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TITLE: 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 under 2 years. The diagnosis is established by a multidisciplinary team based on history, physical examination, imaging, and laboratory findings. Since 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 as well as rib and other fractures inconsistent with the provided mechanism of trauma. The work-up must exclude those 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 the following processes are causative in the constellation of injuries of AHT: cerebral sinovenous thrombosis, hypoxic 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. 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 will reduce confusion by recommending to judges and jurors, the tools necessary to distinguish genuine evidence-based opinions of the relevant medical community from legal argument or etiological speculation unwarranted by the clinical findings, medical evidence and evidenced-based literature.

Keywords: Abusive head trauma, Child abuse, Children, Computed tomography, Infants, Magnetic resonance imaging, Mimics, Unsubstantiated theories.

Executive summary:

This consensus statement supported by Society for Pediatric Radiology (SPR), European Society of Pediatric Radiology (ESPR), American Society of Pediatric Neuroradiology (ASPNR), American Academy of Pediatrics (AAP), European Society of Neuroradiology (ESNR) and American Professional Society on the Abuse of Children (APSAC) 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 organization's 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 under 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 or impact, etc.) so that the current best and most inclusive term is AHT, as advanced by the American Academy of Pediatrics (AAP).

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 doesn't 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 due to acute rebleeding into a previously asymptomatic chronic collection. In addition, subdural hematoma is uncommonly seen in the setting of benign enlargement of the subarachnoid spaces (BESS) and when 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 past 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 generally accepted in the relevant medical community. When pivotal medical testimony is contradictory, the message to the courts, the 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, 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 argument or etiological speculation unwarranted by the clinical findings, medical evidence and evidenced-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 around the country defense attorneys and the medical witnesses who testify for them have been disseminating inaccurate and dangerous messages often repeated by the 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 defense accompanying 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. Their testimony is based on clinical expertise and peer reviewed evidence in the medical literature. 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 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, supported by the SPR Child Abuse Committee and endorsed by the Board 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) reviews and synthesizes relevant scientific data. 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 has 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 2 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 allow misconceptions to perpetuate in the courtroom?
7. What can be done to provide the court accurate information about the state of medical knowledge in AHT?

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

When data from head trauma in children less than 2 years old is evaluated, 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 to 2 months of age [9].

Terms used to describe this form of head injury have evolved as scientific data has advanced [10] (Table 1 with references 11-16). This abusive form of head trauma occurs most frequently with the other forms of abuse and less often in isolation [17].

In 1946, Caffey [11] described 6 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" (PITS).

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 refers to the earlier work of Ommaya et al. [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 were 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 causative mechanism and significantly questions 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 and/or 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 over 60 years [34], "with over 1000 peer-reviewed clinical medical articles written by over 1000 medical authors from more than 25 different countries" [2]. Inflicted brain injuries are multifactorial in origin. It is the role of physicians to determine if the injuries and/or 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 are false and misleading legal rhetoric without any factual support in the statement or in any other statement from the AAP.

2. What are 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 neurological concerns, or discovery during the work-up as a sibling of an abused child. The clinical findings may include neurological signs and symptoms such as irritability/lethargy, altered mental status, seizures, respiratory compromise and apnea, 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 may 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 suffer from non-fatal injuries may have nonspecific symptoms for several hours or more before developing either seizures or coma, while others may remain relatively asymptomatic. 65% of AHT cases may present with neurological abnormality while the remainder may present with nonspecific symptoms [38]. This lack of specificity and other factors may lead to inaccurate diagnosis unless the evaluating physician understands the broad clinical spectrum of AHT [39].

Kemp et al. [40] described a range of clinical certainty in the diagnosis of AHT based on the identification of certain injuries and their severity. This certainty 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 3 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 less than 2 years old, 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%).

3. How is the diagnosis of AHT made?

The diagnosis of AHT is made like any other medical diagnosis by considering all the information acquired via clinical history, physical examination, 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 feet) 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 feet (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 was 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 or effects of repeated injury was not considered [33]. We need to develop a better understanding of these critical differences to develop better biomechanical studies, approximating real life situations, that will 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 children less than 5 years of age per year. 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% (5/1902) were due to falls from less than three stories [47]. In a review of 24 in-hospital newborn falls from less than one-meter height, 2 babies had non depressed linear parietal fractures and 2 babies without skull fracture had infratentorial SDH which was felt to represent 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, majority of these are benign occurrences with no significant neurological dysfunction. Linear skull fracture, associated epidural hemorrhage, focal contusion and rarely small focal SDH or SAH may 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 neurological 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

A meticulous examination for external bruises and tenderness should be performed. 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 it is possible that soft tissue injury including scalp hematomas may only be evident at autopsy [88].

