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REVIEW

Consensus statement on abusive head trauma in infants and young children

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Abstract

Abusive head trauma (AHT) is the leading cause of fatal head injuries in children younger than 2 years. A multidisciplinary team bases this diagnosis on history, physical examination, imaging and laboratory findings. Because the etiology of the injury is multifactorial (shaking, shaking and impact, impact, etc.) the current best and inclusive term is AHT. There is no controversy concerning the medical validity of the existence of AHT, with multiple components including subdural hematoma, intracranial and spinal changes, complex retinal hemorrhages, and rib and other fractures that are inconsistent with the provided mechanism of trauma. The workup must exclude medical diseases that can mimic AHT. However, the courtroom has become a forum for speculative theories that cannot be reconciled with generally accepted medical literature. There is no reliable medical evidence that the following processes are causative in the constellation of injuries of AHT: cerebral sinovenous thrombosis, hypoxic–

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ischemic injury, lumbar puncture or dysphagic choking/vomiting. There is no substantiation, at a time remote from birth, that an asymptomatic birth-related subdural hemorrhage can result in rebleeding and sudden collapse. Further, a diagnosis of AHT is a medical conclusion, not a legal determination of the intent of the perpetrator or a diagnosis of murder. We hope that this consensus document reduces confusion by recommending to judges and jurors the tools necessary to distinguish genuine evidence-based opinions of the relevant medical community from legal arguments or etiological speculations that are unwarranted by the clinical findings, medical evidence and evidence-based literature.

Keywords Abusive head trauma · Child abuse · Children · Computed tomography · Consensus statement · Infants · Magnetic resonance imaging · Mimics · Unsubstantiated theories

Executive summary

This consensus statement, supported by the Society for Pediatric Radiology (SPR), European Society of Paediatric Radiology (ESPR), American Society of Pediatric Neuroradiology (ASPNR), American Academy of Pediatrics (AAP), European Society of Neuroradiology (ESNR), American Professional Society on the Abuse of Children (APSAC), Swedish Paediatric Society, Norwegian Pediatric Association and Japanese Pediatric Society addresses significant misconceptions about the diagnosis of abusive head trauma (AHT) in infants and children. It builds on 15 major national and international professional medical societies' and organizations' consensus statements confirming the validity of the AHT diagnosis. The statement also exposes the fallacy of simplifying the diagnostic process to a "triad of findings" — a legal argument and not a medically valid term.

AHT is the leading cause of fatal head injuries in children younger than 2 years and is responsible for 53% of serious or fatal traumatic brain injury cases. The etiology of injury is multifactorial (shaking, shaking and impact, impact, etc.) so that the current best and most inclusive term is AHT, as advanced by the American Academy of Pediatrics.

No single injury is diagnostic of AHT. Rather the multiplicity of findings including evidence of intracranial and spinal involvement, complex retinal hemorrhages, rib and other fractures inconsistent with the provided mechanism of trauma, as well as the severity and age of the findings provide clues to the diagnosis. Subdural hematoma is the most frequently identified intracranial lesion but brain parenchymal injury is the most significant cause of morbidity and mortality in this setting. There is a high incidence of ligamentous cervical spine injury among victims of inflicted injury. However, it is important to emphasize that absence of ligamentous injury does not exclude AHT. In suspected cases of AHT, alternative diagnoses must be considered and when appropriate explored. The question to be answered is, "Is there a medical cause to explain all the findings or did this child suffer from inflicted injury?"

Despite courtroom arguments by defense lawyers and their retained physician witnesses, there is no reliable medical evidence that the following processes are precise mimics or causative in the constellation of injuries characteristic of AHT: cerebral sinovenous thrombosis, hypoxic–ischemic injury, lumbar puncture or dysphagic choking/vomiting. There is also no substantiation, at a time remote from birth, of the proposal that birth-related subdural hemorrhages can result in sudden collapse, coma or death caused by acute rebleeding into a previously asymptomatic chronic collection. In addition, subdural hematoma is uncommon in the setting of benign enlargement of the subarachnoid space (BESS), and when subdural hematoma is present, AHT should be considered.

The diagnosis of AHT is a medical diagnosis made by a multidisciplinary team of pediatricians and pediatric subspecialty physicians, social workers and other professionals based on consideration of all the facts and evidence. AHT is a scientifically non-controversial medical diagnosis broadly recognized and managed throughout the world. When diagnosed, it signifies that accidental and disease processes cannot plausibly explain the etiology of the infant/child's injuries. A diagnosis of AHT is a medical conclusion, not a legal determination of the intent of the perpetrator or, in the false hyperbole of the courtroom and sensationalistic media, "a diagnosis of murder."

The question in civil and criminal court cases involving allegations of unwitnessed abuse is the quality of the medical evidence and the integrity and expertise of the medical witness's testimony. Over the last decade, the courtroom has become a forum for medical opinions on the etiology of infant/child head injuries that runs the gamut from the well-founded evidence-based conclusions of multidisciplinary medical teams to speculative theories that cannot be reconciled with the medical evidence that is generally accepted in the relevant medical community. When pivotal medical testimony is contradictory, the message to the courts, the news media and the general public about infant injuries and safe caregiving is often confusing and inaccurate.

Professional medical societies use consensus statements to communicate general physician acceptance on a particular topic. These statements are vetted by the membership and designed to help physicians, news media and the public distinguish accurate medical information from non-evidence-based or "courtroom-only" causation theories. The formal

dissemination of this information via a consensus statement is intended to help courts improve the scientific accuracy of their decisions involving vital public health issues. Consensus statements reduce confusion by recommending to judges and jurors the tools necessary to distinguish genuine evidence-based opinions of the relevant medical community from legal arguments or etiological speculations that are unwarranted by the clinical findings, medical evidence and evidence-based literature.

Introduction

This consensus statement addresses significant misconceptions and misrepresentations about the diagnosis of abusive head trauma (AHT) in infants and young children. Major national and international professional medical societies and organizations have consistently confirmed the validity of the AHT diagnosis, its classic features and its severity [1–4].

