This is a repository copy of *Consensus statement on abusive head trauma in infants and young children*.

1   TITLE: Consensus statement on Abusive head trauma in infants and young children.

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128   **Abstract:**

129   Abusive Head Trauma (<u>AHT</u>) is the leading cause of fatal head injuries in children under 2

130   years. The diagnosis is established by a multidisciplinary team based on history, physical

131   examination, imaging, and laboratory findings. Since the etiology of the injury is multifactorial

132   (shaking, shaking and impact, impact etc) the current best and inclusive term is AHT.   There is

133   no controversy concerning the medical validity of the existence of AHT with multiple

134   components including subdural hematoma, intracranial and spinal changes, complex retinal

135   hemorrhages as well as rib and other fractures inconsistent with the provided mechanism of

136   trauma. The work-up must exclude those medical diseases that can mimic AHT. However, the

137   courtroom has become a forum for speculative theories that cannot be reconciled with "generally

138   accepted" medical literature. There is no reliable medical evidence the following processes are

139   causative in the constellation of injuries of AHT: cerebral sinovenous thrombosis, hypoxic

140   ischemic injury, lumbar puncture, or dysphagic choking/vomiting. There is no substantiation, at

141   a time remote from birth, that an asymptomatic birth related subdural hemorrhage can result in

142   rebleeding and sudden collapse. A diagnosis of AHT is a medical conclusion, not a legal

143   determination of the intent of the perpetrator or "a diagnosis of murder".   We hope that this

144   consensus document will reduce confusion by recommending to judges and jurors, the tools

145   necessary to distinguish genuine evidence-based opinions of the relevant medical community

146   from legal argument or etiological speculation unwarranted by the clinical findings, medical

147   evidence and evidenced-based literature.

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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

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175 considered and when appropriate explored. The question to be answered is "Is there a medical

176 cause to explain all the findings or did this child suffer from inflicted injury?"

177 Despite courtroom arguments by defense lawyers and their retained physician witnesses,

178 there is no reliable medical evidence that the following processes are precise mimics or causative

179 in the constellation of injuries characteristic of AHT: cerebral sinovenous thrombosis, hypoxic

180 ischemic injury, lumbar puncture or dysphagic choking/vomiting.   There is also no

181 substantiation, at a time remote from birth, of the proposal that birth-related subdural

182 hemorrhages can result in sudden collapse, coma or death due to acute rebleeding into a

183 previously asymptomatic chronic collection. In addition, subdural hematoma is uncommonly

184 seen in the setting of benign enlargement of the subarachnoid spaces (BESS) and when present,

185 AHT should be considered.

186 The diagnosis of AHT is a medical diagnosis made by a multidisciplinary team of

187 pediatricians and pediatric subspecialty physicians, social workers and other professionals based

188 on consideration of all the facts and evidence. AHT is a scientifically non-controversial medical

189 diagnosis broadly recognized and managed throughout the world. When diagnosed, it signifies

190 that accidental and disease processes cannot plausibly explain the etiology of the infant/child's

191 injuries. A diagnosis of AHT is a medical conclusion, not a legal determination of the intent of

192 the perpetrator or, in the false hyperbole of the courtroom and sensationalistic media, "a

193 diagnosis of murder."

194 The question in civil and criminal court cases involving allegations of unwitnessed

195 abuse is the quality of the medical evidence and the integrity and expertise of the medical

196 witness's testimony. Over the past decade, the courtroom has become a forum for medical

197 opinions on the etiology of infant/child head injuries that runs the gamut from the well-founded

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198 evidence-based conclusions of multidisciplinary medical teams to speculative theories that

199 cannot be reconciled with the medical evidence generally accepted in the relevant medical

200 community. When pivotal medical testimony is contradictory, the message to the courts, the

201 media and the general public about infant injuries and safe caregiving is often confusing and

202 inaccurate.

203   Professional medical societies use consensus statements to communicate general

204 physician acceptance on a particular topic. These statements are vetted by the membership and

205 designed to help physicians, media and the public distinguish accurate medical information from

206 non evidence based or "courtroom-only" causation theories. The formal dissemination of this

207 information via a consensus statement is intended to help courts improve the scientific accuracy

208 of their decisions involving vital public health issues. Consensus statements reduce confusion by

209 recommending to judges and jurors, the tools necessary to distinguish genuine evidence-based

210 opinions of the relevant medical community from legal argument or etiological speculation

211 unwarranted by the clinical findings, medical evidence and evidenced-based literature.

212

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

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236 is none [6, 7]. The straw man "triad" argument ignores the fact the AHT diagnosis typically is

237 made only after careful consideration of all historical, clinical and laboratory findings as well as

238 radiologic investigations by the collaboration of a multidisciplinary team.

