531,594,596,600]

Hypoxic-Ischemic Injury

Hypoxic-ischemic change is a form of secondary brain injury (ie, brain injury that encompasses a reduction in the supply of oxygen [hypoxia] and/or diminished blood supply to the brain) that is frequently reported in AHT series and results in substantial axonal injury burden.[122,517,519] Mechanisms of hypoxic-ischemic change are multifactorial and include concussive-like causes of apnea resulting from cervico-medullary injury, aspiration attributable to muted airway reflexes, perturbation of autoregulation with altered cerebral blood flow, excitotoxicity, altered neurometabolic demands after injury, and seizures in acutely injured brain tissue.[601] Diffuse axonal injury of vascular and ischemic origin is the most frequently reported brain finding across AHT autopsy series.[122,519,528,542,547,583,602]

One small retrospective study found comparable APP staining profiles in cerebral white matter of AHT and hypoxic-ischemic injured cases but brainstem axonal staining only in AHT cases, suggesting cerebral damage in both groups resulted from hypoxia.[519] Another well-annotated neuropathologic series of 37 children found widespread hypoxic-ischemic injury with cerebral edema as the most common finding.[122] A population-based cohort study of perinatal asphyxia in neonatal deaths of preterm and term infants found APP staining patterns could not be reliably

distinguished from mechanical axonal injury, although none of these infants had a known history of trauma.[603] The apparent contradictory findings in some series including reports of dTAI in some AHT cases but not controls underscores the lack of standardized diagnostic criteria applied to dTAI across series as well as methodologic issues that can relate to inadequate age-matched controls, confounding, and insufficient brainstem data.[520]

Cortical and Subcortical Contusions

Because of the immaturity of the infant brain, blunt head trauma at younger than 5 months may be associated with subcortical white matter contusional lacerations, with a predilection for the frontal, occipital, and temporal lobes.[604] Occasionally, these extend into overlying cortex as cortical tears or become distended by blood to form intraparenchymal hemorrhages. Data regarding the frequency of subcortical contusional lacerations in fatal AHT are limited.[269,517,518] One larger descriptive series of 16 infants younger than 5 months with contusional lacerations included 9 infants with head trauma history; the remaining infants had autopsy findings either definitive or suspicious for trauma.[604] Evidence to support nontraumatic etiologies of these lesions, such as obstructed superficial venous drainage, is limited.[605]

Sinovenous Thrombosis

Secondary sinovenous thrombosis may occur as a consequence of venous injury following accidental or inflicted head trauma.[543] Further, it is not unusual to find thrombosed venous sinuses at autopsy examination secondary to raised ICP and relative stasis of venous blood flow.

Postmortem Ocular Findings

Postmortem examination of the eye and orbital tissues can reveal abnormalities not appreciated on antemortem clinical examinations. Such findings include hemorrhages of the optic nerve sheath, extraocular muscles, and orbital fat in cases of child fatality and suspected AHT.[606–608] A protocol to assist forensic pathologists in removal of the orbital contents for standardized examination has been described.[533] Optic nerve sheath hemorrhage is not always accompanied by retinal hemorrhage and can also be seen in patients with nAHT. It tends to be most prominent anteriorly and does not necessarily extend the length of the optic nerve.[143,609–611] Optic nerve sheath hemorrhage frequently involves multiple layers around the optic nerve. The location can be subdural (78%), intradural (61%), or subarachnoid (26%) or involve multiple compartments.[607,612] The extent of the optic nerve sheath hemorrhage does not necessarily correlate with the severity of intraocular findings.[610,613,614]

Retinal findings in nonfatal AHT cases are described earlier in detail in the section on ocular findings.

TAKEAWAY POINTS

1. Autopsies are performed as part of the investigation of a child fatality suspected to be attributable to injury to determine the cause and manner of death.
2. SDH, SAH, RH, cerebral edema, hypoxic-ischemic injury, and traumatic injury are the most common autopsy findings in AHT.
3. Pathologists are alerted to the possibility of AHT when the autopsy findings do not comport with the available history.
4. Detailed examination of the central nervous system, orbital contents, and dura are important components of the autopsy in AHT.
5. Optic nerve sheath hemorrhages are less specific for AHT than intraocular hemorrhages, perimacular retinal folds or macular retinoschisis.

OUTCOMES

Research in outcomes in AHT spans many domains. These include series assessing short- and long-term cognitive, behavioral, and motor outcomes with and without comparison to accidental injuries; predictors of outcome based on acute imaging features; predictors of outcome based on acute presentation factors; visual outcomes; post-traumatic epilepsy; and social and legal outcomes.

