73278-1

SUPERIOR COURT OF WASHINGTON
FOR SNOHOMISH COUNTY

THE STATE OF WASHINGTON, Plaintiff, v. MICHAEL J. MORRIS, Defendant.	73278-1 No. 09-1-01071-9 STATE'S MOTION TO TRANSFER MOTION FOR RELIEF FROM JUDGMENT

I. MOTION

The State of Washington moves for an order transferring the defendant's Motion for Relief from Judgment to the Court of Appeals, for consideration as a personal restraint petition. This motion is based on CrR 7.8(c)(2) and the following memorandum.

II. FACTS

A.M., born April 2009, lived with her mother Brittany Morris, her father, the defendant, Michael Morris, and her sister T.M. in Brier in April and May 2009. T.M. is 18 months older than A.M. The defendant worked outside the home while Ms. Morris was the girls' primary caregiver. 6/1/11 RP 147-149.

During the first six weeks of A.M.'s life the defendant expressed anger and frustration toward A.M. On one occasion when he was asked to care for A.M. he

2220

became angry because he wanted to play video games with his friend instead. The defendant spoke roughly toward A.M. and used profanity in reference to her when she cried. Ms. Morris also noticed the defendant playing roughly with A.M. on another occasion. 6/1/11 RP 101-102; 6/2/11 RP 134.

On May 29, 2009 a neighbor, Ms. Cheralyn Orkiz, cared for A.M. while Ms. Morris took T.M. to the doctor. During that time A.M. acted like a normal baby, sleeping, and playing with a mobile once she awoke. Ms. Orkiz had seen A.M. almost daily since she was born. A.M. acted no different than she had on prior occasions when Ms. Orkiz saw her. 6/1/11 RP 79-84. When Ms. Morris picked A.M. up she took her home. After dinner Ms. Morris fixed A.M. a bottle and left her in the defendant's care while she took T.M. to another doctor's appointment. 6/1/11 RP 79-84, 156-157.

Within 10-12 minutes after Ms. Morris and T.M. left, the defendant raced over to the Orkiz's home with A.M. in his arms. The defendant was panicked and told the Orkiz's that something was wrong with A.M. and he needed to get her to a hospital. Mr. Orkiz noticed that A.M. was having trouble breathing. On the way to the hospital the defendant told Mr. Orkiz that A.M. had suddenly started vomiting out of her mouth and nose. A.M. did not vomit on the way to the hospital. 6/1/11 RP 126-130, 158.

Ms. Orkiz notified Ms. Morris what happened. Ms. Morris met the defendant and A.M. at the hospital where she took A.M. into the emergency room. A.R. was limp and her skin was blue. It appeared she was not breathing. 6/1/11 RP 90, 161-163.

A.M. was immediately examined by Dr. Borromeo. He noticed her breathing was shallow so he first gave her oxygen but ultimately intubated her. A CAT scan showed she had a subdural hemorrhage. Due to her condition A.M. was transferred to Harborview Hospital. 6/1/11 RP 11-24.

A.M. was seen by a team of doctors at Harborview including Dr. Kenneth Feldman, an expert in assessing child abuse. A.M. was given a number of tests that showed she had subdural bleeding and profuse retinal hemorrhages. Dr. Feldman examined A.M. He observed that A.M. had seizures and a recent history of apnea. He got a history from Ms. Morris, reviewed all of the test results, and consulted with other doctors who treated A.M. He considered all of the alternative reasons for her condition and concluded that A.M. had suffered from non-accidental trauma. This opinion was shared by all the other medical professionals who treated A.M. Dr. Feldman believed the trauma likely resulted from a high level of acceleration/deceleration force. Dr. Feldman stated that the constellation of A.M.'s injuries is highly specific for abuse. Dr. Feldman also reviewed A.M.'s medical reports from examinations after her release from the hospital and concluded that the resulting impairment from her injuries would be permanent. 6/2/11 RP 96-109, 117-119, 130-131, 135-138, 152-157; 6/3/11 RP 5-16, 19-21.

Dr. Erin Herlihy, a pediatric ophthalmologist examined A.M. on June 1 and again on July 17. On the first examination Dr. Herlihy found that blood separated all of the layers of A.M.'s retinas, the optic nerve was obscured by the profuse amount of blood in her eyes, and that A.M. had no electrical activity in her eyes. At the second examination Dr. Herlihy found A.M.'s visual behavior subnormal for a child her age. Dr. Herlihy reviewed A.M.'s blood tests and ruled out blood disorders and metabolic disorders, as well as meningitis, that could have caused the kind of retinal hemorrhages observed in A.M.'s eyes. After ruling out any medical condition that could have caused the extensive damage to A.M.'s retinas Dr. Herlihy concluded that A.M. suffered severe head trauma that involved "some shearing injury, acceleration/deceleration force, something that would cause a shearing type of injury to tear blood vessels." In her opinion the nature of the injury could not be caused by an accidental fall from a bed or minor jostling. 6/6/11 RP 279, 290-95, 310-22.

The defendant spoke to a CPS worker and later to Officer Murphy. The defendant told the CPS worker that A.M. choked when he was feeding her. The defendant explained that he panicked and ran across the street to the Orkiz's home. The defendant told Officer Murphy that he performed CPR on A.M. when she started vomiting as she was feeding. He stated A.M. may have been injured when her head bobbed up and down as he ran with her across the street because he had not supported her head. While writing his statement the defendant re-contacted the officer to state that he remembered that he also shook A.M. when she vomited, first a little, and then more forcefully. 6/2/11 RP 25,31, 37-42.

The defendant was charged by amended information with one count of assault of a child first degree. The State alleged that the victim was a family or household member as defined by RCW 10.99.020 and the aggravating factor that the defendant knew or should have known that the victim was particularly vulnerable and incapable of resistance, RCW 9.94A.535(3)(b). Ex. 1. The defendant was found guilty at trial. The jury answered found the victim was a family or household member and the aggravating factor had been proved. Ex. 2.

The Court of Appeals affirmed the defendant's conviction but remanded for clarification of community custody conditions. The mandate issued on July 19, 2013. Motion App A[1]. The court clarified the conditions of community custody on October 24, 2013. App. D.

III. ISSUE

Should this case be transferred to the Court of Appeals for consideration as a personal restraint petition?

IV. ARGUMENT

Motions to vacate judgment can be either resolved by this court on the merits or transferred to the Court of Appeals. The standards governing this choice are set out in CrR 7.8(c)(2):

> The court shall transfer a motion filed by a defendant to the Court of Appeals for consideration as a personal restraint petition unless the court determines that the motion is not barred by RCW 10.73.090 and either (i) the defendant has made a substantial showing that he or she is entitled to relief or (ii) resolution of the motion will require a factual hearing.

The provisions of this rule are mandatory. If the requirements for transfer are satisfied, the court may not decide the motion – even if the motion is clearly unfounded. State v. Smith, 144 Wn. App. 860, 184 P.3d 666 (2008).

Under this rule, this court should resolve three issues: (1) Is the motion barred by RCW 10.73.090? (2) Has the defendant made a substantial showing that he or she is entitled to relief? (3) Will resolution of the motion require a factual hearing?

[1] Attachments to this motion are designated as exhibits. Attachments to the defendant's motion are designated as Motion App. __.

A. THE DEFENDANT'S MOTION IS NOT TIME BARRED.

RCW 10.73.090(1) sets a time limit on motions to vacate judgments and other forms of "collateral attack." Such a motion must be filed within one year after the judgment becomes final. Since the judgment in the present case was appealed, it became final on July 19, 2013, the day that the appellate mandate was issued. RCW 10.73.090(3)(b). The present motion was filed on July 18, 2014. It was filed within the time limit.

B. THE DEFENDANT HAS NOT MADE A SUBSTANTIAL SHOWING OF ENTITLEMENT TO RELIEF.

The defendant raises two grounds for relief, both related to the medical testimony produce through Dr. Feldman. First, the defendant states that the medical testimony was unreliable and misleading, and should not have been admitted under ER 702. He argues that admitting that evidence violated his due process right to a fair trial. Second the defendant argues that he received ineffective assistance of counsel when counsel either did not seek to exclude Dr. Feldman's testimony or did not confront Dr. Feldman's testimony "to prevent the jury from relying on untrustworthy testimony." Motion at 9.

1. <u>Biomechanical Studies Challenging The Opinion That A.M.'s Injuries Resulted From Abusive Head Trauma That Existed Before Trial Do Not Demonstrate Extraordinary Circumstances Justifying a New Trial.</u>

CrR 7.8(b) lists five reasons why a court may relive a party from a final judgment. The defendant relies on CrR 7.8(b)(5) "any other reason justifying relief from the operation of the judgment." Motion at 2. This basis is limited; it is reserved for extraordinary circumstances that include fundamental and substantial irregularities in the court's proceedings or irregularities extraneous to the court's actions. State v. Smith, 159 Wn. App. 694, 700, 247 P.3d 775 (2011). "CrR 7.8(b)(5) will not apply when the circumstances used to justify the relief existed at the time the judgment was entered." Id.

A defendant was entitled to relief pursuant to CrR 7.8(b)(5) when the court considered alternatives to confinement as a factor when imposing a sentence, and those alternatives were later eliminated by the county as a result of budget constraints. Id. However CrR 7.8(b)(5) did not justify relief when the reason asserted for relief existed and was knowable prior to sentencing. State v. Florencio, 88 Wn. App. 254, 945 P.2d 228 (1997), review denied, 134 Wn.2d 1026 (1998) (When a defendant's criminal history entered under a different name was discovered after sentencing CrR 7.8(b)(5) did not justify resentencing the defendant in order to include the new criminal history in the defendant's offender score.), State v. Cortez, 73 Wn. App. 838, 721 P.2d 660 (1994) (A defendant who was informed at the time he pled guilty that his conviction may subject him to deportation was not entitled to vacate his conviction pursuant to CrR 7.8(b)(5) when he was later subject to deportation as a result of that conviction).

Trial in this case was held between May 27 and June 10, 2011. At trial Dr. Feldman testified that in making a diagnosis doctors relied on experience, the child's history, and literature in the field to know "what are the normal consequences of accidents, what are the characteristics of normal injuries and illness, what are the characteristics of abusive injuries." 9/2/11 RP 116-117. Dr. Feldman discussed several good studies that indicated retinal bleeding, subdural bleeding and the brain injury A.M. suffered were typically the result of whiplash forces which could be caused by a variety of mechanical forces. He referenced a recent study by Vinchon that used confessions as a baseline to distinguish injuries observed in cases of non-accidental trauma versus those involving accidental injuries. He noted that there were studies that retinal hemorrhages and apnea are two salient factors for abusive head trauma. 6/3/11 RP 5-6, 12, 16. Dr. Feldman testified that after looking at A.M.'s history and all the possible causes for her injuries is opinion was that she suffered abusive head trauma. He specifically testified that he did not know how A.M. received her injuries because he was not present at the time it occurred. 6/2/11 RP 156-157; 6/3/11 RP 13, 16-17.

The first ground for relief, that the medical evidence produced through Dr. Feldman was unreliable, is based largely on reference to materials written prior to the trial in this case. The defendant cites four biomechanical studies to argue that "it has been repeatedly shown that shaking does not achieve the level of fore necessary to inflict the injuries at issue." Motion at 18. Each of those studies predated the trial by many years:

1. Duhaime et al, <u>The Shaken Baby Syndrome, a Clinical, Pathological and Biomechanical Study</u>, 66 J. Neurosugery 409 (1987) (motion, appendix C)

2. A.K. Ommaya et. al. <u>Biomechanics and Neuropathology of Adult and Paediatric Head Injury</u>, 16 Br. J. Neurosurgery 220, 2002) (motion appendix D)

3. Bandak, <u>Shaken Baby Syndrome: A Biomechanics Analyhsis of injury Mechanisims</u>, 151 Forensic Sci. Int. 71 (2005) (motion appendix G).

4. Prange et al <u>Anthropomorphic Simulations of Falls, Shakes, and Inflicted Impacts in Infants</u>, 99 J. Neurosurg. 143 (2003) (motion appendix H).

Dr. Feldman acknowledged that there is a debate as to whether shaking alone could cause those injuries without some other signs of neck trauma. He specifically discussed the study by Bandak cited by the defendant. Dr. Feldman stated the study was flawed for two reasons. First, the manner in which he conducted his study did not measure the static loads caused by whiplash force. Second, Bandak's calculations were an oversimplification that other well-respected biomechanical engineers had likewise criticized. 6/3/11 RP 7, 22-24. Dr. Feldman also discussed an article by Donohoe that purported to be an evidence based review of the literature regarding shaking as a mechanism of injury. Dr. Feldman noted that the article violated all of the principles of evidence based review, and it was therefore not an authoritative work to rely on. 6/3/11 RP 25-27. Dr. Feldman explained that despite the literature that challenged shaking as a mechanism of injury child abuse, experts continued to consider shaking as a potential cause of abusive head trauma because of the extensive experience with that diagnosis coupled with the confessional data that had to be considered at least partially legitimate. 6/3/11 RP 28-29.

Materials appended to the defendant's motion for new trial support this testimony. The authors of <u>Biomechanics of Head Trauma in Infants and Young Children</u> acknowledge that "many researchers believe that shaking alone can cause SDH (subdural hemorrhage), retinal hemorrhage, and death." Motion App. O page 361. In his book chapter <u>The Case for Shaking</u>, Mark Dias stated "what is abundantly clear, however, from multiple clinical observations is that shaking, with or without impact, plays an important role in the pathogenesis of infant abusive head injuries." Motion App. P at 370.

The defendant also cites a study published in 2011, after he was tried on this charge. Lloyd et al, Biomechanical Evaluation of Head Kinematics During Infant Shaking Versus Pediatric Activities of Daily Living, Journal of Forensic Biomechanics (2011). It does not alter the conclusions in the earlier biomechanics studies. Nor does it alter the body of authority that concludes that shaking may cause sufficiently rapid acceleration/deceleration to result in abusive head trauma. See e.g. Dias, The Case for Shaking, Child Abuse and Neglect, Diagnosis, Treatment and Evidence (2011) (motion app. P). Dias stated that in spite of biomechanical studies, evidence from clinical observations and the number of confessions to shaking resulting in stereotypical injuries that are so frequently encountered in abusive head trauma and rarely found in accidental injury show that is evidentiary basis for shaking as a mechanism of injury. Another book chapter cited by the defendant, Abusive Head Trauma, Hymel and Deye, also address the controversy, stating that a considerable body of literature supporting the conclusion that isolated shaking can injure or kill babies has recently been challenged as "an unproven myth." Motion App. BB at 349.

The defendant does not present evidence that there has been a sudden dramatic shift in the scientific community, unknowable at the time of trial, that so discredits Dr. Feldman's testimony that it should have been excluded under ER 702 thereby constituting an extraordinary circumstance justifying relief under CrR 7.8(b)(5). The evidence supporting and challenging a diagnosis of abusive head trauma and possible mechanisms of injury is the same today as it was before trial. Biomechanical studies do not support the conclusion that the defendant should be entitled to a new trial pursuant to CrR 7.8(b)(5).

2. <u>Information Obtained After the Trial Does Not Constitute An Extraordinary Circumstance that Justifies A New Trial Under CrR 7.8(b)(5)</u>

The defendant also supports his motion with two pieces of information obtained after the trial. He points to an epidemiological report prepared by Dr. Freeman. Motion App. Q. He also points to a subsequent injury sustained by A.M.

The report from Dr. Freeman was generated in 2014 and was based on information from the Kid's Inpatient Database (KID) gathered between 2000 and 2009. Report page 7. This information certainly could have been obtained and Dr. Freeman's analysis conducted before trial. It therefore does not qualify as a reason to grant a new trial under CrR 7.8(b).[2]

The subsequent injury sustained by A.M. was not knowable before trial. However it does not support the conclusion that the diagnosis in the defendant's case was wrong, or should have been excluded on the basis that it was unreliable. Notably the defense asked the court to not rule on the motion, and therefore the court did not grant a motion for new trial on that basis. Ex. 4 (Sub 159). The defendant misrepresents the medical findings from A.M.'s subsequent hospitalization when he states that the Missouri doctors concluded that "abuse seemed implausible." The emails from the deputy prosecutor to trial counsel state the doctors in Missouri believed that A.M.'s injuries in 2011 were the result of acute abusive trauma, unrelated to the prior incident of "shaken baby syndrome." Motion App T, U. Considering the social history the doctors could not suggest who may have caused A.M.'s injuries. The police department therefore closed the case because they were unable to identify a suspect. Motion App. V. This unrelated event, although tragic, has no bearing on whether the

[2] A discussion of Dr. Freeman's report as it relates to the ineffective assistance of counsel claim is covered in section B.3.b.

defendant caused his daughter's injuries in the present case. Nor does it shed any light on the defendant's challenge to Dr. Feldman's testimony as unreliable.

3. Defense Counsel's Strategic Choices Regarding Her Response To The Medical Evidence Are Not a Basis to Grant the Defendant a New Trial.

A defendant's Sixth Amendment right to counsel includes the right to effective assistance of counsel. Strickland v. Washington, 466 U.S. 668, 686, 104 S.Ct. 2052, 80 L.Ed.2d 674 (1984). CrR 7.8(b)(5) may justify relief if the defendant shows that he received ineffective assistance of counsel. State v. Martinez, 161 Wn. App. 436, 440-441, 253 P.3d 445, review denied, 172 Wn.2d 1011 (2011).

A defendant who alleged he received ineffective assistance of counsel as a basis on which to obtain a new trial must establish both that counsel's performance was deficient, and he was thereby prejudiced. Strickland, 466 U.S. at 686. Defense counsel's performance is deficient if it falls below an objective standard of reasonableness based on a consideration of all of the circumstances. State v. McFarland, 127 Wn.2d 322, 334-335, 899 P.2d 1251 (1995). A defendant is prejudiced by counsel's deficient performance if there is a reasonable probability that, except for counsel's unprofessional errors, the result of the proceeding would have been different. Id. A reasonable probability is a probability sufficient to undermine confidence in the outcome of the trial. Strickland, 466 U.S. at 694.

Courts are highly deferential when assessing counsel's performance and will indulge in a strong presumption that counsel acted reasonably. State v. Thomas, 109 Wn.2d 222, 226, 743 P.2d 816 (1987). The defendant must identify the acts or omissions that he alleges do not constitute reasonable professional judgment. Strickland, 466 U.S. at 690. The court determines whether in light of all the circumstance the identified acts or omissions were outside the wide range of professionally competent assistance. Id.

The defendant must overcome the presumption that under the circumstances of the case the challenged action "might be considered sound trial strategy." Strickland, 466 U.S. at 689. Strategic decisions made after a thorough investigation of law and facts relevant to plausible options are virtually unchallengeable. Id. at 690. Where counsel has done less than a complete investigation her conduct is reasonable to the extent that reasonable professional judgment justifies limitation on the investigation. Id. at 691. The court has a duty to "entertain the range of possible" reasons counsel may have had for performing as she did. Cullen v. Pinholster, ___ U.S. ___, 131 S.Ct. 1388, 1407, 179 L.Ed.2d 557 (2011).

The defendant argues that his trial attorney's performance was deficient because she failed to failed to be familiar with the biomechanical literature and history of shaken baby syndrome/abusive head trauma, failed to prevent Dr. Feldman from testifying, and failed to confront Dr. Feldman at trial. Motion at 8-9, 27-28. None of these bases support finding counsel rendered ineffective assistance.

a. Counsel Investigated The Relevant Literature

The defendant does not provide an affidavit from his trial attorneys, or any other evidence, that they did not investigate the biomechanical literature that he relies on or that they were unfamiliar with scientific literature addressing abusive head trauma including shaking as a potential mechanism. His failure to do so is not surprising since the record demonstrates that counsel was familiar with the relevant literature.

Prior to trial defense counsel had interviewed Dr. Feldman. 6/3/11 RP 68. At trial counsel cross examined Dr. Feldman regarding the biomechanical research done by Ommaya in the 1960's involving the use of monkeys and Caffey in the 1970's involving babies. 6/3/11 RP 39-40. Dr. Feldman was questioned about the sector of biomechanical physicians who question the conclusions regarding the kind of injury to infants caused by whiplash forces. Counsel questioned Dr. Feldman about the biomechanical study by Bandak and the 1987 study by Duhaime that he relies on to support his motion for new trial. 6/31/11 RP 41-44, Motion, App C, G. Counsel also questioned Dr. Feldman about the evidence based studies conducted by Leetsma in 2002 and Donohoe wherein each concluded that shaking alone could not account for head injuries in young children. 6/3/11 RP 46.

Counsel also questioned Dr. Feldman about non-abusive causes for A.M.'s injuries and literature that discussed those causes. Specifically counsel explored the symptoms of meningitis and how those symptoms correlated to some of A.M.'s physical findings. She also discussed the possibility of a hypoxic ischemic event. 6/3/11 RP 54-60, 65-67. This line of cross examination was a prelude to the testimony the defense presented through Dr. Barnes and Dr. Gabaeff. 6/7/11 RP 375-405; 6/8/11 RP 492-506, 548-578.

Counsel also questioned Dr. Feldman about other studies that bore on the defense. She asked the doctor about a 2008 study by Rooks that concluded 46% of asymptomatic newborns had subdural hematomas. 6/3/11 RP 62. She also questioned him about a case review conducted by Dr. Adamsbaum that Dr. Feldman had referred her to that looked at cases of confessed abuse. She questioned the doctor about a lucid interval and the possibility of a false confession. 6/3/11 RP 94-99.

This record demonstrates defense counsel did an extensive amount of research into the literature that supported and refuted the diagnosis of abusive head trauma that included shaking as a mechanism of injury. The defendant fails to show counsel's performance was deficient on the basis that she failed to investigate the relevant medical and biomechanical literature.

b. Defense Counsel's Chosen Defense Was a Strategic Decision Made After Full Investigation. The Defendant Was Not Prejudiced When Defense Counsel Did not Move to Exclude Dr. Feldman's Testimony Because That Motion Would Have Been Denied.

When a trial attorney's conduct can be characterized as legitimate trial strategy or tactics, that conduct cannot serve as a basis for a claim that the defendant received ineffective assistance of counsel. State v. McNeal, 145 Wn.2d 352, 362, 37 P.3d 280 (2002). Exceptional deference is given when evaluating trial counsel's strategic decisions. In re Davis, 152 Wn.2d 647, 714, 101 P.3d 1 (2004). Trial counsel's decision to forgo a motion to suppress evidence may be a legitimate trial tactic where there is reason to believe the motion would be unsuccessful or the motion is unfounded. State v. Nichols, 161 Wn.2d 1, 15, 162 P.3d 1122 (2007), McFarland, 127 Wn.2d at 336-337. Since it is apparent that trial counsel had investigated the relevant literature it is reasonable to conclude that counsel concluded that a motion to suppress Dr. Feldman's testimony would not be successful. Counsel therefore made a strategic decision to challenge Dr. Feldman's testimony through cross examination and by presenting expert witnesses who could offer an alternative diagnosis for A.M.'s injuries.

For that same reason the defendant cannot show that trial counsel's decision to forgo a motion to exclude Dr. Feldman's testimony prejudiced him. Expert testimony is admissible if it meets the Frye standard for admissibility and if it qualifies for admission under ER 702, ie. whether the witness qualifies as an expert and whether the witnesses' testimony would be helpful to the trier of fact. State v. Copeland, 130 Wn.2d 244, 255-256, 922 P.2d 1304 (1996).

Under the Frye standard expert testimony is admissible if the underlying principles of that testimony have been generally accepted in the scientific community. Id. at 255. The Frye test applies where either the theory or method of arriving at the data relied on is so novel that it is not generally accepted by the relevant scientific community. Anderson v. Azko Nobel Coatings, Inc, 172 Wn.2d 593, 611, 260 P.3d 857 (2011). It does not require complete unanimity among scientist in order for a court to find the test has been met. Copeland, 130 Wn.2d at 270.

At trial the State introduced evidence that shaking as a mechanism for abusive head trauma is generally accepted in the scientific community. The State offered as exhibits position papers from the American Academy of Pediatrics[3], the American Academy of Ophthalmology,[4] an article published in the American Journal of Forensic Medicine and Pathology written as a position paper for the National Association of Medical Examiners and a document from the Centers for Disease Control. Each of these accepted shaking as a mechanism for injury in abusive head trauma. Ex. 5 (Trial Exhibits 28, 29, 30, 31). In addition to these groups 13 other organizations have

[3] The AAP membership consists of 62,000 primary care pediatricians, pediatric medical sub-specialists, and pediatric surgical specialists. http://www.aap.org/en-us/about-the-aap/aap-facts/Pages/AAP-Facts.aspx

[4] The AAO is the largest national membership association of eye doctors. The academy has over 7,000 members. http://www.aao.org/aao/about/index.cfm

publicly acknowledged that abusive head trauma is a valid medical diagnosis. Narang, M.D., J.D., A Daubert Analysis of Abusive Head Trauma/Shaken Baby Syndrome, 11 Hous. J. Health L. & Pol'y 505-633 (2011), Ex. 6 at pp. 574-576.

In the materials that the defendant relies on several authors also agree that there is a considerable body of literature that supports the conclusion that shaking can injure or kill babies. Motion App. B, Hymenl and Deye, Abusive Head Trauma, p. 349; Motion App. Y, Bhardwaj et. al, A Systematic Review of the Diagnostic Accuracy of Ocular Signs in Pediatric Abusive Head Trauma, p. 983; Motion App E page 1409 (the 2009 policy statement from the AAP introduced by the State as Exhibit 28). Other courts have ruled that shaken baby syndrome is a generally accepted diagnosis in the medical field. State v. Leibhart, 662 N.W. 2d 618, 628 (Neb 2003), State v. Lopez, 412 S.E.2d 390, 393 (S.C. 1991), State v. McClary, 541 A.2d 96, 102 (Conn. 1988).

Trial counsel initially sought a Frye hearing before evidence of shaken baby syndrome could be admitted. Motion App K. In that motion the defense argued that while the diagnosis had general acceptance at one time, recent advancement called into question whether it was still generally accepted. Id. Counsel did not pursue the motion after discussions with the assigned prosecutor revealed the State did not seek to introduce evidence A.M. had been diagnosed with that syndrome. Instead, defense counsel moved in limine to prevent the State from introducing evidence that diagnosis was made. The court granted that motion. 5/27/11 RP 62-64.

Given evidence that there had been longstanding acceptance in the relevant scientific communities regarding abusive head trauma, the physical findings associated with that diagnosis, and the relatively recently published policy statements accepting shaking or shaking with impact as a mechanism for those injuries counsel acted reasonably when she chose the strategy that she did. It was likely that given the available evidence that the trial court would have found the diagnosis was generally accepted in the scientific community, despite evidence some scientists questioned that diagnosis. Instead she successfully excluded evidence that may have been inflammatory, particularly in light of the defendant's admission to repetitively shaking A.M. when she vomited and choked.

Scientific evidence that passes muster under Frye must also be admissible under ER 702. Copeland, 130 Wn.2d at 256. The first prong of that test requires the court to determine whether the witness qualifies as an expert. Id. A witness may be qualified as an expert by knowledge, skill, experience, training, or education. ER 702. Evidence produced at trial established that Dr. Feldman is an eminently qualified child abuse expert. He had been practicing pediatric medicine for 40 years by the time of trial. He had worked as a child abuse specialist for 28 years and had consulted in over 1000 cases of suspected child abuse. He has duel board certifications in pediatrics and in child abuse pediatrics. He had written more than 50 peer reviewed articles, some which dealt with abuse. One article addressed the presence of cervical spine injury related to whiplash type forces. He had written 20 book chapters relating to issues of child abuse. He was an assistant editor of a periodical that reviewed articles pertinent to child abuse. He had given over 100 talks to doctors and other professionals about childhood injuries and distinguishing between accidental and non-accidental injury. 6/2/11 RP 99-112; 6/3/11 RP 24-25. As trial counsel undoubtedly understood had she attempted to challenge Dr. Feldman's testimony on the basis of his qualifications as a child abuse expert she would not have been successful. Counsel reasonably chose not to do so.

Expert testimony must also be helpful to the trier of fact. Expert testimony that is relevant to resolve an issue fact is helpful to the trier of fact. State v. Greene, 139 Wn.2d 64, 73, 984 P.2d 1024 (1999), cert denied, 529 U.S. 1090 (2000). It is also helpful if it concerns matters that are beyond the common knowledge of the average layperson and are not misleading. State v. Groth, 163 Wn. App. 548, 546, 261 P.3d 183 (2011), review denied, 173 Wn.2d 1026 (2012).

To prove the defendant guilty of assault of a child first degree the jury had to determine whether the defendant assaulted A.M. and thereby recklessly inflicted great bodily harm. RCW 9A.36.120. Whether A.M.'s injuries were the result of disease or trauma, and if trauma, whether inflicted or accidental, were issues the jury was required to resolve when deliberating whether the defendant was guilty.

An analysis of the medical findings was certainly beyond the understanding of the average layperson. And Dr. Feldman's testimony was not misleading. He fully explained the evidence that he relied on when forming his opinion. He did not testify as to how A.M. was injured; only that there was a body of authority that established subdural hematomas with the kind of retinal hemorrhages A.M. had were highly indicative of abusive head trauma. He testified about the general belief in the scientific community that shaking or shaking with impact could cause those injuries. He explained the literature that supported that belief as well as the literature that disputed it. The jury was fully informed of all of the relevant facts.

The defendant primarily argues that Dr. Feldman's testimony would not assist the trier of fact because it was unreliable, citing many of the biomechanical studies Dr. Feldman addressed in his testimony. Unreliable testimony is not helpful to the trier of fact. Lakey v. Puget Sound Energy, Inc. 176 Wn.2d 909, 918, 296 P.3d 860 (2013). In this sense expert testimony is not reliable if the expert fails to follow the methodology generally accepted in the relevant scientific community. Id. at 919.

Generally speaking the methodology for diagnosing child abuse and specifically abusive head trauma is the same methodology used for any other medical diagnosis. It includes determining the chief complaint, or symptoms the patient suffers from, reviewing a comprehensive medical history, including a history of the events surrounding the presenting symptoms, and a physical examination. The doctor then considers all the possible causes for the presenting symptoms, and goes through an inferential and deductive process to diagnose the patient. In some contexts the doctor will consult other medical and social services specialists to obtain the most complete information possible in making the diagnosis. Narang, Ex. 6 at 571-573. One of the articles relied on by the defendant advocates this method for diagnosing abusive head trauma. Motion App. BB at 352. This method, the differential diagnosis, has been recognized by courts in Washington as a valid basis on which a medical professional may offer an opinion about causation. Anderson, 172 Wn.2d at 610.

This is the exact process that Dr. Feldman followed when consulted on A.M.'s case. Dr. Feldman reviewed A.M.'s medical and social records, and took a history from A.M.'s mother. He conducted a physical examination of A.M. and recommended additional tests and sought additional medical consultations including an ophthalmology consultation and an MRI. 6/2/11 RP 117-157. Because the methodology Dr. Feldman employed in diagnosing abusive head trauma is an accepted method for any diagnosis his testimony was reliable and therefore admissible.

The defendant's argument is based on a claim that the science behind Dr. Feldman's diagnosis was unreliable and therefore his diagnosis was unreliable and should have been excluded. He focuses on two things; (1) biomechanical research that challenges the belief that shaking can cause the injuries observed in A.M., and (2) materials that challenge the studies that have linked a statistical probability of subdural hematomas and retinal hemorrhages to a diagnosis of abusive head trauma.

The defendant's argument is flawed because it attempts to impose the Daubert test for reliability on this analysis. Motion at 20, n. 14. In Daubert the Supreme Court rejected the Frye test when it concluded FRE 702 had superseded the test for general acceptance in the scientific community. Daubert v. Merrel Dow Pharmaceuticals, Inc, 509 U.S. 579, 587, 113 S.Ct. 2786, 125 L.Ed.2d 469 (1993). Instead the court found the trial court had the duty to determine whether scientific testimony was reliable. Id. at 589. The court set out a nonexclusive list of four factors for trial judges to consider when evaluating whether proffered testimony met that standard; (1) whether the theory or technique can be or has been tested, (2) whether the theory or technique has been subjected to peer review and publication, (3) whether there was a known or potential rater of error, and (4) whether there is general acceptance in the relevant scientific community Id. at 593-594. Washington has not adopted this analysis. Copeland, 130 Wn.2d at 259. Instead the court has found that where the scientific community has accepted a particular methodology and the expert follows that methodology, then questions relating to the validity of the experts' conclusions go to the weight of that evidence and not the admissibility. Id. at 270-71, Lakey, 176 Wn.2d at 920.

Even if the Daubert standard was applicable to admission of Dr. Feldman's testimony the court would likely have admitted that testimony. As Dr. Narang persuasively argues the science behind a diagnosis of abusive head trauma meets that four part test. Ex. 6 at 539-558, 576-588.

The amount of scientific research that has been conducted on abusive head trauma, both as to its characteristic findings and its potential causes, is extensive. Dr. Narang outlined many studies that have found subdural hematomas and retinal hemorrhages are highly associated with non-accidental trauma. Narang, Ex. 6 at 539-540, 578 (referencing the numerous studies in multiple disciplines that have studied abusive head trauma). The vast majority of those studies support Dr. Feldman's diagnosis.

A number of materials that the defendant relies on support the validity of Dr. Feldman's testimony. In Motion App. E the defendant provides the 2009 position paper from the American Academy of Pediatrics which recognized that shaking with or without impact is a recognized mechanism of injury. In Motion App. P at 370, the author reviews the relevant literature and concludes "[w]hat is abundantly clear, however, from multiple clinical observations is that shaking, with or without impact, plays an important role in the pathogenesis of infant abusive brain injuries." Motion App X is the study described by Dr. Feldman at trial. 6/3/11 RP 16. There the researchers studied cases of confessed abuse and known accidents to conclude that the combined findings of subdural hematomas and retinal hemorrhages had an extremely high predictive value for inflicted head trauma. Vinchon et. al, Confessed Abuse Versus Witnessed Accidents in Infants; Comparison of Clinical, radiological, and Ophthalmological Data in Corroborated Cases at 643-644. Motion App. Y concluded that intraocular hemorrhages, especially those that were bilateral, extensive, and multilayered, as observed in A.M., were highly specific for abusive head trauma. Bhardwaj et al, A Systematic Review of the Diagnostic Accuracy of Ocular Signs in Pediatric Abusive Head Trauma. In Motion App. BB the authors recognized that head injury including subdural hematomas can result from rapid acceleration-deceleration. Hymel and Deye, Abusive Head Trauma at 351.

The defendant ignores these authorities and relies on several biomechanical studies to argue that Dr. Feldman's opinion was unreliable because it ruled in shaking which the defendant asserts could not have caused A.M.'s injuries. Motion at 17-19. One study authored by Bandak, Motion App. G, was criticized by other biomechanical scientist due to mathematical errors. Ex. 7 Margulies, Prang, Myers Letter to the Editor Shaken Baby Syndrome: A flawed biomechanical analysis.[5] The other two studies relied on by the defendant do not categorically discredit either Dr. Feldman's conclusions or all of the other scientific work that does recognize shaking, or shaking with impact, can cause the kind of injuries observed in A.M. As Dr. Feldman discussed, studies using anthropomorphic dummies do not establish how an infant brain responds to repetitive sheer forces. 6/3/11 RP 27. At least one recent study using animal models that approximate the stiffness of the human cervical spine has produced the same kind

[5] The defendant appends to his motion Mr. Bandak's study and a response to the letter to the editor. He does not include the letter to the editor criticizing his calculations. Two of the scientists that criticized Bandak's work wrote articles that the defendant relies on to support his position that biomechanical studies discredit the diagnosis of abusive head trauma caused by shaking.

of injuries found in abusive head trauma by shaking, i.e. subdural hematomas and retinal hemorrhages. Ex. 6 Narang, at 569, n. 451. Two biomechanical scientists that the defendant relies upon make clear that while biomechanical studies are an important part of any scientific study of the causes of head trauma they are not the final word on causation. Rather scientists should compare the data with clinical studies to answer that question. Motion App. O, Margulies and Coats Biomechanics of Head Trauma in Infants and Young Children, at 361. For these reasons biomechanical studies do not undermine the wealth of literature and clinical experience that does accept shaking or shaking with impact as a mechanism for abusive head trauma.

The defendant further argues that the evidence base for shaking as a mechanism of injury is weak. Researchers have relied on cases in which perpetrators have confessed to shaking as a mechanism for abusive head injury to determine whether there are characteristic injuries that are predictive for abuse. Motion App X, Vinchon et al, Ex. 8, Maguire et. al, Which Clinical Features Distinguish Inflicted from Non-Inflicted Brain Injury ? A systematic Review, Ex. 9, Maguire et. al, Estimating the Probablity of abusive Head Trauma: A Pooled Analysis (2011), Ex. 10 Piteau et al, Clinical and Radiographic Characteristics Associated with Abusive and Non-abusive Head Trauma: A Systematic Review (2012). The defendant criticized the use of confessions as a method of confirming abusive injury arguing that "perpetrator admissions are hardly scientific and are a known source of wrongful convictions." Motion at 25. But the confessions relied on came from judicial proceedings and therefore were considered reliable by all of the scientist in the cited studies. Because the evidence presented was based on scientific methodology, the scientists conducting the studies, and not lawyers or judges, should be the ones to determine what reliable methodology is and what is not.

Further, the defendant's reference to the Innocence Project's statistic that 30% of the cases in which the defendant had been exonerated through DNA evidence involved perpetrator confessions or guilty pleas does not support the claim that confessions are an unreliable measure to confirm cases of abuse in those studies. The statistic does not provide any insight into the number of cases in which confessions or guilty pleas were made because the perpetrator was actually guilty. As Dias observed:

> It is striking, however, that perpetrators consistently volunteer the same mechanism of injury—shaking the infant—when they are asked to describe what happened. <u>This common and consistent admission by the perpetrator to shaking the infant (whether or not cranial impact injury is described or discovered by physical examination, radiographic studies, or autopsy) overwhelmingly suggests that shaking is an important component of infant abusive TBI and is, in fact, sufficient to cause the intracranial injuries found in AHT. To suggest otherwise (as required by the biomechanical evidence) would require that every confessed perpetrator has to have been</u> consistently and universally <u>lying about the same phenomenon, something that defies logic and common sense.</u>

Motion App. P at 369-370 (emphasis in the original)

The defendant also argues that Dr. Feldman's testimony was unreliable because it was speculative. He argues that injury threshold and level of force are inherent components to determine causation. Motion at 23. Since Dr. Feldman acknowledged that biomechanical studies could not yet determine those facts he argues that Dr. Feldman's testimony that it required a dramatic force with high levels of acceleration and deceleration to cause was unsupported by any reliable information. The defendant does not explain why an exact measure of force or threshold of injury is necessary to conclude a particular victim's injuries were the result of abuse rather than accident. This argument again ignores all the studies that have been conducted regarding abusive head trauma and the general acceptance in the scientific community for the diagnosis of abusive head trauma with shaking or shaking and impact as a mechanism of injury that has been cited in this motion and in Dr. Narang's well written article discussing why the diagnosis meets the Daubert standard for admissibility. It also ignores what Dr. Feldman actually testified to; he did not testify that A.M.'s injuries were the result of shaking because he was not present at the time she was injured. 6/3/11 RP 16-17.

The defendant also challenges Dr. Feldman's opinion that A.M. suffered abusive head trauma on the basis that it was not a differential diagnosis, but a differential etiology. The defendant asserts that while a differential diagnosis is reliable, a differential etiology is not relying on Bowers v. Norfolk S. Corp, 537 F.Supp.2d 1343, 1360 (M.D. Ga. 2007), affirmed, 300 Fed. Appx. 700 (11^{th} Cir. 2008). He claims that Dr. Feldman's diagnosis was a differential etiology because it encompassed the cause manner of injury, i.e. abusive head trauma.

Dr. Feldman's diagnosis that A.M. suffered from abusive head trauma is far different from the diagnosis at issue in Bowers. In Bowers the federal district court ruled on a motion in limine to exclude a doctors "independent" medical exam of the plaintiff at the plaintiff's request in a personal injury action against his employer. The witness was a doctor who did not treat the plaintiff but instead was what the court characterized as a "hired gun" for litigation purposes. Id. at 1354. The doctor had not articulated an accepted premise from which he based his conclusion. Id. at 1355. The court found the doctor's extrapolations from these unstated premises were unfounded. Id. at 1355-56. The doctor did not consider evidence which could have accounted for alternative causes of the plaintiff's injuries. Id. at 1356. Nor was the doctor aware of relevant scientific literature bearing on the issue of causation. Id. It appeared to the court that the doctor simply took the plaintiff's explanation for what happened and adopted it as his own opinion. Id. Finally the doctor admitted on cross examination that he applied no scientific analysis or methodology to come to his conclusion on causation. Id. at 1358. Given these circumstances the court rejected the argument that the doctor's testimony was reliable because it was a differential diagnosis, concluding instead that the opinion was a differential etiology. The court distinguished the two on the basis that a differential diagnosis was reliable because it was performed to treat the patient with potential consequences in the event of a misdiagnosis. A differential etiology was not reliable because there were no attendant risks to getting paid for testifying to an opinion in court. Id. at 1361.

Unlike the diagnosis at issue in Bowers, Dr. Feldman's diagnosis was a diagnosis meant to treat the patient. Abusive head trauma "has long been recognized as a clinically valid medical diagnosis." Ex. 6 Narang, Ex. 11 Al-Holou et al, abstract Nonaccidential head injury in children. Historical vignette. The cause of her injuries was an important factor to that treatment. Whether her injuries were the result of accidental or inflicted trauma the diagnosis gave people guidance on what measures were needed to avoid further injury to A.M. Cf. State v. Sims, 77 Wn. App. 236, 239, 890 P.2d 521 (1995) (Statements identifying an abuser are admissible under the medical diagnosis and treatment exception to the hearsay rule because it is relevant to prevent the recurrence of injury.) Researchers have acknowledged the potentially dire consequences of a misdiagnosis; a child may be returned to an abusive household or kept from his or her family unjustifiably. Ex. 9 at 551. Dr. Feldman was not paid by any interested party, ostensibly to render a particular opinion as a hired gun; he was a salaried employee of Children's Hospital. 6/3/11 RP 35-36. As his direct and cross examination amply demonstrate Dr. Feldman was well versed in the scientific literature both supporting and challenging that diagnosis. He based his diagnosis on scientific principles that had been generally accepted in the medical community.