Ocular findings in AHT include orbital and lid ecchymosis, subconjunctival hemorrhage, anisocoria and dysconjugate 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]. There are a number of conditions which have been associated with retinal hemorrhages, but the above quoted description is highly suspicious of AHT [87] (Table 2). The retina is multilayered

and traumatic retinoschisis occurs from vitreo-retinal traction suffered 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 as they can fade rapidly. Generally, intraretinal hemorrhages clear rapidly, whereas preretinal hemorrhages may persist for many weeks [94]. The presence of too numerous to count intraretinal hemorrhages may 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, eye examinations should be completed as soon as possible after admission, preferably within 24-48 hours [94].

Laboratory studies and imaging

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

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 multisequence MRI of the brain, cervical, thoracic and lumbar spine as soon as feasible. Children

who are intact neurologically can be first imaged using MR [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 due to accidental versus AHT [25, 32, 46, 72, 76-77, 79, 83, 105-110]. 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) EDH are more common following accidental trauma; 3) SDH are far more common following AHT; and 4) subarachnoid, intra-parenchymal and intraventricular hemorrhage are equally common in both AHT and accidental trauma [25, 32, 46, 72, 76-77, 79, 83, 105-109].

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 [110, 111]. The inflicted injury (acceleration/deceleration +/- impact) may 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) [112, 113]. SDH may have a mixed attenuation at presentation (Table 3). Mixed attenuation SDH are found with greater prevalence in AHT than in accidental head trauma [110]. In the review, by Bradford et al. [111], of 105 confirmed AHT cases, intracranial

SDH was identified in 92% of cases. On the initial diagnostic CT study, the SDH was of homogeneously hyper-attenuation in 28% of cases, mixed-attenuation in 58% of cases and homogeneously hypo-attenuation in 14% of cases. In the cases with homogeneously hyper-attenuation SDH on the initial CT, the first hypo-attenuation component was seen between 0.3 days and 16 days after injury and the disappearance of the last hyper-attenuation 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 [114]. The injury may be direct mechanical injury such as contusion, direct axonal injury, laceration or parenchymal hematoma or indirect in nature resulting from hypoxia and ischemia [114]. MRI is more sensitive than CT in delineation of parenchymal injures. Timing parenchymal and extraaxial injury can be challenging and because injuries evolve over time, repeat MRI is frequently indicated.

Venous injury is strongly associated with AHT. It is commonly seen at the junction of bridging vein and superior sagittal sinus complex and is considered to be the source of SDH [110, 115]. Choudhary et al. [115] 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 due to trauma, has also been proposed as an etiology [116, 117]. 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 [118]. Imaging of bony cervical spine is infrequently positive (0.3-2.7%) in children investigated for suspected child abuse [119]. Non bony spinal abnormalities have, however, been identified in up to 2/3 of victims of AHT, in both clinical and autopsy series [118, 120-121]. Choudhary et al. [120] has shown on MRI that 78% of these infants have spinal findings, mostly ligamentous and up to 75% have spinal subdural hematoma which tracks down from the posterior fossa [118, 120, 122]. It is apparent that cervical, thoracic and lumbar MRI should be added to the diagnostic work-up when there is evidence of intracranial injury. Prior to knowledge of the ligamentous injury, those who denied the existence of the shaken baby mechanism used "lack of spinal injury" to boost their unfounded theory [123-125]. However, it is important to emphasize that absence of ligamentous injury doesn't exclude AHT.

4. Unsubstantiated alternative theories proffered in the court [110]

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 of subdural hematoma and appropriate medical evaluation is required for all children.

As 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 judgement. In this section, we shall discuss selected current theories that have

been proffered as causative bases for AHT and 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, 126]. Most of these unsubstantiated alternative theories just focus on one aspect of the range of injuries seen in AHT whilst conveniently ignoring other injuries which 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 or visceral injury.

The theories have included association with common procedures such as lumbar puncture, common symptoms such as cough to uncommon clinical presentations such as cerebral sinovenous thrombosis (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 [127-129]. 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 [130].

Similarly, sustained cough, choking or 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, vomiting etc can potentially cause increased intracranial and retinal venous pressure, by impeding thoracic venous return, leading to traumatic venous rupture with retinal hemorrhage and SDH [131, 132].