Cognitive and Behavioral Outcomes and Their Predictors

Mortality in AHT is high compared with head trauma in general in this age group, averaging about 20% among various series.[43,615] Although incidence is higher in infants younger than 1 year, mortality is higher in children between 12 and 23 months of age.[616] Survivors have variable outcomes, with a significant percentage having cognitive and behavioral difficulties, averaging about one-third with "good" outcomes and two-thirds having deficits ranging from mild to severe.

Specific outcomes scales and categories vary among reports, but as a general rule, "good" or "mild" outcomes signify no or mild deficits that do not interfere in a major way with activities of daily living or require major modifications in schooling; "moderately" affected children have needs that require educational adaptations or include physical limitations; and "severe" outcomes include difficulties with activities of daily living. Using these general categories, about one third of children with abusive head trauma are severely disabled, including those who are nonverbal, nonambulatory, and with severe brain atrophy on imaging, even many years after injury.[100,418,599,615,617–619] Of note, of those children who initially seem only mildly or moderately affected, deficits may become more apparent with age, as cognitive and behavioral demands of school and socialization increase.[617,620–625]

Several studies comparing outcomes between children with inflicted and accidental injuries show worse outcomes

overall in children in the inflicted category. These include both short-term and longer-term outcomes in the cognitive, verbal, motor, and behavioral domains.[100,384,387,626] Despite significant deficits, however, children with inflicted injuries have been found to have similar health-related quality of life to that of healthy children when assessed by parent questionnaire when children are about 2 years of age.[625]

Imaging predictors of worse outcome include larger areas of loss of grey/white differentiation, hemispheric hypodensity or signal change on CT or MRI, and larger areas of diffusion abnormality on MRI.[91,107,384,600,627,628] Acute predictors of worse cognitive outcomes include early seizures, older age, need for intubation, metabolic perturbations including base deficit, and having a male nonparental perpetrator.[388,481,629–633]

A small number of studies have assessed the results of acute intervention or rehabilitation. Decompressive hemicraniectomy, most often performed for children with a predominantly unilateral subdural hemorrhage and significant mass effect or associated hemispheric brain injury, may help children with inflicted injury as well as children with accidental trauma. But as in other series, the AHT group has worse outcomes, likely reflecting more severe injuries overall.[48,634] Similarly, rehabilitation and early intervention services appear to benefit young children with inflicted and accidental injuries, although those in the former group, especially when injured at younger ages, do worse overall.[626,635]

Visual Outcomes

Visual outcome is good (ie, visual acuity of 20/40 or better) in one-third to one-half of survivors.[145,211] Most intraretinal hemorrhages resolve spontaneously without sequelae. However, serious visual impairment occurs in 25% to 30% of children surviving AHT.[145] The main causes of visual impairment are nonclearing vitreous hemorrhage, which can cause deprivation amblyopia, induced myopia, and refractive amblyopia; retinal scarring or fibrosis, possibly secondary to retinoschisis; optic nerve atrophy; and cortical visual impairment from brain injury.[141,636–638] Direct optic nerve injury from acceleration-deceleration forces within the orbit is the most likely cause of the atrophic damage to the optic nerve. Because papilledema occurs rarely and is commonly transient, it is not a common cause of optic atrophy. Trans-synaptic degeneration and retinal injury are also not likely causes of optic atrophy. In addition to central vision loss, patients may experience peripheral visual field loss, color vision impairment, decreased contrast sensitivity, decreased binocularity, and secondary amblyopia.

RHs are not a major cause of visual loss unless the fovea is directly involved.[145] If the fovea is obscured for a prolonged period of time, vision loss from amblyopia may result. Preretinal dome-shaped hemorrhages can break into the vitreous, usually within 3 to 4 weeks after the injury, causing a dense vitreous hemorrhage that can interfere with vision and may cause amblyopia and induce high axial elongation and myopia in young children. Vitrectomy may be indicated in patients with dense vitreous hemorrhage, although these patients rarely have good visual recovery.[639]

Strabismus develops in up to 30% of children who experience AHT.[640] Sixth nerve palsy, nystagmus, and ophthalmoplegia have been reported. Other ophthalmic sequelae include retinal detachment, dislocation or opacification of the crystalline lens (ectopia lentis and traumatic cataract), and phthisis.[640] Bilateral retinal nonperfusion may also occur in AHT survivors and may lead to optic nerve or retinal neovascularization. Children with positive eye findings require regular follow-up by an ophthalmologist. Visual potential in children with AHT may be limited by retinal, optic nerve, and central nervous system injuries. Children also often have neurologic, cognitive, and psychologic disabilities. With these limitations, the correction of refractive errors and treatment of amblyopia can help maximize the child's visual potential and visual learning ability.[619,640] For children in whom vision cannot be improved, auditory and tactile learning can be promoted through early intervention programs.