Recently, denialism of child abuse has become a significant medical, legal and public health problem. In courtrooms in the United States defense attorneys and the medical witnesses who testify for them have been disseminating inaccurate and dangerous messages that are often repeated by the news media. Instead of arguing that there is reasonable doubt that physicians made a mistake in this case, they are arguing that child abuse is routinely overdiagnosed. The deliberate dissemination of this misinformation will deter caregivers from seeking medical services for infants and children — even in cases where there has been no abuse or neglect. The accompanying defense message — that shaking an infant cannot cause serious injury — will create the additional risk of encouraging dangerous or even life-threatening caregiver behavior. The majority of the expert witnesses practice evidence-based medicine; they base their testimony on clinical expertise and peer-reviewed evidence in the medical literature. However in some legal AHT cases, defense arguments (frequently supported by opinion testimony provided by a small group of medical witnesses) have offered a scientific-sounding critique of the AHT diagnosis by offering a laundry list of alternative causation hypotheses [5]. Efforts to create doubt about AHT include the deliberate mischaracterization and replacement of the complex and multifaceted diagnostic process by a near-mechanical determination based on the "triad" — the findings of subdural hemorrhage, retinal hemorrhage and encephalopathy [1]. This critique has been sensationalized in the mass media in an attempt to create the appearance of a "medical controversy" where there is none [6, 7]. The straw man "triad" argument ignores the fact that the AHT diagnosis typically is made only after careful consideration of all historical, clinical and laboratory findings as well as radiologic investigations by the collaboration of a multidisciplinary team.

This consensus statement reviews and synthesizes relevant scientific data. This statement is supported by the SPR Child Abuse Imaging Committee and endorsed by the boards of directors of the Society for Pediatric Radiology (SPR), European Society of Paediatric Radiology (ESPR), American Society of Pediatric Neuroradiology (ASPNR), American Academy of Pediatrics (AAP), European Society of Neuroradiology (ESNR), American Professional Society on the Abuse of Children (APSAC), Swedish Paediatric Society, Norwegian Pediatric Association and Japanese Pediatric Society. This statement is derived from an empirical assessment of the quality and accuracy of the medical literature and addresses the threshold question of when such literature is generally medically accepted in the pediatric health care community. This review of the medical literature also considers the court admissibility and the reliability of expert medical opinions based on such literature. The contributing board-certified physician authors each have one or more pediatric subspecialty board certifications from the American Board of Radiology or the American Board of Pediatrics or American Board of Neurosurgery (all member organizations of the American Board of Medical Specialties) or Royal College of Radiologists (UK) or equivalent boards in Greece and Italy. Additionally, all authors have 10–40 years of individual clinical experience diagnosing and treating children. The non-physician author is a law professor with nearly two decades of experience researching and writing on the appropriate use of child abuse evidence in court.

We address the following questions:

1. What are the causes of head injury in infants and young children? Why has AHT terminology evolved (shaken baby syndrome, battered child, abusive head trauma, etc.)?
2. What are the presenting features of AHT?
3. How is the diagnosis of AHT made?
4. What unsubstantiated alternative diagnoses are being proffered in the court?
5. What is the role of the multidisciplinary child protection team in the determination of AHT?
6. What are the issues that perpetuate misconceptions in the courtroom?
7. What can be done to provide the courts accurate information about the state of medical knowledge in AHT?

Etiology of head trauma in infants and young children and nomenclature of abusive head trauma (AHT)

When data are evaluated from head trauma in children younger than 2 years old, AHT is recognized as the leading cause of fatal head injuries and is responsible for 53% of the serious or fatal traumatic brain injury cases [8]. The peak incidence of

fatal AHT is at 1–2 months of age [9]. Terms used to describe this form of head injury have evolved as scientific data have advanced [10] (Table 1 with references [11–16]). This abusive form of head trauma occurs most frequently with other forms of abuse and less often in isolation [17].

In 1946, Caffey [11] described six children with chronic subdural hematoma and fractures of the long bones. Two of the six children had retinal hemorrhages. Multiple authors subsequently confirmed this association [18–21]. In 1962, Kempe et al. [12] coined the term "battered-child syndrome" to include "discrepancy between clinical findings and historical data. … subdural hematomas with or without fractures of the skull … even in the absence of fractures of the long bones." Caffey [13] in 1972 suggested the term "parent-infant traumatic stress syndrome."

In 1972 and again in 1974, Caffey [14, 15] postulated that the practice of "whiplash shaking and jerking of abused infants are common causes of the skeletal as well as the cerebrovascular lesion." He referred to the earlier work of Ommaya and Yarnell [22] and that of Guthkelch [23] to show the effects of rotational acceleration/deceleration of whiplash as the etiology of subdural hematomas. This mechanism explains why there are frequently no external marks of injury and also provides a reason for the retinal hemorrhages found in abused children [24–26]. In these papers, Caffey mentioned that whiplash/shaking may cause "protracted, repeated breath holding spells which may be similarly damaging to the brain" and was prescient to theories and data published decades later regarding hypoxic–ischemic injury associated with AHT [14, 15, 27–29]. Of note, whiplash/shaking has been repeatedly reaffirmed by confessions of perpetrators in which violent shaking was the most commonly reported mechanism of injury (68–100%) [30–32].

In 1987, Duhaime et al. [16] postulated that based on clinical, pathological data and biomechanical models, rotational acceleration/deceleration whiplash injuries do not provide enough force to account for the severe injuries of these children and that in severe cases blunt trauma must be involved. From this article, the term shaken baby/shaken impact emerged. There still remains discussion over whether shaking alone or shaking with blunt trauma is necessary for the injuries of these abused children, but confessional evidence is quite striking that shaking alone can cause AHT [30–32]. Dias [33] made the case that shaking alone can be a causative mechanism and significantly questioned the validity of the biomechanical model of Duhaime et al. [16]. In 2016, Narang et al. [3] documented that both AHT and shaken baby syndrome (SBS) are generally accepted diagnoses in the medical community. Currently, the medical literature and overwhelming clinical experience and judgment demonstrate that AHT can be caused by shaking alone, shaking with impact, or blunt impact alone.

In 2009, the Committee on Child Abuse and Neglect of the American Academy of Pediatrics issued a statement recommending the medical use of the term abusive head trauma (AHT) [10]. This policy statement did not negate the mechanism of shaking as a significant mechanism of injury but instead merely clarified that the term "shaking" alone was not inclusive of the full range of injury mechanisms. AHT is the most comprehensive term for the intracranial and spinal lesions in abused infants and children. In various forms, AHT has been in the modern medical literature for more than 60 years [34], "with over 1,000 peer-reviewed clinical medical articles written by over 1,000 medical authors from more than 25 different countries" [2]. Inflicted brain injuries are multifactorial in origin. It is the role of physicians to determine whether the injuries and the history for the injuries are suspicious for AHT and whether the child should be evaluated by a multidisciplinary child protection team with the goal of protecting the child. We note that the repeated defense counsel argument that the 2009 AAP statement constitutes a rejection of the medical evidence for shaking as a mechanism of infant injury is false and misleading legal rhetoric without any factual support in the statement or in any other statement from the AAP.