The defendant's challenge to Dr. Feldman's testimony is similar to the challenge raised to actuarial evidence in Sexually Violent Predator Cases. The Court recognized that actuarial analyses have some drawbacks and that there were arguments for and against their reliability as a predictive tool. In re Thorell, 149 Wn.2d 724, 753, 72 P.3d 708 (2003), cert denied, 541 U.S. 990 (2004). Nevertheless the court found sufficient scientific support to find them reliable. Challenges to the reliability of the science behind that evidence went to the weight, and not the admissibility. Id. at 756, In re Halgren, 124 Wn. App. 206, 220, 98 P.3d 1206 (2004), affirmed, 156 Wn.2d 795 (2006). While the scientific study of abusive head trauma is ongoing, like the actuarial evidence in SVP cases, any data gaps go to the weight and not the admissibility.

The defendant also presents a report from Dr. Freeman who conducted an epidemiology study based on the KID database hospital discharge reports from 2000-2009. Because no affidavit from counsel was supplied it is unknown if trial counsel investigated this avenue to challenge Dr. Feldman's testimony. In the absence of an affidavit the court should not presume that counsel did not investigate an epidemiology study, or made a reasonable strategic decision that investigation was unnecessary.

Even if counsel had not investigated this avenue to challenge Dr. Feldman's testimony the defendant was not prejudiced. The database from which Dr. Freeman drew his conclusions was general; it did not consider the difference between discharge reports from general hospitals and those that specialized in pediatric care. A study that was conducted to determine whether there was a difference in child abuse diagnosis depending on the type of hospital treating the child concluded that general hospitals dramatically underreported child abuse as compared to children's hospitals that specialized in pediatric care. Ex. 12 Trokel et. al, Variation in the Diagnosis of Child Abuse in Severely Injured Infants. In addition to this flaw in Dr. Freeman's analysis, the report fails to take into account all of the physical findings that pointed to a diagnosis of abusive head trauma. He noted that A.M. had difficulty breathing (which the doctors treating her called apnea), retinal hemorrhages, retinoschisis, and subdural bleeding (SDH). Motion App. Q at 4. He analyzed the significance of the retinal injury and SDH separately and together. Id. at 8. But he failed to account for the apnea and seizures, two findings which other experts have found significant in diagnosing abusive head trauma. Ex. 9 at 554-557 (concluding that the presence of apnea and seizures in conjunction with intercranial injury significantly increased the likelihood of abusive head trauma.) If counsel had obtained Dr. Freeman's analysis before trial and presented his testimony, it would not have supported a motion to suppress Dr. Feldman's testimony. Nor given the analytical gaps in his analysis can it be said with any confidence that it would have completely undermined Dr. Feldman's testimony, or any of the other evidence presented that supported the defendant's conviction.

Because a challenge to the admissibility of Dr. Feldman's testimony would not have been successful, the defendant has not shown that he was prejudiced when his attorney did not challenge the admission of his testimony. He has also not established prejudice because other evidence, that the defendant does not challenge, independent of Dr. Feldman's testimony supports the conclusion that A.M. suffered abusive head trauma. Dr. Herlihy, a pediatric ophthalmologist, testified that A.M. had extensive hemorrhages in her eyes; all layers of A.M.'s retinas were completely disconnected by blood. This indicated a severe traumatic injury to her eyes. 6/6/11 RP 292-294. She ruled out any disease or organic cause for A.M.'s injuries. 6/6/11 RP 310-315. She concluded that A.M.'s injuries were the result of severe trauma caused by acceleration/deceleration action. 6/6/11 RP 316. Even in the absence of Dr. Feldman's testimony that A.M.' injuries were the result of abusive head trauma there was sufficient evidence from Dr. Herlihy's testimony for a jury to conclude that A.M.'s injuries were caused by abuse.

c. The manner in which counsel chose to cross-examine Dr. Feldman was a strategic choice that does not constitute deficient performance.

The defendant also argues that trial counsel performed deficiently in that she failed to adequately challenge Dr. Feldman's testimony. He argues counsel failed to question Dr. Feldman about (1) the Donohoe article and the basis for the author's choice of data on which he based his conclusions, (2) the circumstances surrounding the confessions utilized in the Vinchon study, (3) the nature of the studies reviewed in the Bhardwaj article, and (4) articles that suggested a lucid interval could occur after injury.

The conduct of cross examination is a strategic decision within the control of the attorney. In re Stenson, 142 Wn.2d 710, 735, 16 P.3d 1 (2001), quoting ABA standards for criminal justice, 4-.5.2 (2d ed. Supp. 1986). A defendant is not entitled to a new trial on the basis that counsel's cross examination was deficient if counsel's performance fell within the range of reasonable representation or the defendant fails to show that the testimony that would have been elicited on cross-examination could have overcome the evidence against the defendant. State v. Johnson, 143 Wn App. 1, 20, 177 P.3d 127 (2007).

Here counsel explored numerous studies bearing on Dr. Feldman's conclusions, questioned the validity of confessions as potentially false and therefore an inaccurate basis for testing the reliability of physical findings in concluding a patient suffered from abusive head trauma, and explored the subject of lucid intervals. The defendant presents no evidence that had counsel more thoroughly explored the criticisms of the Donohue article that it would have resulted in discrediting those criticisms. See Ex. 6 Narang at 534. The defendant fails to show counsel performed deficiently and was thereby prejudiced by the strategy she chose in cross examining Dr. Feldman.

C. THE DEFENDANT IS NOT ENTITLED TO A FACTUAL HEARING.

When a defendant collaterally challenges his conviction he bears the burden to prove that he was actually prejudiced by any claimed constitutional error. In re Hews, 99 Wn.2d 80, 87, 660 P.2d 263 (1983). When a personal restraint petitioner alleges ineffective assistance of counsel he need only prove prejudice to the degree articulated in Strickland, and not the heightened standard of "actual and substantial" prejudice for other constitutional errors. In re Monschke, 160 Wn. App. 479, 491, 251 P.3d 885 (2010). As to each non-constitutional error the defendant must show that the claimed error constitutes a fundamental defect which inherently results in a complete miscarriage of justice. In re Cook, 114 Wn.2d 802, 813, 792 P.2d 506 (1990).

If the defendant makes a prima facie showing of actual prejudice, but the merits of his contentions cannot be determined solely on the record, the court should hold a full hearing on the merits. In re Rice, 118 Wn.2d 876, 885, 828 P.2d 1086, cert denied, 506 U.S. 958 (1992). The parties have fully briefed the issues in this case and provided the court with extensive scientific literature that addresses the issues as well as the transcript of the trial. The merits of the defendant's claims can be determined from the record. For the reasons outlined above the defendant has not shown that had defense counsel sought to exclude Dr. Feldman's testimony either pursuant to Frye or under ER 702 that a court would have granted that motion. Counsel's decision to withdraw her Frye motion and challenge Dr. Feldman's testimony through cross examination and by presenting experts who testified to alternative, non-traumatic, explanations for A.M.'s injuries was a reasonable strategic choice. For that reason he has not demonstrated that he received ineffective assistance of counsel. He has not made a prima facie showing that he is entitled to relief. The court should not grant him an evidentiary hearing.

V. CONCLUSION

This motion is not time barred. The defendant has not made a substantial showing of entitlement to relief. There is also no need for a factual hearing. Under CrR

7.8(c)(2), the motion should be transferred to the Court of Appeals for consideration as a personal restraint petition.

Respectfully submitted on October 24, 2014.

MARK K. ROE
Snohomish County Prosecuting Attorney

By: Kathleen Webber
KATHLEEN WEBBER, WSBA # 16040
Deputy Prosecuting Attorney

Exhibits States Motion to transfer CrR 7.8 motion for new trial

1. Information
2. Verdict forms, Judgment and Sentence
3. Order amending Judgment and Sentence
4. Order on defense motion for a new trial
5. trial exhibits 28, 29, 30, 31
6. Narang, M.D. J.D., A Daubert Analysis of Abusive Head Trauma/Shaken Baby Syndrome, 11 Hous. J. Health L. & Pol'y 505-633 (2011)
7. Margulies, Prang, Myers Letter to the Editor Shaken Baby Syndrome: A flawed biomechanical analysis
8. Maguire et al, Which Clinical Features Distinguish Inflicted from non-Inflicted Brain Injury? A systematic Review (2009)
9. Maguire et al, Estimating the Probability of Abusive Head Trauma: A Pooled Anlaysis (2011)
10. Piteau et. al., Clinical and Radiographic Characteristics Associated with abusive and Non-abusive Head Trauma: a Systematic Review (2012)
11. Al-Holou et al, abstract Nonaccidental head injury in children. Historical vignette.
12. Trokel et. al, Variation in the Diagnosis of Child Abuse in Severely Injured Infants

FILED

2011 MAY -6 PM 3: 19

SONYA KRASKI
COUNTY CLERK
SNOHOMISH CO. WAS

SUPERIOR COURT OF WASHINGTON
FOR SNOHOMISH COUNTY

THE STATE OF WASHINGTON, Plaintiff, v. MORRIS, MICHAEL J. Defendant.	No. 09-1-01071-9 1st AMENDED INFORMATION

Aliases:

Other co-defendants in this case:

Comes now MARK K. ROE, Prosecuting Attorney for the County of Snohomish, State of Washington, and by this, his Information, in the name and by the authority of the State of Washington, charges and accuses the above-named defendant(s) with the following crime(s) committed in the State of Washington:

ASSAULT OF A CHILD IN THE FIRST DEGREE (DOMESTIC VIOLENCE), committed as follows: That the defendant, on or about the 29th day of May, 2009, then being a person of eighteen years age or older, did intentionally assault A.M. (DOB: 4/15/09), a child who at the time of the assault was under the age of thirteen years, and did recklessly inflict great bodily harm; proscribed by RCW 9A.36.120, a felony; and the victim was a family or household member as defined by RCW 10.99.020; and the crime was aggravated by the following circumstance: the defendant knew and should have known that the victim was particularly vulnerable and incapable of resistance, as provided by RCW 9.94A.535(3)(b).

MARK K. ROE
PROSECUTING ATTORNEY

JARETT A. GOODKIN, 25399
Deputy Prosecuting Attorney

DATED this 6th day of May, 2011 at the Snohomish County Prosecuting Attorney's Office.

EXHIBIT 1

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Address: 965 CEDAR ST	POPLAR BLUFF	MO	63901
HT: 508	DOB: 09/27/1988		SID: WA
WT: 163	SEX: M		FBI:
EYES: Brown	RACE: White		DOC:
HAIR: Brown	DOL: s100024004		DOL STATE: MO
ORIGINATING AGENCY: BRIER POLICE DEPARTMENT			AGENCY CASE#: 0900398

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FILED

2011 JUL -6 PM 4:00

SONYA KRASKI
COUNTY CLERK
SNOHOMISH CO. WASH

INELIGIBLE TO CARRY FIREARMS

SUPERIOR COURT OF WASHINGTON
FOR SNOHOMISH COUNTY

THE STATE OF WASHINGTON, Plaintiff, v. MORRIS, MICHAEL J. Defendant. SID: WA If no SID, use DOB:	No. 09-1-01071-9 JUDGMENT AND SENTENCE [X] Prison [ ] Jail One Year or Less [ ] First Time Offender [ ] Special Drug Offender Sentencing Alternative [X] Clerk's action required, firearm rights revoked, ¶ 5.5 [X] Clerk's action required, ¶¶ 2.1, 4.1, 4.3, 4.5, 5.2, 5.3 [ ] Clerk's action required, ¶ 5.6 (use of motor vehicle) [ ] Restitution Hearing set, ¶ 4.3

Date 7/7/11 Clerk 5 CC Jail 3
CC PA 1 CC SCSO 1 DPA Stm Y/N

I. HEARING

1.1 A sentencing hearing was held and the defendant, the defendant's lawyer and the (deputy) prosecuting attorney were present.

II. FINDINGS

2.1 **CURRENT OFFENSE(S).** The defendant was found guilty on June 13, 2011, by jury-verdict of:

COUNT	CRIME	RCW	CLASS	INCIDENT #	DATE OF CRIME
I	First Degree Assault of a Child (Domestic Violence)	9A.36.120, 10.99.020 & 9.94A.535(3)(b)	A	BRI 0900396	5/29/09

as charged in the First Amended Information.

The jury returned a special verdict ~~or the court made a special finding~~ with regard to the following:

[ ] See ¶ 4.1 regarding findings in relation to Drug Offender or Parenting Sentencing Alternative.

[ ] The defendant used a firearm in the commission of the offense(s) in Count(s) ________. RCW 9.94A.602, 9.41.010, 9.94A.533.

[ ] The defendant used a deadly weapon other than a firearm in the commission of the offense(s) in Count(s) ________. RCW 9.94A.602, 9.94A.533.

[ ] The defendant committed the offense in Count(s) ________ with sexual motivation. RCW 9.94A.835.

[ ] Count(s) ________ Violation of the Uniform Controlled Substances Act (VUCSA), RCW 69.50.401 and RCW 69.50.435, took place in a school, school bus, within 1000 feet of the perimeter of a school grounds or within 1000 feet of a school bus route stop designated

[X] The victim was particularly vulnerable pursuant to RCW 9.94A.535(3)(b).

ORIGINAL

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✓added to JIS

EXHIBIT 2

by the school district; or in a public park, in a public transit vehicle, or in a public transit stop shelter; or in or within 1000 feet of the perimeter of a civic center designated as a drug-free zone by a local government authority, or in a public housing project designated by a local governing authority as a drug-free zone.

[ ] The defendant committed a crime involving the manufacture of methamphetamine including its salts, isomers, and salts of isomers, **when a juvenile was present in or upon the premises of manufacture** in Count(s) ____________________. RCW 9.94A.605, 69.50.401, 69.50.440.

[ ] Count(s) __________________ is (are) a **criminal street gang-related felony offense** in which the defendant compensated, threatened, or solicited a **minor** in order to involve that minor in the commission of the offense. RCW 9.94A.833.

[ ] Count(s) __________________ is (are) the crime of **unlawful possession of a firearm** and the defendant was a **criminal street gang member** or associate when the defendant committed the crime. RCW 9.94A.702, 9.94A.___.

[ ] The defendant committed **vehicular assault** proximately caused by driving a vehicle while under the influence of intoxicating liquor or drug or by operating a vehicle in a reckless manner. The offense is, therefore, deemed a violent offense. RCW 9.94A.030.

[ ] Count(s) __________________ involve(s) **attempting to elude a police vehicle** and during the commission of the crime the defendant endangered one or more persons other than the defendant or the pursuing law enforcement officer. RCW 9.94A.834.

[ ] Count(s) __________________ is (are) a felony in the commission of which the defendant used **a motor vehicle**. RCW46.20.285.

[ ] The defendant has a **chemical dependency** that has contributed to the offense(s) in Count(s) __________________. RCW 9.94A.607.

[X] The crime charged in Count(s) ___I___ involve(s) **domestic violence**. RCW 10.99.020.

[ ] The offense in Count(s) ____________________ was (were) **committed in a county jail or state correctional facility**. RCW 9.94A.533(5).

[ ] Count(s) __________________ involve(s) **kidnapping in the first degree, kidnapping in the second degree, or unlawful imprisonment** as defined in Chapter 9A.40 RCW, where the victim is a minor and the offender is not the minor's parent. RCW 9A.44.130.

[ ] Count(s) _________ and _________ merge. *(See ¶ 3.2 for dismissal of specific count.)*

[ ] Counts __________________ **encompass the same criminal conduct** and count as one crime in determining the offender score. RCW 9.94A.589.

[ ] **Other current convictions** listed under different cause numbers used in calculating the offender score are *(list offense and cause number)*:

2.2 **CRIMINAL HISTORY.** Prior convictions constituting criminal history for purposes of calculating the offender score are (RCW 9.94A.525):

	CRIME	DATE OF SENTENCE	SENTENCING COURT (County & State)	A or J (Adult or Juvenile)	TYPE OF CRIME
1	None				

[ ] The defendant committed Count(s) __________________ while on community custody (adds one point to score). RCW 9.94A.525.

[ ] The court finds the following prior convictions are one offense for purposes of determining the offender score (RCW 9.94A.525):

[ ] The following prior convictions are not counted as points but as enhancements pursuant to RCW 46.61.520:

2.3 SENTENCING DATA.

COUNT NO.	OFFENDER SCORE	SRA LEVEL	STANDARD RANGE (not including enhancements)	*PLUS ENHANCEMENTS	TOTAL STANDARD RANGE (including enhancements)	MAXIMUM TERM
1	0	XII	93 to 123 months		93 to 123 months	LIFE

*(F) Firearm, (D) Other deadly weapons, (V) VUCSA in a protected zone, (VH) Vehicular Homicide, See RCW 46.61.520, (JP) Juvenile Present, (CSG) Criminal Street Gang Involving Minor, (AE) Endangerment While Attempting to Elude.

2.4 [X] EXCEPTIONAL SENTENCE. Substantial and compelling reasons exist which justify an exceptional sentence [X] above [ ] below the standard range for Count(s) I or [ ] within the standard range for Count(s) ______ but served consecutively to Count(s) ______.

[ ] The defendant and State stipulate that justice is best served by imposition of an exceptional sentence above the standard range and the court finds that exceptional sentence furthers and is consistent with the interests of justice and the purpose of the Sentencing Reform Act.

[X] Aggravating factors were [ ] stipulated by the defendant, [ ] found by the court after the defendant waived jury trial, [X] found by jury by special interrogatory. [ ] Findings of fact and conclusions of law are attached in Appendix 2.4. [X] The jury's interrogatory is attached. The prosecuting attorney [X] did [ ] did not recommend a similar sentence.

2.5 ABILITY TO PAY LEGAL FINANCIAL OBLIGATIONS. The court has considered the total amount owing, the defendant's past, present and future ability to pay legal financial obligations, including the defendant's financial resources and the likelihood that the defendant's status will change. The court finds that the defendant has the ability or likely future ability to pay the legal financial obligations imposed herein. RCW 9.94A.753.

[ ] The following extraordinary circumstances exist that make restitution inappropriate (RCW 9.94A.753(5)):

______

[ ] The defendant has the present means to pay costs of incarceration. RCW 9.94A.760.

2.6 PROSECUTOR'S RECOMMENDATION. The prosecutor's recommendation was as follows:

171(*) months on Count I ______ months on Count IV

______ months on Count II ______ months on Count V

______ months on Count III ______ months on Count VI

(*) 123 months + 48 months for vulnerable victim aggravator

Terms on each count to run:

[ ] concurrently with or [ ] consecutively to each other

[ ] concurrently with or [ ] consecutively to the terms imposed in Cause No(s). ______

III. JUDGMENT

3.1 The defendant is GUILTY of the counts and charges listed in Paragraph 2.1.

3.2 [ ] The court DISMISSES Count(s) ______________________.

3.3 [ ] The defendant was found NOT GUILTY of Count(s) ______________________.

IV. SENTENCE AND ORDER

IT IS ORDERED:

4.1 **CONFINEMENT OVER ONE YEAR.** The court sentences the defendant to total confinement as follows:

CONFINEMENT. RCW 9.94A.589. A term of total confinement in the custody of the Department of Corrections (DOC):

147 months on Count I	________ months on Count IV
________ months on Count II	________ months on Count V
________ months on Count III	________ months on Count VI

[ ] The confinement time on Count(s) ____________ includes ____________ months as enhancement for [ ] Firearm [ ] Deadly Weapon [ ] VUCSA in a Protected Zone [ ] Manufacture of Methamphetamine with Juvenile Present [ ] other ________________.

Actual term of total confinement ordered is 147 (*) months.

(*) 123 months plus 48 months for aggravating factor - See ¶2.4

All counts shall be served concurrently, except for the portion of those counts for which there is an enhancement as set forth above at ¶ 2.3, and the following counts which shall be served consecutively:

____.

The sentence herein shall run consecutively to the sentence in cause number(s) ________________

____

and consecutive to any sentence which was imposed before the date of violation for the offenses in this cause number. The sentence shall run concurrently to the sentence in cause numbers ____________ ______________________. RCW 9.94A.589.

Confinement shall commence immediately unless otherwise set forth here: ____________________

____.

CREDIT FOR TIME SERVED. The defendant shall receive credit for time served prior to sentencing if that confinement was solely under this cause number. RCW 9.94A.505(6). The time served shall be computed by the jail unless the credit for time served prior to sentencing is specifically set forth by the court:

____.

[ ] **WORK ETHIC PROGRAM.** RCW 9.94A.690, RCW 72.09.410. The court finds that the defendant is eligible and is likely to qualify for work ethic program. The court recommends that the defendant serve the sentence at a work ethic program. Upon completion of work ethic program, the defendant shall be released on community custody for any remaining time of total confinement, subject to the conditions in ¶ 4.2. Violation of the conditions of community custody may result in a return to total confinement for the balance of the defendant's remaining time of total confinement.

4.2 [X] **COMMUNITY CUSTODY.** RCW 9.94A.701. The defendant shall serve the following term of community custody *(12 months for crimes against a person, drug offenses, or offenses involving the unlawful possession of a firearm by a street gang member or associate; 18 months for violent offenses; and 36 months for serious violent offenses)*:

Count I for a period of	36	months	Count IV for a period of	______	months
Count II for a period of	______	months	Count V for a period of	______	months
Count III for a period of	______	months	Count VI for a period of	______	months

and the conditions ordered are set forth below. The combined term of community custody and confinement shall not exceed the statutory maximum.

The defendant shall report to DOC, 8625 Evergreen Way, Suite 100, Everett, Washington 98208 not later than 72 hours after release from custody.

While on community custody, the defendant shall (1) report to and be available for contact with the assigned community corrections officer as directed; (2) work at DOC-approved education, employment and/or community restitution; (3) notify DOC of any change in the defendant's address or employment; (4) not consume or possess controlled substances except pursuant to lawfully issued prescriptions; (5) not own, use, or possess firearms or ammunition; (6) pay supervision fees as determined by DOC; (7) perform affirmative acts necessary to monitor compliance with orders of the court as required by DOC; and (8) abide by any additional conditions imposed by DOC under RCW 9.94A.704 and .706. The residence location and living arrangements are subject to the prior approval of DOC while on community custody.

[ ] The defendant shall not consume any alcohol.

[X] The defendant shall have no contact with A.M. ____________. [X] See ¶ 4.5.

[ ] The defendant shall remain [ ] within [ ] outside of a specific geographical boundary, to wit: ______________________________.

[ ] The defendant shall participate in the following crime-related treatment or counseling services: ______________________________.

[X] The defendant shall participate in the following: [ ] State certified domestic violence treatment program [ ] chemical dependency evaluation [X] ~~mental health~~ evaluation [ ] anger management program, and fully comply with all recommended treatment. psychological

[X] The defendant shall comply with the following crime-related prohibitions: no contact with minors unless supervised by an adult approved by ~~[illegible]~~ CCO. Brittany Morris may not supervise contact.

Court Ordered Treatment: If any court orders mental health or chemical dependency treatment, the defendant must notify DOC and the defendant must release treatment information to DOC for the duration of incarceration and supervision. RCW 9.94A.562.

4.3 LEGAL FINANCIAL OBLIGATIONS. Defendant shall pay to the clerk of the court:

PVC	[X] $500	Victim assessment	RCW 7.68.035
CRC	$ [X] waived	Court costs, including	RCW 9.94A.030, .505; 10.01.160
		Criminal filing fee $ ____ FRC	
		Witness costs $ ____ WFR	
		Sheriff service fees $ ____ SFR/SFS/SFW/SRF	
		Jury demand fee $ ____ JFR	RCW 10.46.190
		Other $ ____	
PUB	[ ] $962 [X] waived	Fees for court appointed attorney	RCW 9.94A.760
WFR	$	Court appointed defense expert and other costs	RCW 9.94A.760
FCM	[ ] $1,000 [ ] $2,000	Fine RCW 9A.20.021; [ ] VUCSA additional fine deferred due to indigency	RCW 69.50.430
CDF/LDV FCD/NTF/SAD/SDI	$	Drug enforcement fund of $ ____	RCW 9.94A.760
CLF	[ ] $100	Crime lab fee [ ] suspended due to indigency	RCW 43.43.690
EXT	$	Extradition costs	RCW 9.94A.505
RTN/RJN	$	Emergency response costs (Vehicular Assault, Vehicular Homicide, DUI only, $1000 maximum)	RCW 38.52.430
	[X] $100	Biological Sample Fee (for offenses committed after 07-01-2002)	RCW 43.43.7541
PDV	[ ] $100	Domestic Violence Penalty (for offenses committed after 06-04-2004 – maximum $100)	RCW 10.99.080
	$	Other costs for: ____	
	$ 600.00	TOTAL	RCW 9.94A.760

[X] **RESTITUTION.** The above total does not include all restitution or other legal financial obligations, which may be set by later order of the court. An agreed restitution order may be entered. RCW 9.94A.753.

- [ ] A restitution hearing shall be set for ____.
- [X] Defendant waives any right to be present at any restitution hearing (*sign initials*): ____.
- [ ] Defendant waives any right to a restitution hearing within 6 months. RCW 9.94A.750.

[ ] A separate Restitution Order is being entered contemporaneously with this Judgment and Sentence.

[ ] The Department of Corrections (DOC) or clerk of the court shall immediately issue a Notice of Payroll Deduction. RCW 9.94A.7602, RCW 9.94A.760(8).

All payments shall be made in accordance with the policies of the clerk and on a schedule established by the Department of Corrections, commencing immediately, unless the court specifically sets forth the rate here of not less than:

$ 50.00 per month commencing 60 days after release. RCW 9.94A.760.

All payments shall be made within 240 months of [ ] release of confinement; [X] entry of judgment; [ ] other ____.

The defendant shall report to the clerk of the court or as directed by the clerk to provide financial and other information requested. RCW 9.94A.760(7)(b).

[ ] In addition to the other costs imposed herein the Court finds that the defendant has the means to pay for the cost of incarceration and is ordered to pay such costs at $100.00 per day (not to exceed $100 per day) unless another rate is specified here ____. RCW 9.94A.760(2).

[X] The defendant shall pay the costs of services to collect unpaid legal financial obligations. RCW 36.18.190.

[X] The financial obligations imposed in this judgment shall bear interest from the date of the judgment until payment in full, at the rate applicable to civil judgments. RCW 10.82.090. An award of costs on appeal against the defendant may be added to the total legal financial obligations. RCW 10.73.

4.4 [X] **DNA TESTING.** The defendant shall have a biological sample collected for purposes of DNA identification analysis and the defendant shall fully cooperate in the testing. The appropriate agency shall be responsible for obtaining the sample prior to the defendant's release from confinement. RCW 43.43.754.

[ ] **HIV TESTING.** The Health Department or designee shall test and counsel the defendant for HIV as soon as possible and the defendant shall fully cooperate in the testing. The defendant, if out of custody, shall report to the HIV/AIDS Program Office at 3020 Rucker, Suite 106, Everett, Washington 98201 within one (1) business day of entry of this order to arrange for the test. RCW 70.24.340.

4.5 **NO CONTACT.**

[X] The defendant shall not have contact with A.M. (D.O.B. 4/15/09) (*) _(name, DOB)_ including, but not limited to, personal, verbal, telephonic, written or contact through a third party ~~until~~ for life _(date)_ (not to exceed the maximum statutory sentence). EVEN IF THE PERSON WHO THIS ORDER PROTECTS INVITES OR ALLOWS CONTACT, YOU CAN BE ARRESTED AND PROSECUTED. ONLY THE COURT CAN CHANGE THIS ORDER. YOU HAVE THE SOLE RESPONSIBILITY TO AVOID OR REFRAIN FROM VIOLATING THIS ORDER.

(*) unless permitted by a court ordered parenting plan

[X] A separate post conviction Domestic Violence No Contact Order, Anti-Harassment Order, or Sexual Assault Protection Order [ ] was filed at the time of entry of the plea of guilty/guilty verdict [X] is filed contemporaneously with this Judgment and Sentence. _(Entry of a separate order makes a violation of this no contact sentencing provision also punishable as a criminal offense, and the order will be entered into the law enforcement database.)_

[ ] The pre-trial Domestic Violence No Contact Order, Anti-Harassment Order, or Sexual Assault Protection Order entered on ____________ is hereby terminated.

4.6 **OTHER.** ____

4.7 **OFF-LIMITS ORDER.** (Known drug trafficker). RCW 10.66.020. The following areas are off limits to the defendant while under the supervision of the county jail or Department of Corrections: ____________.

4.8 Unless otherwise ordered, all conditions of this sentence shall remain in effect notwithstanding any appeal.

V. NOTICES AND SIGNATURES

5.1 **COLLATERAL ATTACK ON JUDGMENT.** Any petition or motion for collateral attack on this Judgment and Sentence, including but not limited to any personal restraint petition, state habeas corpus petition, motion to vacate judgment, motion to withdraw guilty plea, motion for new trial or motion to arrest judgment, must be filed within one year of the final judgment in this matter, except as provided for in RCW 10.73.100. RCw 10.73.090.

5.2 **LENGTH OF SUPERVISION.** For an offense committed prior to July 1, 2000, the defendant shall remain under the court's jurisdiction and the supervision of the Department of Corrections for a period up to 10 years from the date of sentence or release from confinement, whichever is longer, to assure payment of all legal financial obligations unless the court extends the criminal judgment an additional 10 years. For an offense committed on or after July 1, 2000, the court shall retain jurisdiction over the offender for the purposes of the offender's compliance with payment of the legal financial obligations, until the obligation is completely satisfied, regardless of the statutory maximum for the crime. RCW 9.94A.753(4); RCW 9.94.A.760 and RCW 9.94A.505(5).

5.3 **NOTICE OF INCOME-WITHHOLDING ACTION.** If the court has not ordered an immediate notice of payroll deduction in paragraph 4.1, you are notified that the Department of Corrections may issue a notice of payroll deduction without notice to you if you are more than 30 days past due in monthly payments in an amount equal to or greater than the amount payable for one month. RCW 9.94A.7602. Other income-withholding action under RCW 9.94A may be taken without further notice. RCW 9.94A.7606.

5.4 **VIOLATION OF JUDGMENT AND SENTENCE/COMMUNITY CUSTODY VIOLATION.**
(a) Any violation of a condition or requirement of sentence is punishable by up to 60 days confinement for each violation. RCW 9.94A.633.

(b) If you have not completed your maximum term of total confinement and you are subject to a third violation hearing and DOC finds that you committed the violation, DOC may return you to a state correctional facility to serve up to the remaining portion of your sentence. RCW 9.94A.714.

5.5 **FIREARMS. You may not own, use or possess any firearm unless your right to do so is restored by a superior court in Washington State, and by a federal court if required. You must immediately surrender any concealed pistol license.** *(The clerk of the court shall forward a copy of the defendant's driver's license, identicard, or comparable identification to the Department of Licensing along with the date of conviction or commitment.)* RCW 9.41.040, 9.41.047.

(Pursuant to RCW 9.41.047(1), the Judge shall read this section to the defendant in open court.)

The defendant is ordered to forfeit any firearm he/she owns or possesses no later than ____________ to ______________________________ *(name of law enforcement agency)*. RCW 9.41.098

5.6 **MOTOR VEHICLE.** If the court found that you used a motor vehicle in the commission of the offense, then the Department of Licensing will revoke your driver's license. The clerk of the court is directed to immediately forward an Abstract of Court Record to the Department of Licensing, which must revoke your driver's license. RCW 46.20.285.

5.7 **CERTIFICATE OF DISCHARGE.**
(a) If you are under the custody and supervision of the Department of Corrections, the court will not issue a Certificate of Discharge until it has received notice from Department of Corrections and clerk's office that you have completed all requirements of the sentence and satisfied all legal financial obligations. RCW 9.94A.637.

(b) If you are not under the custody and supervision of the Department of Corrections, the court will not issue a Certificate of Discharge until it has received verification from you that you have completed all sentence conditions other than payment of legal financial obligations and the clerk's office that you have satisfied all legal financial obligations.

5.8	**RIGHT TO APPEAL.** If you plead not guilty, you have a right to appeal this conviction. If the sentence imposed was outside of the standard sentencing range, you also have a right to appeal the sentence. You may also have the right to appeal in other circumstances. This right must be exercised by filing a notice of appeal with the clerk of this court within 30 days from today. If a notice of appeal is not filed within this time, the right to appeal is IRREVOCABLY WAIVED. If you are without counsel, the clerk will supply you with an appeal form on your request, and will file the form when you complete it. If you are unable to pay the costs of the appeal, the court will appoint counsel to represent you, and the portions of the record necessary for the appeal will be prepared at public expense.

5.9	**VOTING RIGHTS STATEMENT.** I acknowledge that I have lost my right to vote because of this felony conviction. If I am registered to vote, my voter registration will be cancelled. My right to vote is provisionally restored as long as I am not under the authority of DOC (not serving a sentence of confinement in the custody of DOC and not subject to community custody as defined in RCW 9.94A.030). I must re-register before voting. The provisional right to vote may be revoked if I fail to comply with all the terms of my legal financial obligations or an agreement for the payment of legal financial obligations. My right to vote may be permanently restored by one of the following for each felony conviction: a) a certificate of discharge issued by the sentencing court, RCW 9.94A.637; b) a court order issued by the sentencing court restoring the right, RCW 9.92.066; c) a final order of discharge issued by the indeterminate sentence review board, RCW 9.96.050; or d) a certificate of restoration issued by the governor, RCW 9.96.020. Voting before the right is restored is a class C felony, RCW 29A.84.660. Registering to vote before the right is restored is a class C felony, RCW 29A.84.140.

5.10 OTHER. A hearing is scheduled for July 28, 2011, at 9:00a.m. in Dept 9 for defense to argue a motion for Stay.

DONE in Open Court and in the presence of the defendant this date: 15 July 2011

JUDGE
Print name: RONALD L. CASTLEBERRY

JARETT A. GOODKIN WSBA 25399 Deputy Prosecuting Attorney	CASSIE C. TRUEBLOOD WSBA 37829 Attorney for Defendant	MICHAEL J. MORRIS Defendant

Interpreter signature/Print name: ______________________

I am a certified interpreter of, or the court has found me otherwise qualified to interpret, the ______________ language, which the defendant understands. I translated this Judgment and Sentence for the defendant into that language. Cause No. of this case: 09-1-01071-9.

I, Sonya Kraski, Clerk of this Court, certify that the foregoing is a full, true and correct copy of the Judgment and Sentence in the above-entitled action, now on record in this office.

WITNESS my hand and seal of the said Superior Court affixed this date: ______________

Clerk of said County and State, ______________________, Deputy Clerk

IDENTIFICATION OF DEFENDANT

SID Number: WA
(If no SID, take fingerprint card for State Patrol)

Date of Birth: 09/27/1988

FBI Number:

Local ID Number:

PCN Number:

DOC Number:

Alias name, SSN, DOB:

Race: White

Ethnicity:
[ ] Hispanic
[ ] Non-Hispanic

Sex: M

Height: 508

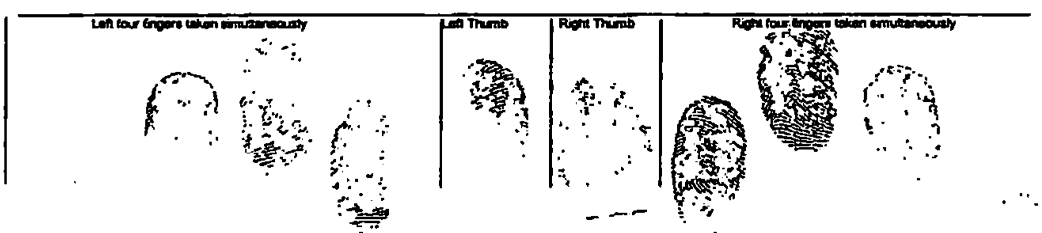
Weight: 163

Hair: Brown

Eyes: Brown

FINGERPRINTS: I attest that I saw the same defendant who appeared in court on this document affix his or her fingerprints and signature thereto. Clerk of the Court: ______, Deputy Clerk.
Dated: 7/1/11

DEFENDANT'S SIGNATURE: ______
ADDRESS: DOC



ORDER OF COMMITMENT

FILED
2011 JUL -6 PM 4:01
SONYA KRASKI
COUNTY CLERK
SNOHOMISH CO. WASH

THE STATE OF WASHINGTON to the Sheriff of the County of Snohomish, State of Washington, and to the Secretary of the Department of Corrections, and the Superintendent of the Washington Corrections Center of the State of Washington:

WHEREAS, MICHAEL J. MORRIS has been duly convicted of the crime(s) of Count 1: Assault of a Child in the First Degree as charged in the 0 Information filed in the Superior Court of the State of Washington, in and for the County of Snohomish, and judgment has been pronounced against him/her that he/she be punished therefore by imprisonment in such correctional institution under the supervision of the Department of Corrections, Division of Prisons, as shall be designated by the Secretary of the Department of Corrections pursuant to RCW 72.02.210, for the term(s) as provided in the judgment which is incorporated by reference, all of which appears of record in this court; a certified copy of said judgment being endorsed hereon and made a part thereof; Now, Therefore,

THIS IS TO COMMAND YOU, the said Sheriff, to detain the said defendant until called for by the officer authorized to transfer to the custody of the Superintendent for the Washington State Department of Corrections or his designee for transport to either the Washington Corrections Center at Shelton, Washington or Washington Corrections Center for Women at Purdy, Washington and this is to command you, the said Superintendent and Officers in charge of said Washington Corrections Center to receive from the said officers the said defendant for confinement, classification, and placement in such corrections facilities under the supervision of the Department of Corrections, Division of Prisons, as shall be designated by the Secretary of the Department of Corrections.

And these presence shall be authority for the same. HEREIN FAIL NOT.

WITNESS the Honorable **RONALD L. CASTLEBERRY**, Judge of the said Superior Court and the seal thereof, this 1st day of July, 2011.

Sonya Kraski
CLERK OF THE SUPERIOR COURT

By [signature]
Deputy Clerk

Filed in Open Court
6-13, 20 11
SONYA KRASKI
COUNTY CLERK
By ________
Deputy Clerk

IN THE SUPERIOR COURT OF THE STATE OF WASHINGTON
FOR THE COUNTY OF SNOHOMISH

STATE OF WASHINGTON	CASE NO. 09-1-01071-9
Plaintiff,	VERDICT FORM A
v.	
MICHAEL J. MORRIS Defendant.	

We, the jury, find the defendant, Michael J. Morris,

GUILTY of the crime of assault of a child in the first degree as charged.
(write in "not guilty" or "guilty")

DATED this 13 day of JUNE, 2011.

Presiding Juror

ORIGINAL

125

Filed in Open Court
6-13, 2011
SONYA KRASKI
COUNTY CLERK
By [signature]
Deputy Clerk

IN THE SUPERIOR COURT OF THE STATE OF WASHINGTON
FOR THE COUNTY OF SNOHOMISH

STATE OF WASHINGTON Plaintiff, v. MICHAEL J. MORRIS Defendant.	CASE NO. 09-1-01071-9 SPECIAL VERDICT FORM 1

We, the jury, answer the question submitted by the court as follows:

Were Michael J. Morris and A.M. members of the same family or household?

ANSWER: Yes
(Write "yes" or "no")

Date: June 13th 2011

[signature]
Presiding Juror

ORIGINAL

127

Filed in Open Court
6-13, 2011
SONYA KRASKI
COUNTY CLERK
By B McDougall
Deputy Clerk

IN THE SUPERIOR COURT OF THE STATE OF WASHINGTON
FOR THE COUNTY OF SNOHOMISH

STATE OF WASHINGTON	CASE NO. 09-1-01071-9
Plaintiff,	SPECIAL VERDICT FORM 2
v.	
MICHAEL J. MORRIS Defendant.	

We, the jury, having found the defendant guilty of assault of a child in the first degree or the lesser included offense of assault of a child in the second degree, return a special verdict by answering as follows:

Did the defendant know, or should the defendant have known, that the victim was particularly vulnerable or incapable of resistance?

ANSWER: YES
(Write "yes" or "no")

DATED this 13 day of JUNE, 2011.

PRESIDING JUROR

ORIGINAL

128

FILED
2011 JUL 28 PM 12: 13
SONYA KRASKI
COUNTY CLERK
SNOHOMISH CO. WASH

SUPERIOR COURT OF WASHINGTON
FOR SNOHOMISH COUNTY

THE STATE OF WASHINGTON, Plaintiff, v. MORRIS, MICHAEL J. Defendant.	No. 09-1-01071-9 ORDER AMENDING JUDGMENT AND SENTENCE

THIS MATTER having come on regularly before the undersigned Judge of the above court on the motion of plaintiff to amend the Judgment and Sentence entered in the above entitled matter on JULY 6, 2011.

AND THE COURT having considered the records and files herein and being fully advised;

NOW THEREFORE, IT IS HEREBY ORDERED that in Paragraph 4.1 of the Judgment and Sentence entered in the above entitled matter on JULY 6, 2011 be and the same hereby is amended to read in part as follows:

- "Actual term of total confinement ordered is 147 months. 123 months plus 24 months for aggravating factor – see Paragraph 2.4"

All other provisions of the Judgment and Sentence remain in force and effect.

IT IS FURTHER ORDERED that the attached Special Verdict Form 2 shall be incorporated as part of the Judgment and Sentence filed on July 6, 2011, as previously indicated in paragraph 2.4 of that Judgment and Sentence.