Social and Legal Outcomes

Less than one-third of AHT cases are accompanied by a confession by a perpetrator.[30] A 1986 study showed that in fatal cases of child abuse, a minority lead to a conviction in the legal system, and in 78% of cases, no jail time was served.[641] With respect to disposition, of children diagnosed with inflicted head injuries in England, 50% were in foster care or had been adopted. Thirty-two percent lived with their birth mother, and 18% lived with extended family.[418] Physical abuse is associated with a variety of long-term psychological dysfunctions when abused children become adults.[642] However, overall, there is a paucity of data on the true long-term effects of early child abuse from the social, legal, and economic perspectives throughout the lifespan of children with early inflicted head injuries, and these remain target areas for new research.[643]

Economic Outcomes

There is some evidence that children who have experienced AHT or other forms of abuse early in life have a higher risk of detrimental long-term socioeconomic consequences. These consequences include negative socioeconomic effects on the abused children but also on families, caregivers, communities, and societies overall.[644,645] For this reason, some authors have suggested that prevention measures may be considered cost-effective when compared against the high

societal and economic consequences for children who sustain AHT early in life.[645]

Motor Outcomes

AHT may cause persistent motor complications including hemiplegia, quadriplegia, and/or fine motor deficits.[646] Several longitudinal studies have measured long-term motor outcomes at 5 or more years following abusive head trauma. Although less common than behavioral or vision problems, fine and gross motor difficulties have been reported in between 25% and 60% of patients.[617,623,624,647,648] Gross motor impairments are primarily spastic hemi- or quadriplegia. These children require ongoing care with pediatric rehabilitation specialists. Factors that predict higher likelihood of long-term disability include the severity of initial presentation (intensive care unit admission, longer hospital stay, lower GCS score, respiratory compromise), extent of injury (cranial fracture, higher brain lesion burden, cerebral edema, seizures) and lower socioeconomic status.[623,648,649] Importantly, multiple studies have found disability incidence or severity was worse at longer follow-up.[647,648] Therefore, long-term longitudinal care of all children after AHT is important, even for patients with initial good recovery.[647]

Post-traumatic Epilepsy

Many children will go on to experience post-traumatic epilepsy (PTE) as a long-term sequela after AHT (10%-40%).[617,623,624,647,650–652] PTE is a long-term seizure disorder defined as having 2 or more unprovoked seizures following traumatic brain injury and is independent of the presence of seizures during the acute period of AHT. For many of these patients, epilepsy is described as intractable, or refractory to multiple antiepileptic medications.[623,651] Although children with PTE following AHT may have multiple seizure types, 1 retrospective study of 10 years of electronic health records at a single center identified hypomotor seizures as the most common seizure type.[651] Severity of injury and having seizures early during the acute presentation and hospitalization for AHT are risk factors for PTE, with the latter increasing the likelihood of PTE by over thirty-fold.[650,651] Although seizure prophylaxis with antiseizure medications can prevent early onset seizures, this practice has not been found to prevent later onset PTE.[653] PTE is notably more frequently found in patients with AHT than accidental or noninflicted head trauma.[623,651]

TAKEAWAY POINTS

1. As a group, children with AHT, in general, have worse outcomes including higher mortality than children in a similar age range with accidental trauma. This likely reflects an increased severity of injury overall in this group.
2. Cognitive and behavioral outcomes range from good to severely impaired. These outcomes may become more apparent as children become older and have more cognitive demands such as school.
3. Although RHs generally clear rapidly, approximately half of children with AHT may have long-term visual impairments attributable to both ocular and cortical injury.
4. Social, legal, and economic outcomes are less well studied but suggest long-term consequences in many children and families after AHT.
5. Fine and gross motor impairments are common after AHT and contribute to significant long-term disability. Similar to cognitive and behavioral deficits, motor deficits may become more apparent over time.
6. Some children may go on to develop PTE.

PREVENTION

Introduction

AHT prevention efforts have targeted various outcomes: improved caregiver or provider knowledge about the dangers of shaking or AHT in general, improved caregiver knowledge about infant crying, reducing infant crying, improved caregiver emotional regulation, and reduction in AHT incidence rates. Current empiric evidence demonstrates efficacy of prevention programs to affect caregiver knowledge and behavior with regards to AHT, infant crying, and emotional-behavioral responses to infant crying. However, replicable effect on AHT incidence rates is lacking.