The presenting features of AHT

The clinical presenting features of AHT include severe head injury; death; less severe trauma with an unexplained mechanism; unsuspected finding on imaging or assessment for macrocephaly, developmental delay, seizures or other neurologic concerns; or discovery during the workup as a sibling of an abused child. The clinical findings might include neurologic signs and symptoms such as irritability/lethargy, altered mental status, seizures, respiratory compromise and apnea,

Table 1 Nomenclature for inflicted, non-accidental trauma in infants and children

1946	Caffey [11]	Multiple fractures in long bones of infants suffering from chronic subdural hematoma
1962	Kempe [12]	Battered child syndrome
1972	Caffey [13]	Parent-infant traumatic stress syndrome
1972,1974	Caffey [14, 15]	Whiplash shaking baby syndrome
1987	Duhaime [16]	Shaken-impact syndrome
2009	Christian [10]	Abusive head trauma

fractures, varying degrees of pattern marks or bruises in unusual locations, vomiting and poor feeding [35].

Children with fatal head injuries have altered mental status immediately after the injury [36]. However on rare occasions young victims of fatal head trauma present with Glasgow coma scale (GCS) of >12 for a short time before death, although GCS is a very rough guide of normalcy in the youngest age group [36, 37]. There is no evidence that children with fatal head trauma have prolonged asymptomatic lucid intervals prior to neurologic collapse. Some victims of AHT who sustain non-fatal injuries have nonspecific symptoms for several hours or more before developing either seizures or coma, while others remain relatively asymptomatic. Sixty-five percent of AHT cases present with neurologic abnormality while the remainder present with nonspecific symptoms [38]. This lack of specificity and other factors can lead to inaccurate diagnosis unless the evaluating physician understands the broad clinical spectrum of AHT [39].

Kemp et al. [40] described the predictive power of different neuroradiologic features to aid in the distinction of AHT from other causes. The clinical certainty for AHT is higher for children with more severe presentations or with multiple findings [17, 41]. Several characteristic findings have most frequently been identified in AHT including subdural hematoma (SDH), brain parenchymal injuries, retinal hemorrhages and rib fractures [2, 10, 41, 42]. In the review by Maguire et al. [41], any combination of three or more of the significant diagnostic features yielded a positive predictive value of 85%. Kelly et al. [43], in their review of referrals to a child protection team over a 20-year period, reported that in children younger than 2 years the characteristics of particular interest for AHT included no history of trauma (90%), no external evidence of impact to the head (90%), complex skull fractures with intracranial injury (79%), subdural hemorrhage (89%) and hypoxic–ischemic injury (97%).

How the diagnosis of AHT is made

The diagnosis of AHT is made like any other medical diagnosis, by considering all the information acquired via clinical history, physical examination, and laboratory and imaging data.

History

Inconsistency of the presenting history with the clinical findings is a concern for child maltreatment including AHT. Therefore, detailed history including a follow-up history once the acute illness has been addressed is vital to diagnostic accuracy [44, 45]. The two most common histories provided in cases of confirmed AHT are a low-height fall (of less than 4–6 ft) and no specific history of trauma [46]. Severe head injury or moderate to large non-focal SDH are rarely consistent with a history of a short fall of less than 4 ft [47].

There are significant limitations with published biomechanical studies evaluating falls including a lack of complete biofidelic integrity [48–51]. The data for injury thresholds in these studies were derived from adult primates undergoing single, non-impact accelerations [48–51]. The differences in intrinsic material properties of the infant skull, brain, cerebrospinal fluid (CSF) and blood vessels versus an adult human or primate were not considered, nor were the effects of repeated injury [33]. We need to develop a better understanding of these critical differences to develop better biomechanical studies approximating real-life situations in order to provide more accurate and reliable information.

Review of extensive literature demonstrates that severe intracranial injury from short falls is rare, and the predictions from any biomechanical study/model should not deviate too much from established extensive real-life data to be considered valid [25, 47, 52–86]. For example, Chadwick et al. [52] in their study of short falls demonstrated a mortality of 0.48 per million per year in children younger than 5 years. A review of 26 studies of accidental falls from various heights [25, 72–85] involving 1,902 children found 23 fatal injuries, of which only 0.26% (5/1,902) were from falls less than three stories [47]. In a review of 24 in-hospital newborn falls from less than 1 m height, 2 babies had non-depressed linear parietal fractures and 2 babies without skull fracture had infratentorial SDH, which was thought to be birth-trauma-related SDH and unrelated to the fall. All the babies had a normal or benign physical examination post fall and had normal findings on examination at discharge [86].

Review of the extensive literature informs us that mortality from short falls is extremely rare, and the majority of these are benign occurrences with no significant neurologic dysfunction. Linear skull fracture, associated epidural hemorrhage, focal contusion and rarely small focal SDH or subarachnoid hemorrhage might be seen on imaging, but significant intracranial hemorrhage, parenchymal contusion or diffuse hypoxic–ischemic injury is uncommon in contrast to findings seen in AHT. When significant neurologic dysfunction or mortality does occur with short falls, it is related to a large extra-axial hematoma or vascular dissection and secondary stroke [33, 52].

Physical examination and importance of ocular findings

Clinicians should perform a meticulous examination for external bruises and tenderness. Bruises to the head and face have been associated with AHT, and patterns of injury consistent with grabbing, choking and blunt trauma should be sought [69, 87]. The absence of external trauma to the head and neck is common, however, and sometimes

soft-tissue injuries including scalp hematomas are only evident at autopsy [88].