DATED this 28 day of July, 2011.

JUDGE

Presented by:

JARETT A. GOODKIN, 25399
Deputy Prosecuting Attorney

Approved for Entry; Notice of Presentation Waived:

CASSIE C. TRUEBLOOD, 37829
Attorney for Defendant

EXHIBIT 3

FILED

2013 OCT 24 PM 4:24

SONYA KRASKI
COUNTY CLERK
SNOHOMISH CO. WASH

IN THE SUPERIOR COURT OF THE STATE OF WASHINGTON
IN AND FOR THE COUNTY OF SNOHOMISH

THE STATE OF WASHINGTON, Plaintiff, v. MORRIS, MICHAEL J., Defendant.	No. 09-1-01071-9 ORDER AMENDING JUDGMENT AND SENTENCE

THIS MATTER having come on regularly before the undersigned Judge of the above court on the motion of plaintiff to amend the Judgment and Sentence entered in the above entitled matter on October 24, 2013;

AND THE COURT having considered the records and files herein and being fully advised;

NOW THEREFORE, IT IS HEREBY ORDERED that Paragraph 4.5 of the Judgment and Sentence entered in the above entitled matter on July 1, 2011, be and the same hereby is amended to read in part as follows:

4.5 NO CONTACT.

[X] The defendant shall not have contact with A.M. (DOB 4/15/09) *(name, DOB)* including, but not limited to, personal, verbal, telephonic, written or contact through a third party until for life *(date)* (not to exceed the maximum statutory sentence). EVEN IF THE PERSON WHO THIS ORDER PROTECTS INVITES OR ALLOWS CONTACT, YOU CAN BE ARRESTED AND PROSECUTED. ONLY THE COURT CAN CHANGE THIS ORDER. YOU HAVE THE SOLE RESPONSIBILITY TO AVOID OR REFRAIN FROM VIOLATING THIS ORDER. ⑧

[x] The defendant shall complete a psychological evaluation as previously ordered. The court clarifies that the evaluation should be of a parenting nature similar to the type done in divorce/dependency proceedings.

⑧ Mr. Morris may petition this court for this condition to be lifted after A.M. reaches the age of majority. A separate DV no contact order is being entered contemporaneously with this order amending the judgment and sentence.

SCJO # 1004

√ nco already in JIS per #217

216

All other provisions of the Judgment and Sentence remain in force and effect.

DONE IN OPEN COURT this 24th day of October, 2013.

Judge Ronald L. Castleberry

Presented by:

JARETT A. GOODKIN, 25399
Deputy Prosecuting Attorney

Approved for entry, copy received:

CASSIE C. TRUEBLOOD, 37829
Attorney for Defendant

MICHAEL J. MORRIS
Defendant

IN THE COURT OF APPEALS OF THE STATE OF WASHINGTON
DIVISION I

FILED
2013 JUL 22 PM 12:48
SONYA KRASKI
COUNTY CLERK
SNOHOMISH CO. WASH

STATE OF WASHINGTON, Respondent, v. MICHAEL J. MORRIS, Appellant.	)	No. 67495-1-I MANDATE Snohomish County Superior Court No. 09-1-01071-9 ***Court Action Required***

THE STATE OF WASHINGTON TO: The Superior Court of the State of Washington in and for Snohomish County.

This is to certify that the opinion of the Court of Appeals of the State of Washington, Division I, filed on February 11, 2013, became the decision terminating review of this court in the above entitled case on July 19, 2013. This case is mandated to the Superior Court from which the appeal was taken for further proceedings in accordance with the attached true copy of the opinion.

Court Action Required: The sentencing court or criminal presiding judge is to place this matter on the next available motion calendar for action consistent with the opinion.

c: Jarett A. Goodkin
Casey Grannis
Kathleen Webber
Michael J. Morris
Hon. Ronald Castleberry

SEAL OF THE COURT OF APPEALS • STATE OF WASHINGTON •

IN TESTIMONY WHEREOF, I have hereunto set my hand and affixed the seal of said Court at Seattle, this 19th day of July, 2013.

RICHARD D. JOHNSON
Court Administrator/Clerk of the Court of Appeals, State of Washington, Division I.

53

209

FILED

2011 JUL 28 PM 12: 13

SONYA KRASKI
COUNTY CLERK
SNOHOMISH CO. WASH

IN THE SUPERIOR COURT OF THE STATE OF WASHINGTON
IN AND FOR THE COUNTY OF SNOHOMISH

THE STATE OF WASHINGTON, Plaintiff, v. MORRIS, MICHAEL J. Defendant.	No. 09-1-01071-9 ORDER ON DEFENSE MOTION FOR NEW TRIAL

THIS MATTER having come on regularly before the undersigned Judge of the above court on the motion of [ ] State [X] defendant [ ] court for new trial:

AND THE COURT having considered the records and files herein and being fully advised;

Now Therefore,

IT IS HEREBY ORDERED that the court grants defense request for court not to rule of the motion for new trial.

DONE IN OPEN COURT this 28th day of July, 2011.

Judge

Presented by:

JARETT A. GOODKIN, 25399
Deputy Prosecuting Attorney

Approved for entry, copy received:

CASSIE C. TRUEBLOOD, 37829
Attorney for Defendant

MICHAEL J. MORRIS
Defendant

159

EXHIBIT 4

American Academy of Pediatrics
DEDICATED TO THE HEALTH OF ALL CHILDREN

POLICY STATEMENT

Abusive Head Trauma in Infants and Children

Organizational Principles to Guide and Define the Child Health Care System and/or Improve the Health of All Children

Cindy W. Christian, MD, Robert Block, MD, and the Committee on Child Abuse and Neglect

ABSTRACT

Shaken baby syndrome is a term often used by physicians and the public to describe abusive head trauma inflicted on infants and young children. Although the term is well known and has been used for a number of decades, advances in the understanding of the mechanisms and clinical spectrum of injury associated with abusive head trauma compel us to modify our terminology to keep pace with our understanding of pathologic mechanisms. Although shaking an infant has the potential to cause neurologic injury, blunt impact or a combination of shaking and blunt impact cause injury as well. Spinal cord injury and secondary hypoxic ischemic injury can contribute to poor outcomes of victims. The use of broad medical terminology that is inclusive of all mechanisms of injury, including shaking, is required. The American Academy of Pediatrics recommends that pediatricians develop skills in the recognition of signs and symptoms of abusive head injury, including those caused by both shaking and blunt impact, consult with pediatric subspecialists when necessary, and embrace a less mechanistic term, abusive head trauma, when describing an inflicted injury to the head and its contents. *Pediatrics* 2009;123:1409–1411

www.pediatrics.org/cgi/doi/10.1542/peds.2009-0408

doi:10.1542/peds.2009-0408

All policy statements from the American Academy of Pediatrics automatically expire 5 years after publication unless reaffirmed, revised, or retired at or before that time.

Abbreviation
AHT—abusive head trauma

PEDIATRICS (ISSN Numbers: Print, 0031-4005; Online, 1098-4275).

HISTORY

The recognition of child abuse in modern medicine began in the 19th century, with the work of the French forensic physician Ambroise Tardieu,[1,2] who described a wide array of physical and sexual injuries to children, including meningeal hemorrhage and brain injuries in fatally abused infants. More than 80 years later, American physicians began describing the clinical and radiologic manifestations of child abuse. Pediatrician and radiologist John Caffey[3,4] first described the association of chronic subdural hemorrhages and long-bone fractures in 1946, but it was not until 1972 that he published a seminal paper describing the radiologic and clinical features attributed to shaking injuries. Ludwig and Warman[5] first published the term "shaken baby syndrome" in their review of 20 infants and young toddlers injured by shaking, none of whom showed evidence of impact injury to the head. In 1987, Duhaime et al[6] reported that victims of fatal shaken baby syndrome, and many of those who survived their trauma, showed evidence of blunt impact to the head at the time of diagnosis. The importance of impact in acceleration/deceleration injury was supported by their basic biomechanical models, and they concluded that most serious abusive head injuries required an impact to the head. The relative importance of impact as a contributor to the head injury sustained by abused children became a source of controversy. Biomechanical modeling has since been used to both support and refute the contributions of shaking or impact to abusive head trauma (AHT).[7,8] In reality, all models and theories have known limitations, and many clinicians and researchers acknowledge that precise mechanisms for all abusive injuries remain incompletely understood.[9] Efforts to better understand the mechanisms and causations of injury have improved the gathering of objective data in the clinical realm. Case investigations, including meticulous medical history taking, examinations, and medical workups, have expanded and improved. Medical diseases that can mimic the presentation of AHT are recognized, and screening is performed when indicated. Social welfare, law enforcement, and legal professionals have become better educated about AHT. Clinical research has expanded, and biomechanical modeling of injuries has improved.

Case histories clearly support the conclusion that shaking occurs in some injury scenarios. Shaking was the most commonly reported mechanism of injury described in a series of AHT cases in which perpetrators admitted abuse (68% of 81 cases).[10] Shaking alone was described in 32 cases, and only 4 of the victims showed evidence of impact injury. Although this indicates incomplete admission to the injury mechanism in some cases, the commonality of a described shaking mechanism along with the infrequency of impact evidence supports shaking as an important mechanism of AHT. In addition, blunt impact trauma or impact combined with shaking can result in infant head injuries.[11] In severe and fatal cases, concomitant cervical spine injury can sometimes be found.[12] Secondary brain injury resulting from hypoxia, ischemia, and metabolic cascades contributes to poor outcomes.[13,14] Shaken baby

EXHIBIT 5

syndrome is a subset of AHT. Injuries induced by shaking and those caused by blunt trauma have the potential to result in death or permanent neurologic disability, including static encephalopathy, mental retardation, cerebral palsy, cortical blindness, seizure disorders, and learning disabilities. Medical and biomechanical research, clinical and pathologic experience, and radiologic advances have improved our understanding of the range of mechanisms that contribute to brain injury from AHT, yet controversy remains.

DISCUSSION

Few pediatric diagnoses engender as much debate as AHT, in part because of the social and legal consequences of the diagnosis. The diagnosis can result in children being removed from their homes, parents losing their parental rights, and adults being imprisoned for their actions. Controversy is fueled because the mechanisms and resultant injuries of accidental and abusive head injury overlap, the abuse is rarely witnessed, an accurate history of trauma is rarely offered by the perpetrator, there is no single or simple test to determine the accuracy of the diagnosis, and the legal consequences of the diagnosis can be so significant.[15] Because the civil and criminal justice systems are often involved in cases of AHT, the scientific debates related to mechanism and causation of injury often are argued during courtroom proceedings. On occasion, the courtroom allows for scientific theory to be confirmed or refuted,[16] but in reality, the American justice system is not designed to determine scientific truth but, rather, to balance contested facts and bring closure to a dispute. Medical terminology should accurately reflect the medical diagnosis. The term "shaken baby syndrome" has become synonymous in public discourse with AHT in all its forms.[17] The term is sometimes used inaccurately to describe infants with impact injury alone or with multiple mechanisms of head and brain injury and focuses on a specific mechanism of injury rather than the abusive event that was perpetrated against a helpless victim. Legal challenges to the term "shaken baby syndrome" can distract from the more important questions of accountability of the perpetrator and/or the safety of the victim. The goal of this policy statement is not to detract from shaking as a mechanism of AHT but to broaden the terminology to account for the multitude of primary and secondary injuries that result from AHT, some of which contribute to the often-permanent and significant brain damage sustained by abused infants and children.

The term "shaken baby syndrome" has become recognized by the public; prevention strategies for curtailing the incidence of AHT have been developed and researched, and some states have mandated shaken baby syndrome education for parents of all newborn infants.[18] Because it may not be obvious to parents that shaking can be harmful to infants, the newborn nursery is an appropriate venue for this education. The American Academy of Pediatrics supports prevention efforts that reduce the frequency of AHT and recognizes the utility of maintaining the use of the term "shaken baby syndrome" for prevention efforts. Just as the public commonly uses the term "heart attack" and not "myocardial infarction," the term "shaken baby syndrome" has its place in the popular vernacular. However, for medical purposes, the American Academy of Pediatrics recommends adoption of the term "abusive head trauma" as the diagnosis used in the medical chart to describe the constellation of cerebral, spinal, and cranial injuries that result from inflicted head injury to infants and young children.

THE ROLE OF THE PEDIATRICIAN

As mandated reporters of suspected child abuse and neglect, pediatricians carry the burden of recognizing and responding to medical manifestations of AHT. The diagnosis is sometimes obvious, but injuries in many symptomatic infants are unrecognized by unsuspecting physicians.[19] In addition, physicians do not always report to child welfare agencies injuries that are highly suspicious for abuse, which puts children at further risk for injury.[20,21] To protect abused infants and prevent future severe neurologic injury, pediatricians must remain cognizant of the possibility of AHT in infants who present with both subtle and overt neurologic symptoms and take seriously the ethical and legal mandates to report suspected child abuse to governmental agencies for investigation. Pediatricians also have a responsibility to consider alternative hypotheses when presented with a patient with findings suggestive of AHT. A medical diagnosis of AHT is made only after consideration of all the clinical data. On some occasions, the diagnosis is apparent early in the course of the evaluation, because some infants and children have injuries to multiple organ systems that could only be the result of inflicted trauma. On other occasions, the diagnosis is less certain, and restraint is required until the medical evaluation has been completed. However, as physicians, we have an obligation to make a working diagnosis, as we do with many other diagnoses, and take the legally mandated steps for further investigation when indicated. Pediatricians often find it helpful to consult a subspecialist in the field of child abuse pediatrics to ensure that the medical evaluation has been complete and the diagnosis is accurate. Subspecialists in radiology, ophthalmology, neurosurgery, neurology, and other fields should also be consulted when necessary to ensure a complete and accurate evaluation. When child protective services or law enforcement is involved in an investigation, the pediatrician is required to interpret medical information for nonmedical professionals in an understandable manner that accurately reflects the medical data. Pediatricians also have a responsibility to the family of the abused child. The diagnosis of child abuse has enormous social, psychological, and legal implications for families. The role of the pediatrician is not to apportion blame or investigate potential criminal activity but to identify the medical problem, treat the child's injuries, and offer honest medical information to parents and families. Finally, pediatricians can work to prevent AHT by supporting prevention efforts in the community and in practice. Pediatricians may help prevent AHT by providing anticipatory guidance to new parents about the dangers of shaking or impact and providing methods for dealing with the frustration of a crying infant. They can also stress the importance of leaving a young infant or toddler in the care of adults whom the parents trust will not harm their child and can participate in comprehensive community-based prevention efforts. AHT commonly results in permanent neurologic damage and carries tremendous family and societal costs. With an aim toward prevention, the American Academy of Pediatrics recommends the following.

RECOMMENDATIONS

1. Pediatricians should be alert to the signs, symptoms, and head injury patterns associated with AHT.
2. Pediatricians should know how to begin a thorough and objective medical evaluation of infants and children who present for medical care with signs and symptoms of potential AHT. Consultants in radiology, ophthalmology, neurosurgery, and other subspecialties are important partners in the medical evaluation and can assist in interpreting data and reaching a diagnosis.
3. Pediatricians should consider consulting a subspecialist in the field of child abuse pediatrics to ensure that the medical evaluation of the patient has been complete and that the diagnosis is accurate.
4. Pediatricians should use the term "abusive head trauma" rather than a term that implies a single injury mechanism, such as shaken baby syndrome, in their diagnosis and medical communications.
5. Pediatricians should continue to educate parents and caregivers about safe approaches to calming and coping with crying infants and the dangers of shaking, striking, or impacting an infant's head.

COMMITTEE ON CHILD ABUSE AND NEGLECT, 2008–2009

Carole Jenny, MD, MBA, Chairperson
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Emalee Flaherty, MD
Roberta A. Hibbard, MD
Rich Kaplan, MD, Section Member
*Robert Block, MD, Past Chairperson

LIAISONS

David L. Corwin, MD
American Academy of Child and Adolescent Psychiatry
Janet Saul, PhD
Centers for Disease Control and Prevention

STAFF

Tammy Piazza Hurley

*Lead authors

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AMERICAN ACADEMY OF OPHTHALMOLOGY
The Eye M.D. Association

[redacted]

INFORMATION STATEMENT

Abusive Head Trauma/Shaken Baby Syndrome
June 2010

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The following physicians, state that they have no significant financial or other relationship with the manufacturer of any commercial product or provider of any commercial service discussed in the material he contributed to this publication or with the manufacturer or provider of any competing product or service:

Alex V. Levin MD, MHSc
Brian Forbes, MD, PhD
Randell Alexander, MD, PhD
Carole Jenny, MD, MBA

Individual Disclosures:
The following disclosures have been reviewed and deemed not to present a bias or conflict of interest with the material that follows.

Gil Binenbaum, MD, MSCE: (S) National Eye Institute

C = consultant fee, paid advisory boards, or fees for attending a meeting
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Abusive Head Trauma/Shaken Baby Syndrome

INFORMATION STATEMENT

Abusive Head Trauma/Shaken Baby Syndrome
May 2010

Although most eye injuries in childhood are a result of accidents, a significant portion result from physical abuse by adults. Child abuse, including physical abuse, sexual abuse, neglect, and emotional abuse, is a pervasive problem in our society, with an estimated 1,256,600 million confirmed victims per year in the United States alone (http://www.acf.hhs.gov/programs/opre/abuse_neglect/natl_incid/index.html). Physically abusive behavior by a parent or other caregiver usually reflects temporary loss of control during a period of anger or stress rather than premeditated cruelty. Lack of knowledge of normal child development and the proper way to care for or discipline a child are also frequent contributing factors.

A reliable history is often difficult to obtain when child abuse has occurred. Suspicion should be aroused when repeated accounts of the circumstances of injury or histories obtained from different individuals are inconsistent or when the events described seem to conflict with the extent of injuries (e.g., bruises on multiple aspects of the head after a fall) or with the child's developmental level (e.g., a 2 month old rolling off a bed or a 9 month old climbing out of a high chair). Any physician who suspects that child abuse might have occurred is required by law in every US state and Canadian province to report the incident to a designated governmental agency. Once this obligation has been discharged, the ophthalmologist may be an important contributor to understanding the case but probably is best advised to leave the full investigation of the situation to appropriate specialists or other authorities.

The presenting sign of child abuse involves the eyes in approximately 5% of cases, and ocular manifestations are detected in the course of evaluating many others. Blunt trauma inflicted with fingers, fists, or implements such as belts or straps is the usual mechanism of nonaccidental injury to the ocular adnexa or anterior segment. Periorbital ecchymosis, subconjunctival hemorrhage, and hyphema should raise suspicion of recent abuse if the explanation provided is less than completely plausible, particularly in infants. Cataract or lens dislocation may be signs of injury or trauma inflicted in the past. A majority of rhegmatogenous retinal detachments that occur in childhood have a traumatic origin; abuse should be suspected when such a finding is encountered in a child without a history of injury or an apparent predisposing factor such as high myopia. Certain signs of trauma include ruptured globe, orbital fracture, vitreous base detachment and retinal commotio. Trauma should also be considered in the differential diagnosis, of unilateral optic atrophy, hyphema, and periocular ecchymosis, in the absence of another clear explanation.

Shaken Baby Syndrome

Shaken Baby Syndrome is a subset of abusive head trauma characterized by repetitive acceleration-deceleration forces with or without blunt head impact resulting in a unique complex of ocular, intracranial, and sometimes other injuries, usually in infants. Because the essential features were identified in the early 1970s, it has become widely recognized as one of the most serious manifestations of child abuse.

Abusive Head Trauma/Shaken Baby Syndrome

Victims of shaking injury are almost always under 5 years old and usually under 12 months old. When a reliable history is available, it typically involves a parent or other caregiver who shook an inconsolably crying baby out of anger and frustration. Often, however, the only information provided is that the child's mental status deteriorated or that seizures or respiratory difficulty developed. The involved caregiver may relate that an episode of relatively minor trauma occurred, such as a fall from a bed. Even without a supporting history, the diagnosis of Shaken Baby syndrome can still be made with confidence on the basis of characteristic clinical findings in the absence of a valid history or of an identified pathologic process that could present with similar signs and symptoms, such as a metabolic disease or clotting disorder. Such alternative diagnoses are usually readily identifiable by history, laboratory/radiologic testing, and clinical examination, including the appearance of the retina. Answers to important questions concerning the timing and circumstances of injury and the identity of the perpetrator sometimes cannot be inferred from medical evidence alone.

The infant's head is particularly vulnerable to the effects of repeated acceleration-deceleration because of its relatively large mass in relation to the body and poor stabilization by neck muscles. Intracranial injury in shaken infants frequently includes subdural hematoma, unilateral or bilateral over the cerebral convexities, or in the interhemispheric fissure. Displacement of the brain in relation to the skull and dura mater ruptures bridging vessels, leading to subdural hemorrhages. Evidence of subarachnoid bleeding may often be apparent. Although on initial scans the brain can be normal, in many cases cerebral parenchymal damage is manifest on neuroimaging, acutely as edema, ischemia, or contusion, and in later stages as atrophy. Repetitive stresses and strains within the brain cause direct brain damage, which is magnified by subsequent cerebral edema hypoxic-ischemic damage. Some authorities, citing the frequency with which shaken baby syndrome victims also show evidence of having received blows to the head, think that impact is an essential component of this syndrome, although in many cases, no sign of impact is found.

Ocular Involvement

The most common ocular manifestation of shaking injury, present in approximately 85% of cases, are retinal hemorrhages. The absence of retinal hemorrhage does not rule out abuse. Rarely, retinal hemorrhage can occur without intracranial hemorrhage. Preretinal, nerve fiber layer, deeper intraretinal (dot and blot), or subretinal localization may be seen. Hemorrhages tend to be concentrated in or near the posterior pole, but frequently are so extensive that they occupy nearly the entire fundus. Retinal hemorrhages in shaken infants resolve over a period ranging from several days, to 1-2 weeks, rarely to several months, depending upon location and severity. For example, superficial flame hemorrhages often resolve quickly, whereas preretinal and subretinal hemorrhage take longer to resolve. Retinal hemorrhages cannot be dated with any precision. Vitreous hemorrhage can also develop, usually as a secondary phenomenon resulting from migration of blood that was initially intraretinal or preretinal. Occasionally, the vitreous becomes almost completely opacified by dispersed hemorrhage within a few days of injury. A vitrectomy should be considered if a vitreous hemorrhage does not clear as the development of amblyopia or other complication such as myopia are likely to result.

Some eyes of shaken infants show evidence of retinal tissue disruption in addition to hemorrhage. Full-thickness perimacular folds in the neurosensory retina, typically with circumferential orientation around the macula that creates a crater-like appearance, are highly characteristic. Splitting of the retina (traumatic retinoschisis), either deep to the nerve fiber layer or superficial (involving only the internal limiting membrane), creates partially blood-filled cavities of considerable extent, usually in the macular region. Full-thickness retinal breaks and detachment are rare. Schisis cavities usually flatten out within a few weeks of injury, but can persist indefinitely. Although similar findings have been reported rarely in fatal crush injuries and fatal motor vehicle accidents, such histories are readily apparent and would allow rapid identification.

A striking feature of Shaken Baby syndrome is the frequent lack of external evidence of trauma. The ocular adnexa and anterior segments appear entirely normal. Occasionally, the trunk or extremities show bruises representing the imprint of the perpetrator's hands. In some cases, rib fractures or characteristic metaphyseal fractures of the long bones result from forces generated during grasping of a limb or the perpetrator's hands encircling the thorax during the application of acceleration-deceleration forces. It must be kept in mind, however, that many shaken babies are also victims of other forms of abuse. Signs of impact to the head must be carefully sought, abdominal trauma specifically considered, and a radiologic skeletal survey performed in all cases of suspected abusive head trauma in this age range.

When extensive retinal hemorrhage accompanied by perimacular folds and schisis cavities are found in association with intracranial hemorrhage or other evidence of trauma to the brain in an infant without another clear explanation, abusive head trauma can be diagnosed with confidence regardless of other circumstances. Retinal hemorrhages without other ocular findings strongly suggests that intracranial injury has been caused by shaking, but alternative possibilities such as a coagulation disorder, normal birth hemorrhages, fulminant meningitis, and leukemia should be considered as well. Severe accidental head trauma (e.g. sustained in a fall from a second-story level or a motor vehicle collision) is infrequently accompanied by retinal hemorrhage, which is rarely extensive and usually confined to the posterior pole. Retinal hemorrhage is rare and has never been documented to be extensive following cardiopulmonary resuscitation by trained personnel. Terson syndrome appears to be rare in childhood and not likely to be associated with extensive multilayered retinal hemorrhage. Likewise, increased intracranial pressure or hypoxia are not known to be associated with extensive retinal hemorrhage. Papilledema is uncommon in Shaken Baby syndrome. Retinal hemorrhages resulting from birth trauma are common in newborns but do not persist beyond 1 month of age unless the hemorrhages are preretinal, subretinal or in the vitreous.

Currently, there is abundant evidence from multiple sources (perpetrator confessions, clinical studies, postmortem studies, mechanical models, animal models and finite element analysis) that repetitive acceleration-deceleration with or without head impact is injurious, and the primary cause of retinal hemorrhage in victims of *Shaken Baby synd*rome is vitreo-retinal traction. The well formed vitreous of infants and young children is very firmly attached to retinal blood vessels, the peripheral retina and the macula. Other factors such as increased intrathoracic pressure, increased intracranial pressure, brain trauma induced coagulopathy, hypoxia and anemia may have some role in modulating the appearance of retinal hemorrhage but are not likely to play a major pathogenic role, especially for extensive hemorrhage or schisis. Increased intracranial pressure, which can have many causes, does not lead to extensive retinal hemorrhages in the absence of abuse.

Abusive Head Trauma/Shaken Baby Syndrome

Ophthalmologists should be prepared to promptly respond to requests for consultation in cases of suspected abusive head injury. Ophthalmology consultations are appropriate when there is suspicion of abusive head injury based on other relevant findings or a history of witnessed events that might result in such injury. In addition, consultation may be appropriate for children who experience sudden unexplained life threatening events, including seizures and apnea. Even within the first 24 hours after injury hemorrhages may start to resolve or even worsen to some degree. Although photodocumentation is not required, it may later aid in providing recollection of the findings. Clinical notes that carefully detail (with or without hand drawn figures) the number, extent, pattern, type, and laterality of all ocular findings are essential. Findings should be communicated with the responsible pediatric child abuse teams that are found in many institutions. In the absence of such support, the ophthalmologist must honor their legal obligation to report a suspicion of child abuse via appropriate state or provincial reporting pathways. Protocols have been published for postmortem examination of the eyes and orbital tissues.

Prognosis

Up to one third of children with head trauma die from their injuries. Poor visual and pupillary response have been correlated with a higher risk of mortality. Survivors often suffer permanent impairment, ranging from severe retardation and quadriparesis to mild learning disability and motor disturbances. The most common cause of visual loss is cortical injury followed by optic atrophy. Dense vitreous hemorrhage, usually associated with deep traumatic retinoschisis, carries a poor prognosis for both vision and life.

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Alex V. Levin MD, MHSc, Wills Eye Institute, Philadelphia, PA
Randell Alexander, MD, PhD, University of Florida-Jacksonville, Jacksonville, FL
Gil Binenbaum, MD, MSCE, Children's Hospital of Philadelphia/University of Pennsylvania School of Medicine, Philadelphia, PA
Brian Forbes, MD, Ph.D, Children's Hospital of Philadelphia/ University of Pennsylvania School of Medicine, Philadelphia, Pennsylvania
Carole Jenny, MD, MBA, Warren Alpert Medical School of Brown University, Providence, Rhode Island

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Position Paper on Fatal Abusive Head Injuries in Infants and Young Children

Mary E. Case, M.D., Michael A. Graham, M.D., Tracey Corey Handy, M.D., Jeffrey M. Jentzen, M.D., and James A. Monteleone, M.D.: the National Association of Medical Examiners Ad Hoc Committee on Shaken Baby Syndrome

This article represents the work of the National Association of Medical Examiners Ad Hoc Committee on shaken baby syndrome. Abusive head injuries include injuries caused by shaking as well as impact to the head, either by directly striking the head or by causing the head to strike another object or surface. Because of anatomic and developmental differences in the brain and skull of the young child, the mechanisms and types of injuries that affect the head differ from those that affect the older child or adult. The mechanism of injury produced by inflicted head injuries in these children is most often rotational movement of the brain within the cranial cavity. Rotational movement of the brain damages the nervous system by creating shearing forces, which cause diffuse axonal injury with disruption of axons and tearing of bridging veins, which causes subdural and subarachnoid hemorrhages, and is very commonly associated with retinal schisis and hemorrhages. Recognition of this mechanism of injury may be helpful in severe acute rotational brain injuries because it facilitates understanding of such clinical features as the decrease in the level of consciousness and respiratory distress seen in these injured children. The pathologic findings of subdural hemorrhage, subarachnoid hemorrhage, and retinal hemorrhages are offered as "markers" to assist in the recognition of the presence of shearing brain injury in young children.

Key Words: Abusive head injury—Shaken baby syndrome—Head injury—Inflicted injuries.

The original charge to this ad hoc committee was to produce a position paper on shaken baby syndrome. This terminology was taken by the committee to refer generally to the area of abusive head injury in young children. However, because the term *shaken baby syndrome* has taken on such controversy, this article will address the topic of abusive head injury in young children. In several areas of this article, the term *marker* is used when describing the importance of identifying the presence of subdural, subarachnoid, and retinal hemorrhages. The term *marker* indicates a grossly observable sign to signify the possible existence of diffuse axonal injury that is not grossly evident. Use of the term *marker* does not imply that such hemorrhages cannot exist without such an association but is intended to remind us to be alert to the possibility.

Head injuries account for up to 80% of fatal child abuse injuries at the youngest ages (1). Blunt force impact as well as vigorous shaking may play a role in the pathogenesis of these injuries (2). This article describes the state of knowledge concerning the pathogenesis, clinical features, and pathologic changes of fatal abusive head injuries in young children. The intent is to inform the practicing pathologist about the proper recognition, interpretation, and clinical correlation of these injuries.

Caffey's description of whiplash shaking of infants in the early 1970s introduced the concept that serious and even fatal head injury could be inflicted by a caretaker through shaking (3,4). Caffey described injuries characterized by subdural and/or subarachnoid hemorrhages, brain swelling, and reti-

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From the Department of Pathology, Division of Forensic Pathology, (M.E.C., M.A.G.) and Department of Pediatrics (J.A.M.), St. Louis University Health Sciences Center, St. Louis, Missouri; Office of the Chief Medical Examiner of the State of Kentucky (T.C.H.); and Office of the Medical Examiner of Milwaukee County, Milwaukee, Wisconsin (J.M.J.), U.S.A.

Address correspondence and reprint requests to Mary Case, M.D., Saint Louis University, Health Science Center, 1402 South Grand Blvd., St. Louis, MO 63104-8298, U.S.A.

Editor's note: The Board of Directors of the National Association of Medical Examiners charged the authors of this article with writing a position paper on the shaken baby syndrome. This article was the result. The manuscript was reviewed by three reviewers on the Board of Editors of the *American Journal of Forensic Medicine and Pathology*. They believed that while it was worthy of publication, it should not be published as a position paper because of the controversial nature of the subject. The Board of Directors responded to this opinion by stating that position papers always deal with controversial subjects.

nal hemorrhage without injuries that would indicate impact, such as facial bruises, scalp bruises, or skull fractures. Since that time, experts in many scientific fields have investigated whether such apparently innocent practices as tossing a baby into the air and other playful maneuvers might cause brain damage by a similar shaking mechanism. Currently, it is generally accepted that such playful practices do not result in injuries to the young child's brain. The type of shaking that is thought to result in significant brain injury involves holding the child by the thorax or an extremity and violently shaking the child back and forth, causing the head to forcefully whiplash forward and backward with repeated accelerations and decelerations in each direction.

Abusive head injury commonly occurs in response to prolonged crying and often is inflicted by a caregiver with limited patience or experience in handling a child. Some individuals who admitted to shaking children as a mechanism of injury have stated that shaking would stop the babies from crying. These assailants have actually used this practice to stop babies from crying on previous occasions without any visible adverse result.

When shaking is the mechanism of injury, the arms and legs of the child may also be violently flung about during the shaking, causing injuries to the long bones. Most frequently affected are the tibia, distal femora, and proximal humeri. These lesions were originally described as bucket-handle and corner fractures and were thought to be caused by the indirect forces of the shaking. These injuries are currently recognized as fractures through the most immature portion of the metaphyseal primary spongiosa and appear radiographically as separation of portions (corner) or of the entire disk (bucket handle) of metaphyseal bone, depending on the radiographic plane (5,6). Many babies with fatal abusive head injuries do not demonstrate any external injury, although in about 25% to 50% of cases, such injuries are evident on external examination (7–10). It is important that a careful search is made to identify any injury to the body, such as a bruise or abrasion. Grasping the child by the arms or thorax may result in bruises in these locations, but children may be grasped in this manner without leaving bruises. Likewise, ribs may be fractured while grasping the child around the thorax. At autopsy, close attention should be directed to the rib cage. Fractured ribs should be removed, decalcified, and examined microscopically. Posterior rib fractures are highly specific for abuse. Ribs are the most common bones fractured in association with other abusive injuries of children who die of fatal child abuse (11,12). Infants may sustain abusive head injury of less than fatal outcome and may sustain injuries to the brain that will later be reflected in degrees of mental retardation or slowness, learning disorders, seizures, blindness, or irritability. Of infants who receive abusive head injuries, approximately 7% to 30% die; 30% to 50% have significant cognitive or neurologic deficits; and 30% have a chance of full recovery (13–15). Lethal abusive head injury is not confined to infants. Children as old as 4 or 5 years can be fatally head injured by abuse, although the great majority are under 2 years of age, and most are under 12 months of age (16). Adults may also sustain head injuries by shaking, with findings identical to those found in shaken infants. Pounder described a slightly built, short, 30-year-old prisoner who was grasped by the shoulders and violently shaken. When he died 3 days later, his autopsy demonstrated subdural, subarachnoid, and retinal hemorrhages with diffuse axonal injury (17).

MECHANISMS OF INJURY

Appreciation of the unique characteristics of young children's head injuries requires an understanding of the developmental differences in the skull, brain, and neck before the age of about 4 years. Injuries to the young child's brain are unique in that the trauma occurs to an organ that is in the process of maturing; the mechanisms, the thresholds of injury, and the types of injuries differ from those that affect the older child or adult. The primary features unique to the young child include the thinness and pliability of the skull; the rapid growth of the brain and skull, resulting in a large heavy head; the softness of the brain, which is composed primarily of neurons without dendritic connections; the paucity of the myelin sheath of axons; the relative flatness of the skull base; the undeveloped neck muscles; and the subarachnoid space, which is large in its extent but shallow in depth (18,19). Because of the unique characteristics of the developing skull and brain, children under the age of 4 or 5 years are particularly vulnerable to a type of brain injury that is best described as shearing injury. Shearing injury implies a distortion of the brain shape that elongates it in an anterior–posterior dimension with resulting shifting apart of adjoining brain structures. Impact to the immature brain is more likely to produce shearing injury rather than the typical brain contusions that might occur in older children and adults (19). Impact force is more effectively transferred through the thin pliant skull and across the large and shallow subarachnoid space of a young child's head. The paucity of myelination, the large number of neurons without glial or dendritic connections, and the small axonal size predispose the young brain to shearing injury by creating a soft consistency. The large heavy head mounted on the weak neck of the young child produces instability of the head, which allows greater movement of the head and brain when acted on by acceleration–deceleration forces. Last, the shallow skull base allows the young child's brain to rotate more readily in response to head acceleration or deceleration than occurs after the skull base has developed more prominent bony ridges and concavities (7,19,20). The mechanical forces that are operative in head injury are primarily translational and rotational (angular). Translational forces produce linear movement of the brain, a type of movement that is quite benign (21,22). The trivial falls that children sustain in falling from furniture and even down stairs primarily involve translational forces. Although such falls may occasionally result in a skull fracture, these incidents are generally very benign and do not result in loss of consciousness, neurologic deficit, or death (23–26). Rotational forces are generated by either impact or nonimpact inertial mechanisms, such as whiplash shaking, which produce sudden acceleration or deceleration of the head. Rotational forces applied to the head cause the brain to turn abruptly on its central axis or its attachment at the brainstem–cerebral junction. Extensive clinical and experimental data have demonstrated that such rotational movements of the brain result in a type of injury referred to as *shearing injury* or *diffuse axonal injury* (23–29). To keep pace with the accelerating skull responding to rotational forces, the brain moves within the cranial cavity, and because of the nonuniformity and different consistencies of the brain structures, strains occur at the junctures between cortex and subcortical white matter, white matter and deep gray matter, and lateral extensions across the midline of the brain (corpus callosum and superior and middle cerebellar peduncles). Disruptions of the axonal processes occur at these junctures as the peripheral parts of the brain rotate farther or more rapidly than the inner, deeper, more relatively fixed parts of the brain. Lesser forces disrupt the most external junctures, whereas greater forces disrupt deeper junctures down to the deep gray matter of the basal ganglia, thalamus, and finally the rostral brainstem. In young children, either an impact or a shaking mechanism may result in diffuse axonal injury, when significant, because both impart rotation to the brain (7,19,20). After the age of 4 or 5 years, the most common cause of diffuse axonal injury is the motor vehicle crash.

Some experimental evidence suggests that shaking alone may not be sufficient to produce the angular acceleration necessary to create fatal shear injury (7). The particular model used in the latter study to determine how much force could be transmitted by shaking to an infant's head utilized as a model a doll with a rubber neck, and the force considered necessary to produce shear injury was derived from studies of adult primates. Neither of these experimental circumstances necessarily truly resembles the immature human infant skull and brain (29). In favor of shaking as a possible mechanism, many forensic pathologists have experience based on confessions by perpetrators or witnesses of how these injuries were inflicted, as well as on autopsies in which no impact site is found on the scalp or skull. However, this experience must be received with some caution. Perpetrators may not remember, or later may not be willing, to fully describe their actions. Children may be violently shaken, then forcefully thrown rather than just placed down gently. Impacts may not be reflected on the scalp if the striking surface is padded or if it is broad and firm. A child's scalp is very elastic and stretches on impact. Not all impacts are registered as hemorrhage in the galea. In the vast majority of cases, it is not possible to definitely characterize children's head injuries as being caused by either pure impact or pure shaking because the pathologic changes in the brain are identical in cases in which either of these two mechanisms has been suggested (7). If there are focal injuries, such as skull fractures, scalp bruises, or subgaleal hemorrhage, an impact can be assumed, but coexistent shaking cannot be excluded. In the absence of signs of an impact, however, shaking alone should not be presumed because there may well have been an impact that cannot be identified (30). Subarachnoid and subdural hemorrhages should be appreciated as markers of brain displacement by angular force and the possibility of accompanying diffuse axonal injury (24,31). In young children, both impact and shaking produce these pathologic findings, which should be appreciated as markers for the underlying problem in the brain: the diffuse axonal injury (32).

SUBDURAL HEMORRHAGE

The grossly and microscopically identifiable pathologic changes in young children's rotation or acceleration–deceleration head injuries include subdural hemorrhage, subarachnoid hemorrhage, and retinal lesions, including hemorrhages and schisis. Subdural hemorrhage results from tearing of bridging veins, which extend from the cortical surface to the dural venous sinuses (Fig. 1). These rather transparent veins tear when they are stretched as the brain moves within the subdural space of the cranial cavity (23,33–35). Subdural hemorrhages occur most frequently over the convex cerebral surfaces, especially posteriorly within the interhemispheric sulcus, and may be either unilateral or bilateral, although more commonly they are bilateral (Fig. 2). If the subdural hemorrhage is associated with a skull fracture, it need not be on the same side as the fracture. Subdural hemorrhage is probably uniformly present in cases of shearing injury but is evident at autopsy in about 90% to 98% of cases (7,36). Small amounts of interhemispheric blood that can be detected by computed tomography (CT) may not be seen at autopsy (5,37). Very thin layers of subdural blood over the cerebral convexities may not be visible on CT but can be found at autopsy. Magnetic resonance imaging (MRI) is able to detect a much greater number of subdural hemorrhages than can CT; however, many critically ill young children are not able to undergo MRI (19). As a result, studies that report data on nonfatal cases of abusive head injury find that about 80% to 85% of patients have subdural hemorrhage (38). At autopsy, the subdural hemorrhage may consist of only 2 to 3 ml of blood and may not be observed if the prosector does not personally inspect the subdural space as the calvarium is being removed (Fig. 3). Extreme caution should be taken to not misinterpret as premortem subdural hemorrhage the blood draining from the dural sinuses when these are incised at autopsy. The importance of subdural hemorrhage is typically not that of a space-occupying mass lesion producing increased intracranial pressure and the consequences of tentorial herniation, although some hemorrhages are large enough to bring about these complications. Rather, it is important as a marker of brain movement within the cranial cavity and may accompany shearing injury. Subdural bleeding may continue and accumulate to some extent if the child experiences postinjury survival. At autopsy, large subdural hemorrhages resulting in part from postinjury accumulation have been observed. Even a small amount of subdural hemorrhage indicates that brain displacement has been produced, which may have caused some shearing brain injury.

FIG. 1. Thirteen-week-old infant with normal brain and intracranial spaces demonstrating bridging vein (arrow) arising from left cerebral convexity (right).

FIG. 2. Nine-month-old infant with fracture of right parietal calvarium showing bilateral acute subdural hemorrhages over the cerebral convexities.

SUBARACHNOID HEMORRHAGE

Subarachnoid hemorrhage occurs in patches over the cerebrum, especially over the parasagittal cerebral convexities (Fig. 4). It is present in virtually all

FIG. 3. Eleven-week-old infant with small amounts of acute subdural hemorrhage over both cerebral convexities.

fatal cases, although it may be very small and difficult to identify, especially on the interhemispheric surfaces (Fig. 5). Subarachnoid hemorrhage arises from tearing of arachnoid vessels at the same time bridging veins are torn, because the bridging veins are surrounded by an arachnoid sheath as they cross the subdural space to enter the inner dural layer and finally the dural sinuses. Tearing of bridging veins usually produces both subdural and subarachnoid hemorrhages.