239 This consensus statement, supported by the SPR Child Abuse Committee and endorsed by the

240 Board of Directors of the Society for Pediatric Radiology (SPR), European Society of Paediatric

241 Radiology (ESPR), American Society of Pediatric Neuroradiology (ASPNR), American Academy

242 of Pediatrics (AAP), European Society of Neuroradiology (ESNR) & American Professional

243 Society on the Abuse of Children (APSAC) reviews and synthesizes relevant scientific data. This

244 statement is derived from an empirical assessment of the quality and accuracy of the medical

245 literature and addresses the threshold question of when such literature is generally medically

246 accepted in the pediatric health care community. This review of the medical literature also

247 considers the court admissibility and the reliability of expert medical opinions based on such

248 literature. The contributing board-certified physician authors each has one or more pediatric

249 subspecialty board certifications from the American Board of Radiology or the American Board

250 of Pediatrics or American Board of Neurosurgery (all member organizations of the American

251 Board of Medical Specialties) or Royal College of Radiologists (UK) or equivalent boards in

252 Greece and Italy. Additionally, all authors have 10–40 years of individual clinical experience

253 diagnosing and treating children. The non-physician author is a law professor with nearly 2

254 decades of experience researching and writing on the appropriate use of child abuse evidence in

255 court.

256 We address the following questions:

257 1. What are the causes of head injury in infants and young children? Why has AHT terminology
258   evolved (shaken baby syndrome, battered child, abusive head trauma, etc.)?
259 2. What are the presenting features of AHT?

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260   3. How is the diagnosis of AHT made?

261   4. What unsubstantiated alternative diagnoses are being proffered in the court?

262   5. What is the role of the multidisciplinary child protection team in the determination of AHT?

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

264   7. What can be done to provide the court accurate information about the state of medical

265   knowledge in AHT?

266   **1. Etiology of head trauma in infants and young children and nomenclature of abusive**

267   **head trauma (AHT)**

268   When data from head trauma in children less than 2 years old is evaluated, AHT is recognized as

269   the leading cause of fatal head injuries and is responsible for 53% of the serious or fatal

270   traumatic brain injury cases [8]. The peak incidence of fatal AHT is at 1 to 2 months of age [9].

271   Terms used to describe this form of head injury have evolved as scientific data has

272   advanced [10] (Table 1 with references 11-16). This abusive form of head trauma occurs most

273   frequently with the other forms of abuse and less often in isolation [17].

274   In 1946, Caffey [11] described 6 children with chronic subdural hematoma and fractures of

275   the long bones. Two of the six children had retinal hemorrhages. Multiple authors subsequently

276   confirmed this association [18-21]. In 1962, Kempe et al. [12] coined the term "battered-child

277   syndrome" to include "discrepancy between clinical findings and historical data….subdural

278   hematomas with or without fractures of the skull…even in the absence of fractures of the long

279   bones." Caffey [13] in 1972 suggested the term "parent-infant traumatic stress syndrome"

280   (PITS).

281   In 1972 and again in 1974, Caffey [14, 15] postulated that the practice of "whiplash shaking and

282   jerking of abused infants are common causes of the skeletal as well as the cerebrovascular

283   lesion". He refers to the earlier work of Ommaya et al. [22] and that of Guthkelch [23] to show

284   the effects of rotational acceleration/deceleration of whiplash as the etiology of subdural

285 hematomas. This mechanism explains why there were frequently no external marks of injury

286 and also provides a reason for the retinal hemorrhages found in abused children [24-26]. In these

287 papers, Caffey mentioned that whiplash/shaking may cause "protracted, repeated breath holding

288 spells which may be similarly damaging to the brain" and was prescient to theories and data

289 published decades later regarding hypoxic ischemic injury associated with AHT [14-15, 27-29].

290 Of note, whiplash/shaking has been repeatedly reaffirmed by confessions of perpetrators in

291 which violent shaking was the most commonly reported mechanism of injury (68% -100%) [30-

292 32].

293   In 1987, Duhaime et al. [16] postulated that based on clinical, pathological data and

294 biomechanical models, rotational acceleration/deceleration whiplash injuries do not provide

295 enough force to account for the severe injuries of these children and that in severe cases blunt

296 trauma must be involved. From this article, the term shaken baby/shaken impact emerged.

297 There still remains discussion over whether shaking alone or shaking with blunt trauma is

298 necessary for the injuries of these abused children but confessional evidence is quite striking that

299 shaking alone can cause AHT [30-32]. Dias [33] made the case that shaking alone can be

300 causative mechanism and significantly questions the validity of the biomechanical model of

301 Duhaime et al. [16]. In 2016, Narang et al. [3] documented that both AHT and Shaken baby

302 syndrome (SBS) are generally accepted diagnoses in the medical community. Currently, the

303 medical literature and overwhelming clinical experience and judgment demonstrate that AHT

304 can be caused by shaking and/or shaking with impact or blunt impact alone.