Improved Knowledge About the Dangers of Shaking or AHT in General

At least 25 studies have examined 12 different initiatives aimed at raising caregiver awareness about AHT and/or the dangers of shaking.[654] The Period of PURPLE Crying program is a multifaceted, hospital-based postpartum program that focuses on parental education about infant sleeping, crying, and soothing behaviors. The program also provides educational materials to caregivers describing AHT and the dangers of shaking. Barr et al found that participants exposed to these educational materials showed a significant increase in knowledge about the risks of shaking an infant.[655] Implementation of the Period of PURPLE Crying program in various countries has confirmed the effect of improved caregiver knowledge about the dangers of shaking.[656–658]

Similarly, the Shaking Your Baby is Just Not the Deal educational program developed in Australia has also demonstrated improved caregiver knowledge about the risks/dangers of shaking.[659] When implemented in other countries, this program has also shown replicable results.[660,661] Several other hospital-based parent education programs,

provided in maternity wards, have also demonstrated improvements in caregiver knowledge about the dangers of shaking and AHT.[662,663] These include Love Me…Never Shake Me, The Hand Project: More Hugs, No Shakings, and the Colombia Prevention Program, to name a few. Thus, empiric evidence adequately demonstrates that hospital-based caregiver education programs do improve caregiver knowledge about the dangers of shaking and AHT.

Improved Knowledge About Infant Crying

Another prevention tactic has been to educate caregivers about normal infant crying patterns, given that infant crying is a well-documented triggering event for AHT.[664] Again, there are numerous programs that have studied the effect on caregiver knowledge of infant crying patterns.[657,659,662,665] The most widely implemented and studied has been the Period of PURPLE Crying program discussed earlier. The Period of PURPLE Crying program provides a 12-minute DVD along with a booklet that addresses the normality of crying, suggests strategies to comfort the infant, reinforces the idea that such strategies will not always work, and describes the reasons why inconsolable crying is frustrating. In addition to its demonstrated improvement of caregiver knowledge about the dangers of shaking, the program has showed a significant increase in caregiver knowledge regarding infant crying patterns.[655,657,665]

Reduction of Infant Crying and Modification of Caregiver Behavior or Emotional Regulation

Although knowledge is an important outcome of prevention programs, several initiatives have targeted behavior modifications (either infant or caregiver) as primary outcomes. These initiatives have included efforts to reduce infant crying and efforts to modify caregiver response to the stress/frustration of infant crying.

With regard to reduction in infant crying, at least 7 studies have been published on 5 different initiatives. The REST Routine for Infant Irritability program is a nurse home-visit program aimed at reducing an infant's level of arousal by promoting synchrony in the parent-infant dyad.[666] In a study that included 121 infants between the ages of 2 and 6 weeks, parents of infants were randomly distributed among the experimental group, receiving the REST Routine intervention, and the control group, receiving standard well child care intervention. Keefe et al found that infants in REST Routine group were crying 1.7 hours less per day compared with the control group, and at the end of intervention, 61.8% of infants in the REST Routine group and 28.8% of infants in the control group reduced the crying average to less than 1 hour per day.

Another initiative, The Happiest Baby, involves teaching caregivers a 5-step method to calm infants and stop crying on the basis of the assumption that actions mimicking conditions in the womb will trigger a calming reflex. McRury and Zolotor evaluated the effectiveness of this intervention by randomly assigning 35 parents to 2 conditions: 1) a group that received a video that taught The Happiest Baby method by mail; and 2) a control group that received a video (by mail) with guidelines for standard child care.[667] After 12 weeks of monitoring, McRury and Zolotor found no differences for amount of infant crying and parental stress between the two groups. A similar intervention, the Baby Business intervention, also underwent a randomized controlled trial (RCT) and did not demonstrate reduction in infant crying.[668]

A couple of other methods to reduce infant crying—swaddling and acupuncture—have also been studied. Van Sleuwen et al studied an initiative in which parents were educated about proper swaddling and sleep/play interactions via nursing clinic visits and telephone contact. Parents were randomly assigned to experimental and control groups.[669] Swaddling had no added benefit in the total group, but a small but significant effect was shown in infants aged 1 to 7 weeks. A separate RCT comparing swaddling versus massage in infants with neonatal cerebral insults has shown that swaddling significantly decreases the amount of crying compared with massage.[670]

Landgren et al explored the effects of acupuncture on reducing infant crying by randomly dividing 81 infants with colic into 2 groups.[671] An experimental group underwent 6 sessions of a "minimal, standardised acupuncture" (ie, a sterilized, disposable acupuncture needle inserted at an approximate depth of 2 mm at point LI4 of the hand's first dorsal interossal muscle and left in place for 2 seconds) for 3 weeks. The control group experienced similar pretreatment efforts (ie, nurse soothing and comforting efforts) but without the use of acupuncture. After the intervention, parent surveys reported a significant reduction in the number of hours of crying for both groups, but the reduction was greater in the experimental (acupuncture) group.