Ocular findings in AHT include orbital and lid ecchymosis, subconjunctival hemorrhage, anisocoria and disconjugate eye movements and retinal hemorrhages. Retinal hemorrhages are an important finding in AHT and when abuse is suspected, a prompt complete examination including full indirect opthalmoscopic examination through a dilated pupil should be obtained [87]. The incidence of retinal hemorrhage in AHT is approximately 85% [89, 90]. "Hemorrhages that are too numerous to count, multilayered and extending to the ora serrata are specific" [91]. A number of conditions have been associated with retinal hemorrhages, but this quoted description is highly suspicious for AHT [87] (Table 2; also see reference [92]). The retina is multilayered and traumatic retinoschisis occurs from vitreo-retinal traction sustained from repeated rapid acceleration/deceleration forces [93]. Deep splits of the retina and even focal retinal detachment can occur. Retinal folds are hypopigmented ridges, usually around the macula. In the absence of severe documented head trauma, retinal folds and retinoschisis are more specific for AHT [93]. These types of retinal lesions do not occur from birth trauma or papilledema (papilledema occurs in 10% of AHT) [87].

A prompt evaluation for retinal hemorrhages is important because they can fade rapidly. Generally, intraretinal hemorrhages clear rapidly, whereas preretinal hemorrhages might persist for many weeks [94]. The presence of too-numerous-to-count intraretinal hemorrhages might indicate that trauma occurred within a few days prior to examination, whereas the presence of preretinal with no or few intraretinal hemorrhages suggests days to weeks since trauma [94]. To identify these patterns accurately, the health care team should complete eye examinations as soon as possible after admission, preferably within 24–48 h [94].

Laboratory studies and imaging

Although the history and physical examination are paramount, appropriate use of laboratory studies and imaging is vital for accurate diagnosis and treatment. Recent papers discuss the evaluation of bleeding and bone diseases when there is a suspicion of abuse [95, 96]. Skeletal survey following current guidelines should be performed for all children with potential AHT, particularly those younger than 2 years [4]. In older children, long-bone fractures can be more reliably suspected in the presence of extremity tenderness, swelling or refusal to bear weight.

For an acutely ill child with neurologic impairment, an optimal imaging strategy involves initial unenhanced CT with 3-D reformatted images of the calvarium [97], followed by a full multi-sequence MRI of the brain and the cervical, thoracic and lumbar spine as soon as feasible. Children who are intact neurologically can be imaged with MR first [98–101]. Suspicion of AHT warrants comprehensive imaging, and the decision rule developed from a network of emergency departments regarding the use of imaging in low-risk blunt head trauma does not apply when there are concerns for AHT [102–104]. Intracranial bleeding is common in AHT and often presents as subdural hematoma. Magnetic resonance imaging of the brain and spine with a variety of sequences is useful in characterizing extra-axial bleeds and defining cerebral contusion, laceration and other parenchymal brain injuries.

A number of comparative studies in young children have elucidated the statistical differences in the types and severity of intracranial injuries from accidental versus abusive head trauma [25, 32, 43, 46, 72, 76, 77, 79, 83, 105–108]. These studies collectively demonstrate that: (1) skull fractures are equally as common following accidental trauma and AHT, but the complex skull fractures are more common following AHT; (2) epidural hematomas are more common following accidental trauma; (3) subdural hematomas are far more common following AHT; and (4) subarachnoid, intraparenchymal and intraventricular hemorrhage are equally common in both AHT and accidental trauma [25, 32, 43, 46, 72, 76, 77, 79, 83, 105–107].

Subdural hematoma is the most commonly observed intracranial lesion (in up to 90%) in young infants with AHT and is most commonly parafalcine in location [109, 110]. The inflicted injury (acceleration/deceleration +/- impact) can lead to tearing of convexity bridging veins at the junction of the bridging vein and superior sagittal sinus. Additionally, rupture of the arachnoid membrane allows cerebrospinal fluid to enter the subdural space, mixing with subdural blood (hematohygroma) [111, 112]. SDH might have a mixed attenuation at presentation (Table 3). Mixed-attenuation subdural hematomas are found with greater prevalence in AHT than in accidental head trauma [109]. In a review by Bradford et al. [110], of 105 confirmed AHT cases, intracranial SDH was identified in 92% of cases. On the initial diagnostic CT study, the SDH was of homogeneous hyperattenuation in 28% of cases, mixed attenuation in 58% of cases and homogeneous hypoattenuation in 14% of cases. In the cases with homogeneous hyperattenuation SDH on the initial CT, the first hypoattenuated component was seen between 0.3 days and 16 days after injury and the disappearance of the last hyperattenuated component was identified between 2 days and 40 days after injury. For these reasons, precise estimation of age of the mixed-attenuation SDH on the initial CT should be avoided.

While SDH is the most frequent intracranial lesion in AHT, parenchymal brain injury is the most significant cause of morbidity and mortality [113]. The injury might be direct mechanical injury such as contusion, direct axonal injury, laceration or parenchymal hematoma or indirect in nature, resulting from hypoxia and ischemia [113]. MRI is more sensitive than CT in delineation of parenchymal injures. Timing parenchymal and

Table 2 Processes associated with retinal bleeding (modified from Levin et al. [87])

Injury or condition	Discussion
Accidental trauma	Few in number except in very severe trauma, usually limited to posterior pole, predominantly intraretinal and pre-retinal, extremely rare (most studies <3% incidence) after short falls except if there has been an epidural hemorrhage or occipital impact
Birth	Between 19.2% and 37.3% incidence in vaginal birth, 6% incidence after C-section
Motor vehicle crash or severe crush injury	Easily determined by history
Cardiopulmonary resuscitation	Extremely rare, few in number, posterior pole
Extracorporeal membrane oxygenation (ECMO)	5 of 37 (13%) ECMO patients had retinal hemorrhage
Prematurity	Retinal hemorrhage occurs at the peripheral circumferential demarcation between the vascularized and avascular retina
Intracranial hypertension or papilledema	Small number of retinal hemorrhages on or around the optic disc
Coagulopathy/anemia	Uncommon, few in number, posterior pole severe anemia and usually thrombocytopenia required, often with cotton wool spots[a]
Meningitis	More often if coagulopathy or sepsis is present. Only severe retinal hemorrhage if purulent meningitis, otherwise few in number, posterior pole
Ruptures aneurysm/arteriovenous malformation	May have severe extensive retinal hemorrhage; vascular malformation easily recognized on neuroimaging
Hypoxia	Few in posterior pole
Menkes disease	Causes blue sclera
Galactosemia	Vitreous hemorrhages reported
Glutaric aciduria	Rarely occurs and is confined to posterior pole

[a] Rare in critically ill children with fatal accidental trauma, severe coagulopathy sepsis and myeloid leukemia [92]

extra-axial injury can be challenging, and because injuries evolve over time, repeat MRI is frequently indicated.