305   In 2009, the Committee on Child Abuse and Neglect of the American Academy of

306 Pediatrics issued a statement recommending the medical use of the term abusive head trauma

307 (AHT) [10]. This policy statement did not negate the mechanism of shaking as a significant

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308   mechanism of injury but instead merely clarified that the term "shaking" alone was not inclusive

309   of the full range of injury mechanisms. AHT is the most comprehensive term for the intracranial

310   and spinal lesions in abused infants and children. In various forms, AHT has been in the modern

311   medical literature for over 60 years [34], "with over 1000 peer-reviewed clinical medical articles

312   written by over 1000 medical authors from more than 25 different countries" [2]. Inflicted brain

313   injuries are multifactorial in origin. It is the role of physicians to determine if the injuries and/or

314   the history for the injuries are suspicious for AHT and whether the child should be evaluated by

315   a multidisciplinary child protection team with the goal of protecting the child. We note that the

316   repeated defense counsel argument that the 2009 AAP statement constitutes a rejection of the

317   medical evidence for shaking as a mechanism of infant injury are false and misleading legal

318   rhetoric without any factual support in the statement or in any other statement from the AAP.

319

320   **2. What are the presenting features of AHT?**

321 The clinical presenting features of AHT include severe head injury, death, less severe trauma

322   with an unexplained mechanism, unsuspected finding on imaging or assessment for

323   macrocephaly, developmental delay, seizures or other neurological concerns, or discovery during

324   the work-up as a sibling of an abused child. The clinical findings may include neurological signs

325   and symptoms such as irritability/lethargy, altered mental status, seizures, respiratory

326   compromise and apnea, fractures, varying degrees of pattern marks or bruises in unusual

327   locations, vomiting and poor feeding [35].

328   Children with fatal head injuries have altered mental status immediately after the injury

329   (36). However, on rare occasions, young victims of fatal head trauma may present with Glasgow

330   Coma Scale (GCS) of >12 for a short time before death, although GCS is a very rough guide of

331   normalcy in the youngest age group [36, 37]. There is no evidence that children with fatal head

332   trauma have prolonged asymptomatic lucid intervals prior to neurologic collapse. Some victims

333   of AHT who suffer from non-fatal injuries may have nonspecific symptoms for several hours or

334   more before developing either seizures or coma, while others may remain relatively

335   asymptomatic. 65% of AHT cases may present with neurological abnormality while the

336   remainder may present with nonspecific symptoms [38]. This lack of specificity and other factors

337   may lead to inaccurate diagnosis unless the evaluating physician understands the broad clinical

338   spectrum of AHT [39].

339   Kemp et al. [40] described a range of clinical certainty in the diagnosis of AHT based on

340   the identification of certain injuries and their severity. This certainty is higher for children with

341   more severe presentations or with multiple findings [17, 41]. Several characteristic findings

342   have, most frequently, been identified in AHT including subdural hematoma (SDH), brain

343   parenchymal injuries, retinal hemorrhages and rib fractures [2, 10, 41, 42]. In the review by

344   Maguire et al. [41], any combination of 3 or more of the significant diagnostic features yielded a

345   positive predictive value of 85%. Kelly et al. [43] in their review of referrals to a child protection

346   team over a 20-year period, reported that in children less than 2 years old, characteristics of

347   particular interest for AHT included no history of trauma (90%), no external evidence of impact

348   to the head (90%), complex skull fractures with intracranial injury (79%), subdural hemorrhage

349   (89%) and hypoxic-ischemic injury (97%).

350

351   **3.   How is the diagnosis of AHT made?**

352   The diagnosis of AHT is made like any other medical diagnosis by considering all the

353   information acquired via clinical history, physical examination, laboratory and imaging data.

354   **History**:

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355 Inconsistency of the presenting history with the clinical findings is a concern for child

356 maltreatment including AHT. Therefore, detailed history including a follow-up history once the

357 acute illness has been addressed is vital to diagnostic accuracy [44, 45]. The two most common

358 histories provided in cases of confirmed AHT are a low-height fall (of less than 4-6 feet) and no

359 specific history of trauma [46]. Severe head injury or moderate to large non-focal SDH are rarely

360 consistent with a history of a short fall of less than 4 feet (47).

361 There are significant limitations with published biomechanical studies evaluating falls including

362 a lack of complete biofidelic integrity [48-51]. The data for injury thresholds in these studies was

363 derived from adult primates undergoing single, non-impact accelerations [48-51]. The

364 differences in intrinsic material properties of the infant skull, brain, cerebrospinal fluid (CSF)

365 and blood vessels versus an adult human or primate or effects of repeated injury was not

366 considered [33]. We need to develop a better understanding of these critical differences to

367 develop better biomechanical studies, approximating real life situations, that will provide more

368 accurate and reliable information.

369 Review of extensive literature demonstrates that severe intracranial injury from short falls

370 is rare, and the predictions from any biomechanical study/model should not deviate too much

371 from established extensive real life data to be considered valid [25, 47, 52-86]. For example,

372 Chadwick et al. [52] in their study of short falls demonstrated a mortality of 0.48 per million

373 children less than 5 years of age per year. A review of 26 studies of accidental falls from various

374 heights [25, 72-85] involving 1,902 children, found 23 fatal injuries, of which only 0.26%

375 (5/1902) were due to falls from less than three stories [47]. In a review of 24 in-hospital newborn

376 falls from less than one-meter height, 2 babies had non depressed linear parietal fractures and 2

377 babies without skull fracture had infratentorial SDH which was felt to represent birth trauma

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378 related SDH and unrelated to the fall. All the babies had a normal or benign physical

379 examination post fall and had normal findings on examination at discharge [86].