RETINAL HEMORRHAGES

Retinal lesions are observed in 70% to 85% of young children with severe rotational brain injuries (32,39). Currently, their pathogenesis is not precisely understood. Their presence highly correlates with rotational head injury, and they are greatly overrepresented among cases of nonaccidental trauma in young children. Possible mechanisms to account for retinal hemorrhages include increased pressure transmitted to the central retinal vein from increased intrathoracic or intracranial pressure, direct trauma to the retina from being struck by the vitreous moving within the eye, and traction on the retina by the movement of the vitreous pulling away from the retina. The retinal hemorrhages seen in abusive head injuries are similar to those that are frequently observed in full-term neonates after vaginal delivery. In neonates, the hemorrhages appear to be consequent to increased intrathoracic or intracranial pressure from squeezing of the thorax during the passage through the birth canal. Most of the neonatal retinal hemorrhages completely resolve by 5 or 6 days, although a few persist longer (40,41). In children older than 30 days who have retinal hemorrhages, the great majority have abusive head injuries.

Ophthalmologic findings in abused children include peripheral retinal hemorrhages associated with retinal detachments, retinal tears, and large numbers of retinal hemorrhages (39). There may be other internal eye injuries in these children, consisting of vitreous bleeding and retinal folds. There is

FIG. 4. Seventeen-week-old infant with large (70–80 ml) acute subdural hemorrhage over right cerebral convexity demonstrating patches of subarachnoid hemorrhage over both parasagittal regions, greater on the right than on the left.

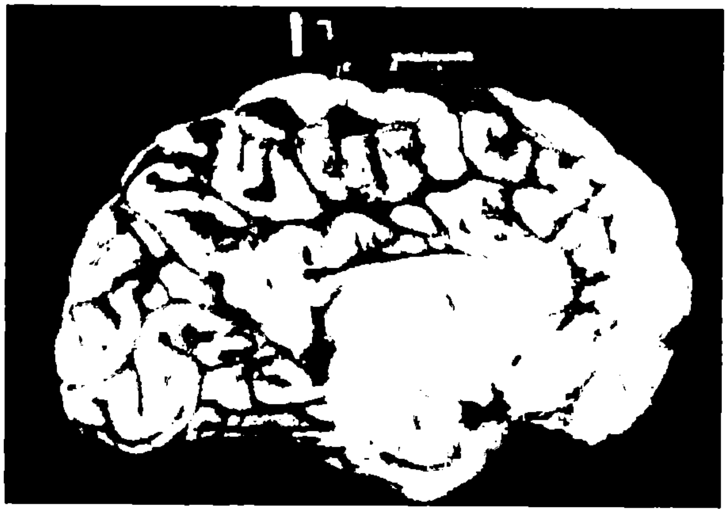

FIG. 5. Five-month-old infant with large bilateral acute subdural hemorrhages over cerebral convexities showing patches of subarachnoid hemorrhage on the mesial surface of the right cerebral hemisphere.

evidence that increasing severity of trauma to the head directly correlates to severe eye damage, beginning with subhyloid and intraretinal hemorrhages and progressing to retinal detachment and finally choroidal and vitreous hemorrhage (42). In children with very severe accidental head injury, (e.g., from a car accident), retinal hemorrhage is occasionally found (26). The retinal hemorrhages associated with nonaccidental head injuries tend to be bilateral, although they may be unilateral, multiple, and extensive and reach far into the periphery of the retina (39). Nontraumatic causes of retinal hemorrhages include bleeding disorders, sepsis, meningitis, vasculopathies, increased intracranial pressure, and, very rarely if ever, cardiopulmonary resuscitation (43–47). Retinal hemorrhages that occur in association with increased intracranial pressure are found at the posterior pole of the retina around the optic disc and are accompanied by papilledema (39).

Optic nerve hemorrhage is observed in association with inflicted head injuries in children but is not specific for those injuries. Optic nerve hemorrhage is hemorrhage in the perineural area. These hemorrhages are seen commonly whenever subdural hemorrhage is found in the cranial cavity, although there is not necessarily a direct connection between the subdural compartment of the orbital sheath and the subdural compartment of the intracranial cavity (42). Optic nerve hemorrhage can be seen in some cases of increased intracranial pressure that are not related to any form of trauma (48).

DIFFUSE BRAIN INJURY

Diffuse brain injury consists of tears of axonal processes and small blood vessels and, rarely, more extensive tissue tears (49,50). The areas of predilection are the corpus callosum; the subcortical white matter, especially of the superior frontal gyri; the periventricular areas; and the dorsolateral quadrants of the rostral brainstem. The axonal disruptions result in microscopic lesions that may be visible by light microscopy after 18 to 24 hours as retraction bulbs or varicosities. Retraction bulbs are accumulations of axoplasm, which appear on hematoxylin and eosin staining as pink bulbs. They are observed as the axoplasm of the disrupted axons accumulates at the damaged end and creates a bulbous enlargement. These axonal lesions are very difficult to see in young children because of the small size of the axonal processes. Immunohistochemical stains for β-amyloid precursor protein may allow demonstration of axonal injury as early after survival as 2 hours (51,52). The blood vessel tears of diffuse brain injury may be visible grossly as linear streaks or punctate hemorrhages, which vary from less than 1 mm up to many centimeters if bleeding continues for several days (31). However, these hemorrhages are very seldom seen in young children with diffuse brain injury because the blood vessels in young children are very elastic and do not readily tear even when adjacent axonal processes are torn.

In some (rare) cases of diffuse axonal injury in children under 1 year of age, parenchymal tears are

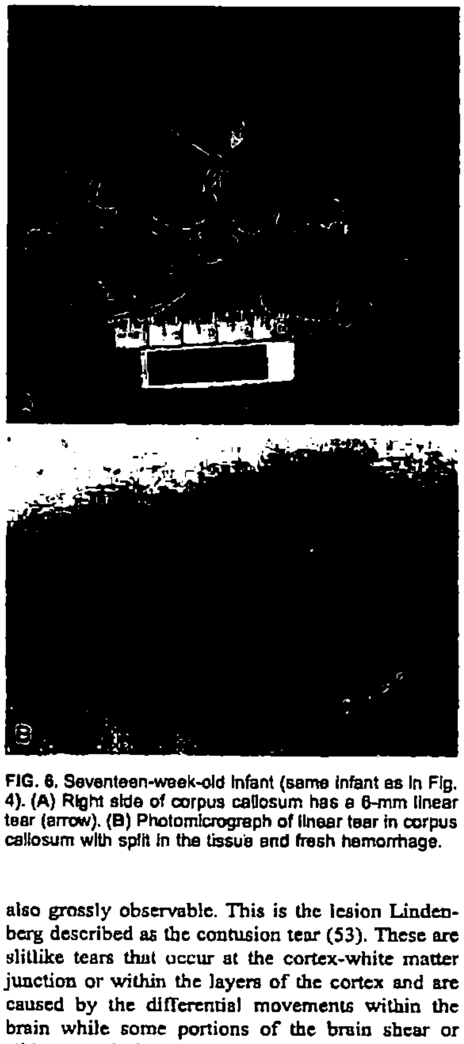

FIG. 6. Seventeen-week-old infant (same infant as in Fig. 4). (A) Right side of corpus callosum has a 6-mm linear tear (arrow). (B) Photomicrograph of linear tear in corpus callosum with split in the tissue and fresh hemorrhage.

also grossly observable. This is the lesion Lindenberg described as the contusion tear (53). These are slitlike tears that occur at the cortex-white matter junction or within the layers of the cortex and are caused by the differential movements within the brain while some portions of the brain shear or slide apart during differential rotation of the brain tissues (Figs. 6 and 7). Contusion tears are rare, and when seen they are accompanied by the usual markers of diffuse axonal injury, the subarachnoid and subdural hemorrhages in the usual locations (33). Care must be taken not to misinterpret a cross-section through the depth of a sulcus as a contusion tear. It is also prudent to take care to not mistake artifacts created by the process of handling or cutting the brain as true tears. Contusion tears should not be diagnosed on the basis of finding only a microscopic tear without other evidence of diffuse axonal injury.

It is not usually possible to morphologically establish the existence of diffuse axonal injury in young children by demonstrating the classic pathologic changes of retraction bulbs, tissue tears, or intraparenchymal hemorrhages, although these findings may be demonstrated on occasion (33,54). Many of these children die too soon after injury for these pathologic changes to be established. For this reason, it is important to appreciate the markers of shearing injury to identify these cases as diffuse axonal injury.

BRAIN SWELLING

Shearing injuries in young children are accompanied by various degrees of brain swelling. The swelling may not be apparent at autopsy in infants with brief survival intervals. Initially, CT may demonstrate progressive brain swelling and decreased ventricular size without other lesions being visible. The swelling is probably related both to direct injury to the axonal processes, causing localized edema, and to generalized swelling caused by changes in vascular permeability and autoregulation (5). Some investigators have postulated that hypoxia occurring when a child is shaken and becomes apneic accounts for the underlying cerebral insult and brain swelling (55). However, hypoxia does not explain why the injury is sometimes more unilateral than bilateral, the atrophy that develops in the brains in children who survive, or that the appearance of these brains at autopsy is not typical of hypoxic injury.

TIMING OF INJURIES

Timing of the head injury is often an important issue because most abusive injuries occur only in the presence of the individual who injured the child and who may not provide an accurate history. Studies in children dying of accidental head injuries indicate that children with diffuse injury show an immediate decrease in the level of consciousness (16,56). Studies in children with nonaccidental head injuries also indicate that they show an immediate decrease in their level of consciousness at injury (36). Individuals sustaining diffuse brain injury of moderate to severe degree become symptomatic immediately (24,31,49). Young children with moderate to severe degrees of diffuse brain injury would certainly include those in whom there is a significant neurologic outcome or death. Correlations of clinical and experimental observations on

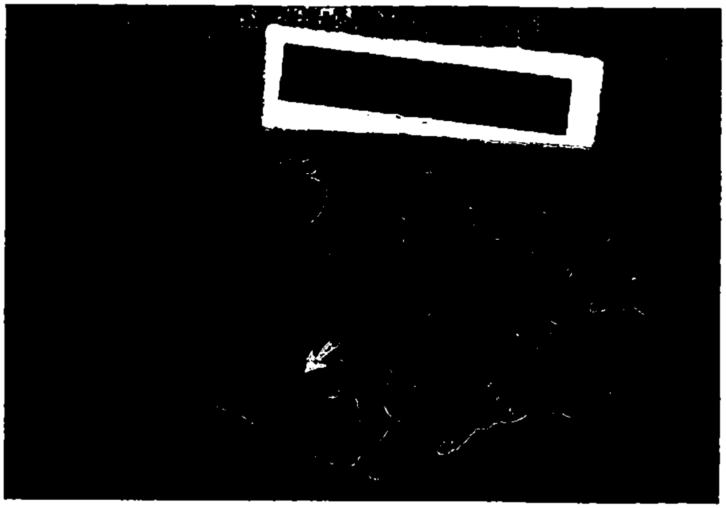

FIG. 7. Seven-month-old infant with depressed skull fracture of left parietooccipital calvarium with small acute subdural hemorrhage over the posterior aspects of both cerebral convexities demonstrating a 3- to 4-mm contusion tear (arrow) in left orbital subcortical white matter.

cerebral concussion and traumatic unconsciousness demonstrate that progressively deeper disconnections of axonal processes affecting the deep gray matter and rostral brainstem are the cause of the unconsciousness in these children, not that the unconsciousness is the result of increasing intracranial pressure or hypoxia (24). Symptoms experienced by these severely injured children include an immediate decrease in the level of consciousness (either lethargy or unconsciousness); respiratory irregularity, difficulty, or apnea; and frequently seizures. The respiratory difficulty in these children may be related to damage to the lower brainstem (medullary) centers of respiratory control. The timing with which respiratory difficulty develops is not very precise, but it is concurrent with or follows the decrease in level of consciousness in these children, who cannot survive for many hours without ventilatory support. It is not currently possible to predict the exact length of time such an injured child may survive.

Children who sustain repetitive episodes of mild diffuse brain injury may gradually accumulate brain damage and acquire neurologic deficiencies (14). The exact timing of such mild additive injuries is not possible. There is no reason to believe that remote shearing lesions would make the brain more susceptible to new shearing injury. Mild injuries unaccompanied by loss of consciousness are not usually brought to medical attention. Some mild shearing injuries are manifested as seizures and clinically present difficult diagnostic problems, because there is no currently available method to demonstrate the underlying pathologic changes of the shear injury until more severe degrees of diffuse axonal damage have been sustained and can be recognized by the markers of subdural or subarachnoid hemorrhage on CT or MRI (38).

CHRONIC SUBDURAL HEMATOMA

Rebleeding after trivial injury or spontaneous rebleeding from a preexisting chronic subdural hematoma should not be offered as an explanation for the presence of acute subdural blood lacking obvious demonstration of such an old subdural membrane (57). The pathogenesis of subdural bleeding has become better delineated after more than 20 years' experience with CT. The classic multilayered chronic subdural hematoma is currently considered a unique type of hemorrhage for several reasons. A chronic subdural hematoma very rarely follows severe head injury in a previously normal person, in whom an acute subdural hemorrhage transforms by aging to become a chronic subdural membrane. Instead, the blood of the acute subdural hemorrhage in these head injuries is readily resolved or rapidly organized (58–60). The resorption of subdural blood tends to be even more rapid and more complete in children than in adults (19). The development of the classic multilayered chronic subdural hematoma results from venous bleeding under low pressure and requires the potential for the subdural space to enlarge without a significant increase in pressure. The factors that promote such a development within a low-pressure intracranial space exist only in specific categories of people, such as those with brain atrophy (i.e., the elderly and those with alcoholism), those with hydrocephalus who have been treated by placement of a ventricular shunt, or those with traumatic encephalomalacia (59). In children with glutaric aciduria type 1, frontotemporal atrophy develops, and occasionally subdural hemorrhage without trauma develops on that basis (61). Minor trauma in these specific categories of patients may result in tearing of bridging veins and small amounts of subdural hemorrhage, which induces an ingrowth of granulation tissue from the dura. This granulation tissue contains fragile capillaries, which may produce microbleeds leading to enlargement of the hematoma. The further evolution of these hematomas is determined mostly by the nature of the vascular neomembrane formed in these patients. The expansion of these hematomas also appears to be related to the excessive activation of both the clotting and the fibrinolytic systems in the subdural fluid (62–64). A young child whose subdural hemorrhage subsequently organizes into a membrane composed of large vascular channels at risk for rebleeding would have been symptomatic before the time of rebleeding, because there would have been a preexisting brain abnormality. The signs and symptoms that would be expected before rebleeding include seizures, macrocephaly, anorexia, lethargy, headache, and apnea (60).

About 20% to 30% of asymptomatic neonates have small amounts of subarachnoid and subdural hemorrhage during delivery. The resolution of this blood may result in the presence of small numbers of dural macrophages containing hemosiderin and, sometimes, small fibrous patches consisting of a few layers of granulation tissue on the dura. These patches of thin membrane or scattered macrophages are not at risk of rebleeding with trivial trauma. Proof of rebleeding of a chronic subdural membrane should be based on the demonstration of a chronic subdural membrane that is grossly evident at autopsy, followed by microscopic confirmation of the vascularized membrane, and should not be based solely on the microscopic finding of fragments of fibrous tissue or a few macrophages containing hemosiderin.

The dura is a tough, fibrous, bilayered membrane overlying the arachnoid. It consists of an inner layer (menigeal) and an outer layer (periosteal). The periosteal layer serves as the periosteum of the inner table of the skull. The dura of young children, particularly along the basilar skull sutures, is a very cellular structure, which contains growing fibrous tissue along with numerous hematopoietic cells, including macrophages, many of which normally contain hemosiderin. The appearance of the normal young dura may be misinterpreted as having a thin chronic subdural membrane by microscopists who are not familiar with looking at these young duras, who may not be able to tell the inner from the outer dural surface, and who may incorrectly believe a chronic membrane to be the cause of acute subdural hemorrhage.

INTERPRETATION OF INJURY

The distinction between nonaccidental and accidental head injury in children is an area of concern for pathologists as well as other medical specialties. Fatal accidental shearing or diffuse brain injuries require such extremes of rotational force that they occur only in obvious incidents such as motor vehicle accidents. Besides vehicular accidents, other fatal accidental childhood head injuries tend to involve crushing or penetrating trauma, which is readily evident. These injuries tend to be the result of falling from considerable heights (greater than 10 feet) or having some object penetrate the head. There are distinctions between head injuries that are truly accidental and those that are abusive. The incidence of isolated subdural/subarachnoid hemorrhage as the only gross finding in fatal accidental head injuries in young children is less than 2%, compared with the 90% to 98% incidence of these hemorrhages associated with abusive head injuries (Case ME, unpublished autopsy studies). The trivial home accidents that children so frequently sustain are associated with primarily translational forces and not with the rotational forces necessary to develop tearing of bridging veins, which would produce subdural hemorrhage or other shearing injury (26,32,65–71). In low falls of less than about 8 feet, witnessed by uninvolved and nonbiased individuals, about 1% to 2% of children sustain a narrow simple linear skull fracture. In a small proportion of the children who sustain these skull fractures, an epidural hemorrhage will develop. These epidural hemorrhages are not accompanied by a decrease in the level of consciousness at the time of injury. If the epidural bleeding continues and produces significant increased intracranial pressure, there may be a subsequent decrease in the level of consciousness attributable to tentorial herniation.

It is essential that a meticulous autopsy examination be performed in all cases of possible injury to children. When subdural and/or subarachnoid hemorrhage is found at autopsy, the brain must be thoroughly examined to exclude the possibility of other causes of bleeding in these spaces. Although berry aneurysms are uncommon in young children, they may occur. Vascular malformations may also occur in young children and cause hemorrhage in intracerebral and intracerebellar subarachnoid and subdural areas. The distribution of bleeding in aneurysms and arteriovenous malformations is unlikely to resemble that of head injury, but these malformations need to be excluded by careful examination of the brain (72,73).

CONCLUSION

Anatomic and developmental differences of the brain and skull of children under the age 4 or 5 years make the head injuries and mechanisms of injury that affect these children different in certain respects from those occurring after that age. Inflicted head injuries in these young children usually create shearing injuries of the brain and blood vessels, resulting in diffuse axonal injury and subdural, subarachnoid, and retinal hemorrhages. The pathologic findings of subdural and subarachnoid hemorrhages and very, frequently, retinal hemorrhages are the most common findings by which these rotational head injuries in young children are identified at autopsy. Recognition of the underlying mechanism of the rotational brain or shearing injury is important to an understanding of the clinical course of these children, particularly with respect to the decrease in the level of consciousness and respiratory distress demonstrated after injury.

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PREVENTING SHAKEN BABY SYNDROME

A Guide for Health Departments and Community-Based Organizations

A part of CDC's "Heads Up" Series

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
CENTERS FOR DISEASE CONTROL AND PREVENTION

"Preventing Shaken Baby Syndrome: A Guide for Health Departments and Community-Based Organizations" is a publication of the Centers for Disease Control and Prevention, National Center for Injury Prevention and Control. See www.cdc.gov/injury.

TABLE OF CONTENTS

Shaken Baby Syndrome is a Preventable Public Health Problem

Shaken Baby Syndrome (SBS) is often seen as a crime, but it is also a preventable public health problem. State and local health departments and community-based organizations (CBOs) have ties to broader resources and programs, and strong connections in communities. Both health departments and CBOs can take a leading role in reframing the issue and engaging all members of a community to take part in the prevention of SBS.

The purpose of this guide from the Centers for Disease Control and Prevention (CDC) is to help your organization identify your role and to take action to protect infants from SBS. It outlines steps to implement evidence-based intervention strategies, to integrate specific education messages into existing programs for new parents, caregivers, professionals, and the general public, and to engage in activities that impact policy development that are effective in preventing SBS.

Together with local and national partners, your organization can make a difference in preventing SBS, so that all children can have safe, stable, and nurturing relationships and a better chance to live to their full potential.

THE FACTS:

What You Need to Know about Shaken Baby Syndrome

Understanding the facts helps build awareness, and ultimately affects how we take action. Using your contact with parents and other community members to promote the facts, risk factors and triggers, and ways to prevent SBS is the first step in addressing it as a public health problem that can be prevented, and not just a crime to be punished.

SBS, a form of abusive head trauma (AHT) and inflicted traumatic brain injury (ITBI), is a preventable and severe form of physical child abuse. It results from violently shaking an infant by the shoulders, arms, or legs. SBS may result from both shaking alone or from impact (with or without shaking).[1] The resulting whiplash effect can cause bleeding within the brain or the eyes.[2]

The American Academy of Pediatrics (AAP) and the Centers for Disease Control and Prevention (CDC) report that maltreatment causes a high rate of injury and death among infants.[4,5] SBS can cause death, mental retardation or developmental delays, cerebral palsy, severe motor dysfunction, blindness, and/or seizures.

SBS Basics[3]:

- SBS is a leading cause of child abuse deaths in the United States. Nearly all victims of SBS suffer serious health consequences and at least one of every four babies who are violently shaken dies from this form of child maltreatment.
- Babies (newborn to 4 months) are at greatest risk of injury from shaking.
- Inconsolable crying is a primary trigger for shaking a baby.

[1] Carbaugh SF. Understanding shaken baby syndrome. Adv Neonatal Care 2004;4(2):105–16

[2] Centers for Disease Control and Prevention. National Center for Injury Prevention and Control, Division of Violence Prevention. Shaken Baby Syndrome Tip Sheet [online] [cited 2010 March 1]. Available from URL: http://www.cdc.gov/healthmarketing/entertainment_education/tips/shaken_baby.htm

[3] Carbaugh SF. Understanding shaken baby syndrome. Adv Neonatal Care 2004;4(2):105–115.

[4] Centers for Disease Control and Prevention. Nonfatal Maltreatment of Infants [online] [cited 2010 March 1]. Available from URL: www.cdc.gov/mmwr/preview/mmwrhtml/mm5713a2.htm

[5] American Academy of Pediatrics Committee on Child Abuse and Neglect. Shaken baby syndrome: Rotational cranial injuries—technical report. Pediatrics 2001; 108(1):206–10 [online]. [cited 2010 March 1]. Available from URL: aappolicy.aappublications.org/cgi/reprint/pediatrics;108/1/206.pdf

FOCUSING ON PREVENTION

Research shows that shaking most often occurs in response to a baby crying or other factors that can trigger the person caring for the baby to become frustrated or angry.

The fact is that crying—including long bouts of inconsolable crying—is normal developmental behavior in infants. The problem is not the crying, it's how caregivers respond to it. Picking up a baby and shaking, throwing, hitting, or hurting him or her is never an appropriate response.

Everyone, from caregivers to bystanders, can do something to prevent SBS. Giving parents and caregivers tools that can help them cope if they find themselves becoming frustrated while caring for a baby are important components of any SBS prevention program (see Appendix B: *Prevention Tips for Parents and Caregivers*).

As a public health professional, you play a key role in reinforcing prevention through helping people understand the dangers of violently shaking a baby, the risk factors and the triggers for it, and ways to lessen the load on stressed-out parents and caregivers, all of which may help to reduce the number of cases of SBS.

UNDERSTANDING THE CONSEQUENCES

It is important to understand that SBS is the result of violent shaking that leads to a brain injury, which is much like an adult may sustain in repeated car crashes. The forceful shaking that causes SBS is child abuse; it does not happen in normal play. Claims of perpetrators that the highly traumatic internal injuries characterized by SBS resulted from "playing with the baby" are false. While jogging an infant on your knee or tossing him or her in the air can be very risky, the injuries that result from SBS are not caused by these types of activities.

Approximately one in four victims die, but there is a high risk of serious and long-term health consequences for those who live. SBS can potentially result in the following consequences:

- Death,
- Blindness,
- Mental retardation or developmental delays (any significant lags in a child's physical, cognitive, behavioral, emotional, or social development, in comparison with norms) and learning disabilities,
- Cerebral palsy,
- Severe motor dysfunction (muscle weakness or paralysis),
- Spasticity (a condition in which certain muscles are continuously contracted—this contraction causes stiffness or tightness of the muscles and may interfere with movement, speech, and manner of walking), and
- Seizures.

LEARNING WHAT TO LOOK FOR

SBS Signs and Symptoms

Babies, newborn to one year (especially babies ages 2 to 4 months) are at greatest risk of injury from shaking.[6] SBS injuries are not always visible. However, babies with SBS may display some outward signs.[7,8] Parents, family members, caregivers, or others in close and regular contact with an infant should seek medical attention right away if they notice any of the signs and symptoms listed below.

- Significant changes in sleeping patterns or inability to be awakened,
- Vomiting (more than usual),
- Convulsions or seizures,
- Increasing irritability,
- Uncontrollable crying,
- Inability to be consoled, and
- Inability to nurse or eat.

In more severe cases, babies may be:

- Unresponsive
- Unconscious

Babies should be taken to the emergency department immediately if they are experiencing any of these *severe* signs and symptoms of SBS listed above.

[6] Carbaugh SF. Understanding shaken baby syndrome. Adv Neonatal Care 2004 4(2):105–16.
[7] Miehl NJ. Shaken baby syndrome. J Forensic Nurs 2005 1(3):111–7
[8] Carbaugh SF. Understanding shaken baby syndrome. Adv Neonatal Care 2004 4(2):105–16

Factors that Put an Infant at Risk

The following factors increase an infant's risk of being shaken,[9,10,11] particularly when combined with a parent or caregiver who's not prepared to cope with caring for a baby:

- Being less than 1 year of age,
  - Babies less than 1 year of age are at the greatest risk, but SBS has been reported in children up to 5 years of age.
  - Babies (especially babies ages 2 to 4 months) are particularly at risk of injury from shaking, because they are small in relation to the size of adults who may pick them up and shake them, and they tend to cry more frequently and longer than older babies.
- Infant prematurity or disability,
- Being one of a multiple birth,
- Inconsolable and/or frequent crying,
- Prior physical abuse or prior shaking, and
- Most SBS victims are male.

Factors that Can Increase Parents' or Caregivers' Risk for Harming a Baby

Most SBS perpetrators are parents and their partners, with the majority of the perpetrators being the male parent or partner. The following factors increase a parent's or caregiver's risk of shaking a baby, particularly when combined with not being prepared to cope with caring for a baby.[12]

- Frustration or anger resulting from an infant's crying,
- Being tired,
- Having limited anger management or coping skills,
- Limited social support,
- Young parental age,
- Unstable family environment,

[9] Hoffman JM. A case of shaken baby syndrome after discharge from the newborn intensive care unit. Adv Neonatal Care 2005;5(3):135–46

[10] Black DA, Heyman RE, Smith Slep AM. Risk factors for child physical abuse. Aggress Violent Behav 2001;6(2–3):121–88

[11] Keenan HT, Runyan DK, Marshall SW, Nocera MA, Merten DF, Sinal SH. A population-based study of inflicted traumatic brain injury in young children. JAMA 2003;290(5):621–6

[12] Mraz MJ. Shaken baby syndrome. J Forensic Nurs 2005;1(3):11–7

- Low socioeconomic status,
- Unrealistic expectations about child development and child-rearing,
- Rigid attitudes and impulsivity,
- Feelings of inadequacy, isolation, or depression,
- Being a victim or witness to intimate partner violence, or
- Negative childhood experiences, including neglect or abuse.

Diagnosing SBS

Health care providers may be alerted to a possible SBS injury by *any* of the following:[13]

- Any infant or young child who presents with a history that is not plausible or consistent with the presenting signs and symptoms,
- The presence of a new adult partner in the home,
- A history of delay in seeking medical attention,
- A previous history or suspicion of abuse,
- The absence of a primary caregiver at the onset of injury or illness,
- Physical evidence of multiple injuries at varying stages of healing, or
- Unexplained changes in neurologic status, unexplained shock, and/or cardiovascular collapse.

There are a number of diagnostic tools that health care providers can use to assess the possibility of SBS in injured babies. In addition to a thorough history and physical exam, including ophthalmologic examination, physicians may use computerized tomography, magnetic resonance imaging, skeletal surveys, and other medical tests to diagnose SBS.

[13] Mehl NJ. Shaken baby syndrome. J Forensic Nurs 2005;1(3):116–7.

GETTING STARTED

Knowing what needs to be done and prioritizing where to start can often be a challenging task. Start by using a prevention model, rooted in behavioral science, as a practical tool to create an effective strategy. The model can guide decisions to identify your organization's role in the issue, align activities with your mission, and leverage your reach to the community to maximize impact.

BUILDING A FRAMEWORK FOR PREVENTION

Prevention requires understanding the factors that influence violence. CDC uses a four-level social-ecological model to better understand violence and the effect of potential prevention strategies.[14] This model considers the complex interplay between individual, relationship, community, and societal factors and is more likely to sustain prevention efforts over time than any single intervention.

The model serves as a practical framework to build your strategy. The activities, messages, partnership, and policy suggestions outlined in this guide already build upon each of the four levels of the framework. Additionally, you can also use the model as a reference or guide to assess ways to use resources and reach audiences at various levels.

SOCIETAL COMMUNITY RELATIONSHIP INDIVIDUAL

[14] Dahlberg LL, Krug EG. Violence a global public health problem. In: Krug E, Dahlberg LL, Mercy JA, Zwi AB, Lozano R, eds. World Report on Violence and Health. Geneva, Switzerland: World Health Organization; 2002:1-56.

Or, it can be used to help build a more feasible plan by identifying appropriate partners with more optimal resources to address multiple levels.

Individual-level strategies are ones that can be aimed at changing parents' or caregivers' knowledge and skills.

Relationship-level strategies are ones that are aimed at trying to change the interactions between people—parents and children, parents and other caregivers, parents and health care providers, bystanders, and parents.

Community-level strategies are those that are aimed at modifying the characteristics of settings that give rise to violence or that protect against violence (e.g., address social and economic factors; access and availability of parental support programs, early child care, respite care centers).

Societal-level strategies are aimed at changing cultural norms surrounding parenting, as well as laws and policies aimed at supporting parents.

LEARNING ABOUT AND FROM EXISTING PROGRAMS

Start with what is available, feasible, and has the greatest level of evidence of effectiveness. There are a number of existing SBS prevention programs and resources available that can be implemented. Balance the options below to build on programs you already have, or to reach audiences you already work with.

Many of these programs strive to raise awareness about SBS, educate parents and other caregivers about the serious effects of SBS-related injuries, and inform them about infant crying behavior and safe ways to reduce and prevent SBS injuries. These strategies address the multiple levels of the prevention model. Examples of common prevention strategies include:

- Coordinated hospital-based primary prevention programs targeting parents of newborns.
- Home visits for new parents (home visits bring community resources to families in their homes; health professionals provide information, health care, psychological support, and other services that can help people to be more effective parents and caregivers).
- Anticipatory guidance at well-baby visits in pediatric practice and/or health clinics.
- School prevention programs for junior high and high school students providing students with an understanding of child maltreatment issues, anger management techniques, and child care skills. For example, the National Center on Shaken Baby Syndrome offers a school-based program for junior and senior high school students, which teaches students about the "medical aspects of shaking injuries, combined with basic anger management and child care skills. The program reaches young adults as potential caregivers and future parents. Teaching students how frustration can lead to shaking helps them understand the importance of appropriate coping skills."[15]
- Programs targeting males, especially new fathers, with information and resources for providing safe and nurturing care for their new infants and safe strategies for coping with frustration caused by crying babies.[16]
- Professional education and trainings for doctors, nurses, social workers, and others providing family services, and
- Educational print and video materials provided at the time of delivery, as well as at pediatric offices and prenatal classes.

This is Important

When possible, it is important to use evidence-based practice as the foundation for any intervention. Evidence-based practice, as defined by the Institute of Medicine (IOM), is a combination of three factors:

1. Best research evidence
2. Best clinical experience, and
3. Consistency with patient values.[17]

[15] National Center for Shaken Baby Syndrome [online]. [cited 2010 March 1] Available from URL: www.dontshake.com/Audience.aspx?categoryID=10&PageName=Schoolbased.htm
[16] National Center for Shaken Baby Syndrome, Dads 101 [online] [cited 2010 March 1] Available from URL www.dontshake.com/Audience.aspx?categoryID=10&PageName=Dads101.htm
[17] Committee on Quality of Health Care in America. Institute of Medicine. Crossing the quality chasm: A new health system for the 21st century. (2001). National Academy Press. Washington, DC

Upstate New York Shaken Baby Syndrome Education Program

The California Evidence-Based Clearinghouse for Child Welfare (CEBC) has designated the Upstate New York Shaken Baby Syndrome Education Program as having *Promising Research Evidence*.[18] Developed by Dr. Mark S. Dias and colleagues, this hospital-based, parent education program provides information to parents at the time of birth about the effects of violent shaking, as well as alternatives for responding to crying infants. As part of this intervention, nurses were asked to have both parents read a pamphlet, view a video titled *Portrait of Promise: Preventing Shaken Baby Syndrome*, and sign a commitment statement to indicate that they received and understood the information before their baby was discharged from the hospital.

Findings from follow-up telephone surveys with parents 7 months after the birth suggest that more than 95 percent of the parents remembered receiving the information. In addition, after 5 years, this effort resulted in significantly decreased incidence of abusive head trauma among children ages 36 months and younger.[19] The program, which began in December 1998, has been adopted by many states and hospitals across the nation.

As part of CDC-funded research, Dr. Dias is replicating a revised version of the program in Pennsylvania. The components of this evaluation include:

1) Time series analysis of reported cases in Pennsylvania to examine the trends in SBS cases before, during, and after the program is introduced, and comparing results,
2) Time series analysis of hospital discharge data in Pennsylvania and surrounding states,
3) Randomized trial in 31 counties in central Pennsylvania to evaluate the effectiveness of providing additional information at pediatric well visits, and
4) An economic analysis to examine program costs and benefits.

[18] California Evidence-Based Clearinghouse for Child Welfare [online] [cited 2010 March 1] Available from URL: www.cachildwelfareclearinghouse.org/scientific-rating-scale.php#rating3.

[19] Dias MS, Smith K, deGuehery K, Mazur P, Li V, Shaffer ML. Preventing abusive head trauma among infants and young children: A hospital-based, parent education program. Pediatrics 2005;115(4):e470–7.

Period of PURPLE Crying[a]

The CEBC has designated the Period of PURPLE Crying® program as having *Promising Research Evidence*. This program was developed by Dr. Ronald Barr and colleagues at the National Center on Shaken Baby Syndrome (NCSBS). Two randomized controlled trials of the program showed increases in knowledge about crying and SBS and "walk away behavior when the mother was frustrated."[20]

As part of CDC-funded research, Dr. Desmond K. Runyan is testing the program in North Carolina. The state-wide program will educate parents about normal infant crying patterns, how to respond to crying, and the dangers of shaking. The information will be disseminated in a manner designed to reach every new parent three times. Nurses will educate parents of every newborn at all 86 hospitals/birthing centers in North Carolina, using discussion, a video, and a booklet about infant crying. In addition:

- County health departments, pediatricians, and family physicians will provide a dose of the intervention in prenatal classes and/or at 2-week well-child checks; and
- The NCSBS and the University of North Carolina School of Journalism and Mass Communications will develop a statewide media campaign that addresses social norms about shaking and reinforces program messages directly and through family and friends.

Period of PURPLE Crying®

P - Peak Pattern
Crying peaks around 2 months, then decreases.

U - Unpredictable
Crying for long periods can come and go for no reason.

R - Resistant to Soothing
The baby may keep crying for long periods.

P - Pain-like Look on Face

L - Long Bouts of Crying *Crying can go on for hours.*

E - Evening Crying
Baby cries more in the afternoon and evening.

Source: National Center on Shaken Baby Syndrome

[a] National Center on Shaken Baby Syndrome. Randomized Controlled Trials on the Effectiveness of the PURPLE Materials: Parallel Studies in the state of Washington (USA) and the province of British Columbia (Canada) [online]. [cited 2010 March 14]. Available from URL: http://www.dontshake.org/sbs.php?topNavID=4&subNavID=32&subnav_1=172&navID=176

Period of PURPLE Crying® (continued)

The multi-level evaluation strategy for the project includes:

1) Active surveillance of traumatic brain injuries in all Pediatric Intensive Care Units,
2) Anonymous surveys of parents on discipline practices before and after the intervention,
3) Time series analysis to examine the trends in abusive head trauma cases before, during, and after the program is introduced and comparing results,
4) Process evaluation of the delivery of the messages,
5) Nested case-control study of abusive head trauma cases, compared to other hospitalized children with brain injury and the entire cohort of children under age 2, and
6) An economic analysis to examine program costs and benefits.

Reviews and ratings used to determine the research evidence for other interventions related to child welfare are located on Web sites, such as the National MCH Center for Child Death Review, **www.childdeathreview.org**, the RAND Promising Practices Network on Children, Families and Communities, **www.promisingpractices.net**, and the California Evidence-Based Clearinghouse for Child Welfare, **www.cachildwelfareclearinghouse.org**.

GETTING the MESSAGE OUT:

The Right Message, Right Person, Right Time

CREATING EFFECTIVE MESSAGES

Prevention messages are often drowned out in the thousands of messages seen and heard by people every day. That's why it is important to focus your message to make sure it reaches the right audience for the right results. Since all ideas and words do not resonate with all audiences, it is important to tailor specific messages to each audience to make them effective.

Using messages to communicate about SBS prevention can range from developing a complex campaign to using a few appropriate messages in your existing programs and activities. No matter the size of your effort, using the example messages below consistently with the right audiences can help reframe SBS as a preventable public health problem, and can be used to address each of the four levels of the prevention framework.

To prevent child maltreatment, it is important to focus on making people aware of what to normally expect as a child develops, how to support parents, and steps they can take in different situations.[21] Because crying is one of the primary triggers for shaking a baby, messages that teach how to cope with crying may be most effective. Messages should also be specific and focus on awareness and action. It is important to make people not only aware of SBS and their role in preventing it, but also

[21] Wesley B. Reframing the issue: A new child maltreatment prevention message. N C Med J 2005;65(5):367–9

Health communication tools are available to help you plan, design, implement, and evaluate an SBS prevention communication campaign. Below are some examples:

- *Adding Power To Our Voices: A Framing Guide for Communicating About Injury (www.cdc.gov/injury)*
- *CDCynergy: Violence Prevention Edition (www.cdc.gov/ncipc/dvp/CDCynergy/CDCynergy.htm)*
- *National Cancer Institute's Making Health Communication Programs Work. (www.cancer.gov/pinkbook)*

give them specific and feasible actions for prevention. Messages should also match activity goals and can be used to:

- Increase awareness of SBS in a particular audience.
- Help make an audience more open to participating in programs or taking specific action for prevention.
- Help support policies.
- Help decisionmakers, such as policymakers, partners, or organizational leadership, understand SBS as a preventable issue that needs their support.

The example messages below can help guide you in addressing key audiences with the power to prevent SBS: parents, caregivers, bystanders, and health care providers.

Example Messages for Parents and Other Caregivers:

1) Crying is normal for babies.
   a) Crying is one way babies communicate.
   b) Excessive crying is a normal phase in infant development.
      - Babies cry most between 2 and 4 months.
      - Prolonged, inconsolable crying generally lessens when babies are around 5 months old.[22]
      - Most babies who cry a great deal are healthy and stop crying spontaneously.
   c) You are not a bad parent if your baby continues to cry after you have done all you can to calm him or her.
   d) Remember, this will get better.

[22] St James-Roberts I. Effective services for managing infant crying disorders and their impact on the social and emotional development of young children. In: Tremblay RE, Barr RG, Peters RDeV, eds. Encyclopedia on Early Childhood Development [online]. 2004:1–6. Available from URL: http://www.child-encyclopedia.com/pages/PDF/StJames-RobertsANGxp.pdf

2) When a baby cries, there are steps you can take to try to comfort him or her.

   a) Check for signs of illness or discomfort like a dirty diaper, diaper rash, teething, fever, or tight clothing.
   b) Assess whether s/he is hungry or needs to be burped.
   c) Rub his/her back, gently rocking him/her; offer a pacifier; sing or talk; take a walk using a stroller or a drive in a properly-secured car seat.
   d) Call the doctor if you think the child is ill.
   e) Remember you are not a bad parent or caregiver if your baby continues to cry after you have done all you can to calm him or her.

3) When you feel frustrated, angry, or stressed while caring for your baby, take a break.

   a) Call a friend, relative, neighbor, or a parent helpline for support.
   b) Put your baby in a crib on his or her back, make sure the baby is safe, and then walk away for a bit, checking on him or her every 5 to 10 minutes.
   c) Remember, this will get better.

4) Be aware of signs of frustration and anger in yourself and others caring for your baby:

   a) See a health care professional if you have anger management or other behavioral concerns.
   b) Ensure others caring for your baby see a health care professional if they easily become angry or frustrated around your baby.

Focusing on Positive Parenting

Focus on promoting protective factors in your SBS prevention messages. Messages that encourage positive and protective factors through parenting skills can create a positive norm of good parenting. Because young children experience the world through their relationships with parents and other caregivers,[23] protective factors at both the family and community levels provide a buffer for children at risk for abuse or neglect. Scientific evidence shows that a supportive family environment is a key protective factor against abuse or neglect. Specifically, *safe, stable, and nurturing relationships* between children and adults protect against maltreatment and other adverse exposures occurring during childhood that compromise health over the lifespan.

Example Messages for Bystanders

1) Crying is normal for babies.
   a) Excessive crying is a normal phase in infant development.
   b) Sometimes babies cry for no apparent reason.
   c) It is not always possible to console a crying infant.