Venous injury is strongly associated with AHT. It is common at the junction of the bridging vein and superior sagittal sinus complex and is considered the source of SDH [109, 114]. Choudhary et al. [114] found that nearly 70% of children with AHT had some sort of venous abnormality. Findings consisted of cortical vein injury (44%) and mass effect on cortical draining veins or dural sinuses (69%). Specifically, disruption of bridging veins at their insertion into the superior sagittal sinus is a common source of SDH in AHT. Rupture of smaller intradural vessels resulting in subdural hemorrhage, likely caused by trauma, has also been proposed as an etiology [115, 116]. Trauma of both types, accidental and AHT, causes venous injury including intracranial venous thrombosis.

Young infants are at an increased risk of upper cervical spinal injury. Such injury is more likely to be soft-tissue or ligamentous in nature [117]. Imaging of bony cervical spine is infrequently positive (0.3–2.7%) in children investigated for suspected child abuse [118]. Non-bony spinal abnormalities have, however, been identified in up to 2/3 of victims of AHT, in both clinical and autopsy series [117, 119, 120]. Choudhary et al. [119] has shown on MRI that 78% of these infants have spinal findings, mostly ligamentous, and up to 75% have spinal subdural hematoma that tracks from the posterior fossa [117, 119, 121]. It is apparent that cervical, thoracic and lumbar MRI should be included in the diagnostic workup when there is evidence of intracranial injury. Prior to knowledge of the ligamentous injury, those who denied the existence

Table 3 Various appearances of subdural collection as seen on CT [109]

Appearance of subdural collection on CT	Possible time frame
Iso-attenuation	Hyperacute, acute
Hyperattenuation	Acute, early subacute
Mixed hyper- and hypoattenuation	Hyperacute, acute, subacute and chronic
Hypoattenuation	Chronic

of the shaken baby mechanism used "lack of spinal injury" to boost their unfounded theory [122–124]. However, it is important to emphasize that absence of ligamentous injury does not exclude AHT.

Unsubstantiated alternative theories proffered in the court [109]

The determination of whether certain theories are putative explanations for AHT must at least recognize the long and storied medical history of the many etiologies already investigated as reasonable explanations. With those historical investigations as a foundation, trauma has come to be uniformly recognized as the primary etiology of pediatric and adult SDHs [46]. Depending on the health history, clinical presentation and pertinent laboratory testing, there are diseases that are considered in the differential diagnosis of subdural hematoma and appropriate medical evaluation is required for all children.

Because medicine and science are dynamic, it is important to continually evaluate new hypotheses and, consequently, re-evaluate previously confirmed scientific understanding, thus avoiding a rush to judgment. In this section, we discuss selected current theories proffered as causative bases for AHT that reportedly "mimic" the injuries seen. However, the lack of scientific evidence for these assertions underscores the general consensus opinion of pediatricians and pediatric subspecialists against these theories as reasonable explanations for AHT [1, 125]. Most of these unsubstantiated alternative theories just focus on one aspect of the range of injuries seen in AHT while conveniently ignoring other injuries that cannot be explained away. For instance, those postulating cerebral sinovenous thrombosis (CSVT) theory as an alternative diagnosis of AHT focus on retinal hemorrhage and intracranial SDH while they ignore concomitant skeletal injuries, neck injury and visceral injury.

The theories have included association of common procedures such as lumbar puncture and common symptoms such as cough with uncommon clinical presentations such as CSVT or hypoxic–ischemic injuries (HII) in the newborn. The theory of lumbar puncture leading to intracranial hemorrhage precisely mimicking AHT speculates that loss of CSF pressure leads to intracranial hypotension and resultant SDH, but the only evidence provided has been couple of case reports in older children and adult literature [126–128]. Meanwhile lumbar puncture is a routine procedure performed safely across outpatient and inpatient settings without intracranial sequela. Complications from lumbar puncture are rare, and in fact a recent study in adults has documented that an underlying issue such as coagulopathy is typically present when complications arise [129].

Similarly, sustained cough, choking and dysphagic choking have been speculated to cause SDH and retinal hemorrhage mimicking AHT. The theory speculates that any cause of sustained raised intrathoracic pressure such as choking, paroxysmal coughing, gagging or vomiting can cause increased intracranial and retinal venous pressure by impeding thoracic venous return, leading to traumatic venous rupture with retinal hemorrhage and SDH [130, 131]. However a computer model developed to prove this hypothesis was lacking because it did not have a clearly defined threshold for failure of bridging vein in infants and because it was developed from data obtained mostly from adult and animal studies [109, 131]. An isolated case report of SDH in an infant with pertussis has also been cited to support this theory, but this particular case also had a confounding history of a fall a week before presentation, which might have been responsible for the SDH [109, 132]. Additionally, this theory has been negated by prospective studies in 83 infants suffering from pertussis demonstrating no evidence of retinal hemorrhages seen in AHT [133, 134]. Dysphagic choking-type of acute life-threatening event (ALTE) mimicking AHT was described in a Barnes et al. [135] case report and review [136]. The case report has been criticized for failing to disclose the source of information, for the author's role as defense expert witness, for omission and misrepresentations of certain facts and legal outcome, for lacking proper evidence base and for use of inaccurate information to support speculative explanations [137, 138]. ALTE, which has been replaced with the new terminology "brief resolved unexplained events," has been shown to have a low prevalence of retinal hemorrhage or SDH and cannot be considered to be the cause of SDH or retinal hemorrhage [139–141]. Similarly, retinal hemorrhage was not identified in a prospective study of vomiting infants with hypertrophic pyloric stenosis [142]. These prospective studies underline the fact that while the cough/dyphagic choking/vomiting theory is supported by no recent solid evidence base, there are strong prospective studies providing evidence that refutes these theories. In a retrospective study, children who presented with ALTE and subdural hemorrhages were found to be nearly 5 times more likely to have at least one suspicious extracranial injury, supporting the diagnosis of AHT and thereby negating the role of ALTE as a causative mechanism for findings concerning AHT [141].