380   Review of the extensive literature informs us that mortality from short falls is extremely

381 rare, majority of these are benign occurrences with no significant neurological dysfunction.

382 Linear skull fracture, associated epidural hemorrhage, focal contusion and rarely small focal

383 SDH or SAH may be seen on imaging but significant intracranial hemorrhage, parenchymal

384 contusion or diffuse hypoxic ischemic injury is uncommon in contrast to findings seen in AHT.

385 When significant neurological dysfunction or mortality does occur with short falls, it is related to

386 a large extra axial hematoma or vascular dissection and secondary stroke [33, 52].

387 **Physical examination and importance of ocular findings**

388   A meticulous examination for external bruises and tenderness should be performed.

389 Bruises to the head and face have been associated with AHT and patterns of injury consistent

390 with grabbing, choking and blunt trauma should be sought [69, 87]. The absence of external

391 trauma to the head and neck is common however and it is possible that soft tissue injury

392 including scalp hematomas may only be evident at autopsy [88].

393   Ocular findings in AHT include orbital and lid ecchymosis, subconjunctival hemorrhage,

394 anisocoria and dysconjugate eye movements and retinal hemorrhages. Retinal hemorrhages are

395 an important finding in AHT and when abuse is suspected, a prompt complete examination

396 including full indirect opthalmoscopic examination through a dilated pupil should be obtained

397 [87]. The incidence of retinal hemorrhage in AHT is approximately 85% [89-90]. "Hemorrhages

398 that are too numerous to count, multilayered and extending to the ora serrata are specific" [91].

399 There are a number of conditions which have been associated with retinal hemorrhages, but the

400 above quoted description is highly suspicious of AHT [87] (Table 2). The retina is multilayered

16

401 and traumatic retinoschisis occurs from vitreo-retinal traction suffered from repeated rapid

402 acceleration/deceleration forces [93]. Deep splits of the retina and even focal retinal detachment

403 can occur. Retinal folds are hypopigmented ridges usually around the macula. In the absence of

404 severe documented head trauma, retinal folds and retinoschisis are more specific for AHT [93].

405 These types of retinal lesions do not occur from birth trauma or papilledema (papilledema occurs

406 in 10% of AHT) [87].

407   A prompt evaluation for retinal hemorrhages is important as they can fade rapidly.

408 Generally, intraretinal hemorrhages clear rapidly, whereas preretinal hemorrhages may persist

409 for many weeks [94]. The presence of too numerous to count intraretinal hemorrhages may

410 indicate that trauma occurred within a few days prior to examination, whereas the presence of

411 preretinal with no or few intraretinal hemorrhages suggests days to weeks since trauma [94]. To

412 identify these patterns accurately, eye examinations should be completed as soon as possible

413 after admission, preferably within 24-48 hours [94].

414 **Laboratory studies and imaging**

415   While the history and physical examination are paramount, appropriate use of laboratory

416 studies and imaging is vital for accurate diagnosis and treatment. Skeletal survey following

417 current guidelines should be performed for all children with potential AHT, particularly those

418 less than 2 years of age [4]. In older children, long bone fractures can be more reliably suspected

419 in the presence of extremity tenderness, swelling or refusal to bear weight. Recent papers

420 discuss the evaluation of bleeding and bone diseases when there is a suspicion of abuse [95, 96].

421   For an acutely ill child with neurologic impairment, an optimal imaging strategy involves

422 initial unenhanced CT with 3-D reformatted images of the calvarium [97], followed by a full

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

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424   who are intact neurologically can be first imaged using MR [98-101]. Suspicion of AHT

425   warrants comprehensive imaging and the decision rule developed from a network of emergency

426   departments regarding the use of imaging in low risk blunt head trauma does not apply when

427   there are concerns for AHT [102-104]. Intracranial bleeding is common in AHT and often

428   presents as subdural hematoma. Magnetic resonance imaging of the brain and spine with a

429   variety of sequences is useful in characterizing extra-axial bleeds and defining cerebral

430   contusion, laceration and other parenchymal brain injuries.

431   A number of comparative studies in young children have elucidated the statistical

432   differences in the types and severity of intracranial injuries due to accidental versus AHT [25,

433   32, 46, 72, 76-77, 79, 83, 105-110]. These studies collectively demonstrate that: 1) skull

434   fractures are equally as common following accidental trauma and AHT, but the complex skull

435   fractures are more common following AHT; 2) EDH are more common following accidental

436   trauma; 3) SDH are far more common following AHT; and 4) subarachnoid, intra-parenchymal

437   and intraventricular hemorrhage are equally common in both AHT and accidental trauma [25,

438   32, 46, 72, 76-77, 79, 83, 105-109].