Bystanders Are Important

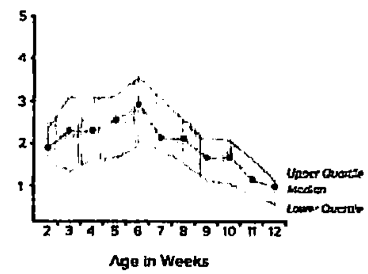
Many parents feel pressured on some level, by others, to make their baby stop crying (CDC 2007 *Healthstyles Survey*). Bystanders are people who are not caring for an infant, but who may be affected by an infant's crying or other behaviors.

2) Support parents and other caregivers of babies.
   a) As appropriate, offer to provide child care so that parents get needed breaks.
   b) Be sensitive and supportive in situations in which parents or caregivers are trying to calm a crying baby.
   c) Communicate to the parent or caregiver that you understand that it is difficult to care for a crying baby and offer to help (this may include giving the parent a helping hand or a break, sharing a parent support helpline number, or just acknowledging that parenting can be difficult and exhausting at times).

[23] Mishi N. Shaken baby syndrome. J Forensic Nurs 2005;1(3):111–7

"Crying Curve"

Infant crying begins to increase around 2 to 3 weeks of age, and peaks around 6 to 8 weeks of age. As illustrated below, it then tapers off when the baby is 3 to 4 months old.



Source: *Crying in Infancy*, T. Berry Brazelton. Reproduced with permission from Pediatrics, Vol. 29, Pages 579-588. Copyright © April 1962 by the AAP

Example Messages for Health Care Providers

1) Remind parents and caregivers that crying is normal for babies.
   a) Explain to parents that excessive crying is a normal phase of infant development.
   b) Share the Crying Curve with parents.

2) Support parents and other caregivers of babies.
   a) During routine pediatric visits, be sure to ask parents how they are coping with parenthood and their feelings of stress.
   b) Assure them that it is normal to feel frustrated at long bouts of crying and a sudden decrease in sleep, but that things will get better.
   c) Give parents the number to a local helpline or other resource for help.
   d) Talk with them about the steps they can take when feeling frustrated with a crying baby, such as putting the baby safely in a crib on his or her back, making sure that he or she is safe, walking away and calling for help or a friend, while checking on the baby every 5 to 10 minutes.
   e) Let parents know what to check for when their baby is crying: signs of illness, fever or other behavior that is unusual, or discomfort like a dirty diaper, diaper rash, teething, or tight clothing, or whether he or she is hungry or needs to be burped.

WORKING WITH THE MEDIA

Messages through the media can have wide reach to multiple audiences. The media can also uniquely help to reframe SBS as a public health problem, rather than just a criminal investigation. CDC has created a companion guide for the media on reporting on SBS. You can share this guide with local media or use it to develop messages when you speak to journalists. The guide, "A Journalist's Guide to Shaken Baby Syndrome: A Preventable Tragedy," as well as radio public service announcements (in English and Spanish) and broadcast-quality video that includes B-roll, full-screen tips, and downloadable scenarios, are available at: **www.cdc.gov/injury.**

Below are some additional steps you can take to work with your local media.

- Proactively build relationships with local radio, print, online, and television outlets that cover family, health, and child maltreatment issues; informing journalists about SBS and your organization's education and prevention activities.
  - Pitch a deskside briefing—a visit with a journalist to brief him or her on the issue of SBS.
  - Hold a media roundtable or virtual roundtable to inform a group of journalists about the issue.
  - Hold a telebriefing, where journalists dial in to learn about SBS from experts, physicians, and/or parents who have a child with SBS.
  - Write an op-ed or article for local newspapers or Web sites.
  - Pitch stories to community affairs programs.
- Contact journalists after a story about SBS is reported in the news to offer the public health perspective, data, prevention tips, experts for interviews and to clarify misinformation as needed (See Appendix B: *Prevention Tips for Parents and Caregivers*). Organizations are best prepared to respond quickly when a story breaks if they have previously developed a media plan that identifies the organization's spokesperson and key prevention messages and talking points.

STRENGTH in NUMBERS:

Building Partnerships to Prevent SBS

COLLABORATING WITH OTHER ORGANIZATIONS AND THE BUSINESS COMMUNITY

Partnerships and collaborations can be critical elements for achieving success. They can be instrumental in expanding your reach to new audiences, augmenting resources, adding outreach channels, facilitating message dissemination within the community, and offering referral sources for your program. For example, when your partners collaborate to deliver the same prevention messages through communication channels used by your target audience, it extends the messages' reach and frequency. Effective messages that are delivered to the same audience through multiple channels are more likely to be remembered and move the audience to take the desired action.

INITIATING PARTNERSHIPS

While every situation and partnership is unique, here are some general steps often used to build partnerships:

1) **Assess your current situation.** Planning your prevention effort should involve a careful analysis of your organizational resources and needs, including staff, funding, facilities, technology, and expertise. This information will help clarify when a potential collaboration with another organization can support your program goals.

2) **Identify potential partners.** The relationship should be mutually beneficial. Identify organizations that support your mission of preventing injuries and improving health and safety for new parents and their babies. Determine how collaboration will mutually support short- and long-term goals. First consider those with which you have successfully partnered in the past. Then consider new partners, such as the media and business communities. These organizations can help you reach new parents with key information and resources and strengthen your advocacy for prevention efforts, or perhaps

combine resources with you and other organizations to develop a collaborative prevention effort in the community supported by a range of like-minded civic groups. For example, an employer's "lunch and learn" program for new parents in a business setting, or building relationships with human resource or employee assistance professionals in large corporations may lead to other opportunities to build community support for parents.

3) **Develop your "pitch."** After strategically selecting groups with which you might work, develop your "pitch," or selling points, and your "ask," that is, what you want the organization or individual to do as part of the collaboration. For example, you may want to ask them to join you in incorporating SBS prevention messages and parent support activities into their ongoing communication activities with parents, caregivers, and other relevant audiences. This will vary based on the resources, needs, and priorities of each organization. In addition, showcase the benefits that your potential partners will gain by collaborating. Be sure to highlight benefits that are most relevant to their values and mission.

4) **Make contact.** Whenever possible, deliver your partnership proposal in person. Consider bringing at least one other person, because different communication styles and demeanors can influence an encounter. However, make sure that your team speaks with one voice, based on the messages you develop. Delivering mixed messages creates confusion and weakens your credibility.

5) **Establish the partnership.** Being credible and offering incentives are important, but these may not be enough. Use your passion to make potential partners believe they should be involved. Describe how your programs and services can make a difference. Share information about the emotional and financial burdens caused by SBS. Underscore how your community will benefit from your collaborative efforts, how others are getting involved, and how even seemingly small contributions can help prevent injury and death. Confirm how the proposed partnership is mutually beneficial. Be specific about what you are asking the organization to do.

6) **Give thanks.** Never forget the power of the phrase "thank you." Acknowledge partnership agreements promptly. Look for creative ways to convey your gratitude to partners often and thank them publicly.

A tool to help you plan your partnership outreach is available in Appendix C.

WORKING TOGETHER: ROLES OF HEALTH DEPARTMENTS AND COMMUNITY-BASED ORGANIZATIONS

Roles for Health Departments in Preventing SBS

As leading agencies in the community, health departments (both state and local) are well positioned to lead SBS prevention activities aimed at all four levels of the prevention framework. They can also forge partnerships to create momentum to help other state or local organizations take action.

SBS prevention activities for local health departments may include:

- Delivering and/or reinforcing SBS prevention messages in health department contacts with expectant and new parents,
- Creating partnerships with local community-based organizations (CBOs) that can provide training, education, message dissemination, and follow-up support to parents identified in health department settings as needing additional resources or information,
- Identifying community leaders and organizations interested in children's health and safety to support SBS programs, such as local hospitals, clinics, pediatric offices, urgent care centers, pharmacists, CBOs, and other health facilities,
- Providing consistent and appropriate prevention messages to the media and other organizations in the community. (Share CDC's "A Journalists Guide to Shaken Baby Syndrome: A Preventable Tragedy" with your local media, available at **www.cdc.gov/injury**),
- Developing a media outreach and response strategy to bring the public health prevention perspective to media coverage of SBS and to position the health department as a resource for information on SBS prevention (see specific suggestions under *Roles for CBOs in Preventing SBS*), and
- Creating and/or disseminating existing SBS education and training materials to share with community organizations that will implement SBS education efforts.

Roles for CBOs in Preventing SBS

CBOs are uniquely positioned to incorporate consistent prevention messages and interventions into their ongoing community programs to address all four levels of the prevention framework. For example, individual-, relationship- and community-level strategies can be directed to new parents, caregivers, health care workers, social workers, and others who provide services to families in existing programs, such as:

- Prenatal classes,
- Parenting classes,
- Postnatal support programs,
- Stress management classes,
- Mentoring activities,
- Home visitation programs,
- Training for babysitters and child care providers,
- Mental health programs, and
- Training programs for health care providers and social workers.

Other SBS prevention activities for CBOs include:

- Forging partnerships with state and local health departments,
- Referring new parents to organizations for information and support,
- Adding information about SBS prevention to existing, regular communications with other community organizations through Web sites, newsletters, and regular mailings,
- Advocating with policymakers and funding bodies for effective policies and resources to support parents in providing safe, stable, nurturing relationships for their infants (see *Taking Public Health Solutions to the Next Level: Impacting Policy*), and
- Working with the media by providing consistent and appropriate messages (see section on *Creating Effective Messages* and CDC's "A Journalists Guide to Shaken Baby Syndrome: A Preventable Tragedy").

IDENTIFYING POTENTIAL PARTNERS

There are many diverse groups you could consider approaching for support, just as there are a variety of ways they might be able to work with you. The chart below offers examples of both:

Type of Organization	*Potential Collaborative Activities to Support SBS Education*
Civic and service organizations	■ Help identify local programs that might incorporate crying and coping education and messages and promote those programs. ■ Disseminate SBS prevention materials to their own members, as appropriate. ■ Disseminate information through existing channels, such as newsletters, flyers, and Web sites. ■ Provide community forums where SBS information can be shared.
Local hospitals	■ Provide crying and coping education materials and messages to new parents. ■ Offer training courses to new and expectant parents. ■ Sponsor health and wellness fairs incorporating crying and coping education information. ■ Incorporate SBS education in health care employee training. ■ Publish messages about crying behavior and coping in hospital wellness publications that are distributed to the public.
Faith-based organizations	■ Include crying behavior and coping messages in newsletters, marriage/parent education classes, daycare facilities, etc. ■ Distribute SBS prevention information at community health fairs and other appropriate forums.
Government programs that serve families and children, such as Women, Infants, and Children (WIC)	■ Include messages and education materials in outreach to new and young parents. ■ Display crying and coping information on bulletin boards and include it as part of regular communication with clients. ■ Put inserts into mailings and/or other types of regular statements. ■ Incorporate crying and coping messages in trainings, job preparation classes, maternity classes, work/life balance sessions, etc.
Large employers	■ Post information on bulletin boards and Web sites. ■ Offer information at employee brown bag lunches and other employee activities for parents. ■ Include messages on pay stubs.

Type of Organization	*Potential Collaborative Activities to Support SBS Education*
Large employers (continued)	■ Include crying and coping messages in employee health fairs and other forums. ■ Ask employee assistance programs to incorporate messages and education materials into their counseling for new parents, and to direct new parents to resources that address "new baby" stress.
Elementary schools, Head Start, nursery, and daycare programs with access to new parents and potential parents-to-be	■ Send flyers home with children. ■ Put information on message boards and in children's school bags. ■ Sponsor family health fairs that incorporate crying and coping education materials.
Community centers, local YMCAs, libraries, support groups for parents, and other local organizations sponsoring children and family activities	■ Incorporate messages and materials into ongoing classes for new and expectant parents, exercise, and stress management classes, etc. ■ Display posters or other information on bulletin boards. ■ Sponsor family wellness days that incorporate crying and coping messages.
Community and (new) parent groups	■ Sponsor an event or gathering focused on crying and coping. ■ Organize a forum for new parent groups that incorporates SBS messages.
Local merchants including: baby retailers; bulk and big box discount retailers; pharmacies; grocery stores; gyms; department stores; shoe stores; home improvement stores; hair salons/barber shops/nail salons/day spas; coffee shops; bookstores; theaters; movie theatres	■ Insert messages in or on shopping packages. ■ Display prevention messages on sales slips. ■ Display brochures, posters, or flyers. ■ Sponsor in-store seminars for new parents as a way to bring them into their facilities for shopping. ■ Include messages on marquees, screens, or message boards. ■ Air targeted public service announcements (PSAs) in movie theatres (PSAs for males during movie genres targeted to young adult males and for new mothers during *mommy movie days*, select days at some theaters when babies are welcomed).
Media	■ Develop and air a PSA campaign (radio/TV/Web). ■ Use on-air talent to host events, appear in PSAs, and serve as spokespersons. ■ Obtain media sponsorships for community events. ■ Provide exclusive stories. ■ Provide ink/air time. *(Please note that partnering with one outlet may preclude work with a competitor and make it hard to pitch stories to other media.)*
State and local health departments	■ Provide appropriate data to define the problem at the local level. ■ Create and disseminate messages to frame SBS as a preventable, public health issue. ■ Apply the prevention framework to intervention strategies. ■ Incorporate SBS prevention messages in programs for and contacts with expectant and new parents.

TAKING PUBLIC HEALTH SOLUTIONS TO THE NEXT LEVEL:

Impacting Policy

POLICY IS AN EFFECTIVE PUBLIC HEALTH INTERVENTION

Policy interventions are important and effective community and societal level strategies for improving the public's health. Policy can be defined many ways, but one definition applicable to public health policy intervention describes policy as: "laws, regulations, formal and informal rules and understandings that are adopted on a collective basis to guide individual and collective behavior."[24] Public health policy interventions are targeted to influence systems development, organizational change, social norms, and individual behavior to promote improvement in the health of a population.

Policy interventions are particularly valuable because they are systems-based and impact populations by changing the context in which individuals take action or make decisions (i.e., making default decisions safe and healthy). The effectiveness of a policy intervention, though, depends on the level of awareness, education, acceptance, funding, implementation efforts, enforcement, and even programs that accompany the creation or passage of the policy.

[24] Wallack L. *Media advocacy: Promoting health through mass communication.* In: Glanz K, Lewis FM, Rimer BK, eds. Health Behavior and Health Education: Theory, Research and Practice. San Francisco, Calif: Jossey-Bass; 1990: 370–386

There are different types and levels of policy; each of them plays an important role in improving the public's health.

- Legislative, which is a law, ordinance, or regulation:
  - Local (city or county),
  - State, and
  - Federal.
- Organizational, which could include the following examples:
  - Local education agencies and/or local schools or school districts,
  - Private hospital or other health care delivery sites (i.e., physicians' offices),
  - Non-governmental organizations,
  - Governmental agencies,
  - Business, industrial, or corporate, and
  - Professional associations or accredited organizations.

Examples of SBS Policy Initiatives

The following are two examples that demonstrate just a few of the multiple levels and types of policy changes that have been used in developing SBS prevention initiatives.

1) Legislation/law at a state level.

"New York passed Bill A08314, which requires all child care providers in New York to receive training on SBS. It is a mandate that all child day care providers be educated and informed on the identification, diagnosis and prevention of SBS. This education is to be added to the training providers already receive on early childhood development, nutrition and statues and regulations toward safety issues."[25] (See Appendix A: *State Initiatives on SBS*, for a list of examples of state legislative initiatives.)

[25] National Center on Shaken Baby Syndrome [online] [cited 2010 March 11] Available from URL: http://www.dontshake.org/sbs.php?topNavID=2&subNavID=318

This example provides guidance on the agency responsible for implementation. The state health department is identified as the agency required to develop and implement an ongoing public information and educational campaign to inform child care providers about harmful effects of SBS.

2) Organizational policy at a local/regional level.

In response to a doubling of SBS admissions in 2004, the administrative leadership of Legacy Health authorized a multidisciplinary task force to study the feasibility of implementing a system-wide SBS prevention program. Though not mandated by either state, this Oregon and Washington hospital system secured grant funding to research, develop, and launch a pilot program. Program design was informed both by patient satisfaction data and other patient care quality initiatives. A successful system-wide roll- out followed within one year, as did the systematic integration of SBS prevention messages throughout the family-centered maternity and pediatric services continuum of care procedures. Mandatory Skills Day training for all 400 obstetric care registered nurses included a 30-minute session on SBS and viewing of a DVD/Booklet.

In 2009, every family from every hospital went home with the *Period of PURPLE Crying®* DVD/Booklet; 90 percent of those families had both seen the DVD and received SBS prevention education from a nurse either at a discharge class or during one-to-one education at the bedside.

YOUR AGENCY/ORGANIZATION'S ROLE IN IMPACTING POLICY

While the two examples above do a good job of illustrating the potential for policy interventions used to prevent SBS at the state government and local organizational levels, it should be noted that neither of them have been rigorously evaluated to determine their effectiveness in preventing SBS. As a matter of fact, there are no current examples of evidence-based policy interventions for prevention of SBS. However, an educational approach has been shown to be effective in preventing SBS and there are a few evidence-informed prevention programs available (see section, *Getting Started: Working Towards Success*). These are important factors to consider when thinking about activities to include in a policy plan or intervention to prevent SBS.

For example, while we may not yet have evidence-based *policy* interventions, there are promising practice program interventions for prevention of SBS with research evidence. Therefore, it may be appropriate to start with the development of policy interventions focused on the implementation of promising practice programs or to begin by identifying an existing state legislative policy or initiative and work to enhance that policy (i.e., include requirement of programs with promising research evidence or include dedicated resources to implement the policy intervention).

Before developing a policy plan or engaging in activities to impact policy related to SBS prevention, it is important to assess the political and social environment of your state or community. This will help your agency define its role and target the type of policy change that would be most effective. Due to the possible restrictions or limitations regarding your organization's activities related to policy (i.e., federal dollars are not allowed to be used to lobby or advocate and/or state health departments may have limitations regarding the level or type of involvement allowed in the legislative process), your agency may not have the capacity or be most appropriate to take the lead for all levels of the policy initiative. This is why it is important to work together with your partners in developing and implementing a policy plan or initiative to prevent SBS (See section, *Strength in Numbers: Building Partnerships to Prevent SBS*).

Regardless of these limitations, there are numerous activities your agency can engage in to participate in developing, implementing, and evaluating policy interventions to prevent SBS. Therefore, whether a CBO or a local or state health department, you have an important role to play in impacting policy interventions that improve the public's health. Finally, remember that successfully implementing a policy intervention takes time and requires being ready when the opportunity presents itself.

Examples of Activities that Impact Policy

Depending on the environment of the community and the role of the agency, an organization or program could engage in any or all of these activities to affect public health through policy.

1) Collecting, analyzing, summarizing, and interpreting data and other scientific-based information relevant to the frequency and seriousness of SBS and its prevention,
2) Proactively disseminating data linked to possible solutions and making sure the data gets into the hands of decisionmakers or those who can influence decisionmakers,
3) Packaging, presenting, or promoting data and information in ways that resonate with the audience and can be used to inform decisionmaking (i.e., cannot assume that data or information can stand alone),
4) Utilizing media and partners to help convey important messages to policymakers and the public,
5) Reviewing and/or drafting potential policies or legislation,
6) Building coalition networks that are able to advocate for policy changes, educating the public, and implementing programs that impact prevention of SBS (See section, *Strength in Numbers: Building Partnerships to Prevent SBS*),
7) Identifying and analyzing existing SBS-related policies in your community or state and working to enhance them,
8) Engaging in awareness efforts and implementation of existing policies that support prevention of SBS,
9) Conducting a cost-benefit analysis related to the burden of SBS and predicting how a science-based prevention effort will impact the cost for an organization or society,
10) Evaluating existing/new policies, including an assessment of effectiveness and cost effectiveness, and
11) Meeting with policymakers to inform or educate on the burden of SBS and what works to prevent SBS.

CDC RESOURCES

National Center for Injury Prevention and Control

Violence prevention is a major focus of the National Center for Injury Prevention and Control (Injury Center). As the lead federal agency for injury prevention and control, CDC's Injury Center works closely with other federal agencies; national, state, and local organizations; state and local health departments; and research institutions.

www.cdc.gov/injury

National Center on Birth Defects and Developmental Disabilities

The National Center on Birth Defects and Developmental Disabilities promotes the health of babies, children, and adults, and enhances the potential for full, productive living by providing positive parenting tips and information on developmental milestones and screening.

www.cdc.gov/ncbddd/child/default.htm

OTHER FEDERAL RESOURCES

Administration for Children and Families

The Administration for Children and Families, within the Department of Health and Human Services, is responsible for federal programs that promote the economic and social well-being of families, children, individuals, and communities.

www.acf.hhs.gov

Administration for Children and Families, Children's Bureau

The Children's Bureau is designed for professionals concerned with child abuse and neglect, child welfare, and adoption.

www.cbexpress.acf.hhs.gov

The Child Welfare Information Gateway

The Child Welfare Information Gateway (a merger of the former National Clearinghouse on Child Abuse and Neglect Information and National Adoption Information Clearinghouse) provides access to information and resources to help protect children and strengthen families.

www.childwelfare.gov

FRIENDS National Resource Center

FRIENDS National Resource Center is a federally mandated Training and Technical Assistance Provider for agencies working to prevent child abuse.

www.friendsnrc.org

ONLINE RESOURCES

American Academy of Pediatrics

The American Academy of Pediatrics (AAP) and its member pediatricians dedicate their efforts and resources to the health, safety, and well-being of infants, children, adolescents, and young adults. The AAP has approximately 60,000 members in the United States, Canada, and Latin America. The AAP develops guidelines on a variety of pediatric health issues and distributes a wide range of patient education materials.

www.aap.org

American Professional Society on the Abuse of Children

The American Professional Society on the Abuse of Children (APSAC) is a nonprofit national organization focused on meeting the needs of professionals engaged in all aspects of services for maltreated children and their families. Especially important to APSAC is the dissemination of state-of-the-art practice in all professional disciplines related to child abuse and neglect.

www.apsac.org/mc/page.do

The California Evidence-Based Clearinghouse for Child Welfare

The California Evidence-Based Clearinghouse for Child Welfare (CEBC) identifies and disseminates information about evidence-based practices relevant to child welfare. The CEBC provides guidance to statewide agencies, counties, public and private organizations, and individuals. This guidance is provided in a simple, straightforward format, reducing the user's need to conduct literature searches, review extensive literature, or to understand and critique research methodology.

www.cachildwelfareclearinghouse.org

Children's Safety Network National Injury and Violence Prevention Resource Center

The Children's Safety Network provides resources and technical assistance to maternal and child health agencies and organizations seeking to reduce unintentional injuries and violence toward children and adolescents. This is one of four Children's Safety Network Resource Centers funded by the Maternal and Child Health Bureau of the U.S. Department of Health and Human Services.

www.childrenssafetynetwork.org

Childhelp® USA

Childhelp® is a national nonprofit organization dedicated to helping victims of child abuse and neglect. Childhelp's approach focuses on prevention, intervention, and treatment. The Childhelp National Child Abuse Hotline, 1-800-4-A-CHILD, operates 24 hours a day, seven days a week, and receives calls from throughout the United States, Canada, the U.S. Virgin Islands, Puerto Rico, and Guam. Childhelp's programs and services also include residential treatment services (villages); children's advocacy centers; therapeutic foster care; group homes; child abuse prevention, education, and training; and the National Day of Hope®, part of National Child Abuse Prevention Month every April.

www.childhelp.org

Child Welfare League of America

The Child Welfare League of America is an association of nearly 800 public and private nonprofit agencies that assist more than 3.5 million abused and neglected children and their families each year with a range of services.

www.cwla.org

Circle of Parents

Circle of Parents provides a friendly, supportive environment led by parents and other caregivers. It is a place where anyone in a parenting role can openly discuss the successes and challenges of raising children.

www.circleofparents.org

FrameWorks Institute

For several years, the FrameWorks Institute has conducted communications research on how people think about children's issues in general, and child development and parenting in particular.

www.frameworksinstitute.org

The International Society for Prevention of Child Abuse and Neglect

The International Society for Prevention of Child Abuse and Neglect's (ISPCAN) mission is to prevent cruelty to children in every nation, in every form: physical abuse, sexual abuse, neglect, street children, child fatalities, child prostitution, children of war, emotional abuse and child labor. ISPCAN is committed to increasing public awareness of all forms of violence against children, developing activities to prevent such violence, and promoting the rights of children in all regions of the world.

www.ispcan.org

National Alliance of Children's Trust and Prevention Funds

The National Alliance of Children's Trust and Prevention Funds is a membership organization that provides training, technical assistance, and peer consulting opportunities to state Children's Trust and Prevention Funds to strengthen efforts to prevent child abuse.

www.msu.edu/user/nactpf/

National Center on Shaken Baby Syndrome

The National Center on Shaken Baby Syndrome has a mission to educate and train parents and professionals, and to conduct research that will prevent shaking and abuse of infants in the United States. It provides help to professionals and parents looking for information, ideas, and answers to questions about SBS.

www.dontshake.org

The National Children's Advocacy Center Child Abuse Library Online

The Child Abuse Library Online of the National Children's Advocacy Center is one of the largest professional collections of published knowledge, educational materials, and resources related to child maltreatment in the United States. It provides training, online services, and annotated bibliographies to organizations and individuals, and offers resource packages to decisionmakers and researchers.

www.nationalcac.org

National Exchange Club

The National Exchange Club (NEC) Foundation is committed to making a difference in the lives of children, families, and our communities through its national project to prevent child abuse. The NEC Foundation's most successful method of countering abuse is by working directly with parents through the parent aide program. The Foundation coordinates a nationwide network of nearly 100 Exchange Club Child Abuse Prevention Centers that use the parent aide program and provide support to families at risk for abuse.

www.preventchildabuse.com

National Indian Child Welfare Association

The National Indian Child Welfare Association (NICWA) addresses the issues of child abuse and neglect through training, research, public policy, and grassroots community development. NICWA improves the lives of American Indian children and families by helping tribes and other service providers implement activities that are culturally competent, community-based, and focused on the strengths and assets of families.

www.nicwa.org

National Maternal and Child Health Center for Child Death Review: Keeping Kids Alive

This organization promotes, supports, and enhances child death review methodology and activities at the state, community, and national levels. It builds public and private partnerships to incorporate Child Death Review (CDR) findings into efforts that improve child health. Building on the extensive knowledge of current CDR programs, the Center actively involves states in its service development.

www.childdeathreview.org/state.htm

National MCH Center for Child Death Review
The National Center for Child Death Review is a resource center for state and local Child Death Review programs, funded by the Maternal and Child Health Bureau. It promotes, supports, and enhances child death review methodology and activities at the state, community and national levels.
www.childdeathreview.org

National Scientific Council on the Developing Child
The National Scientific Council on the Developing Child is a multi-disciplinary collaboration comprising leading scholars in neuroscience, early childhood development, pediatrics, and economics.
www.developingchild.net

Parents Anonymous® Inc.
Parents Anonymous® Inc. is a community of parents, organizations, and volunteers committed to strengthening families and building strong communities; achieving meaningful parent leadership and shared leadership; and leading the field of child abuse and neglect.
www.parentsanonymous.org

Prevent Child Abuse America
Prevent Child Abuse America works to prevent abuse and neglect of our nation's children. Through its chapters in 43 states and its voluntary home visitation services provided by Healthy Families America® in more than 400 communities nationwide, Prevent Child Abuse America helps provide healthy, safe, and nurturing experiences for more than 100,000 families every year.
www.preventchildabuse.org/index.shtml

Promising Practices Network on Children, Families and Communities
The Promising Practices Network (PPN) is a group of individuals and organizations who are dedicated to providing quality evidence-based information about what works to improve the lives of children, families, and communities.
www.promisingpractices.net

Shaken Baby Alliance
The Shaken Baby Alliance collaborates with community agencies and professionals to provide support for victim families (including adoptive and foster parents) of SBS to advocate justice for SBS victims, and to increase awareness of the problem.
www.shakenbaby.com

Zero to Three
The mission of Zero to Three is to support the healthy development and well-being of infants, toddlers, and their families. The organization accomplishes this by informing, educating, and supporting adults who influence the lives of infants and toddlers.
www.zerotothree.org

The organizations and resources listed in this guide are additional resources only. Their inclusion does not imply endorsement by the U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Injury Prevention and Control.

APPENDIX A:

State Initiatives on SBS

Many states have initiated their own SBS programs. Following is a list of state initiatives; some were compiled by the National Association of Children's Hospitals and Related Institutions (NACHRI) and others were located on the respective state's legislative Web site. As information is continually updated, please visit the NACHRI Web site (www.childrenshospitals.net) and state legislative Web sites for the most up-to-date legislation.

California requires health facilities and licensed midwives to provide information and instructional materials about SBS to parents or guardians of newborns. The law also requires the health department to provide instructional materials regarding the medical risks and ways of preventing SBS, if available, free of charge to child care providers upon licensure and at the time of a site visit.[26]

Florida requires hospitals and birthing facilities to provide new parents with SBS brochures.[27]

Hawaii authorizes hospitals that provide medical care to a newborn to provide each parent of the newborn with written educational information, approved by the department of health and provided by nonprofit organizations, about the dangerous effects of SBS and the different methods of preventing SBS.[28]

Illinois established an SBS program to educate parents and primary caregivers about SBS and provides commitment statements.[29]

[26] California State Legislature [online]. [cited 2010 March 1] Available from URL: http://www.leginfo.ca.gov/cgi-bin/waisgate?WAISdocID=26862315299+0+0+0&WAISaction=retrieve.

[27] National Association of Children's Hospitals and Related Institutions [online]. [cited 2010 March 1] Available from URL: http://www.childrenshospitals.net/AM/Template.cfm?Section=Search3&template=/CM/HTMLDisplay.cfm&ContentID=46204.

[28] Hawaii State Legislature [online]. [cited 2010 March 1] Available from URL: http://www.capitol.hawaii.gov/hrscurrent/Vol06_Ch0321-0344A/HRS0321/HRS_0321-0033.htm

[29] National Association of Children's Hospitals and Related Institutions [online]. [cited 2010 March 1] Available from URL: http://www.childrenshospitals.net/AM/Template.cfm?Section=Search3&Template=/CM/HTMLDisplay.cfm&ContentID=46204

Indiana requires that the State Department, with the assistance of the Department of Child Services, establish a program that raises public awareness concerning the causes and nature of childhood hazards, including factors that place parents, guardians, and other caregivers at risk for shaking an infant, risks associated with shaking an infant, and suggestions for preventing SBS.[30]

Iowa requires the Department of Health to establish a statewide SBS prevention program to educate parents and persons responsible for the care of a child about the dangers to children 3 years old or younger caused by shaking babies and infants.[31]

Massachusetts created a hospital-based program for parents of newborns; education and training programs for parents, caregivers, and professionals; support for victims of SBS and their families; and a surveillance and data collection program to measure the incidence of SBS.[32]

Minnesota mandates distribution of SBS information to new parents at hospitals and training for child care providers.[33]

Missouri stipulates that every hospital and birthing center offer all new parents a viewing of an SBS video prior to discharge.[34]

Montana mandates that the Department of Public Health and Human Services develop educational materials that present readily comprehensible information on SBS and post the materials on the department's Web site in an easily accessible format. The SBS educational materials must be distributed by:

- Childbirth educators and staff of pediatric physicians' offices and obstetricians' offices— to an expectant parent who uses the educator's or physician's services,
- Hospitals— to each newborn child's parent before the child is discharged from the facility,

[30] Indiana General Assembly [online] [cited 2009 Aug 14] Available from URL: http://www.in.gov/legislative/ic/code/title16/ar41/ch40.html

[31] The Iowa General Legislature 2009 Senate Journal Archives [online] [cited 2010 March 1] Available from URL: http://search.legis.state.ia.us/NXT/gateway.dll?f=templates&fn=default.htm&vid=...document-legis...

[32] National Association of Children's Hospitals and Related Institutions [online] [cited 2010 March 1] Available from URL: http://www.childrenshospitals.net/AM/Template.cfm?Section=Search3&template=/CM/HTMLDisplay.cfm&ContentID=46204

[33] Ibid.

[34] Ibid

- Service providers under the MIAMI project—to a child's parent during visits conducted in accordance with that project,
- Child care facilities operating in this state—to each of its employees, and
- Groups or entities that offer classes for babysitters.[35]

Nebraska requires that every hospital, birth center, or other medical facility that discharges a newborn child request that each maternity patient and father of a newborn child, if available, view a video presentation and read printed materials about SBS that are approved by the Department of Health and Human Services and sign a form indicating they have viewed and read or refused to view or read the material.[36]

In addition, the Nebraska Department of Health and Human Services shall conduct public awareness activities designed to promote the prevention of Sudden Infant Death Syndrome (SIDS) and SBS.[37] In addition, the Nebraska Department of Health and Human Services shall adopt and promulgate rules and regulations for mandatory training requirements for providers of child care and school-age-care programs. The training requirements for providers of child care programs shall include, but not be limited to, information on SIDS, SBS, and child abuse.[38]

New York requires every hospital and birth center to ask all new parents to view an SBS video and sign a form indicating they have viewed or refused to watch the video. The requirements also mandate training for child care providers on the identification, diagnosis, and prevention of SBS.[39]

The New York State Department of Health shall develop and implement an ongoing public information and educational campaign to inform the general public about brain injuries and other harmful effects that may result from shaking infants and children under five years of age. The program shall include educational and informational materials in print, audio, video, electronic, and other media and public

[35] Montana Legislature [online] [cited 2010 March 1] Available from URL: http://data.opi.mt.gov/bills/2009/billhtml/SB0442.htm

[36] Nebraska Legislature. Nebraska Revised Statute 71-2103 [online]. [cited 2010 March 1] Available from URL: http://www.legislature.ne.gov/laws/statutes.php?statute=7121003000

[37] Nebraska Legislature. Nebraska Revised Statute 71-2104 [online] [cited 2010 March 1] Available from URL: http://www.legislature.ne.gov/laws/statutes.php?statute=7121004000

[38] Nebraska Legislature. Nebraska Revised Statute 43-2606 [online] [cited 2010 March 1] Available from URL: http://www.legislature.ne.gov/laws/statutes.php?statute=s4326026000

[39] National Association of Children's Hospitals and Related Institutions [online] [cited 2010 March 1] Available from URL: http://www.childrenshospitals.net/AM/Template.cfm?Section=Search3&template=/CM/HTMLDisplay.cfm&ContentID=46204.

service announcements and advertisements. In addition, all schools shall be authorized to include, as an integral part of home economics or health education, instruction regarding child development and parental skills and responsibility. The commissioner shall promulgate rules and regulations to establish a curriculum for instruction regarding child development and parental skills and responsibility for the welfare of pupils and the community to be available in school districts. The curriculum may include instruction relating to the consequences and prevention of SBS, which may include the viewing of a video presentation for students in secondary schools.[40]

Ohio requires the Director of Health to establish the SBS education program and requires the Department of Job and Family Services to record in the statewide automated child welfare information system whether a reported case of child abuse involved SBS.[41]

Pennsylvania mandates hospitals to provide parents with free educational materials on SBS, including a voluntary commitment statement.[42]

Rhode Island requires the Department of Health to collaborate with the Department of Children, Youth, and Families and other state agencies serving families and children, the medical community, law enforcement, human service providers, and child advocacy organizations to develop and implement a comprehensive, statewide initiative to reduce death and disability resulting from SBS.[43]

South Carolina requires that the Department of Health and Environmental Control identify and provide videos on the dangers of shaking infants and information on the importance of infant CPR available to hospitals, child care facilities, child care providers, and the Department of Social Services. Hospitals must request that the maternity patient, father, or primary caregiver view the video. Child care facilities must include this video presentation in the training of the facility's caregivers. The Department of Social Services shall make the video and information on infant CPR available to adopting parents and shall request these parents to view the video.

[40]The New York State Legislature [online] [cited 2010 March 1] Available from URL: http://public.leginfo.state.ny.us/bmlcad.cgi?MENU-14277198

[41]General Assembly of the State of Ohio [online] [cited 2010 March 1] Available from URL: http://www.legislature.state.oh.us/bills.cfm?ID=127_SB_144

[42]National Association of Children's Hospitals and Related Institutions [online] [cited 2010 March 1] Available from URL: http://www.childrenshospitals.net/AM/Template.cfm?Section=Search3&template=/CM/HTMLDisplay.cfm&ContentID=45204

[43]State of Rhode Island Office of Law Revision [online] [cited 2010 March 1] Available from URL: http://www.rilin.state.ri.us/PublicLaws/law06/law06547.htm

In addition, the Department of Health and Environmental Control will establish a protocol for health care providers to educate parents or primary caregivers about the dangers of shaking infants and young children and review these dangers with parents or caregivers associated with shaking infants at well-baby visits.[44]

Tennessee requires that the departments of health and human services shall jointly develop information and instructional materials for distribution, free of charge, to health care facilities, midwives, and child care agencies. The information and instructional materials will focus on the risks of shaking infants and young children. The Department of Health shall be the lead agency in developing such information and instructional materials.

The Department of Health shall provide the information and materials free of charge to health care facilities and nurse midwives. Health care facilities will provide the materials free of charge to parents or guardians of each newborn, upon discharge from the health care facility. If a home birth is attended by a nurse midwife, the nurse midwife shall provide the information and instructional materials to the parents or guardians of the newborn. The Department of Human Services shall provide the information and instructional materials free of charge to child care agencies under the jurisdiction of such department upon licensure and at the time of site visits.[45]

Texas requires licensed daycare facilities to receive education on the identification and prevention of SBS.[46]

Utah requires all daycare centers to receive training on SBS prevention, SIDS prevention, coping with crying babies, and brain development.[47]

[44] The South Carolina Legislature [online]. [cited 2010 March 1] Available from URL: http://www.scstatehouse.gov/cgi-bin/query.exe?first=DOC&querytext=shaking&category=Legislation&session=117&conid=4905947&result_pos=0&keyval=1170518.

[45] Michie's Legal Resources [online]. [cited 2010 March 1] Available from URL: http://www.michie.com/tennessee/lpext.dll?f=templates&fn=main-h.htm&cp=tncode.

[46] National Association of Children's Hospitals and Related Institutions [online]. [cited 2010 March 1] Available from URL: http://www.childrenshospitals.net/AM/Template.cfm?Section=Search3&template=/CM/HTMLDisplay.cfm&ContentID=46204.

[47] Ibid.

Virginia requires information on SBS to be made available to maternity patients by nurse midwives, licensed midwives, and hospitals with maternity services.[48]

Washington mandates that the Council for Children and Families conduct a proactive, public information and communication outreach campaign regarding the dangers of shaking infants and young children, and the causes and prevention of SBS.[49]

Wisconsin stipulates that all new parents, prior to discharge, receive information and watch a videotape on the dangers of SBS. School districts are required to educate grades 5, 8, and 11 on SBS. Licensed child care providers are trained regarding SBS, and at-risk families receive SBS education through the Department of Health and Human Services.[50]

[48] Virginia General Assembly [online] [cited 2010 March 1] Available from URL: http://leg1.state.va.us/cgi-bin/legp504.exe?05+ful+CHAP0519.
[49] Washington State Legislature [online] [cited 2010 March 1] Available from URL: http://apps.leg.wa.gov/RCW/default.aspx?cite=43.121.140
[50] National Association of Children's Hospitals and Related Institutions [online]. [cited 2010 March 1] Available from URL: http://www.childrenshospitals.net/AM/Template.cfm?Section=Search3&template=/CM/HTMLDisplay.cfm&ContentID=46204

APPENDIX B:

Prevention Tips for Parents and Caregivers

- Babies cry a lot in the first few months of life and this can be frustrating. But it will get better.
- Remember, you are **not** a bad parent or caregiver if your baby continues to cry after you have done all you can to calm him or her.
- You can try to calm your crying baby by:
  - Rubbing his or her back,
  - Gently rocking,
  - Offering a pacifier,
  - Singing or talking, or
  - Taking a walk using a stroller or a drive with the baby in a properly-secured car seat.
- If you have tried various ways to calm your baby and he or she won't stop crying, do the following:
  - Check for signs of illness or discomfort like diaper rash, teething, or tight clothing.
  - Call the doctor if you suspect your child is ill.
  - Assess whether he/she is hungry or needs to be burped.
- If you find yourself pushed to the limit by a crying baby, you may need to focus on calming yourself. Put your baby in a crib on his or her back, make sure he or she is safe, and then walk away for a bit and call a friend, relative, neighbor, or parent helpline for support. Check on him or her every 5 to 10 minutes.

- Understand that you may not be able to calm your baby and that it is not your fault, nor your baby's. It is normal for healthy babies to cry much more in the first 4 months of life. It may help to think of this as the *Period of PURPLE Crying*® as identified by the National Center for Shaken Baby Syndrome (NCSBS). For more information about the *Period of PURPLE Crying*® and NCSBS, visit **www.dontshake.org**.
- Tell everyone who cares for your baby about the dangers of shaking a baby and what to do if they become angry, frustrated, or upset when your baby has an episode of inconsolable crying or does other things that caregivers may find annoying, such as interrupting television, video games, sleep time, etc.

- Be aware of signs of frustration and anger among others caring for your baby. Let them know that crying is normal and that it will get better.
- Do not leave your baby in the care of someone you know has anger management issues.
- See a health care professional if you have anger management or other behavioral concerns.

APPENDIX C:

A Partnership Planning Tool

This tool may help you plan your outreach to potential partner organizations and individuals.