Hypoxic–ischemic injury is another diagnosis proposed as an etiology of intracranial SDH and retinal hemorrhage, posited by some to precisely mimic AHT [143, 144]. This is based upon Geddes et al.'s [143] unified hypoxia theory, which derived its findings from the commonality between intracranial postmortem findings of pediatric patients who suffered from hypoxia and people with AHT. However, this theory has been refuted by a number of studies in which SDH was not identified on pathology or imaging or either in the clinical context of hypoxic injury [145–148]. Besides, traumatic AHT can be present without hypoxia, and AHT with hypoxic injury can coexist with other clinical findings such as visceral or skeletal injuries

and paraspinal soft-tissue injuries supporting the diagnosis of AHT [117]. Although hypoxia is frequently seen in traumatic injury of the brain, it is likely a comorbid association similar to other traumatic injuries of the brain and spine.

Cerebral sinovenous thrombosis has been proposed as a cause of intracranial injury in children. This unsupported theory proposes that raised intracranial venous pressure resulting from cerebral sinovenous thrombosis leads to bursting of bridging veins resulting in brain parenchymal injury, SDH and retinal hemorrhage similar to the pattern of injuries seen in AHT [114, 149–151]. CSVT is an uncommon disorder in childhood but fortunately has been well reported in the literature and thereby provides us with a robust evidence base to conclusively refute this theory [109, 152–157]. Although CSVT has been associated with parenchymal hemorrhagic infarct, resulting in significant morbidity and mortality, there is no evidence in the literature where primary CSVT thrombosis has been identified as the cause of acute SDH or a presentation with abrupt collapse with prolonged coma in a previously healthy child [114]. CSVT has been identified in situations where it is secondary in nature, consistent with the mechanism of pathology such as iron deficiency anemia or an inherited predisposition toward coagulation and trauma [109, 114]. We should not confuse thrombosis with subcortical hemorrhage; similarly, absence of veins on MR venogram doesn't equate to thrombosis, and demonstration of intraluminal thrombosis is equally important [114].

Subdural hematoma in the setting of benign enlargement of the subarachnoid space (BESS)

Benign enlargement of subarachnoid space (BESS) is common in the setting of macrocephaly in infancy. Although BESS was initially thought to predispose children to SDH with minimal trauma [158], the latest reviews reveal that less than 6% of infants with BESS develop hemorrhagic subdural collections (Table 4, references [158–164]). Most of the published series are lacking because of their variable methods of ascertainment, variable descriptions of the kind of subdural collections — cerebrospinal fluid, hemorrhagic fluid or a mixture of the two — and incomplete assessment for abuse in these cases [162].

Taking only those reports from Table 4, in which the prevalence of BESS has also been documented, a total of 712 cases of BESS were documented, with 38/712 (5.3%) reported to have subdural collection, including 12/712 (1.7%) that were reported to be hemorrhagic in nature. Accidental trauma or abuse was reported in 5/12 (41.7%) of the subdural collections that were hemorrhagic. Besides, up to 50% of children with BESS and SDH may display concomitant important injuries [165]. Overall subdural collections are uncommonly seen in the setting of BESS and assessment to exclude trauma, including AHT, should be performed in those with hemorrhagic and non-hemorrhagic subdural collections, especially in children younger than 2 years.

Birth trauma

The risk factor for intracranial hemorrhage in newborn infants is abnormal labor, as evidenced by a higher rate of traumatic brain injury in infants born by Cesarean section after an abnormal labor and those born with vacuum extraction and forceps as compared to infants born by spontaneous vaginal delivery or delivered by elective Cesarean section [166]. Birth trauma accounts for 1–2% of the mortality in newborns and any significant intracranial injury presents in the immediate postnatal period with significant clinical symptoms such as

Table 4 Subdural hematoma in the setting of benign enlargement of the subarachnoid space (BESS)

Authors	Number of patients with BESS	Number of subdural collections (% of total BESS cases)	Number (% of total BESS) with reported hemorrhagic subdural collections	Other details
Wilms et al. [158] 1993	19	8 (42.1%)	3 (15.8%)	One case of recent trauma with hemorrhagic subdural collection
McKeag et al. [159] 2013	177	4 (2.3%)	4 (2.3%)	1 rib fracture
Tucker et al. [160] 2016	311	18 (5.8%)	1 (0.3%)	Hemorrhagic subdural collection case reported for abuse
Greiner et al. [161] 2013	108	6 (5.6%)	2 (1.9%)	2 reported for abuse
Mcneely et al. [162] 2006	n/a	7 (n/a)	7 (n/a)	Abuse cases were excluded. 2 cases with accidental trauma
Haws et al. [163] 2017	84	2 (2.4%)	2 (2.4%)	n/a
Alper et al. [164] 1999	13	0 (0%)	0 (0%)	n/a

(n/a=not available)

irritability, poor feeding, emesis, apnea or disordered breathing, bradycardia, and seizures or disordered mentation [167–184].

Small birth-related subdural hematomas, most commonly along the tentorium, parietal occipital convexity, retrocerebellar posterior fossa or interhemispheric fissure, are observed in 8–46% of asymptomatic newborn infants [185–187]. This has led to the unsubstantiated theory that rebleeding, months later, in persistent birth-related asymptomatic SDH can present acutely with clinical features mimicking AHT [188]. Rooks et al. [186] in 2008 reported MRI findings within 72 h of birth and serial developmental evaluations of 101 asymptomatic neonates, 79 born by vaginal delivery and 22 by Cesarean delivery. SDH was present in 46 (46%) of the infants, most of whom resolved on follow-up MRI by 1 month and all resolved by 3 months. There were no significant differences in clinical outcomes in this cohort, as compared to the normal population, on serial developmental examinations [186]. Other authors have reported similar findings [187, 189].

To summarize, asymptomatic birth-related subdural hematomas are relatively frequent and resolve in the overwhelming majority of infants within the first 4–6 postnatal weeks, and do not appear to rebleed. If there is significant birth-related trauma, neonates are symptomatic in the immediate postnatal period. In particular, there is no merit to the unsubstantiated proposal that acute collapse, coma or death, occurring months after delivery, is caused by a parturitional SDH with secondary rebleeding.