439   Subdural hematoma is the most commonly observed intracranial lesion (in up to 90%) in

440   young infants with AHT and is most commonly parafalcine in location [110, 111]. The inflicted

441   injury (acceleration/deceleration +/- impact) may lead to tearing of convexity bridging veins at

442   the junction of the bridging vein and superior sagittal sinus. Additionally, rupture of the

443   arachnoid membrane allows cerebrospinal fluid to enter the subdural space mixing with subdural

444   blood (hematohygroma) [112, 113]. SDH may have a mixed attenuation at presentation (Table

445   3). Mixed attenuation SDH are found with greater prevalence in AHT than in accidental head

446   trauma [110]. In the review, by Bradford et al. [111], of 105 confirmed AHT cases, intracranial

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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.

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469   Young infants are at an increased risk of upper cervical spinal injury. Such injury is

470   more likely to be soft tissue or ligamentous in nature [118]. Imaging of bony cervical spine is

471   infrequently positive (0.3-2.7%) in children investigated for suspected child abuse [119]. Non

472   bony spinal abnormalities have, however, been identified in up to 2/3 of victims of AHT, in both

473   clinical and autopsy series [118, 120-121]. Choudhary et al. [120] has shown on MRI that 78%

474   of these infants have spinal findings, mostly ligamentous and up to 75% have spinal subdural

475   hematoma which tracks down from the posterior fossa [118, 120, 122]. It is apparent that

476   cervical, thoracic and lumbar MRI should be added to the diagnostic work-up when there is

477   evidence of intracranial injury. Prior to knowledge of the ligamentous injury, those who denied

478   the existence of the shaken baby mechanism used "lack of spinal injury" to boost their

479   unfounded theory [123-125]. However, it is important to emphasize that absence of ligamentous

480   injury doesn't exclude AHT.

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

482   The determination of whether certain theories are putative explanations for AHT must at

483   least recognize the long and storied medical history of the many etiologies already investigated

484   as reasonable explanations. With those historical investigations as a foundation, trauma has come

485   to be uniformly recognized as the primary etiology of pediatric and adult SDHs [46]. Depending

486   on the health history, clinical presentation and pertinent laboratory testing, there are diseases that

487   are considered in the differential of subdural hematoma and appropriate medical evaluation is

488   required for all children.

489   As medicine and science are dynamic, it is important to continually evaluate new

490   hypotheses and, consequently, re-evaluate previously confirmed scientific understanding, thus

491   avoiding a rush to judgement. In this section, we shall discuss selected current theories that have

20

492 been proffered as causative bases for AHT and that reportedly "mimic" the injuries seen.

493 However, the lack of scientific evidence for these assertions underscores the general consensus

494 opinion of pediatricians and pediatric subspecialists against these theories as reasonable

495 explanations for AHT [1, 126]. Most of these unsubstantiated alternative theories just focus on

496 one aspect of the range of injuries seen in AHT whilst conveniently ignoring other injuries which

497 cannot be explained away. For instance, those postulating cerebral sinovenous thrombosis

498 (CSVT) theory as an alternative diagnosis of AHT, focus on retinal hemorrhage and intracranial

499 SDH while they ignore concomitant skeletal injuries, neck injury or visceral injury.

500   The theories have included association with common procedures such as lumbar

501 puncture, common symptoms such as cough to uncommon clinical presentations such as cerebral

502 sinovenous thrombosis (CSVT) or hypoxic ischemic injuries (HII) in the newborn. The theory

503 of lumbar puncture leading to intracranial hemorrhage precisely mimicking AHT speculates that

504 loss of CSF pressure leads to intracranial hypotension and resultant SDH, but the only evidence

505 provided has been couple of case reports in older children and adult literature [127-129].

506 Meanwhile lumbar puncture is a routine procedure performed safely across outpatient and

507 inpatient settings without intracranial sequela. Complications from lumbar puncture are rare, and

508 in fact a recent study in adults has documented that an underlying issue such as coagulopathy is

509 typically present when complications arise [130].

510   Similarly, sustained cough, choking or dysphagic choking have been speculated to cause

511 SDH and retinal hemorrhage mimicking AHT. The theory speculates that any cause of sustained

512 raised intrathoracic pressure such as choking, paroxysmal coughing, gagging, vomiting etc can

513 potentially cause increased intracranial and retinal venous pressure, by impeding thoracic venous

514 return, leading to traumatic venous rupture with retinal hemorrhage and SDH [131, 132].

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515   However, a computer model developed to prove this hypothesis suffered from lack of a clearly

516   defined threshold for failure of bridging vein in infants and from developing the model from data

517   obtained mostly from adult and animal studies [110, 132]. An isolated case report of SDH

518   present in an infant, with pertussis is also cited to support this theory, but this particular case also

519   had confounding history of a fall a week before presentation, which may have been responsible

520   for the SDH [110, 133]. Additionally, this theory has been negated by prospective studies in 83

521   infants suffering from pertussis demonstrating no evidence of retinal hemorrhages seen in AHT

522   [134, 135]. Dysphagic choking type of ALTE mimicking AHT was described in a Barnes et al.