Overall Purpose of the Partnership

To identify and collaborate with community-based organizations to raise awareness about Shaken Baby Syndrome and to undertake communication and prevention interventions in the community. (Example)

Brief Description of Partnership Outreach Strategies

Participating Individuals and Organizations

Brief Description of Partnership Outreach Strategies

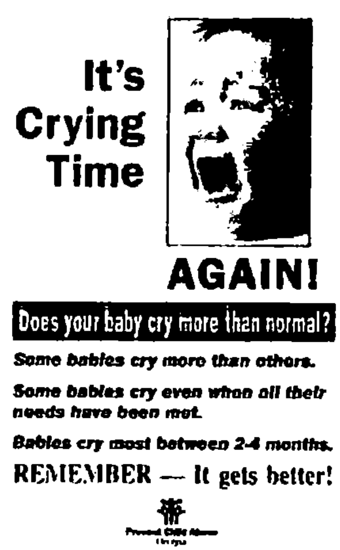
Major Activities/Desired Outcomes of the Partnership

Needed Resources

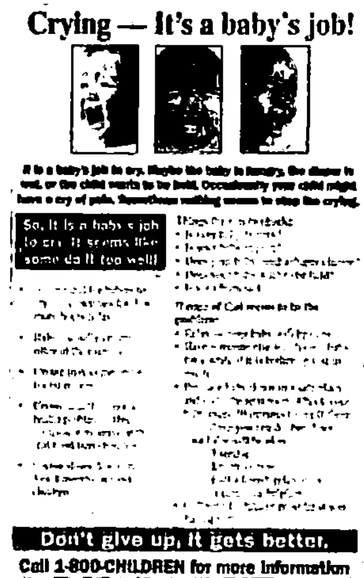
Resource	*Who Provides*	*Details*	*Timing*
Access to new parents			
Communication			
Expertise			
Facility/Venue(s)			
Funding			
Staff			
Other			

Source: These materials were created and produced by Prevent Child Abuse Georgia in collaboration with the Georgia Chapter, American Academy of Pediatrics. For more information or to order these materials, please visit: **www.preventchildabusega.org**.

Front



Back



Front

Babies Cry

Some more than others

Crying is natural.

It's the way babies express themselves.

[illegible]

Crying — It's a baby's job!

Back

Babies Eat, Poop, Sleep, and CRY

- The average newborn cries 2 hours per day.
- Crying increases to 3 hours per day by 3 months of age.
- Babies may cry even when fed, dry, and [illegible].
- Babies usually cry more in the evening.

It's easy to get frustrated
or
to think you've done something wrong.

Crying — It's a baby's job. Your job is to:

[illegible]

Call 1-800-CHILDREN for more information

Front

Crying

"IT" HAPPENS!

[illegible]

Crying last for hours, shaking lasts a lifetime. Stay calm, get help!

Crying — It's a baby's job!

Back

My baby CRIES all the time!

What am I doing wrong?

NOTHING!

Crying is natural.

It's the way babies express themselves.

It doesn't always mean something is wrong.

STRESSED? DEPRESSED? NEED HELP?
TAKE A TIME OUT!
Put your baby in a safe place
and deal with your stress

Call your health care provider if you have any questions.

Call 1-800-CHILDREN for more information

Crying pledge to ______________________

I love you and I promise:

1. To always check to see if you are crying for a reason ____
2. To understand that babies cry sometimes for no good reason ____
3. To be calm if you are simply crying — even if it lasts for hours ____
4. To be proud that you are healthy and strong ____
5. To get help if I feel frustrated ____
6. To teach all of your caregivers about crying and that no one should:
   yell at you ____
   hit you ____
   or shake you ____
7. To sign this pledge and also to have your other caregivers sign it and to keep it for you when you grow up ____

____

signed

Others who love my baby and agree!

____

____

Magnet

These educational resources are provided as example resources only. Their inclusion does not imply endorsement by the U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Injury Prevention and Control.

For more information—as well as radio PSAs and broadcast-quality video that includes B-Roll, full-screen tips, and downloadable scenarios—please visit: **www.cdc.gov/TraumaticBrainInjury**.

"Helping all people live to their full potential"

U.S. Department of Health and Human Services
Centers for Disease Control and Prevention

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A *DAUBERT* ANALYSIS OF ABUSIVE HEAD TRAUMA/SHAKEN BABY SYNDROME

Dr. Sandeep Narang, M.D., J.D.*

Abusive Head Trauma (AHT) has been known over the years by multiple terms—"Whiplash Shaken Baby Syndrome,"[1] "Whiplash Shaken Infant Syndrome," "Shaken Impact Syndrome," "Inflicted Childhood Neurotrauma," "Non-Accidental Trauma," and others. To the lay public, it is most commonly referred to, or recognized as "Shaken Baby Syndrome" (SBS). Irrespective of the vernacular,[2] AHT has long been recognized as a clinically valid medical diagnosis.[3] However, recent legal literature,[4] public media,[5]

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* I am indebted to many for their invaluable assistance in the creation of this document. However, some bear specific recognition. I would specifically like to thank Dr. Betty Spivack, Dr. Chris Greeley, Dr. Alex Levin, Dr. Andy Sirotnak, Dr. Antonia Chiesa, and, most importantly, my friend and mentor, Dr. Don Bross. This article is not only a brief synopsis and testament to the diagnostic genius of our clinical forefathers (Tardieu, Trotter, Caffey, Silverman, Kempe, Guthkelch, and others), but is a salutation of respect and admiration for ALL multidisciplinary colleagues throughout the country who continue to strive for safe, just and equitable outcomes for abused children and their families.

1 This term was one of the earliest descriptive terms of Abusive Head Trauma coined by Dr. John Caffey (often referred to as the Father of Pediatric Radiology). John Caffey, *On the Theory and Practice of Shaking Infants. Its Potential Residual Effects of Permanent Brain Damage and Mental Retardation*, 124 AM. J. DISEASES CHILD. 161, 161–69 (1972).

2 This is not to minimize the recent important shift towards more accurate terminology in describing this medical diagnosis. As noted by one prominent author, "semantic choices play a large role in how concepts spread, are challenged, and evolve. Sometimes what we call something hinders our ability to observe all the available facts clearly and come to a more correct or more encompassing understanding of a particular disease process." *See* Ann-Christine Duhaime, *Calling Things What They Are*, 3 J. NEUROSURGERY: PEDIATRICS 472, 472 (2009).

3 Al-Holou et al., *Nonaccidental Head Injury in Children: Historical Vignette*, 3 J. NEUROSURGERY PEDIATRICS 474, 474 (2009).

EXHIBIT 6

and court decisions have called into question the foundation, and consequent validity, of AHT/SBS as a valid medical diagnosis.[6]

Because of the diagnosis' direct translation and impact in the legal arena, some have gone so far as to champion the cause of its invalidation under philosophical banners of "protection of the innocent" and "justice."[7] Broad assertions and generalizations have been proffered, such as: "the scientific underpinnings of SBS have crumbled over the past decade;"[8] or the medical research underlying SBS is a "flawed science"[9] predicated upon "circular reasoning," "data gaps," and "inconsistency of case definition."[10] Additionally, it has been asserted that "as technology and scientific methodology advanced, researchers questioning the basis for SBS reached a

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[4] *See* Deborah Tuerkheimer, *The Next Innocence Project: Shaken Baby Syndrome and the Criminal Courts*, 87 WASH. U. L. REV. 1, 1 (2009); *see also* Molly Gena, *Shaken Baby Syndrome: Medical Uncertainty Casts Doubt on Convictions*. 2007 WIS. L. REV. 701, 718 (2007).

[5] Emily Bazelon, *Shaken-Baby Syndrome Faces New Questions in Court*, N.Y. TIMES (Feb. 2, 2011), http://www.nytimes.com/2011/02/06/magazine/06baby-t.html?_r=1; Deborah Tuerkheimer, *Anatomy of a Misdiagnosis*, N.Y. TIMES (Sep 20, 2010), http://www.nytimes.com/2010/09/21/opinion/21tuerkheimer.html?ref=opinion; Ari Shapiro, *Foolproof Forensics? The Jury is Still Out*, NPR (Aug. 24, 2009), http://www.npr.org/templates/story/story.php?storyId=112111657.

[6] *See* Cavazos v. Smith, 132 S. Ct. 2, 10 (2011) (per curiam) (Ginsburg, J., dissenting); State v. Edmunds, 746 N.W.2d 590, 596 (Wis. Ct. App. 2008) (granting the defendant/appellant a new trial on the basis defendant presented "newly discovered evidence" of a "significant and legitimate debate in the medical community" regarding Shaken Baby Syndrome, which has emerged in the past ten years); Order Determining Admissibility of Expert Testimony on AHT/SBS at 22-23, Commonwealth v. Davis, No. 04-CR-205 (Ky. Cir. Ct., Apr. 17, 2006); Tuerkheimer, *supra* note 4, at 36 (citing State v. Hyatt, No. 06M7-CR00016-02, (Mo. Cir. Ct Nov. 6, 2007) ("[T]he SBS diagnosis 'appears to have gained considerable acceptance . . . among pediatricians. However, there is substantial, persistent and continuing criticism of this diagnosis among many in the medical and scientific research communities.'"). The American Academy of Pediatrics Section on Child Abuse and Neglect has recently issued a policy statement recommending the use of a more accurate, and less mechanistically constricting, term of "Abusive Head Trauma." *See* Cindy W. Christian et al., *Abusive Head Trauma in Infants and Children*, 123 PEDIATRICS 1409, 1410–11 (2009). Consequently, for the remainder of this article I will refer to the concept of Shaken Baby Syndrome as "Abusive Head Trauma."

[7] *See* Tuerkheimer, *supra* note 4, at 22.

[8] *Id.* at 11.

[9] *Id.* at 12.

[10] *Id.* at 12–13; *see also* Gena, *supra* note 4, at 720.

critical mass."[11]

Despite the assertions, what has not been published thus far is a detailed, critical analysis of the medical literature surrounding AHT, and not only whether that literature meets the Trilogy (*Daubert*, *Joiner*, and *Kumho*) criteria for admissibility of scientific evidence/testimony, but whether that literature is "flawed" and consequently not predicated upon sound scientific and medical principles.[12] Part I of this paper shall examine the Trilogy (*Daubert*, *Joiner*, and *Kumho)* criteria for admissibility of expert testimony/evidence, and the medical and legal quests for sound scientific evidence. Part II of this paper shall explore the issues surrounding the medical diagnosis of AHT. Specifically, we shall review basic statistical principles utilized in critical evaluation of medical/scientific literature and then critically analyze the medical literature involving some of the more common injuries[13] associated with AHT. Finally, Part III of this paper shall assess not only whether the medical literature suffices under *Daubert*, *Joiner*, and *Kumho* scrutiny, but shall briefly examine the contemporary legal

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11 Tuerkheimer, *supra* note 4, at 14; *see also* Gena, *supra* note 4, at 710.

12 The American Academy of Pediatrics provides a general assessment of the topic, but its purpose was not intended to be a critical analysis of the literature on the topic. American Academy of Pediatrics, *Shaken Baby Syndrome: Rotational Cranial Injuries – Technical Report*, 108 PEDIATRICS 206, 206 (2001). One other article has been proffered, and frequently cited by opponents of Abusive Head Trauma, to be a critical review of the literature on the topic. Mark Donohoe, *Evidence-Based Medicine and Shaken Baby Syndrome*, 24 AM. J. FORENSIC MED. & PATHOLOGY 239, 239 (2003). A critical evaluation of that article will be conducted in detail herein below.

13 Abusive Head Injury/Shaken Baby Syndrome entails a wide constellation of symptoms and injuries with varying degrees of severity. The most common injuries associated with this diagnosis are intracranial hemorrhage (most commonly subdural or subarachnoid hemorrhage) and retinal hemorrhages. *See* Antonia Chiesa & Ann-Christine Duhaime, *Abusive Head Trauma*, 56 PEDIATRIC CLINICS N. AM. 317 (2009). While many other injuries are associated with this diagnosis, this paper will focus on the *clinical medical* literature behind the most common injuries—subdural hemorrhage and retinal hemorrhages. A thorough examination of the literature behind all the possible injuries and all potential causes (short falls, biomechanics of head injury, etc.) is simply too broad and beyond the scope of this paper. For a more comprehensive examination of the literature on this topic, I would reference the reader to LORI FRASIER ET AL., ABUSIVE HEAD TRAUMA IN INFANTS & CHILDREN: A MEDICAL, LEGAL, AND FORENSIC REFERENCE (2006). *See also* Lucy Rorke-Adams et al., *Head Trauma*, *in* CHILD ABUSE: MEDICAL DIAGNOSIS & MANAGEMENT 53 (Robert M. Reece & Cindy W. Christian eds. 2009).

issues surrounding admissibility of AHT testimony and proffer some solutions for those issues.

I. THE TRILOGY: *DAUBERT, JOINER,* AND *KUMHO*

A. *Daubert v. Merrell Dow Pharmaceuticals, Inc.*

For many years in the twentieth century, expert testimony on novel scientific evidence was admissible only if the opinion offered was based on a "well-recognized scientific principle or discovery . . . [that was] sufficiently established to have gained general acceptance in the particular field in which it belongs."[14] That standard, enunciated in *Frye v. United States,* was also known as the "general acceptance" test.[15] In 1993, with the Supreme Court's ruling in *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, that standard changed.[16]

In *Daubert,* the Supreme Court evaluated the propriety of a lower court's ruling excluding certain expert testimony in a tort liability case.[17] In the case, Petitioners Jason Daubert and Eric Schuller were minor children born with serious birth defects.[18] They and their parents had sued the respondent, Merrell Dow Pharmaceuticals, alleging that the birth defects were caused by the mother's ingestion of Merrell Dow's drug, Bendectin (an antinausea medication).[19] The Petitioners sought to proffer expert testimony.[20] The district court, applying the "general acceptance" test of *Frye,* denied the admissibility of the petitioner's expert testimony, and granted summary judgment for the respondent.[21] To settle the divisions among the lower courts regarding the proper standard for the admission of expert testimony, the Supreme Court granted

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14 Frye v. United States, 293 F. 1013, 1014 (D.C. Cir. 1923).

15 Daubert v. Merrell Dow Pharm., Inc., 509 U.S. 579, 589 (1993).

16 *Id.*

17 *Id.* at 584–85.

18 *Id.* at 582.

19 *Id.*

20 *Id.* at 583.

21 *Id.* at 584–85.

certiorari.[22]

The Court held unanimously that the *Frye* test had not survived.[23] With regards to the admissibility of expert testimony/evidence, the Court held that Federal Rules of Evidence (FRE) 702 governs, not *Frye*.[24] The *Daubert* court held the text of FRE 702, its drafting history, and prior case law[25] mandated a "liberal" and "relaxed" approach to the admission of expert opinion testimony.[26] The inquiry into admission of expert testimony/evidence was within the province of the trial judge. While the trial judge's inquiry was to be a "flexible one,"[27] the *Daubert* court required trial judges to ensure "that any and all scientific testimony or evidence admitted is not only relevant, but reliable."[28]

With regards to reliability, the *Daubert* Court stated that "[t]he subject of an expert's testimony must be 'scientific... knowledge.'"[29] The Court noted there were definitional differences between science and law on "reliability."[30] But the Court went on to state that "*evidentiary reliability* will be based upon *scientific validity*."[31] The Court enunciated four factors a trial judge could consider in the preliminary assessment of whether proposed testimony was scientifically valid:

1) whether a theory or technique could be (and had been) tested—also known as "falsifiability" or "testability";
2) whether the theory or technique had been subject to peer review and publication;
3) whether there was a known or potential rate of error; and

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22 *Id.* at 585.

23 *Id.* at 589; *id.* at 598 (Rehnquist, C.J., concurring in part and dissenting in part).

24 *Id.*

25 *E.g.* Beech Aircraft Corp. v. Rainey, 488 U.S. 153, 169 (1988).

26 *Daubert*, 509 U.S. at 588.

27 *Id.* at 594.

28 *Id.* at 589.

29 *Id.* at 589–90.

30 *Id.* at 590 n.9.

31 *Id.*

4) whether there was general acceptance in the relevant scientific community.[32]

The Court remarked that these factors were not a "definitive checklist or test," but merely factors for consideration in a trial judge's overall assessment.[33] The Court concluded by stating, "[t]he inquiry envisioned by Rule 702 is, we emphasize, a flexible one . . . . *The focus, of course, must be solely on principles and methodology*, not on the conclusions that they generate."[34]

With regards to relevance, the Court explained that expert testimony cannot assist the trier of fact in resolving a factual dispute, as required by Rule 702, unless the expert's theory is "sufficiently tied to the facts of the case."[35] The Court remarked, "Rule 702's 'helpfulness' standard requires *a valid scientific connection* to the pertinent inquiry as a precondition to admissibility."[36]

B. *General Electric Co. v. Joiner*

In *General Electric Co. v. Joiner*, the Court, in expanding upon the *Daubert* standard, examined and decided two additional, significant issues regarding the admissibility of scientific expert testimony.[37] First, the Court determined the appropriate standard for appellate review of a trial court's determination of admissibility of scientific expert testimony. After establishing an abuse of discretion standard for appellate review,[38] the Court went on to examine a more important issue of whether existing scientific evidence can be generalized to address specific causal relationships.[39]

In *Joiner*, the plaintiff asserted that exposure to polychlorinated biphenyls had promoted the development of his small-cell lung

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32 *Id.* at 593–94.

33 *Id.* at 593.

34 *Id.* at 594–95 (emphasis added).

35 *Id.* at 591.

36 *Id.* at 591–92 (emphasis added)

37 Gen. Elec. Co. v. Joiner, 522 U.S. 136, 138–39 (1997).

38 *Id.* at 141.

39 *See* Joe S. Cecil, *Ten Years of Judicial Gatekeeping Under* Daubert, 95 AM. J. PUB. HEALTH s74, s75 (Supp. 2005).

cancer.[40] The plaintiff argued that *collective* consideration of epidemiologic studies (which, when considered individually and separately, were equivocal), demonstrated a causal relationship.[41] In rejecting this argument, the Court determined the lower court had not abused its discretion in excluding this scientific testimony because there was no logical nexus between the methodology employed by the expert and the expert's conclusion.[42] The Court stated:

> Trained experts commonly extrapolate from existing data. But nothing in either *Daubert* or the Federal Rules of Evidence requires a district court to admit opinion evidence that is connected to existing data *only by the ipse dixit of the expert. A court may conclude that there is simply too great an analytical gap between the data and the opinion proffered.*[43]

C. *Kumho Tire Co. v. Carmichael*

In *Kumho Tire Co. v. Carmichael*, the Court examined the issue of the *extent* of a trial court's "gate-keeping" obligation.[44] Did it extend only to expert testimony based upon "scientific" knowledge or did it also apply to expert testimony based on "technical" and/or "other specialized knowledge"? In unanimously holding that a trial court's "gate-keeping" obligation extended to ALL expert testimony, the Court remarked that Federal Rule of Evidence 702 "makes no relevant distinction between 'scientific' knowledge and 'technical' or 'other specialized' knowledge."[45] Assurance of reliability of expert testimony, whether "scientific" or based upon "technical or other specialized knowledge," was still required.[46]

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[40] *Joiner*, 522 U.S. at 139.

[41] *See* Cecil, *supra* note 39, at s76.

[42] *Joiner*, 522 U.S. at 146–47.

[43] *Id.* at 146 (emphasis added).

[44] Kumho Tire Co. v. Carmichael, 526 U.S. 137, 141 (1999).

[45] *Id.* at 147.

[46] *Id.* at 149.

In grappling with this issue, the Court remarked that there will be witnesses "whose expertise is based purely on experience. . . ."[47] The Court anticipated there would be times when such proffered expert testimony would have to be excluded because the expert's field lacks reliability.[48] But other than citing astrology and necromancy as such excludable disciplines, the Court gave no specific guidance on how a trial court could come to such a conclusion.[49] Instead, the Court proffered general guidance—the "intellectual rigor" test.[50]

The Court noted that the four *Daubert* factors "may or may not be pertinent[: it will all depend] on the nature of the issue, the expert's particular expertise, and the subject of his testimony."[51] The Court concluded that a trial court must exercise its gate-keeping obligation so that the expert, whether relying on "professional studies or personal experience, . . . [will, when testifying, employ] the same level of intellectual rigor" that the expert would use outside the courtroom when working in the relevant discipline.[52] In the words of one legal scholar:

> The Court seems less absorbed in epistemological issues, in formulating general rules for assessing reliability, or in fleshing out

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47 *Id.* at 151.

48 *See id.*

49 *Id.*

50 *See id.* at 152.

51 *Id.* at 150 (quoting Brief for United States as *Amicus Curiae* Supporting Petitioners at 19, Kumho Tire Co. v. Carmichael, 526 U.S. 137 (1999)). Some legal scholars commented that the Court's decision in *Kumho* sought to rectify a bias in *Daubert* towards the "hard sciences" which employ rigorous empirical methods. *See* Paul S. Milich, *Controversial Science in the Courtroom* 43 EMORY L.J. 913, 917 (1994) ("*Daubert* . . . never mentions the psychological sciences, for example, where much of the data is subjective and many of the theories are empirically difficult, if not impossible, to verify"); *see also* Ralph Underwager & Hollida Wakefield, *A Paradigm Shift for Expert Witnesses*, ISSUES IN CHILD ABUSE ACCUSATIONS, Summer 1993, http://ipt-forensics.com/journal/volume5/j5_3_2.htm ("American psychiatry is, by and large, Freudian in its orientation" and "wherever Freudian theory has been subjected to empirical tests, it has either failed, or, at best, been inconclusive as a predictor of human behavior."). Yet psychiatry is a recognized science readily integrated into and accepted by the criminal justice system when issues of mental competency arise.

52 *Kumho*, 526 U.S. at 152.

> the implications of its having singled out testability as the preeminent factor of concern. It appears less interested in a taxonomy of expertise and more concerned about directing judges to concentrate on "the particular circumstances of the particular case at issue." This flexible, nondoctrinaire approach is faithful to the intention of the drafters of the Federal Rules of Evidence . . . .[53]

Essentially, for physicians, the Court's decision in *Kumho* "tethered" the admissibility standard of expert testimony to the standards of medical practice.[54]

D. The Quest for Sound "Scientific Evidence/Testimony"

> *"Science is simply common sense at its best; that is, rigidly accurate in observation and merciless to a fallacy in logic."*[55]
>
> Thomas Henry Huxley

1. *The Legal Perspective*

The objective of law is justice.[56] Yet, justice is not merely the search for dispassionate truth, but dispassionate truth that results in fair and equitable decisions.[57] As the age of science has flourished, science and medicine have increasingly permeated the law and played crucial roles in the courtroom.[58]

In criminal law, the emergence of DNA sampling has resulted in the exoneration of those who were unjustly convicted and has

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53 *See* Margret Berger, *The Supreme Court's Trilogy on the Admissibility of Expert Testimony*, *in* FED. JUDICIAL CTR., REFERENCE MANUAL ON SCIENTIFIC EVIDENCE 9, 21 (2d ed. 2000), www.fjc.gov/public/pdf.nsf/lookup/sciman00.pdf/$file/sciman00.pdf.

54 *See* Jerome Kassirer & Joe Cecil, *Inconsistency in Evidentiary Standards for Medical Testimony: Disorder in the Courts*, 288 JAMA 1382, 1383 (2002).

55 FED. JUDICIAL CTR., REFERENCE MANUAL ON SCIENTIFIC EVIDENCE, at v (2d ed. 2000) (quoting T.H. HUXLEY, THE CRAYFISH: AN INTRODUCTION TO THE STUDY OF ZOOLOGY 2 (1880)).

56 D. Allen Bromley, *Science and the Law*, Address at the 1998 Annual Meeting of the American Bar Association (Aug. 2, 1998).

57 Stephen Breyer, *Introduction*, FED. JUDICIAL CTR., REFERENCE MANUAL ON SCIENTIFIC EVIDENCE 2, 4 (2d ed. 2000), www.fjc.gov/public/pdf.nsf/lookup/sciman00.pdf/$file/sciman00.pdf.

58 *Id.* at 3.

provided greater confidence in the reliability of future convictions.[59] In tort law, courts are constantly confronted with causation or risk of injury determinations, which rely heavily on scientific or medical information.[60] In patent law, cases are heavily immersed in, and decisions frequently hinge upon, technical or scientific information.[61] And, in recent years, the Supreme Court has examined scientific and medical issues ranging from the propriety of statistical sampling techniques in the undercounting of certain identifiable groups on the decennial census,[62] to the constitutionality of a state psychopath statute,[63] to the constitutional question of whether the right to liberty in the Due Process Clause of the Fourth Amendment affords citizens a "right to die."[64]

As our scientific world has grown increasingly complex, courts have become increasingly wary of exposing juries to such potentially confusing evidence. Additionally, courts have recognized the inherent weight and persuasiveness the designation of "scientific evidence" can have in the minds of triers of fact. Bolstering that concern, some research suggests that as evidence becomes more complex and difficult to comprehend, jurors shift their focus to "peripheral indicia of reliability such as the expert's qualifications or demeanor," and are more likely to defer to the expert's opinion rather than forming their own.[65] This deference to

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59 *See id.*

60 *See id.*

61 *Id.*

62 Dep't of Commerce v. U.S. House of Representatives, 525 U.S. 316, 320 (1999); Breyer, *supra* note 57, at 2.

63 Kansas v. Hendricks, 521 U.S. 346, 350 (1997); Breyer, *supra* note 57, at 3.

64 Washington v. Glucksberg, 521 U.S. 702, 722 (1997); Vacco v. Quill, 521 U.S. 793, 797 (1997); Breyer, *supra* note 57, at 3.

65 *See* THE LAW COMM'N, CONSULTATION PAPER 190, THE ADMISSIBILITY OF EXPERT EVIDENCE IN CRIMINAL PROCEEDINGS IN ENGLAND AND WALES: A NEW APPROACH TO THE DETERMINATION OF EVIDENTIARY RELIABILITY, ¶ 2.8 n.6, ¶ 2.28 (2009), www.lawcom.gov.uk/docs/cp190.pdf (discussing how M. Redmayne, in *Expert Evidence and Criminal Justice*, "summarizes research which suggests that as expert evidence becomes more complicated, jurors shift their focus and rely on peripheral indicia of reliability"); *see also id.* at ¶ 2.3 (citing PAUL ROBERTS & A.A.S. ZUCKERMAN, CRIMINAL EVIDENCE 292-96 (2004)).

scientific evidence has been labeled by some courts as the "aura of infallibility."[66] Furthermore, a few recent case reports of wrongful convictions have exacerbated those concerns of juror over-reliance on "scientific evidence."[67]

Nevertheless, in hopes of diminishing the admission of unreliable testimony, courts and legal scholars, both domestic and international, have endeavored to define sound scientific evidence. The *Daubert* Court stated:

> The adjective "scientific" implies a grounding in the methods and procedures of science.... "Science is not an encyclopedic body of knowledge about the universe. Instead, it represents a *process* for proposing and refining theoretical explanations about the world that are subject to further testing and refinement" .... Proposed testimony must be supported by appropriate *validation – i.e., "good grounds,"* based on what is known.[68]

In the words of one learned commentator, evidence is scientifically valid if "it results from sound and cogent reasoning."[69] Other scholars, echoing the Court's decisions in *Daubert* and *Kumho* state, "[i]t is *how* conclusions are reached, not *what* the conclusions are, that makes them 'good science.'"[70] In the words of the Honorable Stephen Breyer, Associate Justice of the Supreme Court:

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66 *See* U.S. v. Addison, 498 F.2d 741, 744 (1974) (The Court stated, "scientific proof may in some instances assume a posture of mystic infallibility in the eyes of a jury of laymen"); *see also* John William Strong, *Language and Logic in Expert Testimony*, 71 OR. L. REV. 349, 367–68 n.81 (1992) ("There is virtual unanimity among courts and commentators that evidence perceived by jurors to be 'scientific' in nature will have particularly persuasive effect.").

67 *See* STEPHEN T. GOUDGE, INQUIRY INTO PEDIATRIC FORENSIC PATHOLOGY IN ONTARIO 531 (Ontario Ministry of the Att'y Gen. 2008); *see also* THE LAW COMMISSION, CONSULTATION PAPER 190, *supra* note 65, at ¶¶ 2.14–2.22 (2009). (citing three recent AHT/SBS cases in England and Wales where criminal convictions were obtained and subsequently overturned on appeal because of "flawed" scientific evidence/testimony). *But see* Neil Vidmar & Shari Seidman Diamond, *Juries and Expert Evidence*, 66 BROOKLYN L. REV. 1121, 1179 (2001) ("Empirical data do not support a view that juries are passive, too-credulous, incompetent, and overawed by the mystique of the expert.").

68 Daubert v. Merrell Dow Pharm., Inc., 509 U.S. 579, 590 (1993) (second emphasis added).

69 Bert Black, *A Unified Theory of Scientific Evidence*, 56 FORDHAM L. REV. 595, 599 (1988); *see also* Bert Black et al., *Science and the Law in the Wake of* Daubert*: A New Search for Scientific Knowledge*, 72 TEX. L. REV. 715, 753 (1994).

70 Clifton T. Hutchinson & Danny S. Ashby, *Redefining the Bases of Admissibility of Expert Scientific Testimony*, 15 CARDOZO L. REV. 1875, 1886 (1994).

> The search is not a search for scientific precision.... A judge is not a scientist, and a courtroom is not a scientific laboratory. But consider the remark made by the physicist Wolfgang Pauli. After a colleague asked whether a certain scientific paper was wrong, Pauli replied, *"That paper isn't even good enough to be wrong!" Our objective is to avoid legal decisions that reflect that paper's so-called science. The law must seek decisions that fall within the boundaries of scientifically sound knowledge.*[71]

In the United Kingdom, the Law Commission recently proposed reformation of English Law with regards to admissibility of expert scientific evidence.[72] After a comprehensive review of the topic, the Commission found the *Daubert* court's analysis and conclusions regarding the admissibility of expert scientific testimony and evidence to be cogent, sound, and, ultimately, convincing.[73] Noting that many judges in England and Wales were already making admissibility decisions based upon the *Daubert* standard, the Commission recommended formal adoption of *Daubert*'s "gate-keeping" role for a trial judge and *Daubert*'s validity-based (reliability and relevance) admissibility test for expert scientific evidence.[74]

Although many have judged the trilogy (*Daubert*, *Joiner* and *Kumho*) to be a laudable attempt to bridge the treacherous crosscurrents of science and law, numerous issues regarding the determination of "sound scientific testimony" have remained unanswered. For example, with regards to the "analytical gap" between research data and expert opinion addressed in *Joiner*,[75] what is a sufficient amount and quality of evidence an expert may rely upon in bridging that "gap" in forming his/her opinion? Are medical textbooks (which are essentially expert treatises) authoritative references upon which experts may rely in forming their opinions? With regards to the "intellectual rigor" test of *Kumho*, what will be the applicable standard of professional practice to apply when, as often occurs in medical practice, multiple disciplines

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71 Breyer, *supra* note 57, at 4 (emphasis added).

72 *See* THE LAW COMM'N, *supra* note 65, at ¶ 1.5.

73 *Id.* at 47.

74 *Id.* at 49–51.

75 Gen. Elec. Co. v. Joiner, 522 U.S. 136, 146 (1997).

are involved? Who determines the applicable standard of professional practice? Individual experts? National organizations? Additionally, some have echoed concerns about the onerous burden *Daubert*'s gate-keeping requirements have placed on the single trial judge.[76] As the Honorable Judge Alex Kozinski of the Ninth Circuit Court of Appeals stated:

> Our responsibility, then, unless we badly misread the Supreme Court's opinion, is to resolve disputes among respected, well-credentialed scientists about matters squarely within their expertise, in areas where there is no scientific consensus as to what is and what is not "good science," and occasionally to reject such expert testimony because it was not "derived by the scientific method." Mindful of our position in the hierarchy of the federal judiciary, we take a deep breath and proceed with this heady task.[77]

Empirical evidence has substantiated Judge Kozinski's concerns. In a 2001 survey of 400 state court judges, 96% of the judges failed to demonstrate even a basic understanding of two of the four *Daubert* criteria.[78] When assessing the concept of "falsifiability," a principle specifically enunciated in *Daubert*, 96% of

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[76] *See* Daubert v. Merrell Dow Pharm., Inc., 509 U.S. 579, 600 (Rehnquist, C.J., concurring in part and dissenting in part). In response to this concern, the Federal Judicial Center, the research and educational arm of the federal judicial system, has published a 1034-page reference source (currently in its third edition) to help federal judges "manage cases involving complex scientific and technical evidence." *See* FEDERAL JUDICIAL CENTER, REFERENCE MANUAL ON SCIENTIFIC EVIDENCE, at xv (3d ed. 2011), http://www.fjc.gov/public/pdf.nsf/lookup/SciMan3D01.pdf/$file/SciMan3D01.pdf. For other comprehensive references on the issues surrounding Science, Law, and Expert testimony, see generally 1 MODERN SCIENTIFIC EVIDENCE: THE LAW AND SCIENCE OF EXPERT TESTIMONY (David L. Faigman et al. eds., 1997); EXPERT EVIDENCE: A PRACTITIONER'S GUIDE TO LAW, SCIENCE, AND THE FJC MANUAL (Bert Black & Patrick W. Lee eds., 1997).

[77] Daubert v. Merrell Dow Pharm., Inc., 43 F.3d 1311, 1316 (9th Cir. 1995). However, trial judges have adapted to the heady responsibility of the trilogy decisions by utilizing innovative case-management techniques, such as court-appointed independent experts or court-appointed scientific panels, to assist with the comprehension of complex scientific information. Furthermore, public and private organizations, such as the American Association for the Advancement of Science (AAAS), have offered trial judges the service of locating impartial, skilled experts at fee-for-service costs. *See Court Appointed Scientific Experts*, AM. ASS'N FOR THE ADVANCEMENT OF SCI., http://www.aaas.org/spp/case/case.htm (last visited Oct. 21, 2011).

[78] Sophia I. Gatowski et al., *Asking the Gatekeepers: A National Survey of Judges on Judging Expert Evidence in a Post-Daubert World*, 25 L. & HUM. BEHAV. 433, 442–47 (2001).

the judges lacked even a basic understanding of this core scientific concept.[79] When asked to comment on the value of *Daubert* to their decision-making process, only 55% of judges found *Daubert* to provide a "great deal" of value.[80] Consequently, the researchers concluded that "[t]he survey findings strongly suggest that judges have difficulty operationalizing the *Daubert* criteria and applying them . . . ."[81]

Expectedly, the courts have grappled with confusion and responded with variable and inconsistent decisions. Some courts have attempted to reduce determinations of sound scientific evidence to "simple all-or-nothing rules, such as . . . doubling . . . the background rate of disease as proof of causality."[82] Some have required peer-reviewed studies[83] or statistical data[84] prior to admitting expert testimony. Some have dismissed case reports as non-scientific,[85] whereas other courts have given them significant weight.[86] Finally, some courts have disallowed expert testimony when such reliance was based primarily upon "animal studies[, have] cautioned against extrapolation of dosage levels, and [have] objected to generalization across similar substances."[87]

Whereas courts once greeted scientific evidence and testimony with deferential respect and relative trust, recent empirical data demonstrates that the legal pendulum has swung the other way. An

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79 *Id.* at 444–45.

80 *Id.* at 443.

81 *Id.* at 452.

82 Kassirer & Cecil, *supra* note 54, at 1384. "This approach was urged by the United States Court of Appeals for the Ninth Circuit when it reconsidered the *Daubert* case." *Id.*

83 *Id.* (citing Jones v. United States, 933 F. Supp 894, 897 (N.D. Cal. 1996)).

84 *Id.* (citing Raynor v. Merrell Pharm. Inc., 104 F.3d 1371, 1375 (D.C. Cir 1997)).

85 *See* Haggerty v. Upjohn Co, 950 F. Supp. 1160, 1165 (S.D. Fla. 1996); Hall v. Baxter Healthcare Corp, 947 F. Supp. 1387, 1411 (D. Or. 1996).

86 *See* Pick v. Am. Med. Sys., Inc., 958 F. Supp. 1151, 1160–62 (E.D. La. 1997); Glaser v. Thompson Med. Co., 32 F.3d 969, 975 (6th Cir. 1994); Cella v. United States, 998 F.2d 418, 426 (7th Cir. 1993).

87 *See* Cecil, *supra* note 39, at s76 (citing Newman v. Motorola Inc., 218 F. Supp. 2d 769, 780–81 (D. Md. 2002); Amorgianos v. Nat'l R.R. Passenger Corp., 137 F. Supp.2d 147, 189 (E.D.N.Y. 2001); Mitchell v. Gencorp Inc., 165 F.3d 778, 782 (10th Cir. 1999)).

"analysis by the Rand Corporation of a sample of 399 published and unpublished federal district court decisions" demonstrated a more restrictive approach by federal courts to the admissibility of scientific testimony and a shift "toward excluding proffered scientific and technical evidence."[88] Additionally, a recent survey of federal judges and attorneys by the Federal Judicial Center "confirmed a shift toward more demanding standards for admissibility" of scientific testimony and evidence.[89] In the words of one learned commentator, "[t]he courts appear to be asserting standards that they attribute to the medical profession, but that are inconsistent and sometimes more demanding than actual medical practice."[90]

2) The Medical Perspective

If the objective of law is justice, then the objective of medicine is to care for the patient. To truly understand the medical perspective, one must understand and accept the canon that medicine is inherently, by its nature, an inexact science.[91] There are aspects of medicine (for example laboratory research), which are more scientific in nature. But the fields of medicine that deal with direct patient interaction, also known as clinical medicine, are not exclusively scientific. The human interaction inherently introduces variables (such as the nuances of effective communication and an individual's behavioral, social, economic, and cultural norms and biases) that are not readily reducible to empirical scientific data and most certainly affect the outcome. The medical provider's judicious interplay of the human variable with the scientific data of the

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[88] *See* Cecil, *supra* note 39, at s75. This data is in contrast to one author's assertion of judicial deference to admissibility of testimony on Abusive Head Trauma/Shaken Baby Syndrome. *See* Tuerkheimer, *supra* note 4, at 42–44.

[89] *See* Cecil, *supra* note 39, at s75.

[90] *See* Kassirer & Cecil, *supra* note 54, at 1382.

[91] *See* Mary Sue Henifin et al., *Reference Guide on Medical Testimony*, *in* FED. JUDICIAL CTR., REFERENCE MANUAL ON SCIENTIFIC EVIDENCE 439, 465 (2d ed., 2000), http://www.fjc.gov/public/pdf.nsf/lookup/sciman00.pdf/$file/sciman00.pdf; *see also* JEROME GROOPMAN, HOW DOCTORS THINK 7 (2007).

human body is what has been termed by many as the art[92] of clinical medicine.[93]

It is important to understand that the designation of an "art" is not a relegation to imprecision or lack of reliability. On the contrary, clinical medical decision-making is grounded in the roots of the scientific method. As Dr. Mark McClellan, Co-Chair of Institute of Medicine's 2007 Annual Meeting, stated, "[physicians'] education includes the scientific basis of health and disease. They have been trained to use scientific literature to compare alternative approaches to diagnosis and treatment. They do their best to stay up-to-date through reading and conferences."[94] Additionally, physicians receive basic training on statistical analysis, often apply those principles to critically evaluate the medical literature, and sometimes pursue advanced degrees in statistical expertise (like biostatistics or epidemiology).

While the cognitive underpinnings of the diagnostic process are rational and scientifically sound, ultimately, "[a]ll diagnostic hypotheses represent probabilistic judgments . . . that have variable probabilities of being correct."[95] Furthermore, physicians are as susceptible as anyone to biases, preconceptions, or "intrusions of emotion," any or all of which can influence clinical judgment and actions.[96] Physicians can, and do, avoid, or at least minimize, errors in cognition by maintaining awareness of the pitfalls of heuristics, and how personal biases and emotional temperature can affect them.[97]

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92 MARK B. MCCLELLAN ET AL., EVIDENCE-BASED MEDICINE AND THE CHANGING NATURE OF HEALTH CARE: 2007 IOM ANNUAL MEETING SUMMARY 94 (Nat'l Acad. of Scis. 2008).

93 Some prefer to refer to this as an "applied science" rather than an "art." *See* Harriet Hall, *The "Art" of Clinical Decision-Making*, SCIENCE-BASED MEDICINE (May 13, 2008), http://www.sciencebasedmedicine.org/index.php/the-art-of-clinical-decision-making/.

94 *See* McClellan et al., *supra* note 92, at 94.

95 *See* Henifin et al., *supra* note 91, at 465.

96 *See* GROOPMAN, *supra* note 91, at 37; *see also* Pat Croskerry, *The Importance of Cognitive Errors in Diagnosis and Strategies to Minimize Them*, 78 ACAD. MED. 775, 775 (2003); Pat Croskerry, *Achieving Quality in Clinical Decision Making: Cognitive Strategies and Detection of Bias*, 9 ACAD. MED. 1184, 1184 (2002).

97 GROOPMAN, *supra* note 91, at 35–36, 39.

Physicians have continually reflected upon the clinical decision-making process, repeatedly assessing its cogency and need for improvement.[98] As technologic advancements in medical informatics occurred in the 1970s and 1980s, large volumes of medical literature were synthesized into computer indices and became available for large-scale statistical analysis.[99] This bred a new type of medical evidence, the systematic review.[100] On the heels of these technologic innovations, and the consequent ability to conduct comprehensive reviews of large volumes of medical literature, the Evidence-Based Medicine (EBM) movement came afoot.[101]

EBM has been characterized by one of its pioneers, Dr. David Sackett, as the "conscientious, explicit, and judicious use of current best evidence in making decisions about individual care."[102] Dr. Harvey Fineberg, President of the Institute of Medicine, recently stated that, "[t]he central notion in EBM [is] the importance of *integrating individual clinical expertise with the best available external evidence.*"[103] This will provide "a helpful framework for providers to navigating uncertainty inherent in patient care."[104] In fact, most healthcare providers strive to be "evidence-based" in their

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[98] *See* Croskerry, *The Importance*, *supra* note 96, at 776; Croskerry, *Achieving Quality*, *supra* note 96, at 1184.