However, a computer model developed to prove this hypothesis suffered from lack of a clearly defined threshold for failure of bridging vein in infants and from developing the model from data obtained mostly from adult and animal studies [110, 132]. An isolated case report of SDH present in an infant, with pertussis is also cited to support this theory, but this particular case also had confounding history of a fall a week before presentation, which may have been responsible for the SDH [110, 133]. Additionally, this theory has been negated by prospective studies in 83 infants suffering from pertussis demonstrating no evidence of retinal hemorrhages seen in AHT [134, 135]. Dysphagic choking type of ALTE mimicking AHT was described in a Barnes et al. [136] case report and also in a review [137]. The case report was criticized for failing to disclose the source of information, the author's role as defense expert witness, omission and misrepresentations of certain facts and legal outcome, lacking proper evidence base and use of inaccurate information to support speculative explanations [138, 139]. ALTE, which has been replaced with the new terminology brief resolved unexplained events (BRUE), 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 [140-142]. Similarly, retinal hemorrhage was not identified in a prospective study of vomiting infants with hypertrophic pyloric stenosis [143]. 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 which 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 thereby negating the role of ALTE as a causative mechanism for findings concerning AHT [144].

Hypoxic ischemic injury (HII) is another diagnosis proposed as an etiology of intracranial SDH and retinal hemorrhage, posited by some to precisely mimic AHT [145, 146]. This is based upon Geddes et al. [145] unified hypoxia theory which derived its findings from the commonality between intracranial postmortem findings of pediatric patients who suffered from hypoxia and patients with AHT. However, this theory has been refuted by a number of studies where SDH was not identified, either on pathology, on imaging or both, in the clinical context of hypoxic injury [147-150]. Besides, traumatic AHT can be present without hypoxia and AHT with hypoxic injury also may coexist with other clinical findings such as visceral, skeletal injuries and paraspinal soft tissue injuries supporting the diagnosis of AHT [118]. Though hypoxia can be seen frequently 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 (CSVT) 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 pattern of injuries seen in AHT [115, 151-153]. 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 [110, 154- 159]. Though it can be 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 presentation with abrupt collapse with prolonged coma in a previously healthy child [115]. CSVT has been identified in situations where it is secondary in nature, consistent with the mechanism of pathology such as iron deficiency anemia, inherited predisposition toward coagulation and

trauma [110, 115]. We should not confuse thrombosis with subcortical hemorrhage, similarly absence of veins on MRV (MR Venogram) doesn't equate to thrombosis and demonstration of intraluminal thrombosis is equally important [115].

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

Benign enlargement of subarachnoid spaces (BESS) is commonly seen in the setting of macrocephaly in infancy. Though initially thought to predispose to SDH with minimal trauma [160], the latest reviews (Table 4) reveals less than 6 % of such patients develop hemorrhagic subdural collections [160-166]. Most of the published series suffer from variable methods of ascertainment, variable description of the kind of subdural collections –cerebrospinal fluid, hemorrhagic fluid, or a mixture of the two – and without complete assessment for abuse in these cases [164].

Taking only those reports from Table 6, 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%) which were reported to be hemorrhagic in nature. Accidental trauma or abuse was reported in 5/12 (41.7%) of subdural collections which were hemorrhagic. Besides, up to 50% of children with BESS and SDH may display concomitant important injuries [167].

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 hemorhagic subdural collections, especially under 2 years of age.

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

583 abnormal labor and those born with vacuum extraction and forceps as compared to infants born
584 by spontaneous vaginal delivery or delivered by elective Cesarean section [168]. Birth trauma
585 accounts for 1-2% of mortality in newborn infants and any significant intracranial injury will
586 present in the immediate postnatal period with significant clinical symptoms such as irritability,
587 poor feeding, emesis, apnea or disordered breathing, bradycardia, seizures or disordered
588 mentation [169-186].
589 Small birth related SDH, most commonly along the tentorium, parietal occipital convexity,
590 retrocerebellar posterior fossa or interhemispheric fissure may be observed in 8-46% of
591 asymptomatic newborn infants [187-189]. This has led to the unsubstantiated theory that
592 rebleeding, months later, in persistent birth related asymptomatic SDH can present acutely with
593 clinical features mimicking AHT [190]. Rooks et al. [188] in 2008 reported MRI findings within
594 72 hours of birth and serial developmental evaluations of 101 asymptomatic neonates, 79 born
595 by vaginal delivery and 22 by Cesarean delivery. SDH was present in 46 (46%) of the infants
596 most of whom resolved on follow up MRI by 1 month and all resolved by 3 months. There were
597 no significant differences in clinical outcomes in this cohort, as compared to the normal
598 population, on serial developmental examinations [188]. Similar findings have been reported by
599 other authors [189, 191].
600 Therefore, to summarize, asymptomatic birth related SDH are relatively frequent and resolve in
601 the overwhelming majority of infants within the first 4-6 post-natal weeks, and do not appear to
602 rebleed. If there is significant birth related trauma, neonates will be symptomatic in the
603 immediate postnatal period. In particular, there is no merit to the unsubstantiated proposal that
604 acute collapse, coma or death, occurring months after delivery, are due to a parturitional SDH
605 with secondary rebleeding.