523   [136] case report and also in a review [137]. The case report was criticized for failing to disclose

524   the source of information, the author's role as defense expert witness, omission and

525   misrepresentations of certain facts and legal outcome, lacking proper evidence base and use of

526   inaccurate information to support speculative explanations [138, 139]. ALTE, which has been

527   replaced with the new terminology brief resolved unexplained events (BRUE), has been shown

528   to have a low prevalence of retinal hemorrhage or SDH and cannot be considered to be the cause

529   of SDH or retinal hemorrhage [140-142]. Similarly, retinal hemorrhage was not identified in a

530   prospective study of vomiting infants with hypertrophic pyloric stenosis [143]. These prospective

531   studies underline the fact that while the cough/dyphagic choking/vomiting theory is supported by

532   no recent solid evidence base, there are strong prospective studies providing evidence which

533   refutes these theories. In a retrospective study, children who presented with ALTE and subdural

534   hemorrhages were found to be nearly 5 times more likely to have at least one suspicious

535   extracranial injury, supporting the diagnosis of AHT thereby negating the role of ALTE as a

536   causative mechanism for findings concerning AHT [144].

22

537   Hypoxic ischemic injury (HII) is another diagnosis proposed as an etiology of intracranial

538   SDH and retinal hemorrhage, posited by some to precisely mimic AHT [145, 146]. This is based

539   upon Geddes et al. [145] unified hypoxia theory which derived its findings from the

540   commonality between intracranial postmortem findings of pediatric patients who suffered from

541   hypoxia and patients with AHT. However, this theory has been refuted by a number of studies

542   where SDH was not identified, either on pathology, on imaging or both, in the clinical context of

543   hypoxic injury [147-150]. Besides, traumatic AHT can be present without hypoxia and AHT

544   with hypoxic injury also may coexist with other clinical findings such as visceral, skeletal

545   injuries and paraspinal soft tissue injuries supporting the diagnosis of AHT [118]. Though

546   hypoxia can be seen frequently in traumatic injury of the brain, it is likely a comorbid association

547   similar to other traumatic injuries of the brain and spine.

548   Cerebral sinovenous thrombosis (CSVT) has been proposed as a cause of intracranial

549   injury in children. This unsupported theory proposes that raised intracranial venous pressure

550   resulting from cerebral sinovenous thrombosis leads to bursting of bridging veins resulting in

551   brain parenchymal injury, SDH and retinal hemorrhage similar to pattern of injuries seen in AHT

552   [115, 151-153]. CSVT is an uncommon disorder in childhood but fortunately has been well

553   reported in the literature and thereby provides us with a robust evidence base to conclusively

554   refute this theory [110, 154- 159]. Though it can be associated with parenchymal hemorrhagic

555   infarct, resulting in significant morbidity and mortality, there is no evidence in the literature

556   where primary CSVT thrombosis has been identified as the cause of acute SDH or presentation

557   with abrupt collapse with prolonged coma in a previously healthy child [115]. CSVT has been

558   identified in situations where it is secondary in nature, consistent with the mechanism of

559   pathology such as iron deficiency anemia, inherited predisposition toward coagulation and

23

560 trauma [110, 115]. We should not confuse thrombosis with subcortical hemorrhage, similarly

561 absence of veins on MRV (MR Venogram) doesn't equate to thrombosis and demonstration of

562 intraluminal thrombosis is equally important [115].

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

564   Benign enlargement of subarachnoid spaces (BESS) is commonly seen in the setting of

565 macrocephaly in infancy. Though initially thought to predispose to SDH with minimal trauma

566 [160], the latest reviews (Table 4) reveals less than 6 % of such patients develop hemorrhagic

567 subdural collections [160-166]. Most of the published series suffer from variable methods of

568 ascertainment, variable description of the kind of subdural collections –cerebrospinal fluid,

569 hemorrhagic fluid, or a mixture of the two – and without complete assessment for abuse in these

570 cases [164].

571 Taking only those reports from Table 6, in which the prevalence of BESS has also been

572 documented, a total of 712 cases of BESS were documented with 38/712 (5.3%) reported to have

573 subdural collection, including 12/712 (1.7%) which were reported to be hemorrhagic in nature.

574 Accidental trauma or abuse was reported in 5/12 (41.7%) of subdural collections which were

575 hemorrhagic. Besides, up to 50% of children with BESS and SDH may display concomitant

576 important injuries [167].

577 Overall, subdural collections are uncommonly seen in the setting of BESS and assessment to

578 exclude trauma, including AHT should be performed in those with hemorrhagic and non

579 hemorhagic subdural collections, especially under 2 years of age.

580 **Birth trauma**

581 The risk factor for intracranial hemorrhage in newborn infants is abnormal labor, as

582 evidenced by a higher rate of traumatic brain injury in infants born by Cesarean section after an

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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.