[99] *See About Us*, THE COCHRANE COLLABORATION, http://www.cochrane.org/about-us (last visited Jan. 24, 2012); *Happy 35th Birthday, MedLine!*, U.S. NAT'L LIBRARY MED., http://www.nlm.nih.gov/news/medline_35th_birthday.html (last updated Oct. 23, 2006) (showing the Medline database was founded in 1971).

[100] *See History of Systematic Reviews*, EPPI-CENTRE, http://eppi.ioe.ac.uk/cms/Default.aspx?tabid=68 (last visited, Jan. 24, 2012).

[101] *See id.*

[102] David Sackett, et al., *Evidence Based Medicine: What It Is and What It Isn't: It's About Integrating Individual Clinical Expertise and the Best External Evidence*, 312 BRIT. MED. J. 71, 71 (1996). The determination of what the "current best evidence" is in a given field requires a critical evaluation of the relevant medical literature, utilizing statistical principles to assess the validity of studies and the conclusions they reach. *See id.* at 72. We will discuss basic principles of statistical analysis herein below when we critically evaluate the "current best evidence" in the field of Abusive Head Trauma. *See also* McClellan et al., *supra* note 92, at *v*.

[103] McClellan et al., *supra* note 92, at *v* (emphasis added).

[104] *Id.*

practice.[105]

Despite an increased focus on "evidence basis" in their practice, "studies repeatedly show marked variability in what healthcare providers actually do in a given [clinical] situation."[106] Many had hoped that EBM would be the panacea to the judicial pains over medical practice guidelines and interpretation of medical evidence. However, as lingering controversies between reputed medical bodies[107] demonstrate, it has not been that panacea.[108] Additionally, there are some areas of medicine, where the evidence is so sparse, that EBM simply cannot be instructive either for Medicine or Law.[109]

Ultimately, the physician must sagely balance his scientific knowledge, underscored by statistical data, his emotional temperature and potential biases, and the myriad complexities that make up the "human" variable. "Statistics cannot substitute for the human being before you; statistics embody averages, not individuals. Numbers can only complement a physician's personal experience . . . ."[110] That is the "Art" of Clinical Medicine. Explicit evidence is only a portion of what physicians do.

II. ABUSIVE HEAD TRAUMA AS A MEDICAL DIAGNOSIS

"Those who cannot remember the past are condemned to repeat it."[111]

George Santayana

[105] William W. Stead & John M. Starmer, *Beyond Expert-Based Practice*, *in* McClellan et al., *supra* note 92, at 94.

[106] *Id.* at 94.

[107] Controversy exists between the American Cancer Society and the United States Preventive Services Task Force on breast cancer and prostate cancer screening guidelines. For further review, the reader should examine the respective societies' websites.

[108] *See* Kassirer & Cecil, *supra* note 54, at 1383.

[109] *Id.*

[110] GROOPMAN, *supra* note 91, at 6.

[111] GEORGE SANTAYANA, THE LIFE OF REASON 284 (Charles Scribner's Sons 1905).

A. History

Much of what we currently know about AHT is the result of decades of meticulous, tireless work by physicians from various disciplines from all over the world.[112] Many of these historical clinicians did not have the benefit of advanced laboratory or radiographic techniques such as coagulation (clotting) studies, CTs or MRIs. They relied only upon their clinical skills and acumen. As time and medical technology have evolved, additional studies have corroborated their clinical suspicions, lending further credence to their clinical acumen.

While it can safely be said that the medical community, and society in general, did not recognize child abuse as a valid entity until the mid-twentieth century, it was a French forensic physician, Auguste Ambroise Tardieu (fig. 1), who penned the first detailed medical description of child abuse in his 1860 publication *Etude Medico-Legale sur les Sevices et Mauvais Traitements Exerces sur des Enfants* (Forensic Study on Cruelty and Ill Treatment of Children; fig. 2).[113] Tardieu was the leading forensic expert of his time, holding prestigious positions such as dean of the faculty of medicine at the University of Paris and president of the French Academy of Medicine.[114] He published works on child physical abuse, child sexual abuse, and child labor laws.[115]

In his 1860 publication, Tardieu detailed thirty-two cases of child abuse, describing bruises of varying colors, skeletal fractures, and subdural hemorrhages (SDHs).[116] Tardieu also described findings of infanticide, including cases without external signs of injury, but where hemorrhage in the brain and collections of blood

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[112] *See* Al-Holou et al., *supra* note 3, at 474.

[113] *Id.* at 475 (citing Ambroise Tardieu, *Etude Medico-Legale sur les Sevices et Mauvais Traitements Exerces sur des Enfants*, 13 ANNALES D'HYGIÈNE PUBLIQUE ET DE MÉDECINE LÉGALE 361-98 (1860)).

[114] *Id.*

[115] *Id.* at 476.

[116] *Id.* at 475.

over the brain were described.[117] In these writings, Tardieu clearly expressed his belief that the abuse was inflicted by parents or caretakers of the child.[118] Although his considerable influence led to revision of French child labor laws, Tardieu's works on child abuse went unappreciated and essentially ignored.[119]

The mid-to-late nineteenth century was a period of significant medical advancements.[120] Secondary to the works of Louis Pasteur and others, Germ theory became the predominant explanation for previously unexplained maladies.[121] Diseases such as scurvy, rickets, and even SDHs, were thought to be infectious.[122] A highly prominent physician, Rudolf Virchow, proposed the theory that SDHs, because they frequently presented with a membrane, were caused by inflammation and infection.[123] He termed this theory "pachymeningitis hemorrhagica interna".[124] Because of Virchow's significant stature within the medical community, and because the theory fit within the greater framework of the prevailing germ theory, the inflammation/infection theory of SDHs ("pachymeningitis hemorrhagica interna") was accepted for many decades.[125]

It was not until the early twentieth century that trauma began to be realized as an important cause of SDHs.[126] While earlier reports of the 20th century (despite a significant lack of evidence) still tended to support infectious or nutritional deficits as the cause of the SDHs,

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117 *Id.*

118 *Id.*

119 *Id.* at 476.

120 *Id.*

121 *Id.*

122 *Id.*

123 *Id.*

124 *Id.* It is one learned scholar's opinion that use of this terminology constricted the open and comprehensive scientific evaluation of the cause of such injuries in many of the earlier cases, resulting in probable misdiagnosis in many cases. *See* Duhaime, *supra* note 2, at 472.

125 Al-Holou et al., *supra* note 3, at 476.

126 *Id.*

later reports began to identify trauma as the *primary* etiology.[127] Additionally, many of those reports documented the association of SDHs, ophthalmic hemorrhages, and sometimes bone lesions in infants.[128]

In 1914, the prominent British neurosurgeon, Wilfred Trotter (fig. 3), published a report declaring trauma as the true cause of SDHs.[129] Trotter was a distinguished and accomplished physician who held many significant positions, not the least of which was his position as private physician to King George V.[130] Frustrated by the term "pachymeningitis hemorrhagica interna," Trotter asserted that the term presumed an infectious or inflammatory etiology and thus was a misleading hypothesis.[131] Trotter stated, "[h]aemorrhagic pachymeningitis is almost if not quite invariably a true traumatic haemorrhage coming from veins torn in their course between the brain and a dural sinus."[132] Trotter's work paved the way for other physicians, especially neurosurgeons, to re-examine the pathophysiology of SDHs.[133] As a consequence, multiple case reports by well-reputed physicians began to question other previously well-recognized causes—syphilis,[134] hydrocephalus,[135] nutritional (scurvy),[136] and other infectious[137]—as the primary

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127 *Id.* at 477.

128 *Id.* at 476; *see also id.* at 481 nn.7, 13, 482 nn.21, 34, 47, 61, 67, 483 nn.74, 86 (citing reports documenting the association of SDHs, ophthalmic hemorrhages, and sometimes bone lesions in infants).

129 *See* Al-Holou et al., *supra* note 3, at 478.

130 *Id.* at 477–78.

131 *Id.* at 478.

132 *Id.* (citing Wilfred Trotter, *Chronic Subdural Haemorrhage of Traumatic Origin, and Its Relation to Pachymeningitis Haemorrhagica Interna*, 2 BRIT. J. SURGERY, 271-91 (1914)).

133 *Id.* at 478.

134 *Id.* at 478 (citing Max. M. Peet & Edgar A. Kahn, *Subdural Hematoma in Infants*, 98 JAMA, 1851-56 (1932)).

135 *Id.* at 478–79 (citing Franc D. Ingraham & Donald D. Matson, *Subdural Hematoma in Infancy*, 24 J. PEDIATRICS 1-37 (1944)).

136 *Id.* at 478 (citing Franc D. Ingraham & Henry L. Heyl, *Subdural Hematoma in Infancy and Childhood*, 112 JAMA. 198-204 (1939)).

137 *See id.*

etiology for SDHs.[138]

Then, in 1946, Dr. John Caffey (considered by many to be the father of pediatric radiology), examined the correlation of SDHs and long bone fractures in a separate field of medicine—radiology.[139] After seeing repetitive cases of injuries over many years, Caffey published a case series of six infants with SDHs and long bone fractures.[140] In none of the six cases was there a historical report of trauma or of systemic disease.[141] Nevertheless, after systematically ruling out all other causes, Caffey concluded that trauma was the most logical etiology for these radiologic findings.[142] Caffey even associated the retinal hemorrhages in several of these cases to trauma.[143] Caffey, however, was reluctant to conclude inflicted injury in these cases.[144]

Secondary to Caffey's work, in 1953, another prominent radiologist, Frederic Silverman, catalogued radiographic signs of what he termed to be the "most common bone 'disease' of infancy": skeletal trauma.[145] In identifying trauma as the most common cause of SDHs and bone fractures in infants, Silverman meticulously ruled out all nutritional and metabolic causes.[146] In the two decades following Caffey's historic article, multiple articles from national and international authors confirmed the association of SDHs with

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[138] As will be discussed herein below, this is not to say that these causes (infectious, nutritional, metabolic, etc.) are no longer considered potential causes of SDHs, just that they are no longer considered the *primary* cause of SDHs. *See id.*

[139] *Id.* at 479.

[140] Al-Holou et al., *supra* note 3, at 479 (citing John Caffey, *Multiple Fractures in the Long Bones of Infants Suffering from Chronic Subdural Hematoma*, 56 AM. J. ROENTGENOLOGY 163–73 (1946)).

[141] Paul K. Kleinman & Paul D. Barnes, *Head Trauma*, *in* DIAGNOSTIC IMAGING OF CHILD ABUSE, 285, 297 (2d ed. 1998).

[142] Al-Holou et al., supra note 3, at 479.

[143] *Id.*

[144] Kleinman & Barnes, *supra* note 141, at 297–98.

[145] *See* Al-Holou et al., *supra* note 3, at 479 (citing F. Silverman, *The Roentgen Manifestations of Unrecognized Skeletal Trauma in Infants*, 69 AM. J. ROENTGENOLOGY RADIUM THERAPY NUCLEAR MED. 413–27 (1953)).

[146] *Id.*

inflicted trauma.[147]

It was not until 1962 that the work of an eminent pediatrician, C. Henry Kempe (fig. 4) and his colleagues (radiologist Frederic Silverman and psychiatrist Brandt Steele) brought the issue of child abuse to the medical and national forefront. In their landmark article, *The Battered-Child Syndrome* (fig. 5), Kempe et al. carefully and thoughtfully described a syndrome of various injuries, including SDHs, that resulted from trauma.[148] However, unlike the vast majority of physicians that preceded them, Kempe et al. concluded that these injuries resulted from the intentional acts of parents or other care-givers.[149] Kempe et al. stated that abuse:

> should be considered in any child exhibiting evidence of fracture of any bone, subdural hematoma, failure to thrive, soft tissue swellings or skin bruising, in any child who dies suddenly, or where the degree and type of injury is at variance with the history given regarding the occurrence of trauma.[150]

In support of their conclusions, the authors had surveyed 71 hospitals nationwide, with a report of over 300 cases in which 33 children had died and 85 had suffered permanent brain damage in one year.[151]

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147 Kleinman & Barnes, *supra* note 141, at 298 (citing F. Burke, et al., *Traumatic Periostitis and Subdural Hematoma*, 12 CLINICAL PROCS. CHILD. HOSP., D.C. 240–46 (1956); P. Josserand, et al., *Un Nouveau Cas D'Hematome Sous-Dural Associe a des Fractures de Membres Chez un Nourrisson*, 15 PEDIATRIE 647–59 (1960); G. Kinley, et al., *Subdural Hematoma, Hygroma, and Hydroma in Infants*, 38 J.PEDIATRICS 667–86 (1951); M.R. Klein, *L'Hematome Sous-Dural Du Nourrisson*, 21 ARCHIVES FRANCAISES DE PEDIATRIE 425–40 (1964); G. Lazorthes, et al., *Les Epanchements Sous-Duraux Du Nourrisson: Discussion Etiopathogenique a Propos de 59 Cas*, 71 PRESSE MED. 1903–05 (1963); M. Lelong et al., *L'Hematome Sous-Dural Chronique du Nourrisson*, 12 ARCHIVES FRANCAISES DE PEDIATRIE 1037–84 (1955); E.F. Lis & G.S. Frauenberger, *Multiple Fractures Associated With Subdural Hematoma in Infancy*, 6 PEDIATRICS 890–92 (1950); J. Meneghello, &, J. Hasbun, *Hematoma Subdural y Fractura de los Huesos Largos*, 22 REVISTA CHILENA DE PEDIATRIA 80–83 (1951); N. Neimann, et al., *Les Enfants Victimes de Services*, 23 PEDIATRIE 861–75 (1968); M.J. Smith, *Subdural Hematoma with Multiple Fractures: Case Report*, 63 AM. J. ROENTGENOLOGY 342–44 (1950)).

148 *See* Henry Kempe et al., *The Battered-Child Syndrome*, 9 CHILD ABUSE & NEGLECT 143, 144 (1985).

149 *See id.* at 143.

150 *Id.*

151 *Id.*

As a consequence of Kempe et al.'s historic work and the general medical community's increasing acceptance of child abuse as a viable medical diagnosis, case reports continued to publish the presence of concurrent SDHs, retinal hemorrhages, and bony lesions in infants, often without external signs of trauma.[152] Finally, in the early 1970s, based upon the work of Wilfred Trotter, numerous case reports, and the experimental biomechanical evidence of Ommaya and his colleagues,[153] a British neurosurgeon, A. Norman Guthkelch, and the father of pediatric radiology, John Caffey, proposed shaking or whiplash injury as the cause of infantile SDHs.[154]

In theorizing that multiple acceleration and deceleration events, caused by head shaking, resulted in the intracranial injuries, Guthkelch stated that, "the relatively large head and puny neck muscles of the infant must render it particularly vulnerable to whiplash injury."[155] Meanwhile, Caffey published a series of case reports identifying the "pattern of concurrent SDHs, [sometimes] bony lesions, and retinal hemorrhages in infants thought to be injured by shaking."[156] In fact, in the words of two learned authors: "It is difficult to comprehend how the common association between SDH and skeletal injuries, and the etiologic factors [trauma] linking the two, could have eluded the scrutiny of all but a handful of physicians and surgeons dealing with children until Caffey reported

152 *See* Al-Holou et al., *supra* note 3, at 480.

153 *Id.* at 478–80 (citing A.N. Guthkelch, *Infantile Subdural Haematoma and its Relationship to Whiplash Injuries*, 2 BRIT. MED. J. 430, 430–31 (1971); A.K. Ommaya et al., *Whiplash Injury and Brain Damage: An Experimental Study*, 204 JAMA 285, 285–89 (1968); A.K. Ommaya & A.E. Hirsch, *Tolerances for Cerebral Concussion from Head Impact and Whiplash in Primates*, 4 J. BIOMECHANICS 13, 13–21 (1971); A.K. Ommaya & P. Yarnell, *Subdural Haematoma After Whiplash Injury*, 2 LANCET 237, 237–39 (1969)).

154 *Id.* at 480.

155 *Id.* (quoting A.N. Guthkelch, *Infantile Subdural Hematoma and Its Relationship to Whiplash Injuries*, 2 BRIT. MED. J. 430, 430–31 (1971)).

156 *Id.* (citing J. Caffey, *On the Theory and Practice of Shaking Infants. Its Potential Residual Effects of Permanent Brain Damage and Mental Retardation*, 124 AM. J. DISEASES CHILD. 161–69 (1972); J. Caffey, *The Parent-Infant Traumatic Stress Syndrome; (Caffey-Kempe Syndrome), (Battered Babe Syndrome)*, 114 AM. J. ROENTGENOLOGY RADIUM THERAPY NUCLEAR MED. 218–29 (1972); J. Caffey, *The Whiplash Shaken Infant Syndrome: Manual Shaking by the Extremities With Whiplash-Induced Intracranial and Intraocular Bleedings, Linked With Residual Permanent Brain Damage and Mental Retardation*, 54 PEDIATRICS 396–403 (1974)).

his historic observations."[157]

B. "A Flawed Science"?[158]

As mentioned earlier, certain legal scholars have asserted that "the scientific underpinnings of SBS have crumbled over the past decade,"[159] that the medical research underlying "SBS is a flawed science"[160] predicated upon "circular reasoning," "data gaps," and "inconsistency of case definition,"[161] and that "as technology and scientific methodology advanced, researchers questioning the basis for SBS reached a critical mass."[162] In order to appropriately assess the sufficiency of the scientific evidence underlying AHT/SBS, some basic statistical concepts must be discussed.

1. *Basic Statistical Principles & Quality of Evidence*

Statistical evidence is an important complement to the practice of clinical medicine. Statistical evidence can offer probabilities and estimations of the risk of disease states in certain patient populations. It can help guide determinations of appropriate and inappropriate diagnostic testing in certain clinical scenarios. Moreover, it can provide empirical support for optimal therapeutic interventions in cases where treatment is warranted. However, statistical evidence cannot substitute for clinical judgment. It is a complement, not a replacement.

The field of statistics generally encompasses collecting, analyzing, presenting, and drawing inferences from data.[163] For the limited purposes of this article, we will review the general statistical

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157 *See* Kleinman & Barnes, *supra* note 141, at 298.

158 *See* Tuerkheimer, *supra* note 4, at 1, 12.

159 *Id.* at 11.

160 *Id.* at 12.

161 *Id.* at 12–13 (quoting Donohoe, *Evidence-Based Medicine*, *supra* note 12); *see also* Gena, *supra* note 4, at 710–14 (quoting Donohoe).

162 *Id.* at 14; *see also* Gena, *supra* note 4, at 710.

163 David H. Kaye & David A. Freedman, *Reference Guide on Statistics*, *in* REFERENCE MANUAL ON SCIENTIFIC EVIDENCE 83, 85 (2nd ed. 2000), http://www.fjc.gov/public/pdf.nsf/lookup/sciman02.pdf/$file/sciman02.pdf.

principles involved in collecting and drawing inferences from data.[164]

a. Collection of Data

It has been stated that "[a]n analysis is only as good as the data on which it rests."[165] The attainment of valid, reliable data is, to a large extent, determined by the design of the study.[166] When the issue is causation, there are three general types of explanatory information provided: anecdotal evidence, observational studies, and controlled experiments.[167] Each of these types of information has its limitations.[168]

Anecdotal reports, while offering information that can be the stimulus for further study, can be misleading and, therefore, are insufficient to conclusively establish association.[169] Observational studies can provide strong evidence of association, but further analysis is necessary "to bridge the gap from association to causation."[170] And controlled experiments, while ideal for determining causation, are often too expensive and cumbersome to undertake.[171] Examples of observational studies include case reports or case reviews, where as examples of controlled experiments include randomized controlled trials (RCTs) or non-randomized

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[164] For a more detailed analysis of statistics and the law, see Panel on Statistical Assessments as Evidence in the Courts, National Research Council, *The Evolving Role of Statistical Assessments as Evidence in the Courts* (Stephen E. Fienberg ed., 1989); MICHAEL O. FINKELSTEIN & BRUCE LEVIN, STATISTICS FOR LAWYERS (2d ed. 2001).

[165] Kaye & Freedman, *supra* note 163, at 90.

[166] *Id.*

[167] *Id.* at 91 ("'Anecdotal evidence' means reports of one kind of event after following another." But, such reports are often chosen "haphazardly or selectively," and do not "demonstrate that the first event causes the second.").

[168] *See id.* at 90–91.

[169] *Id.*; *see also* Haggerty v. Upjohn Co., 950 F. Supp. 1160, 1163–64 (S.D. Fla. 1996) (discussing the use of anecdotal case reports to generate hypotheses about causation).

[170] Kaye & Freedman, *supra* note 163, at 91. As described below, observational studies are susceptible to "confounding variables" and bias. *See id.* at 92. Bias can take many forms (selection, observation, recall, and reporting, to name a few), and can affect both observational and experimental studies.

[171] *See id.* at 91.

controlled studies (such as non-randomized dose finding studies).[172] Because observational studies and controlled experiments are the more reliable types of information,[173] it is important to understand the value of, and distinction between, the two.

"In a controlled experiment, the investigators decide which subjects are exposed to the factor of interest and which subjects go into a control group."[174] In "observational studies, the subjects themselves choose their exposures."[175] Thus, in observational studies, the experimental" (or "treatment") group will most likely differ from the control group "with respect to . . . [many] factors other than the one of primary interest.[176] These many "other factors" are also known as "confounding variables," and could be limitations to the validity of the results if not properly accounted for in the design of the study.[177] "In randomized controlled experiments, investigators assign subjects to [experimental (or "treatment") and] control groups at random."[178] By assigning subjects randomly to either the experimental or control groups, the investigator "tends to balance the groups with respect to possible confounders," thus enhancing the likelihood that the groups are comparable except for the factor of interest (or treatment).[179]

It is noteworthy that "[t]he bulk of the statistical studies . . . [presented] in court are observational, not experimental."[180] Observational studies (*i.e.*, case reports and case reviews) can provide compelling evidence when certain circumstances are present:

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172 *See Glossary*, BMJ, http://clinicalevidence.bmj.com/ceweb/resources/glossary.jsp (last visited Nov. 17, 2011) (defining "case control study" and "observational studies").

173 *See id.*

174 Kaye & Freedman, *supra* note 163, at 92.

175 *Id.*

176 *Id.*

177 *Id.*

178 *Id.* at 93.

179 *Id.* The analytical procedure most commonly used in statistics to control for confounding in observational studies is regression analysis. *See id.* at 94 n.31.

180 *Id.* at 94.

1) When "[t]he association is seen in studies of different types among different groups" ("This reduces the chance that the observed association is due to a defect in one type of study or a peculiarity in one group of subjects.");
2) "[W]hen the effects of plausible confounding variables are taken into account by appropriate statistical techniques;" and
3) When "[t]here is a plausible explanation for the effect of the independent variables."[181]

In general, "observational studies succeed to the extent that their [experimental (or treatment)] and control groups are comparable."[182] If a study is well designed, accounting for confounding variables, it is deemed to be internally valid.[183] However, the generalization of the conclusions of a study, or its "external validity," is a different matter.[184] Finally, a study is "reliable" if its results are reproducible by scientists in separate studies.[185]

In the realm of clinical medicine, observational studies are not just the norm but the cornerstone of medical diagnoses. Almost all well-established, undisputed medical diagnoses have no randomized controlled trials (RCTs) supporting or validating their diagnostic criteria. For example, migraine headaches have an extensive historical basis in the medical literature for evaluation, diagnosis, and therapy. In fact, the International Headache Society lists clear diagnostic criteria for migraine headaches, and provides the most up-to-date medical literature in support of that diagnostic criterion.[186] Yet, throughout the extensive body of medical literature on migraine headaches, there is not one RCT evaluating the diagnostic criteria for migraine headaches, or their validity. But

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181 *Id.* at 95. For example, the evidence that smoking causes lung cancer is largely observational, but still very compelling. *Id.*

182 *Id.* at 94.

183 *Id.* at 96.

184 *Id.* at 96.

185 *Id.* at 102.

186 *See generally* HEADACHE CLASSIFICATION SUBCOMMITTEE OF THE INTERNATIONAL HEADACHE SOCIETY (IHS), THE INTERNATIONAL CLASSIFICATION OF HEADACHE DISORDERS (2d ed., 1st rev. 2005), http://216.25.88.43/upload/CT_Clas/ICHD-IIR1final.pdf.

there is no dispute regarding the validity of migraine headaches as a medical diagnosis.[187] Such is also the case for multiple other well-established, undisputed, common medical diagnoses—viral upper respiratory infections (the common cold), community acquired pneumonia, otitis media (ear infection), depression, and all other psychiatric disorders. In short, the requirement that an RCT is necessary in order to validate diagnostic criteria of a particular medical diagnosis is not only inaccurate but also inconsistent with the vast majority of clinical medicine.[188]

At this point, it is relevant, and important, to examine one piece of medical literature which is often cited by opponents[189] of AHT as evidence of the paucity of sound medical literature on AHT (SBS): "*Evidence-Based Medicine and Shaken Baby Syndrome Part 1: Literature Review, 1966-1998*."[190] In this four-page article, the author proffers "neutrality," and proceeds to educate the reader about properly conducted studies, with sound methodological design, which fall into a "quality of evidence ratings" system.[191] Based upon the author's search of the Medline database, and the Internet via "Internet Explorer," using only the search term "shaken baby syndrome," the author finds only seventy-one articles (in a span of thirty-two years of medical literature) on the topic of AHT (SBS).[192] The author then reduces those seventy-one articles to fifty-four because some of the articles "only peripherally mention" SBS or are somehow "unrelated" to SBS.[193] Of those fifty-four remaining

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[187] *See id.* at 28.

[188] Whereas RCTs are not optimal for diagnostic studies, they are the study of choice for assessing therapies. *See* Jan P. Vandenbroucke, *Observational Research, Randomised Trials, and Two Views of Medical Science*, 5 PLOS MED. 0339, 0340 (2008) ("Randomised controlled trials are rarely used for research to detect or to establish causes of disease, mainly because randomisation is most of the time impossible, but quite fortunately, randomisation is most of the time not needed."); *see also* Alvan R. Feinstein & Ralph I. Horwitz, *Problems in the "Evidence" of "Evidence-Based Medicine,"* 103 AM. J. MED. 529, 529 (1997) ("Randomized trial information is also seldom available for issues in etiology, diagnosis, and prognosis . . . .").

[189] *See* Tuerkheimer, *supra* note 4, at 12 nn.65, 67–70; Gena, *supra* note 4, at 706 n.56.

[190] *See generally* Donohoe, *supra* note 12.

[191] *Id.* at 239–40.

[192] *Id.* at 240.

[193] *Id.*

articles, the author finds only one "randomized control trial" and twenty-six case series (twenty-five retrospective and one prospective), and a total of 307 cases of SBS.[194] Based upon the author's review of this literature, he concludes that in studies conducted before 1999 there exist "*serious data gaps, flaws of logic, [and] inconsistency of case definition*" in SBS; catch-phrases which have been frequently reified in some medical and legal literature.[195] Consequently, the author concludes that "the commonly held opinion that the finding of SDH and RH in an infant was strong evidence of SBS was unsustainable, at least from the medical literature."[196]

Evidence-Based Medicine and Shaken Baby Syndrome Part 1: Literature Review, 1966-1998 is a prime example of poor medical literature, which somehow makes its way into a medical publication. Ironically, the article itself suffers from fatal methodological flaws and data gaps, but professes to assess the methodology of SBS studies and finds "data gaps" in them.[197] It is unclear why, and unacceptable that, the author chooses to conduct his search with the confining search term of "shaken baby syndrome." The author fails to search other common terms such as "inflicted neurotrauma," "non-accidental trauma," "whiplash shaken infant/baby syndrome," or even more general terminology such as "subdural hemorrhage/hematoma" or "retinal hemorrhage."[198] Because of this methodological flaw, as will be demonstrated below, the author misses the vast majority of literature on AHT and even the seminal articles by Guthkelch and Caffey.[199] Additionally, the author offers no critical analysis of any of the articles cited, no assessment of the designs of any of the individual studies, no reference to the statistical information, and no analysis of any of the statistical data

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194 *Id.*

195 *Id.* at 241 (emphasis added); *see also* Tuerkheimer, *supra* note 4, at 12, 32.

196 *See* Donohoe, *supra* note 12, at 241.

197 *Id.*

198 *See id.* at 240.

199 In fact, in the article itself, the author admits missing what he himself considers an "important" study by Jayawant et al. using his own search criteria. *See id.* at 240.

or the inferences drawn from them.[200]

Finally, the author incorrectly uses the quality of evidence ratings system. The author asserts that the best evidence is "Level 1" quality of evidence (RCTs), and this is not found in the diagnostic studies involving AHT/SBS.[201] However, as discussed above, RCTs (the "Level 1" quality of evidence) are NOT appropriate for diagnostic studies. The AHT literature, like many other diagnoses (such as migraine headaches), should not be criticized for the existence of a "higher" level of evidence that is inappropriate to the question being asked. Thus, even the most ardent EBM advocate would admit that the best quality of evidence that can be expected in diagnostic studies is "Level 2" evidence (well-designed case series). And of this, as will be detailed below, there is abundant evidence in the AHT literature.

It is troubling that legal scholars and some courts have relied upon this article as an adequate assessment of the medical literature surrounding AHT.[202] Any future reliance upon this article should be seriously questioned.

b. Drawing Inferences from Data

Upon attainment of data, an investigator must determine what significance should be given to that data. In so doing, the investigator must determine whether the results obtained are attributable to random error.[203] Did "chance" produce the results?[204] Would a different pattern emerge if more data were collected?[205] In assessing the potential impact of chance error, an investigator must consider the precision of the data (i.e., the standard deviation and

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[200] *See* Donohoe, *supra* note 12, at 240–41.

[201] *Id.* at 239–41.

[202] *See* Cavazos v. Smith, 132 S. Ct. 2, 10 (2011) (per curiam) (Ginsburg, J., dissenting). *See generally* Tuerkheimer, *supra* note 4, at 12 & n.70 (citing evidentiary hearing testimony of Patrick Barnes in State v. Edmunds, 746 N.W.2d 590 (Wis. Ct. App. 2008)); Gena, *supra* note 4, at 727.

[203] *See* Kaye & Freedman, *supra* note 163, at 115.

[204] *Id.*

[205] *Id.*

degree of confidence) and the statistical significance (the p-value) of the data.[206]

In assessing precision of the data, a standard deviation (or standard error) gives the investigator an estimate of the magnitude of random error.[207] A standard deviation is a variability range of data from the "mean" of the data.[208] Assuming a normal distribution of data, one standard deviation from the mean of data is commonly understood to encompass 68% of the data.[209] For example, the average height for adult women in the United States is about 64 inches, with a standard deviation of around 3 inches.[210] This means that most women (about 68%, assuming a normal distribution) have a height within 3 inches of the mean (61-67 inches).[211] Two standard deviations from the mean encompass 95% of the data.[212] Thus, in our example with height of adult women in the United States, two standard deviations would be a height within 6 inches of the mean, or 58-70 inches. Since the standard deviation "measures the likely size of the random error[, i]f the standard deviation or error is small, the estimate probably is close to the truth."[213]

Confidence intervals are another manner of expressing reliability in the interval data.[214] Again, assuming a normal distribution curve, a 95% confidence interval indicates a range of data from -2 standard deviations to +2 standard deviations.[215] "A

[206] *Id.* at 116. While posterior probabilities, the applicability of the statistical models, and regression analysis are other important considerations, for the limited purposes of this article, we will focus on precision of data and statistical significance. For a more detailed discussion of the topic, I would guide the reader to Kaye & Freedman, *supra* note 163, at 116–78.

[207] *Id.* at 117.

[208] *Id.* at 115 n.107. The "mean" of data is the average of the data. *Id.* at 114 n.102.

[209] *Id.* at 118.

[210] *Id.* at 174.

[211] *Id.*

[212] *See id.* at 118, 174.

[213] *Id.* at 118.

[214] *Id.* at 118–19.

[215] *Id.* at 118.

high confidence level alone means very little, but a high confidence level for a small [data] interval is impressive, indicating that the random error in the . . . [interval] is low.[216]

In assessing statistical significance, it is important to understand the concept of the *p*-value. The *p*-value is "[t]he probability of getting, just by chance, a test statistic as large as or larger than the observed value."[217] In more simple terms, it is the probability the result obtained is secondary to chance.[218] In social sciences and medicine, this "observed significance level" (the *p*-value) is usually set at 5% (or 0.05) for "statistically significant," 1% (or 0.01) for "moderately high" statistical significance, and 0.1% (or 0.001) for "high or strong" statistical significance.[219] Thus, "[i]f *p* is smaller than 5% [(or 0.05)], the result is said to be 'statistically significant.'"[220] Small *p*-values speak against the hypothesis that the

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[216] *Id.* at 119 (footnotes omitted).

[217] *Id.* at 168.

[218] *See id.* at 122.

[219] *See id.* at 168. *See also id.* at 124 n.142 (quoting Waisome v. Port Auth. N.Y. & N.J., 948 F.2d 1370, 1376 (2d Cir. 1991) ("Social scientists consider a finding of two standard deviations significant, meaning there is about one chance in 20 that the explanation for a deviation could be random . . . ."); Rivera v. City of Wichita Falls, 665 F.2d 531, 545 n.22 (5th Cir. 1982) ("A variation of two standard deviations would indicate that the probability of the observed outcome occurring purely by chance would be approximately five out of 100; that is, it could be said with a 95% certainty that the outcome was not merely a fluke.")).

[220] Kay & Freedman, *supra* note 163, at 168. Computing the *p*-value requires statistical experience and is reserved for those with expertise in statistics and epidemiology. *See id.* at 87, 123. Incidentally, some statisticians point out that a determination of "statistical significance" is not as important as understanding how analysts developed their models. *See id.* at 128. For example:

> If enough comparisons are made, random error almost guarantees that some will yield "significant" findings, even when there is no real effect. Consider the problem of deciding whether a coin is biased. The probability that a fair coin will produce ten heads when tossed ten times is $(1/2)^{10} = 1/1{,}024$. Observing ten heads in the first ten tosses, therefore, would be strong evidence that the coin is biased. Nevertheless, if a fair coin is tossed a few thousand times, it is likely that at least one string of ten consecutive heads will appear.

Id. at 127; *see also id.* at 124, n.140; (citing John C. Bailar III & Frederick Mosteller, *Guidelines for Statistical Reporting in Articles for Medical Journals: Amplifications and Explanations*, *in* MEDICAL USES OF STATISTICS, (2d ed. 1992) ("Merely labeling results as 'significant' or 'not significant' without providing the underlying information that goes into this conclusion is of limited value.").

result can be explained by chance, while large p-values indicate that chance cannot be ruled out as an explanation for the data.[221]

A few other statistical concepts in clinical medicine are important to discuss briefly: "sensitivity," "specificity," "positive predictive value," "negative predictive value," and "odds ratio." "Sensitivity" is "the probability that a test for a disease will give a positive result" when the patient actually has the disease.[222] Put simply, it is actually the chance the condition will be found by the test.[223] "Specificity" is "the probability that a test for disease will give a negative result when the patient does not have the disease."[224] Put simply, it is the chance that someone without the disease will actually have a negative test.[225] "Positive predictive value" is the proportion of patients who have positive test results and actually have the disease or condition.[226] This value is very important in diagnostic testing as it reflects the probability that a positive test reflects the underlying condition being tested.[227] "Negative predictive value" is the "proportion of patients with negative test results who are correctly diagnosed."[228] "An "odds ratio" is a way of comparing whether the probability of a certain event is the same for two groups."[229] "An odds ratio of one implies that the event is equally likely in both groups.[230] An odds ratio greater than one

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221 Kay & Freedman, *supra* note 163, at 122.

222 *Id.* at 172.

223 *See id.*

224 *Id.* at 173. A test with high specificity for a condition will have a low rate of false positives. *See id.* at 172–73

225 *See id.* at 173.

226 FINKELSTEIN & LEVIN, *supra* note 164, at 82.

227 *See id.*

228 Penny F. Whiting et al., *Graphical Presentation of Diagnostic Information*, BMC MED. RESEARCH METHODOLOGY, tbl.1 (Apr. 11 2008), http://www.biomedcentral.com/content/pdf/1471-2288-8-20.pdf ; *see also*, FINKELSTEIN & LEVIN, *supra* note 164, at 83.

229 *Stats: What is an Odds Ratio?*, CHILDREN'S MERCY, http://www.childrens-mercy.org/stats/definitions/or.htm (last visited July 8, 2011).

230 Kaye & Freedman, *supra* note 163, at 167.

implies that the event is more likely in the first group."[231]

2. *The Statistical Evidence*

The peer-reviewed medical literature on the topic of AHT is voluminous. It is somewhat confusing how any author could assert there is a paucity of "quality" medical literature on the topic.[232] In hopes of clarifying and substantiating this matter, this author has compiled a brief bibliography (Appendix A)[233] of the peer-reviewed medical literature on the topic, organized by types of articles in the various subspecialties, so the reader may judge the literature for himself/herself.[234] A critical analysis of the quality of some of that literature will be discussed herein below.

In general, there have been at least two treatises, comprising more than 880 pages, on the topic of AHT.[235] Additionally, there are at least 14 chapters, comprising another approximate 260 pages, on the topic of AHT within larger child maltreatment/abuse texts.[236] In addition to that, there are over 700 peer-reviewed, clinical medical

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[231] *See id.*

[232] *See* Donohoe, *supra* note 12, at 241; *see also* Tuerkheimer, *supra* note 4; Gena, *supra* note 4 (authors who have just "reified" Donohoe's assertions).

[233] This abbreviated bibliography is focused primarily on the literature in the past twelve years, as assertions have been made that there has been a "shifted consensus" in the medical community against the legitimacy of the Abusive Head Trauma diagnosis, which is predicated upon "new research." *See* Tuerkheimer, *supra* note 4, at 15–29.

[234] For a completely comprehensive review of the topic, I would reference the reader to a review of the treatises on the topic (listed herein below in notes 235 & 236) as a starting point, with a subsequent search of the Medline database using broad search terms such as "subdural hemorrhage" or "retinal hemorrhage," with appropriately limiting criteria (i.e., including only children, excluding comments/editorials etc.). Assistance from a medical librarian may be required.

[235] *See* AM. ACAD. OF PEDIATRICS, INFLICTED CHILDHOOD NEUROTRAUMA: PROCEEDINGS OF A CONFERENCE SPONSORED BY DEPARTMENT OF HEALTH AND HUMAN SERVICES, NATIONAL INSTITUTE OF HEALTH, NATIONAL INSTITUTE OF CHILD HEALTH AND HUMAN DEVELOPMENT, OFFICE OF RARE DISEASE, AND NATIONAL CENTER FOR MEDICAL REHABILITATION RESEARCH (Robert M. Reece & Carol E. Nicholson eds., 2003); FRASIER ET AL., *supra* note 13.

[236] *See* CHILD ABUSE AND NEGLECT: DIAGNOSIS, TREATMENT, AND EVIDENCE 35–38, 347–457 (Carole Jenny ed., 2010) (chapters 6, 39–48); Suzanne Starling, *Head Injury in* CHILD MALTREATMENT: A CLINICAL GUIDE AND PHOTOGRAPHIC REFERENCE 37 (Angelo P. Giardino & Randell Alexander eds., 2003); Kleinman & Barnes, *supra* note 141; Rorke-Adams et al., *supra* note 13.

articles,[237] comprising thousands of pages of medical literature, published by over 1000 different medical authors, from at least 28 different countries[238]. on the topic of AHT. Furthermore, the topic of AHT has been examined, studied, and published in the following disciplines: biomechanical engineering, general pediatrics, neonatology, neurology, neurosurgery, nursing, obstetrics, ophthalmology, orthopedics, pathology (including forensic pathology), radiology, and rehabilitative medicine.

With regards to the "quality" of medical literature, it bears remembering that retrospective reviews are not inherently (by the nature of being retrospective or non-randomized) unreliable. It is the design of the review and the quality of the analysis (i.e., accountability for bias, confounding variables, interpretation of data, etc.) that determines the validity of the results. Nevertheless, even with that proviso, there have been at least eight systematic reviews, over fifteen controlled trials, over fifty comparative cohort studies or prospective case series, and numerous well-designed, retrospective case series/reports, comprising thousands of cases, *supporting* the diagnosis of AHT.[239] As will be discussed in detail below, in this author's review of all of the published, peer-reviewed, clinical

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237 In coming to this safe estimation, this author conducted an all language literature search of the Medline database from 1970 to March 2010, using over 15 different keywords/phrases (to include, but not limited to, "shaken baby syndrome," "shaken infant syndrome," "inflicted neurotrauma," "nonaccidental trauma," "subdural hemorrhage," "subarachnoid hemorrhage," and "retinal hemorrhage"). All meta-analyses, practice guidelines, randomized control trials, case reports, comparative studies, controlled clinical trials, historical or classical articles, multicenter studies and technical reports in children under eighteen years of age were included. All reviews, comments, editorials, letters, and news articles were excluded. The restricted searches to the search terms "subdural hemorrhages" and "retinal hemorrhages" by themselves produced over 1000 abstracts and over 500 abstracts, respectively. This author then reviewed over 1000 abstracts from the above searches to gauge applicability to the topic of Abusive Head Trauma, and safely determined that at least 700 articles were pertinent to the topic. Additionally, given the non-comprehensive nature of the search (i.e., the limitation to one database and a non-exhaustive list of keywords/phrases), this author was able to safely conclude that the above-stated number of studies was an underestimate.

238 The different nationalities publishing on this topic include: Argentina, Australia, Belgium, Brazil, Canada, China, Czech Republic, Denmark, Estonia, Finland, France, Germany, Greece, India, Israel, Italy, Japan, Malaysia, Netherlands, New Zealand, Norway, Poland, Russia, Singapore, Spain, Switzerland, United Kingdom, and the United States.