With regard to prevention efforts aimed at modifying caregiver behavioral response or emotional regulation, several initiatives have been studied. These include programs that teach caregivers strategies to reduce their daily stress and programs that educate caregivers on behavioral modification approaches to deal with infant crying.[672–674] In addition to other educational materials discussed earlier, the Period of PURPLE Crying suggests 3 steps to deal with the infant crying: (1) increase responses of holding and walking with the infant in the caregiver's arms, comforting and talking to the infant; (2) if the crying becomes very frustrating, place the infant in a safe place, leave for a few minutes to calm down, and return to see how the infant is; and (3) never shake or hurt the infant.

Several studies examining Period of PURPLE Crying materials have demonstrated a higher frequency of caregiver response of walking away when frustrated.[658,665,672]

Additionally, studies have found that learners exposed to Period of PURPLE Crying materials have also shared knowledge with others on normal infant crying patterns and the risks of shaking infants.[655,658] Finally, Barr et al found that implementation of the Period of PURPLE Crying program diminished pediatric emergency department visits for crying.[675] Similar positive results for caregiver response in walking away to frustration events have been reported for the Take Five Safety Plan for Crying initiative and the All Babies Cry initiative.[673,674]

Reduction in AHT Incidence Rates

A few programs have measured the outcome of intervention effect on AHT incidence rates. Although a couple of programs have demonstrated initial reductions of AHT incidence rates in certain regions, no program has yet to demonstrate replicable results in other regions. In 2005, Dias et al reported on a hospital-based program—Prevent Shaken Baby Syndrome!—implemented in the newborn period in which parents were provided written and video content about the dangers of shaking, discussed the risks of shaking with a maternity nurse, and then gave written affirmation that they received and understood the information.[676] This program initially demonstrated a decrease in AHT incidence rates in some regions of New York.[677] However, larger implementation across the state of Pennsylvania by Dias et al failed to demonstrate a reduction in AHT rates.[678]

Similarly, the Period of PURPLE Crying revealed a decrease in AHT incidence rates in British Columbia[679] but failed to demonstrate a reduction in AHT incidence rates in North Carolina when implemented there.[680]

Some additional studies have examined the effect of economic support as a factor in the reduction of AHT incidence rates. In California, Klevens et al reviewed the effect of paid family leave and the earned income tax credit on AHT rates.[681,682] Using difference-in-difference analysis of state level data from 1995–2011, Klevens et al found a significant decrease in AHT admissions in both <1- and <2-year-olds in California after implementation of a 2004 paid family leave policy. In comparison with 7 states with no such paid family leave, Klevens et al found no significant reduction in hospital admissions for AHT in those states. Using a similar analysis on 14 states with an Earned Income Tax Credit (EITC) and 13 states without one, for the time period 1995 to 2013, Klevens et al found that a refundable Earned Income Tax Credit was associated with a decrease of 3.1 AHT admissions per 100 000 population in children aged <2 years.[681] Although these economic policy effect studies demonstrate possible association, further research is needed.

Finally, in-home visitation programs, such as Nurse-Family Partnership,[683] have demonstrated a long-term decrease in child maltreatment in general. Although such programs have not targeted reductions in AHT rates specifically, their holistic approach to bolstering parenting resilience may be a useful approach in addressing AHT.

TAKEAWAY POINTS

1. There is an evidence base among multiple initiatives that demonstrate AHT prevention programs improve caregiver knowledge on infant crying, the consequences of shaking an infant, and AHT in general.
2. There is an evidence base among several initiatives that demonstrate AHT prevention programs improve caregiver response of walking away when frustrated or agitated by a crying infant.
3. There is a small evidence base that indicates initiatives to reduce infant crying are effective.
4. There is a small evidence base that indicates AHT prevention programs reduce AHT incidence rates.

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APPENDIX A. DEFINITIONS

1. Abusive head trauma: An injury to the skull or intracranial contents of an infant or young child (<5 years of age) attributable to inflicted blunt impact and/or violent shaking (Reference: Centers for Disease Control and Prevention. Pediatric Abusive Head Trauma: Recommended definitions for public health surveillance and research. 2012. Accessed December 18, 2024. https://www.cdc.gov/violenceprevention/pdf/PedHeadTrauma-a.pdf).
2. Acceleration/deceleration: Rate of change of an object's or body's velocity or speed.
3. Accidental head trauma: Injury occurring from mechanisms other than from abusive head trauma or assault.
4. Acute subdural hematoma/hemorrhage: A collection of recent, fresh blood between the arachnoid and the dura, typically (although not always) hyperdense and with a crescent shape on CT. Mixed density may be seen if the collection contains unclotted blood, CSF admixture, and/or active extravasation. On MRI, the acute SDH is iso/hypointense on T1 and very hypointense on T2, GRE, and SW imaging. Although exact timing/dating cannot be determined with absolute certainty, these findings typically characterize hemorrhage that has occurred within hours to a day or so.