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5. Multidisciplinary assessment and long-term outcome

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The medical diagnosis of AHT is made by pediatricians and pediatric subspecialists based on

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medical evaluation. In many children's hospitals, cases are evaluated by an interdisciplinary

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team of specialists that include physicians, nurses, hospital social workers and others. Hospital-

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based multidisciplinary teams have been used in many communities to provide comprehensive

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assessments and services for families for over sixty years. The overriding goal of the work of

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these teams is to diagnose and to treat child abuse and neglect, assess for alternative diagnoses

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when appropriate and to assist in the efforts of the many agencies involved. The Children's

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Hospital Association (formerly the National Association of Children's Hospitals and Related

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Institutions) has released guidelines for team composition and function to aid providing services

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[101, 192]. In addition, in some jurisdictions, multidisciplinary teams of hospital and

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community professionals review injuries, medical history, family and social risk to reach a more

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comprehensive assessment. These hospital-community partnerships are composed in part of

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physicians, nurses, social workers, clergy, psychologists, child protection services, law

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enforcement and other professionals with relevant experience. These multidisciplinary teams can

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review all of the data related to the case from different perspectives to gain a more complete

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understanding of the issues [8, 45, 193-196]. When testimony is presented in a legal setting,

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there has usually been much in-depth consideration of the diagnosis and the probability of the

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correct diagnosis is high.

625

Abusive head trauma is the leading cause of physical abuse fatalities. In a review of child

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abuse fatalities, shaking was identified as a cause or contributor for 45% of the deaths, with

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beating, kicking and chronic battering accounting for the rest [193]. Crying was identified as the

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629 trigger for 20% of deaths, followed by disobedience (6%), domestic arguments (5%), toilet

630 training (4%) and feeding problems (3%) [193]. Infants are significantly more likely to be

631 physically abused when there is caretaker emotional disturbance and violence between caretakers

632 [197]. Unfortunately, when AHT is not prevented, the outcome can be devastating and the

633 financial costs to society extremely high [198] (Table 5). The lifetime cost of estimated 4824

634 cases in 2010 was $13.5 billion [199].

635

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

637 **The Medical Expert Witness**

638   The most recent AAP policy statement on expert witness testimony has reemphasized the

639 fact that expert witness neutrality and professional integrity can be a pivotal factor in civil and

640 criminal child abuse cases [200]. When expert testimony is scientifically reliable, objective and

641 accurate, it provides useful information for the legal factfinder. Ethical and professional norms

642 of responsible expert testimony require physicians to be objective and neutral assessors and

643 conveyors of medical information, which means that they should weigh the scientific merit of

644 their own opinions and conclusions and "present testimony that reflects the generally accepted

645 standard within the specialty or area of practice, including those standards held by a significant

646 minority" [200, 201]. Regrettably, not all medical expert's courtroom testimony falls within

647 these ethical and professional boundaries. A few physicians, including those who do not treat or

648 diagnose children as part of their medical practice, frequently proffer various speculative

649 causation theories (see above) camouflaged as alternative or mimic diagnoses in child

650 maltreatment cases. These medical witnesses run afoul of professional norms and standards and,

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651 when their arguments are repeated by the media, create a grave public health risk by

652 promulgating dangerous misinformation regarding safe infant and childcare.

653

654 **7. What can be done to provide the court accurate information about the state of medical**

655 **knowledge in AHT?**

656 **The admissibility of expert evidence**

657   In current day jurisprudence, admissibility of medical or scientific expert testimony

658 requires some judicial assessment of the "reliability" of that testimony. In some jurisdictions, the

659 standard for assessing admissible expert testimony is the Frye standard (or whether a particular

660 concept or methodology is "generally accepted" in the medical/scientific community); in others,

661 it is a Daubert standard (where judges consider additional criteria other than just "general

662 acceptance", such as testability, peer review and publication and error rate). But, in any legal

663 jurisdiction, the medical precept that is considered "generally accepted" holds significant weight

664 with courts. Unfortunately, courts are generally ill-equipped to measure the general consensus of

665 physician thought on a particular concept, which makes them susceptible to more speculative

666 theories unsupported by the medical evidence and medical literature. Thus, consensus statements

667 present a unique opportunity to provide courts with a way to know general medical thought

668 about a particular medical topic.

669

670 **Professional society consensus statements**

671 **Physician acceptance**

672   Courts should assume that a consensus statement reflects general physician acceptance of a

673 particular precept. Table 6 describes the rigorous process used to construct this type of

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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

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697   symptoms; (3) diagnosis by default; (4) an absence of neck injuries proves AHT did not occur;

698   (5) shifting scientific consensus; (6) an epidemic of copycat false convictions; and, (7) the

699   presumption that confession evidence consistent with infant injuries was coerced (the two papers

700   on confessions from France, in fact offer the perpetrator no reason to confess as leniency cannot

701   be offered via French law) [31, 32]. These arguments are repeatedly raised in court despite the

702   fact that they have never been empirically substantiated or are patently false.