Multidisciplinary assessment and long-term outcome

The medical diagnosis of AHT is made by pediatricians and pediatric subspecialists based on medical evaluation. In many children's hospitals, an interdisciplinary team of specialists that includes physicians, nurses, hospital social workers and others works together to evaluate cases. Hospital-based multidisciplinary teams have existed in many communities to provide comprehensive assessments and services for families for more than 60 years. The overriding goal of the work of these teams is to diagnose and to treat child abuse and neglect, assess for alternative diagnoses when appropriate, and assist in the efforts of the many agencies involved. The Children's Hospital Association (formerly the National Association of Children's Hospitals and Related Institutions) has released guidelines for team composition and function to aid in providing services [101, 190]. In addition, in some jurisdictions, multidisciplinary teams of hospital and community professionals review injuries, medical history, family and social risk to reach a more comprehensive assessment. These hospital–community partnerships are composed of physicians, nurses, social workers, clergy, psychologists, child protection services, law enforcement and other professionals with relevant experience. These multidisciplinary teams can review all of the data related to the case from different perspectives to gain a more complete understanding of the issues [8, 45, 191–194]. Whenever members of these teams present testimony in a legal setting, there has usually been much in-depth consideration of the diagnosis, and the probability of the correct diagnosis is high.

Abusive head trauma is the leading cause of physical abuse fatalities. In a review of child abuse fatalities, the authors identified shaking as a cause or contributor in 45% of the deaths, with beating, kicking and chronic battering accounting for the rest [191]. The authors identified crying as the trigger for 20% of deaths, followed by disobedience (6%), domestic arguments (5%), toilet training (4%) and feeding problems (3%) [191]. Infants are significantly more likely to be physically abused when a caretaker has an emotional disturbance and when there is violence between caretakers [195]. Unfortunately, when AHT is not prevented, the outcome can be devastating (Table 5) and the financial costs to society extremely high [196]. The estimated lifetime cost of 4,824 cases in 2010 was $13.5 billion [197].

The issues that perpetuate misconceptions in the courtroom

The most recent AAP policy statement on expert witness testimony has reemphasized the fact that expert witness neutrality and professional integrity can be pivotal factors in civil and criminal child abuse cases [198]. When expert testimony is scientifically reliable, objective and accurate, it provides useful information for the legal factfinder. Ethical and professional norms of responsible expert testimony require that physicians be objective and neutral assessors and conveyors of medical information, which means that they weigh the scientific merit of their opinions and conclusions and "present testimony that reflects the generally accepted standard within the specialty or area of practice, including those standards held by a significant minority" [198, 199]. Regrettably, not all medical experts' courtroom testimony falls within these ethical and professional boundaries. A few physicians, including those who do not treat or diagnose children as part of their medical

Table 5 Outcomes after abusive head trauma [196]

Death (20-25%)
Spastic hemiplegia or quadriplegia (15–64%)
Intractable epilepsy (11–32%)
Microcephaly with cortico-subcortical atrophy (61–100%)
Visual impairment (18–48%)
Language disorders (37–64%)
Agitation, aggression, tantrums, attention deficits, memory deficits, inhibition or initiation deficits (23–59%)

practice, frequently proffer various speculative causation theories (described in prior sections) camouflaged as alternative or mimic diagnoses in child maltreatment cases. These medical witnesses run afoul of professional norms and standards and, when their arguments are repeated by the news media, create a grave public health risk by promulgating dangerous misinformation regarding safe infant and child care.

What can be done to provide the court accurate information about the state of medical knowledge in AHT

The admissibility of expert evidence

In current day jurisprudence, admissibility of medical or scientific expert testimony requires some judicial assessment of the "reliability" of that testimony. In some jurisdictions, the standard for assessing admissible expert testimony is the *Frye* standard (or whether a particular concept or methodology is "generally accepted" in the medical/scientific community); in others, it is a *Daubert* standard (where judges consider additional criteria other than just "general acceptance," such as testability, peer review and publication and error rate). But in any legal jurisdiction, the medical precept that is considered "generally accepted" holds significant weight with courts. Unfortunately, courts are generally ill-equipped to measure the general consensus of physician thought on a particular concept, which makes them susceptible to more speculative theories unsupported by the medical evidence and medical literature. Thus, consensus statements present a unique opportunity to provide courts with a way to know general medical thought about a particular medical topic.

Professional society consensus statements

Physician acceptance Courts should assume that a consensus statement reflects general physician acceptance of a particular precept. Table 6 describes the rigorous process used to construct this type of statement. Thus, courts can be assured that practice promulgation of consensus statements has been vetted through a process that offers all members a way to contribute to the professional statements of that medical society.

Education of the courts Professional consensus statements can influence the judicial process through interdisciplinary education. Courts need experts to provide general information about infant anatomy, imaging technologies and the interpretation of medical images and laboratory results. To perform their decision-making role, judges and juries must assess the weight of the medical literature and differentiate between persuasive evidence-based medical research and less persuasive or unpersuasive published work (e.g., opinion articles, single case studies or discredited articles). In AHT, pediatricians and pediatric subspecialist physicians can be crucial to a court's accurate understanding of the relevant and reliable medical evidence.

Table 6 Process for developing a consensus statement

a. Topic under society's expertise needs clarification
b. Governing body of a society appoints individuals or a society's committee with expertise on a subject to study the issue and write a statement
c. The appointed group (the writing group) may utilize experts from other medical subspecialties and other professional societies as consultants and authors
d. A draft document is created and reviewed by participating individuals,
e. The document, after modification by this input is sent to the governing body of the specific society for comments
f. With these comments, the writing group revises the document and submits to the governing body for approval
g. The governing body circulates the document to the society membership for comment and if necessary further revisions
h. After this comprehensive creation and review process is completed, the document is published

Experts, through consensus statements, can also help courts identify the medical evidence that reflects scientific knowledge because it is supported by the evidence and has been generally accepted in the relevant field of pediatric medicine. By providing that medical information in a consensus statement, professional medical societies assist courts in identifying testimonial parameters for expert testimony and help judges and juries delineate evidence-based medical knowledge from fringe, speculative, or professionally irresponsible opinions.

Accurate medical evaluation versus non-evidence-based opinions

In cases involving an AHT diagnosis by one or more physicians, defense attorneys and their retained medical witnesses have increasingly challenged longstanding medical consensus that infant shaking can cause brain trauma. Typical defense arguments include: (1) a biased rush to judgment on the diagnosis of abuse; (2) exclusive diagnostic reliance on a "triad" of symptoms; (3) diagnosis by default; (4) an absence of neck injuries, proving AHT did not occur; (5) shifting scientific consensus; (6) an epidemic of copycat false convictions; and (7) the presumption that confession evidence consistent with infant injuries was coerced (in the two papers on confessions from France, in fact, the perpetrators were offered no reason to confess because leniency cannot be offered via French law) [31, 32]. These arguments are repeatedly raised in court despite the fact that they have never been empirically substantiated or are patently false.