5. Animal injury models: Exposing a nonhuman animal to a mechanical phenomenon such as acceleration or force to observe resulting injury so as to understand injury mechanism and threshold.
6. Anthropomorphic test device: A mechanical analogue representing a human that can be used in experiments to simulate potentially injurious events such as falls or motor vehicle crashes to investigate injury risk. ATDs can be equipped with instrumentation to measure acceleration, velocity, force, and other biomechanical outcomes.
7. Autopsy: A medical examination of the body after death that begins with examination of the external surface of the body followed by an examination of the internal organs.
8. Bioengineer: An engineer with expertise in both engineering and life sciences who applies their knowledge of engineering to the human body to better understand injury or disease process.
9. Biofidelity: How well a surrogate or anthropomorphic test device (test dummy) represents its targeted human population in terms of anthropometrics and response to mechanical phenomena (eg, acceleration, force).
10. Biomechanical compatibility: Determines whether the provided history is capable of generating the constellation of injuries and whether the characteristics of injuries are compatible with the provided history.
11. Biomechanical properties: Describes in engineering terms how tissues respond to loading.
12. Biomechanics: Application of mechanical principles to biological systems such as the human body.
13. Bridging veins: Cerebral veins that drain venous blood from the brain surface into the dural venous sinuses
14. Cause and manner of death: The cause of death is the specific disease or injury that initiated the lethal sequence of events. Manner of death is a determination made about the circumstances of death.
15. Center of mass (COM): The location within a body where mass is equally distributed and balanced. The COM of the human body changes as a person moves. The COM of an erect standing person is located at approximately 56% of their height as measured from the soles of their feet within the sagittal plane.
16. Cerebral perfusion pressure: Mean arterial pressure minus mean intracranial pressure.
17. Chronic subdural hematoma/hemorrhage: A collection of nonacute blood between the arachnoid and the dura, typically (although not always) with a crescent shape. On CT, a subacute or chronic SDH will be predominantly iso- or hypodense. On MRI, a subacute SDH will be hyperintense on T1 and will have varying signal intensity on T2. The chronic SDH is slightly hyperintense compared to CSF on both T1- and T2-weighted imaging. FLAIR imaging increases the conspicuity. If rebleeding has occurred in the collection (ie, "chronic recurrent SDH"), the signal may be a variable combination of hypo/iso/hyper-intensity/density on CT and all MR sequences. Internal loculations and septations may be seen on both CT and MRI and these are more conspicuous following intravenous contrast enhancement. Although exact timing/dating cannot be determined with absolute certainty, these findings typically characterize hemorrhage that has occurred in the range of a day or more previously.
18. Computer or computational models: Digitized representations of a human body or regions of a human body and its environment. Models of the human body can be either segmented (rigid body model) or discretized into elements (finite element model); most are 3 dimensional.
19. Computer simulation: Digitized representation of an event such as a fall that includes the includes the fall victim and fall environment.
20. Decompressive craniectomy: A surgical procedure in which portions of the skull are removed (and usually the dura opened widely) to reduce intracranial pressure and/or control dangerous intracranial tissue shifts.
21. Deformation: Change in shape (distortion) of an object under pressure or force.
22. Diffuse traumatic axonal injury: Pattern of white matter injury following rapid acceleration-deceleration characterized by altered consciousness, and the formation of axonal spheroids that contain accumulated amyloid precursor protein.
23. Dura: Tough collagenous connective tissue layer that forms the outer layer of the 3 connective tissue layers covering the brain (dura, arachnoid, and pia mater). The dura is divided into 2 parts: outer periosteal layer adherent to the skull and an inner meningeal layer; the dural venous sinuses are between these layers.
24. Edema: Brain swelling caused by increased fluid accumulation attributable to blood-brain barrier disruption with increased permeability, or intracellular fluid accumulation as a result of cell membrane injury or metabolic derangement, or a combination of both.
25. Epidural hematoma/hemorrhage (EDH): A collection of blood between the skull and dura. On CT, the EDH typically (although not always) has a biconvex shape, an adjacent skull fracture/scalp injury, and classically does not cross sutural margins. (In patients with skull fractures, especially those in children involving the sutures, this rule may not always apply.) The acute EDH appears hyperdense on CT, but may contain hypodense areas representing unclotted blood. As the EDH evolves, it gradually loses its hyperdensity and may appear iso- or hypodense. On MRI, the acute EDH is hypo- or isointense on T1- and very hypointense on

T2-, GRE-, and susceptibility-weighted imaging (SWI). The inwardly displaced dura should be directly visualized on MRI as a thin dark line on all pulse sequences.