703   There is a major flaw propagated in the few articles of those who deny SBS/AHT. It is the

704   erroneous use of the terms "evidence based medicine" and "systematic review "[202]. Because

705   the suggestion that denialist views are supported by the evidence is likely to confuse judges and

706   juries, we address two purported literature reviews: Donohoe in 2003 [203] and Lynoe et al in

707   2017 [204]. Both articles are flawed by "1) improper search and systemic review questions 2)

708   improper criteria for assessing bias and 3) inequitable application of quality of study assessment

709   standards." [138, 205].

710   It is unprecedented that Donohoe's "systematic review" chose to exclude the voluminous

711   literature before 1999 despite the fact that AHT was well described by multiple authors world-

712   wide and the incidence of the disease was quite similar world-wide [206]. In the final analysis,

713   Donohoe uses only 23 articles to reach his erroneous conclusions. As Greeley showed, evidence

714   supporting the AHT medical diagnosis "clearly fits the Bradford Hill criteria for causation"

715   [207]. Similarly, despite the vast medical literature, Lynoe et al. [204] chose to use only 30

716   publications. Narang et al. [205] reveal the severe prejudicial bias of the authors of the Lynoe et

717   al. [204] study. Additional publications have also refuted this report [208-212]. This alternative

718   agenda has no role in true science and can result in infant harm through shaking and neglect,

719   through avoidance of emergency medical intervention.

720   In contrast, a 2016 study published in The Journal of Pediatrics finds a high degree of

721   medical consensus that shaking a young child can cause subdural hematoma, severe retinal

722   hemorrhage, coma or death [3]. The study, which surveyed 628 physicians at 10 leading U.S.

723   children's hospitals, found that 88% of physicians believe that SBS is a valid evidence-based

724   diagnosis and 93% believe that the somewhat more comprehensive diagnosis of AHT is a valid

725   evidence-based diagnosis [3].

726   **AHT is a medical diagnosis not a legal finding of murder**

727   It is increasingly popular for defense lawyers to argue that AHT is a medical diagnosis of

728   murder. This evocative courtroom hyperbole deliberately distorts the judicial process by

729   mischaracterizing the physician expert's role. The medical expert in a child abuse case plays just

730   one role – to help the judge or jury answer the medical question of whether an infant's injuries

731   were most likely caused by abuse or whether they can be plausibly explained by a recognized

732   disease or by one or more of the myriad hypothetical alternative causal explanations typically

733   proffered by the defense. It is absurd to argue that a medical diagnosis proves murder. Medical

734   expert testimony on the etiology of the injury cannot answer the two foundational legal questions

735   of actus reus (latin for guilty act) or mens rea (latin for guilty mind). That is because, even after

736   the factfinder decides that the medical evidence supports a finding that an infant's injuries were

737   inflicted, non-medical evidence is required to determine who committed the act and to determine

738   the level of intent (e.g., knowing, reckless or negligent). "The debate surrounding AHT is neither

739   scientific nor medical but legal" [206]. The denialists have tried to create a medical controversy

740   where there is none.

741   The "diagnosis of murder" argument is obviously wrong because it falsely implies that

742   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.

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788   10.   A professional medical society's consensus statement educates judicial factfinders, the

789   media and the public about "general acceptance", what is accurate medical information and what

790   are non evidence, speculative, or professionally irresponsible etiological hypotheses.

791   11.   The professional society's consensus statement on AHT should help the court recognize

792   unsubstantiated medical expert testimony.

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1309   Table 1. Nomenclature for inflicted, non-accidental trauma in infants and children

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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

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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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1320   Table 3. Various appearances of subdural collection as seen on CT [110]

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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

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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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1330   Table 5. Outcomes after abusive head trauma [198]

1331   Death (20-25%)

1332   Spastic hemiplegia or quadriplegia (15–64%)

1333   Intractable epilepsy (11–32%)

1334   Microcephaly with cortico-subcortical atrophy (61–100%)

1335   Visual impairment (18–48%)

1336   Language disorders (37–64%)

1337   Agitation, aggression, tantrums, attention deficits, memory deficits, inhibition or

1338   initiation deficits (23–59%)

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1342   Table 6. Process for Development of a Consensus Statement

1343   a. Topic under society's expertise needs clarification

1344   b. Governing body of society appoints individuals or society's committee with
1345   expertise on subject to study issue and write a statement.

1346   c. The appointed group (the writing group) may utilize experts from other
1347   medical subspecialties and other professional societies as consultants and
1348   authors

1349   d. A draft document is created and reviewed by participating individuals,

1350   e. The document, after modification by this input is sent to the Governing Body
1351   of the Society for comments

1352   f. With these comments, the writing group revises the document and submits to
1353   the Governing body for approval

1354   g. The Governing body circulates the document to the Society membership for
1355   comment and if necessary further revision

1356   h. After this comprehensive creation and review process is completed, the
1357   document is published

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