606

607 **5. Multidisciplinary assessment and long-term outcome**

608 The medical diagnosis of AHT is made by pediatricians and pediatric subspecialists based on
609 medical evaluation. In many children's hospitals, cases are evaluated by an interdisciplinary
610 team of specialists that include physicians, nurses, hospital social workers and others. Hospital-
611 based multidisciplinary teams have been used in many communities to provide comprehensive
612 assessments and services for families for over sixty years. The overriding goal of the work of
613 these teams is to diagnose and to treat child abuse and neglect, assess for alternative diagnoses
614 when appropriate and to assist in the efforts of the many agencies involved. The Children's
615 Hospital Association (formerly the National Association of Children's Hospitals and Related
616 Institutions) has released guidelines for team composition and function to aid providing services
617 [101, 192]. In addition, in some jurisdictions, multidisciplinary teams of hospital and
618 community professionals review injuries, medical history, family and social risk to reach a more
619 comprehensive assessment. These hospital-community partnerships are composed in part of
620 physicians, nurses, social workers, clergy, psychologists, child protection services, law
621 enforcement and other professionals with relevant experience. These multidisciplinary teams can
622 review all of the data related to the case from different perspectives to gain a more complete
623 understanding of the issues [8, 45, 193-196]. When testimony is presented in a legal setting,
624 there has usually been much in-depth consideration of the diagnosis and the probability of the
625 correct diagnosis is high.

626 Abusive head trauma is the leading cause of physical abuse fatalities. In a review of child
627 abuse fatalities, shaking was identified as a cause or contributor for 45% of the deaths, with
628 beating, kicking and chronic battering accounting for the rest [193]. Crying was identified as the

trigger for 20% of deaths, followed by disobedience (6%), domestic arguments (5%), toilet training (4%) and feeding problems (3%) [193]. Infants are significantly more likely to be physically abused when there is caretaker emotional disturbance and violence between caretakers [197]. Unfortunately, when AHT is not prevented, the outcome can be devastating and the financial costs to society extremely high [198] (Table 5). The lifetime cost of estimated 4824 cases in 2010 was $13.5 billion [199].

6. What are the issues that allow misconceptions to perpetuate in the courtroom?

The Medical Expert Witness

The most recent AAP policy statement on expert witness testimony has reemphasized the fact that expert witness neutrality and professional integrity can be a pivotal factor in civil and criminal child abuse cases [200]. 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 physicians to be objective and neutral assessors and conveyors of medical information, which means that they should weigh the scientific merit of their own 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" [200, 201]. Regrettably, not all medical expert's 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 practice, frequently proffer various speculative causation theories (see above) 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 media, create a grave public health risk by promulgating dangerous misinformation regarding safe infant and childcare.

7. 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

674 statement. Thus, courts can be assured that practice promulgation of consensus statements, have
675 been vetted through a process that offers all members a way to impact the professional
676 statements of that medical society.

677 **Education of the courts**

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

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

692 ***Accurate medical evaluation versus non evidence based opinions***

693 In cases involving an AHT diagnosis by one or more physicians, defense attorneys and
694 their retained medical witnesses have increasingly challenged longstanding medical consensus
695 that infant shaking can cause brain trauma. Typical defense arguments include: (1) a biased rush
696 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 proves 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 (the two papers on confessions from France, in fact offer the perpetrator no reason to confess as 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 "[202]. 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 [203] and Lynoe et al in 2017 [204]. 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." [138, 205].

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 world-wide and the incidence of the disease was quite similar world-wide [206]. In the final analysis, Donohoe uses only 23 articles to reach his erroneous conclusions. As Greeley showed, evidence supporting the AHT medical diagnosis "clearly fits the Bradford Hill criteria for causation" [207]. Similarly, despite the vast medical literature, Lynoe et al. [204] chose to use only 30 publications. Narang et al. [205] reveal the severe prejudicial bias of the authors of the Lynoe et al. [204] study. Additional publications have also refuted this report [208-212]. 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 finds 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 whether they can 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" [206]. 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

743 that disproves the argument's premise, the toxicologist who testifies that the victim was poisoned
744 does not diagnose murder because the court must still decide the *actus reus* (how was the poison
745 ingested?) and the *mens rea* (was the victim's poisoning accidental, negligent, reckless or
746 intentional?).