26. Fall: An event in which there is a change in the person's center of mass (COM) from a higher elevation to a lower elevation. This includes ground-based falls or falls from an elevated surface. When focusing on head injury, fall height is typically measured from the head COM to the impact surface.
27. Fall dynamics: Describes the motion or movement of the human body or segments of the body during a fall.
28. Force: A push or pull that causes an object to change its velocity (speed), shape or direction of motion. In a free fall, when the body impacts the ground, force is applied to the body.
29. Forensic pathologist: A pathologist with specialized training in the investigation of the cause, mechanism, and manner of death, injury, or disease process.
30. Glasgow coma scale (GCS): A practical means for the assessment of impaired consciousness in response to defined stimuli. The scale allows for a numerical assessment of consciousness ranging from 3 to 15 through the assessment of eye opening, verbal response, and motor response either spontaneously or following either verbal or applied painful stimulus. Although initially developed for adults, several pediatric adaptations of the scale have been developed and adopted for the assessment of children.
31. Head drop testing: Dropping a surrogate or PMHS head from an elevated height to ground or onto an object, often for the purposes of measuring acceleration of the head or determining injury outcomes such as skull fracture.
32. Herniation: Abnormal displacement of brain tissue as a result of raised intracranial pressure. Can be caused by pathologic processes that are diffuse (eg, cerebral edema) or focal (eg, hematoma).
33. Household fall: Falls that occur in the course of domestic activities. As applied to infants and toddlers, they usually involve ground-based falls that occur during crawling, standing or walking or falls from furniture such as beds, couches, or changing tables. Most household falls are low energy events with head to ground fall heights of 3 feet or less.
34. Hypoxic-ischemic injury: Brain injury that encompasses a reduction in the supply of oxygen (hypoxia) and/or diminished blood supply to the brain (hypoperfusion or ischemia).
35. Impact: Occurs when an object comes into forcible contact with another object. In a free fall, impact occurs when the body contacts the ground.
36. Inertial loading: Inertia is a body's resistance to change in speed and direction. When force or acceleration are applied to the body to change its speed or direction of motion, the mass of the body tends to resist the motion. This resistance to motion is the inertial loading.
37. Infarct: An area of tissue necrosis caused by ischemia attributable to arterial or venous occlusion.
38. Injury biomechanics: Field of study focusing on the biomechanical behavior of the human body under potentially injurious conditions (eg, acceleration, force) at both the macroscopic and microscopic levels. Often focused on determining whether forces or accelerations applied to the body will cause injury.
39. Injury mechanism: Macroscopic and microscopic analysis of how injuries occur. Describes how forces or accelerations translated into injuries.
40. Injury threshold or tolerance: The level force or acceleration beyond which there is a risk of a specific injury (eg, skull fracture, SDH).
41. Internal limiting membrane (ILM): One of the layers of the retina that forms the structural interface between the retina and the vitreous.
42. Intracranial hypertension: The elevation of intracranial pressure in response to a disturbance of regulatory intracranial pressure mechanisms. As described by the *Monro-Kellie hypothesis*, intracranial volume is fixed. Any increase in 1 of the 3 fixed intracranial contents (brain parenchyma, intravascular blood, and spinal fluid), or the addition of additional mass, must be balanced by reduction in one of these contents if intracranial pressure is to be maintained within a physiologically normal range. Reduction in intracranial spinal fluid spaces through displacement into the spinal subarachnoid space is the common means of physiologic compensation. Pressure in excess of 20 mm Hg is considered elevated and consistent with intracranial hypertension.
43. Intracranial pressure: Pressure inside the cranial cavity.
44. Intraretinal retinal hemorrhages: Presence of blood in and/or between the different retinal layers.
45. Kinematics: Movement or motion of the body or body region (eg, head) during an event.
46. Kinetic energy: Energy of a body in motion. On impact, this energy has the potential to be converted into an injury.
47. Linear or translational acceleration: Rate of change of linear (straight line) velocity (speed).
48. Macula: The retinal region between the major temporal vascular arcades.
49. Mechanical properties: Properties describe the reaction or response to an applied load. Properties include strength, ductility, elastic modulus, and fracture toughness.
50. Medical examiner/coroner: Responsible for conducting investigations into unnatural, sudden or unexpected deaths to determine the cause and manner of death. Medical examiners are usually appointed

officials and often forensic pathologists. Coroners are usually elected officials and not always physicians.