There is a major flaw propagated in the few articles of those who deny SBS/AHT. It is the erroneous use of the terms "evidence-based medicine" and "systematic review" [200].

Because the suggestion that denialist views are supported by the evidence is likely to confuse judges and juries, we address two purported literature reviews: Donohoe in 2003 [201] and Lynoe et al. in 2017 [202]. Both articles are flawed by "(1) improper search and systemic review questions, (2) improper criteria for assessing bias and (3) inequitable application of quality of study assessment standards" [137, 203].

It is unprecedented that Donohoe's "systematic review" chose to exclude the voluminous literature before 1999 despite the fact that AHT was well described by multiple authors worldwide and the incidence of the disease was quite similar worldwide [204]. In the final analysis, Donohoe used only 23 articles to reach his erroneous conclusions [201]. As Greeley [204] showed, evidence supporting the AHT medical diagnosis "clearly fits the Bradford Hill criteria for causation" [205]. Similarly, despite the vast medical literature, Lynoe et al. [202] chose to use only 30 publications. Narang et al. [203] revealed the severe prejudicial bias of the authors of the Lynoe et al. [202] study. Additional publications have also refuted the Lynoe report [206–210]. This alternative agenda has no role in true science and can result in infant harm through shaking and neglect, through avoidance of emergency medical intervention.

In contrast, a 2016 study published in *The Journal of Pediatrics* found a high degree of medical consensus that shaking a young child can cause subdural hematoma, severe retinal hemorrhage, coma or death [3]. The study, which surveyed 628 physicians at 10 leading U.S. children's hospitals, found that 88% of physicians believe that SBS is a valid evidence-based diagnosis and 93% believe that the somewhat more comprehensive diagnosis of AHT is a valid evidence-based diagnosis [3].

AHT is a medical diagnosis, not a legal finding of murder

It is increasingly popular for defense lawyers to argue that AHT is a medical diagnosis of murder. This evocative courtroom hyperbole deliberately distorts the judicial process by mischaracterizing the physician expert's role. The medical expert in a child abuse case plays just one role — to help the judge or jury answer the *medical* question of whether an infant's injuries were most likely caused by abuse or they could be plausibly explained by a recognized disease or by one or more of the myriad hypothetical alternative causal explanations typically proffered by the defense. It is absurd to argue that a medical diagnosis proves murder. Medical expert testimony on the etiology of the injury cannot answer the two foundational *legal* questions of *actus reus* (Latin for guilty act) or *mens rea* (Latin for guilty mind). That is because, even after the factfinder decides that the medical evidence supports a finding that an infant's injuries were inflicted, non-medical evidence is required to determine who committed the act and to determine the level of intent (e.g., knowing, reckless or negligent). "The debate surrounding AHT is neither scientific nor medical but legal" [204]. The denialists have tried to create a medical controversy where there is none.

The "diagnosis of murder" argument is obviously wrong because it falsely implies that medical opinion testimony, by its nature, resolves all legal issues. To cite an analogous example that disproves the argument's premise, the toxicologist who testifies that the victim was poisoned does not diagnose murder because the court must still decide the *actus reus* (how was the poison ingested?) and the *mens rea* (was the victim's poisoning accidental, negligent, reckless or intentional?).

Defense attorneys and few medical witnesses who promulgate scientifically unsubstantiated theories about abuse "mimics" in an effort to manufacture a scientific-sounding controversy run afoul of professional norms and standards, can distort the view of the relevant medical community, and create a grave public health risk by promulgating dangerous misinformation regarding safe infant and child care (i.e. infant shaking is safe). As professional medical societies continue to issue evidence-based consensus statements to help courts, the news media and the public to address these issues, we anticipate that they will also play a greater role in curbing and sanctioning members whose testimony impedes the goals of scientific, adjudicative and public health accuracy.

Conclusions

1. Abusive head trauma (AHT) is the current most appropriate and inclusive diagnostic term for infants and young children who suffer from inflicted intracranial and associated spinal injury. This does not negate the mechanisms of shaking or shaking with impact as a significant mechanism of injury but merely indicates that the term "shaken baby" is not all-inclusive.
2. Lack of history, changing history or the incompatibility of history (i.e. short falls) with the severity of injury raise concerns for possible AHT.
3. Relatively few infants with AHT have isolated intracranial injury without retinal hemorrhages, fractures or other manifestations of child abuse. These children need a comprehensive evaluation to rule out other diseases. However, isolated intracranial injuries occur in a small percentage of children with AHT.
4. No single injury is diagnostic of AHT. A compilation of injuries most often including SDH, complex retinal hemorrhage and/or retinoschisis, rib, metaphyseal or other fractures and soft-tissue injury leads to the diagnosis.
5. Each infant suspected of suffering AHT must be further evaluated for other diseases that might present with similar findings. The question to be answered is, "Is there a medical cause to explain the findings or did this child suffer from inflicted injury?"

6. There is no reliable medical evidence that the following processes cause the constellation of injuries associated with AHT: cerebral sinovenous thrombosis, isolated hypoxic–ischemic injury, lumbar puncture and dysphagic choking/vomiting. There is no reliable evidence to support speculation that long-term consequences of birth-related subdural hematoma can result in later collapse, coma or death from acute rebleeding into a previously asymptomatic chronic subdural hematoma. In addition, subdural hematoma is uncommon in the setting of benign enlargement of the subarachnoid space, and when present, AHT should be considered in the differential diagnosis.
7. After medical diagnosis, in many hospitals a multidisciplinary team provides comprehensive assessment and services to the family, based on consideration of all the facts.
8. There is no controversy about the methodology used to diagnose AHT as a medical disease.
9. AHT is a medical diagnosis unrelated to the legal determination by a judge or jury of a charge of murder. The term "triad" is a legal convention that falsely mischaracterizes a complex AHT diagnosis process.
10. A professional medical society's consensus statement educates judicial factfinders, the news media and the public about "general acceptance," what is accurate medical information and what is non-evidence, speculative or professionally irresponsible etiological hypotheses.
11. The professional societies' consensus statement on AHT should help the court recognize unsubstantiated medical expert testimony.

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Compliance with ethical standards

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