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

756 **Conclusions**

757 1. Abusive head trauma (AHT) is the current, most appropriate and inclusive diagnostic
758 term for infant and young children who suffer from inflicted intracranial and associated spinal
759 injury. This does not negate the mechanisms of shaking or shaking with impact as a significant
760 mechanism of injury but merely indicates that the term "shaken baby" is not all inclusive.

761 2. Lack of history, changing history or the incompatibility of history (i.e. short falls) with
762 the severity of injury raise concerns for possible AHT

763 3. Relatively few infants with AHT have isolated intracranial injury without retinal
764 hemorrhages, fractures or other manifestations of child abuse. These children need a

765 comprehensive evaluation to rule out other diseases. However, isolated intracranial injuries occur
766 in a small percentage of children with AHT.
767 4. There is no single injury that is diagnostic of AHT. It is a compilation of injuries most
768 often including SDH, complex retinal hemorrhage and/or retinoschisis, rib, metaphyseal or other
769 fractures and soft tissue injury which leads to the diagnosis.
770 5. Each infant must be further evaluated for other diseases, that may present with similar
771 findings. The question to be answered is "Is there a medical cause to explain the findings and did
772 this child suffer from inflicted injury?"
773 6. There is no reliable medical evidence that the following processes cause the constellation
774 of injuries associated with AHT: Cerebral sinovenous thrombosis, isolated hypoxic ischemic
775 injury, lumbar puncture and dysphagic choking/vomiting. There is no reliable evidence to
776 support speculation that long term consequences of birth related subdural can result in later
777 collapse, coma or death due to acute rebleeding into a previously asymptomatic chronic
778 subdural. In addition, subdural hematoma is uncommonly seen in the setting of benign
779 enlargement of the subarachnoid space and when present, AHT should be considered in the
780 differential diagnosis.
781 7. After medical diagnosis, in many hospitals, a multidisciplinary team provides
782 comprehensive assessment and services to the family, based on consideration of all the facts.
783 8. There is no controversy about the methodology used to diagnose AHT as a medical
784 disease.
785 9. AHT is a medical diagnosis unrelated to the legal determination by a judge or jury of a
786 charge of murder. The term "triad" is a legal convention that falsely mischaracterizes a complex
787 AHT diagnosis process.

10. A professional medical society's consensus statement educates judicial factfinders, the media and the public about "general acceptance", what is accurate medical information and what are non evidence, speculative, or professionally irresponsible etiological hypotheses.

11. The professional society's consensus statement on AHT should help the court recognize unsubstantiated medical expert testimony.

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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

1314 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 (CPR)	Extremely rare, few in number, posterior pole
Extracorporeal membrane oxygenation (ECMO	5 out of 37 (13 %) of ECMO patients had retinal hemorrhage.
Prematurity	Retinal hemorrhage occur at the peripheral circumferential demarcation between the vascularized and avascular retina
Intracranial hypertension or Papilledema	Small number of retinal hemorrhage 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 ++
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

1315 ++ Rare in critically ill children with fatal accidental trauma, severe coagulopathy sepsis and
1316 myeloid leukemia [92]
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Table 3. Various appearances of subdural collection as seen on CT [110]

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

1325 Table 4. Subdural hematoma in the setting of benign enlargement of the subarachnoid space

1326 (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. [160] 1993	19	8/ (42.1%)	3/ (15.8%)	One case of recent trauma with hemorrhagic subdural collection
Mckeag et al. [161] 2013	177	4/ (2.3%)	4/ (2.3%)	1 rib fracture
Tucker et al. [162] 2016	311	18/ (5.8%)	1/ (0.3%)	Hemorrhagic subdural collection case reported for abuse
Greiner et al. [163] 2013	108	6/ (5.6%)	2/ (1.9%)	2 reported for abuse
Mcneely et al. [164] 2006	n/a	7/ (n/a)	7/ (n/a)	Abuse cases were excluded. 2 cases with accidental trauma
Haws et al. [165] 2017	84	2/ (2.4%)	2/ (2.4%)	n/a
Alper et al. [166] 1999	13	0/ (0%)	0/ (0%)	n/a

1327 (n/a= not available)

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Table 5. Outcomes after abusive head trauma [198]

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%)

Table 6. Process for Development of a Consensus Statement

a. Topic under society's expertise needs clarification
b. Governing body of society appoints individuals or society's committee with expertise on subject to study 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 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 revision
h. After this comprehensive creation and review process is completed, the document is published