51. Multilayered retinal hemorrhages: Retinal hemorrhages that are present at multiple locations in relation to the retina (intraretinal, preretinal, and/or subretinal).
52. Neuropathologist: A pathologist with specialized training in the diagnosis of diseases of the central nervous system, peripheral nervous system, and muscle.
53. Optic atrophy: Optic nerve damage caused by the degeneration of retinal ganglion cell axons.
54. Ora serata: The peripheral termination of the retina at its junction with the ciliary body.
55. Papilledema: Optic disc swelling specifically attributable to raised intracranial pressure.
56. Peripapillary: The region around the optic nerve head or optic disc.
57. Posterior pole: Area of the ocular fundus that encompasses the optic nerve and the macula (posterior location should not be confused with "deep," which refers to the layers of the outer retina closer to the choroid).
58. Preretinal retinal hemorrhages: Blood in the space between the vitreous and the retina.
59. Retinal detachment: A separation between the sensory layers of the retina from the underlying retinal pigment epithelium and choroid.
60. Retinal fold: An area of the retina where it is buckled up, projecting up out of the normal plane of the retina.
61. Retinal hemorrhage (RH): Presence of blood under or between the layers of the retina or between the vitreous and the retina.
62. Retinal vascular arcades: The distal branches of the central retinal artery and vein. There are 4 major arcades or branches, each with arterioles and venules: superotemporal, inferotemporal, superonasal, and inferonasal.
63. Retinoschisis: Splitting of the retinal layers.
64. Rotational or angular acceleration: Rate of change of angular (circular or curved) velocity. Rotational and angular may be used interchangeably.
65. Shear: Force applied parallel to a surface.
66. Short distance fall: An event in which there is a change in the person's COM from a higher elevation to a lower elevation. Typically, this fall height is defined as <1 meter (~3 feet).
67. Skull fracture: A break in the normal integrity of the skull, which may be partial or full thickness, caused by presumed mechanical force.
68. Spinal dorsal root ganglion: Collection of sensory nerve cell bodies on the dorsal roots of spinal nerves located near intervertebral foramina.
69. Strain: Deformation or change in shape of an object (attributable to application of stress) normalized to its original shape.
70. Stress: Force per unit area applied to an object. Strength is the stress at which a material fails.
71. Subarachnoid hemorrhage: Macroscopic blood located between the brain surface and the arachnoid membrane. On CT and MR, the blood in this location will follow the contour of the sulci and cisterns. Acute SAH is hyperdense on CT and hyperintense on FLAIR MR imaging. Subacute SAH may be invisible on CT, although the presence of subtle sulcal "effacement" may occasionally be seen. Chronic SAH, or "hemosiderosis" may be seen on MR as hypointense linear areas of cortical "staining" on GRE- and SW-weighted imaging.
72. Subdural hematohygroma: A collection between the arachnoid membrane and dura, typically (although not always) crescentic in shape containing both blood/blood products and fluid-either cerebrospinal fluid or serum. On CT, it is mixed in attenuation depending on the amount and stage of the blood/blood products, but the hemorrhagic collection typically contains areas of low attenuation fluid and higher attenuation blood. On MRI, acute blood components will be T1-hyperintense and T2-hypointense with hypointense signal on GRE and SWI and be mixed with varying amounts of fluid, typically similar to cerebrospinal fluid in signal on T1- and T2-weighted images.
73. Subdural hygroma: A collection of fluid between the arachnoid membrane and dura, typically (although not always) crescentic in shape. The collection may contain proteinaceous or blood-tinged fluid that is simple and without membranes on imaging. It is low-attenuation on CT, although it is often slightly hyper attenuating to cerebrospinal fluid. On MRI, it may be isointense to slightly hyperintense to cerebrospinal fluid on both T1- and T2-weighted sequences, including on FLAIR (fluid attenuated inversion recovery). These may occur in the traumatic setting with a disruption of the arachnoid membrane with cerebrospinal fluid collecting in the subdural space. It may also occur in the setting of low intracranial pressure with fluid or cerebrospinal fluid seeping into the space or formation following disruption of the arachnoid-dural interface. Because they can result from a disruption of the arachnoid membrane, these collections may occur rapidly following trauma. However, they may also collect in a more delayed timeframe, such as in the setting of decreasing brain volume following a brain parenchymal insult.
74. Subgaleal hemorrhage: A collection of blood between the periosteum and aponeurosis of the scalp caused by ruptured emissary veins that connect the intracranial to scalp veins. It is not confined by the sutures and can extend to involve the entire scalp; thus, leading to a potential significant loss of blood and severe hypovolemia.
75. Subretinal RH: Presence of blood between the retina and the choroid.

76. Surrogate: A mechanical analogue representing a human that can be used in experiments simulating potentially injurious events such as falls, shaking or motor vehicle crashes to investigate injury risk. Surrogates can be equipped with instrumentation to measure acceleration, velocity, force, and other biomechanical outcomes.
77. Thrombosis: Formation of blood clot within blood vessels that can cause downstream tissue ischemia.
78. Vitreoretinal traction: A disorder of the vitreoretinal interface with persistently adherent vitreous exerting tractional pull on the retina.
79. Vitreous hemorrhage: Presence of blood in the vitreous cavity of the eye.

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