239 *See infra* Appendix A.

medical literature (greater than 700 articles), there is *not one* clinical study that demonstrates a greater statistical association of either subdural hemorrhages or retinal hemorrhages with accidental trauma over abusive head trauma. Additionally, since there has been criticism of the questionable "quality" of the medical literature supporting AHT (i.e., a lack of randomized, controlled studies),[240] it is important to note that almost all of the papers "questioning" the validity of AHT (save two or three) are non-randomized, retrospective case series/reports, and without comparative control groups. In fact, many are single case reports.

a. Subdural Hemorrhages

The differential diagnosis (i.e., list of potential causes) for subdural hemorrhages (SDHs) is extensive. A summarized list of those causes is detailed in Appendix B. When traumatic, the mechanism for the SDH is either a contact (or impact) force or an inertial (acceleration-deceleration) force or both.[241] "Contact... [forces] cause damage at the site... [where] contact occurs.[242]" Disruption of the skull's integrity secondary to the contact force can result in a disruption of the underlying blood vessels and consequent development of a hemorrhage.[243] These hemorrhages can be epidural (outside the dura mater), subdural (in the potential space underneath the dura mater), or, sometimes, intradural (within the layers of the dura).[244] In inertial events, the acceleration-deceleration motion of the brain results in strain upon the cortical bridging veins, which exceeds their tolerance levels and subsequently leads to rupture and hemorrhage (subdural and/or subarachnoid).[245]

Although there are many potential causes of SDHs, several

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240 *See* Donohoe, *supra* note 12, at 240–41.

241 *See* Rorke-Adams et al., *supra* note 13, at 61.

242 *Id.*

243 *Id.* at 64.

244 *Id.* at 65 tbl.2.2, 81-84.

245 *Id.* at 61, 63–64.

studies indicate that trauma is the most common cause.[246] In one such prospective study of all infants ages zero to two in the U.K. and the Republic of Ireland, from 1998 to 1999, Hobbs et al. identified 186 infants with SDHs (by CT, MRI, ultrasound, or post-mortem examination).[247] Of the 186 infants with SDHs, 113 (61%) had SDHs caused by trauma, 30 (16%) by infection or other non-traumatic medical cause, and 43 (23%) by an undetermined cause. [248] Of the 113 traumatic SDHs, 106 (94%) were determined to be of non-accidental etiology, and only 7 (6%) were determined to be accidental.[249] Similar results were noted in retrospective reviews by Jayawant et al. in Wales and southwest England from 1993 to 1995, Trenchs et al. in Barcelona, Spain from 1995 to 2005, and Tzioumi & Oates' in the Royal Alexandra Hospital for Children in Australia.[250]

Feldman et al. confirmed a predominance of non-accidental injury over accidental injury as the etiology of SDHs in their 2001 prospective study of 66 children, under age three, with SDHs.[251] Feldman et al. excluded patients that presented with SDHs secondary to known hemorrhagic disease (i.e., bleeding disorder), prior neurosurgical procedure, previously recognized perinatal (i.e., near birth) brain injury, or infection.[252] In efforts to avoid "circularity" concerns, Feldman et al. designed their study such that retinal hemorrhages (RHs) were not a part of the classification

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246 *See, e.g.*, C. Hobbs et al., *Subdural Haematoma and Effusion in Infancy: An Epidemiological Study*, 90 ARCHIVES DISEASE CHILDHOOD 952, 954.

247 *Id.* at 952–53.

248 *Id.* at 953 tbl.2. "Underdetermined cause" combines Hobbs' "Perinatal" and "Undetermined" categories, and "Traumatic SDHs" combines Hobbs' "Accident" and "Abuse" categories. *See id.*

249 *Id.*

250 *See* S. Jayawant et al., *Subdural Haemorrhages in Infants: Population Based Study*, 317 BRIT. MED. J. 1558, 1559, 1561 (1998); Victoria Trenchs et al., *Subdural Haematomas and Physical Abuse in the First Two Years of Life*, 43 PEDIATRIC NEUROSURGERY 352, 352–53, 356 (2007); Dimitra Tzioumi & R. Kim Oates, *Subdural Hematomas in Children Under 2 Years. Accidental or Inflicted? A 10-Year Experience*, 22 CHILD ABUSE & NEGLECT 1105, 1106–07 (1998).

251 Kenneth W. Feldman et al., *The Cause of Infant and Toddler Subdural Hemorrhage: A Prospective Study*, 108 PEDIATRICS 636, 638 (2001) (source also located in Appendix A, "General" literature, prospective article #14).

252 *Id.* at 637.

criteria for intentional injury.[253] In their cohort, Feldman et al. found that of the 66 patients, 39 (59%) patients were confirmed as suffering intentional injury, 15 (23%) were unintentional or accidental, and 12 (18%) were undetermined.[254]

Pathology studies have also confirmed the predominance of trauma, and more specifically non-accidental trauma, as the cause of SDHs.[255] In 2009, Matschke et al., published the results of their fifty-year retrospective review of the causes of death for infants less than one year old.[256] Of 715 infant deaths, only 50 infants (7%) were identified with SDHs.[257] Of those 50 SDHs, 15 (30%) were traumatic, 13 (26%) were secondary to bleeding/clotting disorders, 13 (26%) were perinatal, 4 (8%) were infectious, 4 (8%) were undetermined, and 1 (2%) was secondary to metabolic disease.[258] Of the traumatic SDHs, 14 (93%) were secondary to non-accidental trauma, and only 1 (7%) was accidental.[259] Thus, Matschke et al. concluded that "most . . . [SDHs are] attributable to trauma, with NAHI [(Non-Accidental Head Injuries)] substantially outnumbering accidental injuries . . . ."[260]

Although SDHs are not *specific*[261] for non-accidental injury, several well-designed prospective studies demonstrate a significant and strong association of SDHs with non-accidental/inflicted trauma over accidental trauma.[262] In 1992, Duhaime et al. published

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253 *Id.* at 637-38.

254 *Id.* at 638 tbl.2.

255 *E.g.*, Jakob Matschke et al., *Nonaccidental Head Injury is the Most Common Cause of Subdural Bleeding in Infants < 1 Year of Age*, 124 PEDIATRICS 1587, 1594 (2009) (source also located in Appendix A, "Pathology" literature, retrospective article #20).

256 *Id.* at 1588.

257 *Id.* at 1589.

258 *Id.*

259 *Id.*

260 *Id.* at 1594.

261 As discussed in the statistics section above, the term "specific" in this context is used with regards to its statistical definition; meaning that it is a condition/injury that can produce some false positives with regards to AHT. *See* Kaye & Freedman, *supra* note 163, at 173 (definition of specificity).

262 *E.g.*, A.C. Duhaime et al., *Head Injury in Very Young Children: Mechanisms, Injury Types, and*

the results of their prospective study of 100 patients less than two years of age who suffered head injuries.[263] In efforts to avoid "circularity" concerns, Duhaime et al. used strict criteria for determining "inflicted" injury.[264] The authors excluded retinal hemorrhages (RHs) as a diagnostic criterion, and they only included SDHs that had no history of trauma but had clinical or radiologic findings of blunt impact to the head.[265] Thus, the authors designed an algorithm, which was "deliberately biased to reduce false positives and thus may underestimate the true incidence of child abuse."[266] In Duhaime et al.'s cohort, 76 patients were determined to be from accidental causes and 24 were determined to be "inflicted."[267] Duhaime et al. found that only 3 out of 76 (8%) patients in the accidental group had SDHs, while 13 out of 24 (54%) patients in the "inflicted" group had SDHs.[268] This computed to a *p*-value of less than 0.0002, meaning these findings could have occurred by random chance no more than two times in 10,000 patients.[269] Thus, Duhaime et al. concluded that the relationship between inflicted injury and SDHs was highly statistically significant.[270]

In 2004, Bechtel et al. produced similar results.[271] The authors prospectively studied 82 children, age zero to twenty-four months, who were admitted to Yale New Haven Children's Hospital from

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Ophthalmologic Findings in 100 Hospitalized Patients Younger than 2 Years of Age, 90 PEDIATRICS 179, 183 (1992) (source also located in Appendix A, "General" literature, prospective article #10).

263 *Id.* at 179, 181.

264 *Id.* at 179.

265 *See id.* at 179–80, 184.

266 *Id.* at 180.

267 *See id.* at 181

268 *Id.* at 183.

269 *See id.* at 184. Recall the general statistical principles section above: *p*-value is essentially the likelihood the result is due to chance.

270 *Id.* at 181, 184.

271 *See* Kirsten Bechtel, et al., *Characteristics that Distinguish Accidental from Abusive Injury in Hospitalized Young Children with Head Trauma*, 114 PEDIATRICS 165, 165, 168 (2004) (source also located in Appendix A, "General" literature, prospective article #5).

August 2000 to October 2002 for head trauma.[272] In avoiding "circularity" concerns, the authors classified "inflicted" head injury only if there was clear evidence of head injury and no trauma history provided, if there was a traumatic history incompatible with the developmental capabilities of the infant, if there was a confession of inflicting the injury, if there was a witnessed inflicted injury, or if there was evidence of other physical injuries which were characteristic of inflicted injury (e.g., patterned bruises, etc.).[273] The authors did not include RHs in the diagnostic criterion of "inflicted" injury.[274] Of the eighty-two patients, sixty-seven were determined to be "accidental," and fifteen were determined to be "inflicted."[275] Bechtel et al. found that 12/15 (80%) patients in the "inflicted" head injury group had SDHs, while only 18/67 (27%) patients in the "accidental" head injury group had SDHs.[276] This computed to a *p*-value of less than 0.001.[277] Again, this meant that these findings could have occurred by chance or randomly no more than one in 1,000.[278] Thus, Bechtel et al. also concluded that the association of SDHs with inflicted injury was highly statistically significant.[279]

In 2010, Vinchon et al. published the results of their prospective series of eighty-four patients, from 2001 to 2009, with independent corroboration of head injury.[280] In Vinchon et al.'s cohort, thirty-nine patients were witnessed accidents and forty-five patients were confessed inflicted head injury.[281] Only 17 out of 39 (44%) witnessed accidents had SDHs, while 37 out of 45 (82%) inflicted head injury

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272 *Id.* at 166.

273 *Id.* at 166 tbl.1.

274 *See id.*

275 *Id.* at 166.

276 *Id.* at 167, tbl.3.

277 *Id.*

278 *See id.*

279 *See id.*

280 *See* Matthieu Vinchon et al., *Confessed Abuse Versus Witnessed Accidents in Infants: Comparison of Clinical, Radiological, and Ophthalmological Data in Corroborated Cases*, 26 CHILD'S NERVOUS SYS. 637, 638–39 (2010) (source also located in Appendix A, "General" literature, prospective article #23).

281 *Id.* Confessions were obtained from judicial sources. *Id.* at 638.

patients had SDHs.[282] This computed to a p-value of less than 0.001.[283] As with Duhaime et al. and Bechtel et al., Vinchon et al. concluded that the association SDHs with non-accidental injury was highly statistically significant.[284] Several other well-designed, prospective and retrospective general pediatric studies have found similar results and come to the same conclusion.[285]

Radiology studies have helped to further characterize the appearance of SDHs seen in AHT cases. Multifocal SDHs, interhemispheric SDHs (located between the two hemispheres of the brain), and convexity SDHs (located at the front or back "curves" of the brain) have a stronger statistical association with non-accidental trauma than with accidental trauma.[286] In 2002, Wells et al. published the results of a retrospective review of the CTs of 293 children, under age three, with intracranial hemorrhage at the

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[282] *Id.* at 641 tbl.2.

[283] *Id.*

[284] *See id.* at 639, 641 tbl.2.

[285] See Linda Ewing-Cobbs, et al., *Neuroimaging, Physical, and Developmental Findings after Inflicted and Noninflicted Traumatic Brain Injury in Young Children*, 102 PEDIATRICS 300, 300 (1998); Carla DiScala, et al., *Child Abuse and Unintentional Injuries*, 154 ARCHIVES PEDIATRICS & ADOLESCENT MED. 16, 16 (2000); Kent P. Hymel et al., *Head Injury Depth as an Indicator of Causes and Mechanisms*, 125 PEDIATRICS 712, 715–18 (2010) [hereinafter Hymel et al., *Head Injury Depth*]; Kent P. Hymel et al., *Mechanisms, Clinical Presentations, Injuries, and Outcomes from Inflicted Versus Noninflicted Head Trauma during Infancy: Results of a Prospective, Multicentered, Comparative Study*, 119 PEDIATRICS 922, 922 (2007) [hereinafter Hymel et al., *Mechanisms*]; Heather T. Keenan et al., *A Population-Based Comparison of Clinical Outcome Characteristics of Young Children with Serious Inflicted and Noninflicted Traumatic Brain Injury*, 114 PEDIATRICS 633, 633 (2004); Mark W. Morris et al., *Evaluation of Infants with Subdural Hematoma who Lack External Evidence of Abuse*, 105 PEDIATRICS 549, 549 (2000); M.C. Myhre et al., Traumatic Head Injury in Infants and Toddlers, 96 ACTA PAEDIATRICA 1159, 1159 (2007); Robert M. Reece & Robert Sege, *Childhood Head Injuries*, 154 ARCHIVES PEDIATRICS & ADOLESCENT MED. 11, 11 (2000); Shervin R. Dashti et al., *Current Patterns of Inflicted Head Injury in Children*, 31 PEDIATRIC NEUROSURGERY 302, 302 (1999); Matthieu Vinchon et al., *Accidental and Nonaccidental Head Injuries in Infants: A Prospective Study*, 102 J. NEUROSURGERY: PEDIATRICS 380, 380–81 (2005) (sources also referenced in Appendix A, "General" literature, prospective articles #12, 18, 19 & 21; retrospective articles # 12, 27, 28, & 32; "Neurosurgery" literature, prospective article #3 and retrospective article #5).

[286] Robert G. Wells et al., *Intracranial Hemorrhage in Children Younger than 3 Years*, 156 ARCHIVES PEDIATRICS & ADOLESCENT MED. 252, 253, 254 tbl.2 (2002) (source also referenced in Appendix A, "Radiology" literature, retrospective article #14).

Children's Hospital of Wisconsin from 1991 to 2001.[287] Blinded to the CT findings, an injury was classified as "intentional if there was a confession of abuse, the injuries were incompatible with the stated mechanism of injury, or the caretaker offered no explanation for the injuries."[288] "An injury was classified as "unintentional if it was witnessed by someone other than the caretaker or there were no discrepancies between the described mechanism and the physical findings."[289] Then, blinded to the clinical findings, a pediatric radiologist reviewed the CT findings for the presence and location of intracranial hemorrhage and other intracranial abnormalities.[290] Wells et al. found that 105 out of 148 (71%) intentional injury patients had an interhemispheric SDH, while only 21 out of 109 (19%) unintentional injury patients had an interhemispheric SDH; and, 99 out of 148 (67%) intentional injury patients had a convexity SDH, as compared with 14 out of 109 (13%) unintentional injury patients.[291] For both these injuries, this computed a p-value of less than 0.05.[292] Thus, Wells et al. concluded that there was a statistically significant association with convexity and interhemispheric SDHs and intentional injury.[293] Similar results were produced by Hymel et al. and by Datta et al.[294] Additionally, in the Datta et al. study, there was a statistically significant association with multifocal SDHs and non-accidental injury.[295]

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287 *Id.* at 253.

288 *Id.*

289 *Id.*

290 *Id.*

291 *Id.* at 254 tbls.1 & 2.

292 *Id.* at 255.

293 *See id.* at 255 & tbl.3.

294 *See* S. Datta et al., *Neuroradiological Aspects of Subdural Haemorrhages*, ARCHIVES DISEASE CHILDHOOD 947, 948, 950 (2005); Hymel et al., *Mechanisms*, *supra* note 285, at 928. *But see* Glenn A. Tung et al., *Comparison of Accidental and Nonaccidental Traumatic Head Injury in Children on Noncontrast Computed Tomography*, 118 PEDIATRICS 626, 632 (2006) (showing authors did not find a significant statistical association with interhemispheric SDHs and non-accidental trauma) (source also referenced in Appendix A, "Radiology" literature, comparative article #8).

295 Datta et al., *supra* note 294, at 947–48.

Thus, with regards to the validity and reliability of the statistical evidence on SDHs and AHT, there are several well-designed prospective studies and retrospective reviews. Additionally compelling is that the statistical results are similar along multiple lines of research—pathology, radiology and general pediatrics. All have produced the same results: the significant statistical association of SDHs with non-accidental trauma over accidental trauma. This author's review of the evidence-based medical literature has revealed no published, peer-reviewed clinical studies that conclude differently.

b. Retinal Hemorrhages

The retina is the multi-layered, inner lining of the eye.[296] The posterior pole is the area of the retina that encompasses the major blood vessels, the macula, the fovea, and the optic nerve head (the optic disc).[297] The fovea is the area of the retina where the central visual axis through the pupil falls.[298] The area of retina surrounding the fovea is the macula.[299] These structures are depicted in Figures 6 and 7.

In young children/infants, the vitreous gel that fills the eye is adhered much more strongly to the macula, peripheral retina, and the retinal blood vessels as they course on the retinal surface.[300] This difference in anatomy from the adult eye is relevant to the theory of how RHs are formed (repetitive acceleration-deceleration forces) in the setting of AHT.[301]

"Hemorrhages [can] occur on the surface of the retina (preretinal), under the retina (subretinal), or within the retinal

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296 Alex V. Levin, *Retinal Hemorrhages: Advances in Understanding*, 56 PEDIATRIC CLINICS N. AM. 333, 335 (2009) (source also referenced in Appendix A, "Ophthalmology" literature, review article #5).

297 *Id.*

298 *Id.*

299 *Id.*

300 *Id.*

301 *See id.* at 338.

[layers (intraretinal)]."[302] Hemorrhages can have a certain appearance and size, and can be confined to the posterior pole or extend to the ora serrata (the edges of the retina).[303] "Flame" or "splinter" RHs are hemorrhages that lay in the superficial nerve fiber layer of the retina.[304] "Dot" and "blot" RHs are round and amorphous-shaped hemorrhages within the deeper layers of the retina.[305] An important form of RHs is retinoschisis—where there is splitting of the retinal layers with blood accumulating in the intervening space.[306] Retinoschisis can sometimes be accompanied by circumlinear pleats or folds in the retina at the edges of the schisis.[307] Retinoschisis with pleats or folds is an important finding, because, other than AHT, in children younger than five years it has only been reported in two cases of fatal crush injuries to the head, one case of leukemia, and in cases of severe, fatal motor vehicle accidents.[308]

Mild RHs are generally understood to be a few, dot/blot or flame/splinter-shaped RHs, in the intraretinal or preretinal layers, and confined to the posterior pole.[309] Severe RHs are generally understood to be diffuse, too numerous to count hemorrhages, extending to the periphery of the retina (not confined to the

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[302] *Id.* at 335.

[303] *Id.* at 333, 341.

[304] *Id.* at 335.

[305] *Id.*

[306] *Id.*

[307] *Id.*

[308] *Id.* at 335–36; *see* Gregg T. Lueder et al., *Perimacular Retinal Folds Simulating Nonaccidental Injury in an Infant*, 124 ARCHIVES OPHTHALMOLOGY 1782, 1782–83 (2006) (source also referenced in Appendix A, "Ophthalmology" literature, retrospective article #22); P.E. Lantz et al., *Perimacular Retinal Folds from Childhood Head Trauma*, 328 BRIT MED. J. 754, 754 (2004) (source also referenced in Appendix A, "Pathology" literature, retrospective article #16); Ajay Bhatnagar et al., *Subinternal Limiting Membrane Hemorrhage with Perimacular Fold in Leukemia*, 127 ARCHIVES OPHTHALMOLOGY 1548, 1548 (2009) (source also referenced in Appendix A, "Ophthalmology" literature, retrospective article #4); JD Kivlin et al., *Retinal Hemorrhages in Children Following Fatal Motor Vehicle Crashes: A Case Series*, 126 ARCHIVES OPHTHALMOLOGY 800, 800–01 (2008) (source also referenced in Appendix A, "Ophthalmology" literature, retrospective article #18).

[309] *See*, Levin, *supra* note 296, at 334 box1.

posterior pole), usually involving multiple layers of the retina (intraretinal, preretinal or subretinal), and sometimes accompanied by retinoschisis with or without folds.[310] Mild RHs, severe RHs, and retinoschisis are depicted below in figures 8, 9, 10.

As with SDHs, the differential diagnosis for subdural hemorrhage RHs is extensive. A summarized list of those causes is detailed in Appendix C. Assessing the diagnostic significance of RHs requires the consideration of other medical causes and an understanding of the spectrum of injury patterns observed in accidental trauma. Through the inferential and deductive process of eliminating other potential mechanisms, one recognizes the significant probability that repetitive acceleration-deceleration forces are the causative mechanism of severe RHs.[311]

While several studies demonstrate an association of RHs with birth, several factors distinguish birth-related RHs from the RHs commonly seen in AHT.[312] First, the vast majority of birth-related retinal hemorrhages are intraretinal.[313] Multi-layered RHs, as commonly seen in AHT, have not been reported in the medical literature in association with birth.[314] Second, study of the natural history of birth-related RHs reveals that the vast majority of these RHs resolve by two to four weeks of life.[315] This led one author to conclude that RHs "in infants older than 1 month . . . [are] not likely related to birth".[316] Finally, retinoschisis (splitting of the retina) has

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310 *Id.* at 333, 335.

311 *See* Levin, *supra* note 296, at 337.

312 *See* M. Vaughn Emerson et al., *Incidence and Rate of Disappearance of Retinal Hemorrhage in Newborns*, 108 OPHTHALMOLOGY 36, 36 (2001); Lindsey A. Hughes et al., *Incidence, Distribution, and Duration of Birth-Related Retinal Hemorrhages: A Prospective Study*, 10 J. AM. ASS'N FOR PEDIATRIC OPHTHALMOLOGY & STRABISMUS 102, 102 (2006) (sources also referenced in Appendix A, "Ophthalmology" literature, prospective articles #6 & #13).

313 *See* Emerson et al., *supra* note 312, at 36.

314 *Id.* at 37.

315 *Id.* at 38. There are rare cases of birth-related RHs lasting until six to eight weeks of life. *See id.* There has been no documentation of birth related RHs outside of eight weeks (two months) of life. *See id.*; Hughes et al., *supra* note 312, at 106.

316 *Id.* at 39.

never been reported in association with birth injury.[317]

The commonality, and somewhat similarity, of birth-related RHs and the RHs commonly seen in AHT compels one to consider increased intracranial pressure or increased intrathoracic pressure as potential causative mechanisms for RHs.[318] Additionally, because rib fractures are occasional concurrent injuries in AHT cases, increased intrathoracic pressure is naturally thought to be implicated.[319]

Studies examining the effects of chest compressions in CPR (cardio-pulmonary resuscitation) have failed to demonstrate any severe RHs (the kind seen in AHT).[320] In one such study, Odom et al. prospectively examined the prevalence and character of RHs in patients in a pediatric ICU who had received at least one minute of chest compressions and survived. [321] After excluding patients that had evidence of trauma, documented retinal hemorrhages before CPR, suspicion of child abuse, or diagnosis of near-drowning or seizures, Odom et al. found 43 patients that met inclusion criteria.[322] In fact, "[a]ll of the precipitating events leading to cardiopulmonary arrest occurred in their intensive care unit, eliminating the possibility of physical abuse as an etiology."[323] Of the 43 patients, "[t]he mean duration of chest compressions was 16.4 minutes . . . with 58% lasting between 1 and 10 minutes. Five patients had chest compressions lasting less than 40 minutes, and two patients had open chest cardiac massage. All patients survived their resuscitative

317 *See* Levin, *supra* note 296, at 334 box1.

318 *Id.* at 337.

319 *Id.*

320 *See* James C. Fackler et al., *Retinal Hemorrhages in Newborn Piglets Following Cardiopulmonary Resuscitation*, 146 AM. J. DISEASES CHILDREN 1294, 1295 (1992); M.G.F. Gilliland & Martha Waters Luckenbach, *Are Retinal Hemorrhages Found After Resuscitation Attempts? A Study of the Eyes of 169 Children*, 14 AM. J. FORENSIC MED. & PATHOLOGY 187, 189 (1993); Amy Odom et al., *Prevalence of Retinal Hemorrhages in Pediatric Patients After In-hospital Cardiopulmonary Resuscitation: A Prospective Study*, 99 PEDIATRICS, at *4 (June 2007) (sources also referenced in Appendix A, "Ophthalmology" literature, controlled study #2 & prospective articles #7 & #16).

321 *See* Odom et al., *supra* note 320, at *2.

322 *Id.*

323 *Id.* at *4.

efforts."[324] Odom et al. found small punctate retinal hemorrhages in only one patient.[325] There was no patient with severe RHs.[326] Well-designed studies involving other clinical scenarios that increase intrathoracic pressure, e.g., coughing, vomiting, or seizures, also have failed to demonstrate any of the type of severe RHs commonly seen in AHT.[327]

With regards to increased intracranial pressure as a cause for severe RHs in children, in 2002, Schloff et al. published the results of a prospective study, which was designed to find the incidence of RHs in children with intracranial hemorrhage and increased intracranial pressure (also known as Terson's syndrome).[328] Only children from known non-abuse cases were included in their study.[329] Of the 57 children studied, 27 were from known accidental trauma (MVA's, sports accidents, falls, etc.), 24 from surgeries, and six from other causes (vessel malformations, infection, etc.).[330] Fifty-

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324 *Id.* at *1.

325 *Id.* at *1, *4.

326 *See id.* at *3–*4.

327 *See* A.I. Curcoy et al., *Do Retinal Haemorrhages Occur in Infants with Convulsions?*, 94 ARCHIVES DISEASE CHILDHOOD 873, 874 (2009) (seizures); Michael Goldman et al., *Severe Cough and Retinal Hemorrhage in Infants and Young Children*, 148 J. PEDIATRICS 835, 836 (2006) (coughing); Sandra Herr et al., *Does Valsalva Retinopathy Occur in Infants? An Initial Investigation in Infants with Vomiting Caused by Pyloric Stenosis*, 113 PEDIATRICS 1658, 1660 (2004) (vomiting); M. Mei-Zahav et al., *Convulsions and Retinal Haemorrhage: Should We Look Further?* 86 ARCHIVES DISEASE CHILDHOOD 334, 334–35 (2002) (convulsions); S. Sandramouli et al., *Retinal Hemorrhages and Convulsions*, 76 ARCHIVES DISEASE CHILDHOOD 449, 449–50 (1997) (seizures) Ajai K. Tyagi et al., *Can Convulsions Alone Cause Retinal Haemorrhages in Infants?* 82 BRIT. J. OPHTHALMOLOGY 659, 659–60 (1998) (seizures); (sources also referenced in Appendix A, "Ophthalmology" literature, prospective articles #5, 11, 12, 15, 19, & 23). One other mechanism of retinal hemorrhaging occasionally mentioned is Purtscher's Syndrome. Levin, *supra* note 296, at 337. Purtscher's syndrome is the presence of certain characteristically-patterned RHs (hexagonal with white patches) that occur in adults that suffer severe crush chest injury. *Id.* The particular characteristically-patterned RHs (Purtscher's retinopathy) are rarely seen in AHT and are most likely the result of infarction, fat emboli from broken bones, or inflammation-mediated change. *Id.*

328 *See* Susan Schloff et al., *Retinal Findings in Children with Intracranial Hemorrhage*, 109 OPHTHALMOLOGY 1472, 1472 (2002) (source also referenced in Appendix A, "Ophthalmology" literature, prospective article #20).

329 *Id.* at 1473.

330 *Id.* at 1473 tbl.1.

five out of fifty-seven children (96%) had no evidence of RH.[331] "One . . . [child] had a single dot hemorrhage associated with [a] presumed infectio[n] . . . . The second . . . [child] had three flame and two deeper dot intraretinal hemorrhages."[332] She was the victim of a motor vehicle accident.[333] No child had severe or multi-layered RHs.[334] These results accord with the retrospective review conducted by Morad et al., also published in 2002.[335]

Furthermore, the postulated mechanism of RHs in the setting of increased intracranial pressure—obstruction of venous outflow from the eye (i.e., blood flowing out of the eye, through the head, and back towards the heart)—produces a pattern of hemorrhages that is not the pattern of hemorrhages seen in AHT.[336] The accidental head injury literature also demonstrates no severe RHs, and many of the children in those studies experienced increased intracranial pressure.[337]

On the other hand, several lines of research and analysis point towards acceleration-deceleration forces at the vitreo-retinal interface (remembering, from above, that the anatomy of an infant is

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331 *Id.* at 1473.

332 *Id.* at 1472.

333 *Id.*

334 *Id.* at 1473–74.

335 *See* Yair Morad et al., *Correlation Between Retinal Abnormalities and Intracranial Abnormalities in the Shaken Baby Syndrome*, 134 AM. J. OPHTHALMOLOGY 354, 355–56 (2002); (source also referenced in Appendix A, "Ophthalmology" literature, retrospective article #30).

336 *See* Levin, *supra* note 296, at 338.

337 *See* Yvonne M. Buys et al., *Retinal Findings After Head Trauma in Infants and Young Children*, 99 OPHTHALMOLOGY 1718, 1720 (1992); Cindy W. Christian et al., *Retinal Hemorrhages Caused by Accidental Household Trauma*, 135 J. PEDIATRICS 125, 127 (1999); Dennis L. Johnson et al., *Accidental Head Trauma and Retinal Hemorrhage*, 33 NEUROSURGERY 231, 231–32 (1993); V. Trenchs et al., *Retinal Haemorrhages in Head Trauma Resulting from Falls: Differential Diagnosis with Non-Accidental Trauma in Patients Younger than 2 Years of Age*, 24 CHILD'S NERVOUS SYS. 815, 817 (2008); V. Sturm et al., *Rare Retinal Haemorrhages in Translational Accidental Head Trauma in Children*, 23 EYE 1535, 1540 (2009); Kivlin et al., *supra* note 308, at 803 (sources also referenced in Appendix A, "Ophthalmology" literature, prospective articles #3, 14, & 22; retrospective articles #8, 18, & 36). In the rare instances when RHs were present, there were only a few preretinal or intraretinal RHs confined to the posterior pole. *See, e.g.*, Cindy W. Christian et al., *Retinal Hemorrhages Caused by Accidental Household Trauma*, 135 J. PEDIATRICS 125, 125–27 (1999).

such that the vitreous gel is much more strongly adherent to the retina than in adults) as the causative mechanism for severe RHs.[338] First, "the pattern of hemorrhages . . . [in severe RHs] correlates with the . . . anatomy [of the eye in] the young child where[] the vitreous is most adherent with blood vessels" (in the periphery of the retina, and in the area of the posterior pole where retinoschisis occurs).[339] Second, severe RHs are not commonly seen in single acceleration-deceleration traumatic events (such as motor vehicle accidents and falls).[340] Third, in fatal cases, postmortem studies reveal that the vitreous is often still attached at the top of retinal folds, indicating a traction mechanism.[341] Finally, as will be detailed below, there is an extremely high, statistically significant association of severe RHs with AHT.[342]

In 2005, Vinchon et al. sought to study the diagnostic significance of RHs in cases of child abuse.[343] Their prospective study of 150 children included all children under two years old, who were admitted with head injury over a three year period.[344] Utilizing the strict algorithmic criteria of Duhaime et al. (discussed above) for determining "inflicted" injury, Vinchon et al. identified 57 cases of abuse, 88 eighty-eight accidental cases (household, birth trauma, and traffic accidents), and five undetermined.[345] Retinal data was available for 129 children (56 abuse, 73 accidents).[346] Moderate to severe RHs were found in 37 cases, all of them "abuse".[347] Vinchon et al. found the sensitivity, specificity, and positive predictive value of moderate or severe RHs for abuse to be 66.1%, 100%, and 100%,

338 *See* Levin, *supra* note 296, at 338.

339 *Id.*

340 *Id.*

341 *Id.*

342 *See id.* at 341.

343 *See* Vinchon et al., *supra* note 285, at 380.

344 *Id.* 380–81.

345 *Id.* at 381.

346 *Id.*

347 *Id.* at 382.

respectively.[348]

Vinchon sought to re-examine this data, and its reproducibility, except this time with independent corroboration of head injury, so as to avoid any "circularity" concerns in his design.[349] In 2010, Vinchon et al. published the results of a prospective series of 84 patients who sustained injuries from either witnessed accidents (N=39) or confessed inflicted head injury (N=45; obtained from judicial sources).[350] Of the thirty-nine witnessed accidents, only one patient (2.5%) had moderate or severe RHs—that is the patient had a known impact to his head.[351] Of the 45 confessed inflicted injury patients, 34 (76%) had moderate or severe RHs.[352] Conversely, 34 out of 39 (87%) accident patients had mild or no RHs; and, 10 out of 45 (22%) of the inflicted head injury patients had mild or no RHs.[353] This data is graphically depicted (figure 11) below, and computed to a *p*-value of less than 0.001 (0.1%).[354] In further statistical analysis, Vinchon et al. determined the specificity and positive predictive value of severe RHs for abusive injury to be 97% and 96%, respectively.[355] Vinchon et al. calculated the specificity of SDH, RH and the absence of evidence of impact to be 100% for abusive injury.[356] Thus, Vinchon et al. concluded that, in the absence of ocular impact, severe RHs were specific for inflicted head injury.[357] Similar results have been produced in well-designed prospective and retrospective studies by Pierre-Kahn et al., Bechtel et al., and Reece and Sege.[358]

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348 *Id.*

349 *See* Vinchon et al., *supra* note 280, at 637–38, 644.

350 *See id.* at 637–38.

351 *Id.* at 641 tbl.2.

352 *Id.*

353 *Id.*

354 *Id.* at 640 tbl.1.

355 *Id.* at 642 tbl.4.

356 *Id.* at 642 tbl.4, 643.

357 *Id.* at 644.

358 *See* Vincent Pierre-Kahn et al., *Ophthalmologic Findings in Suspected Child Abuse Victims with Subdural Hematomas*, 110 OPHTHALMOLOGY 1718, 1720 (2003) (source also referenced in

Pathology studies have produced similar results. Riffenburgh studied 197 confirmed child abuse deaths and compared them to 401 controlled patients (deaths secondary to auto accidents, drowning, SIDS).[359] Riffenburgh found 47% of child abuse deaths had RHs whereas only 4% of controls had RHs.[360] This computed to a *p*-value of less than 0.001 (0.1%), and an odds ratio of 18.9 for RHs and abuse.[361] Remembering "odds ratio" from the statistics section above, this means that RHs in abuse is almost nineteen times more likely than RHs in other circumstances (auto accidents, drowning, SIDs, etc).[362] Other authors have published comparable findings.[363]

In 2009, Maguire et al. published the results of their systematic review of all the scientific literature to identify clinical features that distinguished inflicted from non-inflicted brain injury.[364] After reviewing "20 [electronic] databases, websites, references and bibliographies, using over 100 keyword combinations," Maguire et al. identified over 6000 studies, which were relevant to the topic, and reviewed 320.[365] Secondary to strict inclusion criteria (including only those studies that compared the clinical features of inflicted and non-inflicted brain injury with consecutive case ascertainment),

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Appendix A, "Ophthalmology" literature, prospective article #17); Bechtel et al., *supra* note 271, 166–67; Reece & Sege, *supra* note 285, at 13–14.

359 *See* Gaurav Bhardwaj et al., *A Systematic Review of the Diagnostic Accuracy of Ocular Signs in Pediatric Abusive Head Trauma*, 117 OPHTHALMOLOGY 983, 987 tbl.1 (2010) (presenting results of Riffenburgh study) (Bhardwaj source also referenced in Appendix A, "Ophthalmology" literature, systematic review #2; Riffenburgh source also referenced in Appendix A, "Pathology" literature, controlled study #7).

360 *Id.*

361 *Id.*

362 *See id.*; *infra* Part II.B.1.b.

363 *See, e.g.*, Aaron M. Gleckman et al., *Optic Nerve Damage in Shaken Baby Syndrome*, 124 ARCHIVES PATHOLOGY & LABORATORY MED. 251, 252 tbl., 255 (2000) (source also referenced in Appendix A, "Pathology" literature, controlled study #4); Donald L. Budenz et al., *Ocular and Optic Nerve Hemorrhages in Abused Infants with Intracranial Injuries*, 101 OPHTHALMOLOGY 559, 561 (1994) (source also referenced in Appendix A, "Pathology" literature, controlled study #2); Gilliland & Luckenbach, *supra* note 320, at 191.

364 *See* S. Maguire, *Which Clinical Features Distinguish Inflicted from Non-Inflicted Brain Injury? A Systematic Review*, 94 ARCHIVES DISEASE CHILDHOOD 860, 860 (2009) (source also referenced in Appendix A, "General" literature, systematic review article #4).

365 *Id.* at 861, 864 fig.1.

Maguire et al. found 14 studies that met those criteria, representing over 1600 children.[366] Cases were included only if strict definitional criteria for "inflicted" brain injury (i.e., those with witnessed abuse, confessions, legal decisions, or outcome confirmation by multi-agency child protection teams) was met.[367] The authors specifically excluded all studies where the decision of abuse relied solely on clinical features, so as to eliminate concerns for "selection bias" and "circularity."[368]

Conducting a multi-level logistic regression analysis, Maguire et al. found that RHs were "strongly associated with inflicted brain injury, with a positive predictive value of 71% and an odds ratio of 3.504."[369] Again, remembering odds ratios, based upon a comprehensive review of ALL the literature involving RHs, RHs are 3.5 times more likely to occur in inflicted circumstances than non-inflicted ones. The authors concluded, "By producing a multilevel logistic regression of specific clinical features on over 1600 children, we have shown that there is scientific evidence to support the distinction between [inflicted brain injury] and [non-inflicted brain injury] . . . . "This review is the largest of its kind, and offers for the first time a valid statistical probability of [inflicted brain injury] when certain key features are present (e.g., retinal haemorrhages)."[370]

In 2010, Bhardwaj et al. also published a systematic review of the diagnostic accuracy of RHs in AHT.[371] Upon examining three large medical databases, the authors identified 971 articles, and fifty-five met their relevance criteria for grading purposes.[372] Using a published grading checklist (designed to ensure the highest quality of design in studies), Bhardwaj et al. found twenty studies that met

366 *Id.* at 863–64.

367 *Id.* at 861.

368 *Id.*

369 *Id.* at 865.

370 *Id.*; *see* Maguire et al., *supra* note 364, at 865.

371 Bhardwaj et al., *supra* not 359, at 984.

372 *Id.*

inclusion criteria.[373] Similar to the "Quality of Evidence Ratings system" employed by Donohoe (a ratings system that was used to critique the quality of literature behind AHT), Bhardwaj et al. found that the specificity of intra-ocular hemorrhages (RHs) for AHT was 94%.[374] The authors concluded:

> Currently, there is level II evidence from prospective controlled studies, supporting a significant relationship between IOH [(intraocular hemorrhage)] and AHT.... Level I evidence is impossible to achieve in this field, for obvious reasons.... Combined data from prospective studies of head injury indicate that IOH have a specificity of 94% for abuse.[375]

Thus, again, with regards to validity and reliability, there are two systematic reviews (comprising over thirty well-designed clinical studies and thousands of children), several well-designed prospective studies, and numerous retrospective reviews from multiple lines of research, general pediatrics, ophthalmology, and pathology, all of which have produced the same results: the highly significant statistical association of severe RHs with AHT. To this author's review of the evidence based medical literature, there are no published, peer-reviewed clinical studies that conclude differently.

c. Other Statistical Evidence

Well-designed comparative studies have demonstrated a statistically significant worse outcome (for both physical and cognitive functioning) for AHT patients over accidental trauma

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373 *Id.*

374 Bhardwaj, *supra* note 359, at 991. "Level I evidence provides strong support for a statement, and is usually composed of well-performed, randomized controlled-trials or meta-analyses of randomized controlled-trials. Level II evidence provides substantial support for the statement... [and] usually includes observational studies, such as cohort studies and case control studies. Level III indicates a weak body of evidence relying on consensus statements, small noncomparative case series, and individual case reports." *Id.* at 984; *see also* Alex V. Levin et al., *Clinical Report: The Eye Examination in the Evaluation of Child Abuse*, 126 PEDIATRICS 376, 376–77 (2010) (discussing use of intraocular hemorrhage diagnoses in assessing AHT) (source also referenced in Appendix A, "Ophthalmology" literature, systematic review #4).

375 *See* Bhardwaj, *supra* note 359, at 990–91.

patients. In 1997, Haviland and Russell published the results of their comparative retrospective review of the outcomes of fifteen children, under age two, admitted to the pediatric ICU with AHT, and ten children, under age two, admitted to the same pediatric ICU during the same time-frame with known accidental head trauma.[376] Haviland and Russell followed the children for up to three years.[377] Of the AHT group, two patients died.[378] Of the remaining thirteen survivors, seven (54%) showed "severe" (meaning total mental and physical dependence) handicap, four (31%) had "moderate" (meaning partial paralysis, blindness, and developmental delay), one (8%) had "mild" (meaning partial paralysis and seizures), and only one (8%) was considered "normal" at a three-month follow-up.[379] Of the accidental group, one patient died.[380] Of the remaining nine survivors, only one (11%) had severe handicap, one (11%) had mild handicap, and seven were considered normal at discharge.[381] This computed to a p-value of less than 0.01 (1%).[382] Similar results were reproduced by Hymel et al., Vinchon et al., Keenan et al., and Ewing-Cobbs et al.[383]

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376 *See* J. Haviland & R.I. Ross Russell, *Outcome After Severe Non-Accidental Head Injury*, 77 ARCHIVES DISEASE CHILDHOOD 504, 504–05 (1997) (source also found in Appendix A, "General" literature, comparative study #16).

377 *Id.* at 505.

378 *Id.*

379 *Id.* At discharge nine AHT survivors were deemed severe, three fell in the moderate category, and one patient was normal. *Id.* at 506 tbl.3.

380 *Id.* at 505.

381 *Id.* at 505, 506 tbl.4. The article presents conflicting data. The body of the article only accounts for eight of the nine survivors, stating six of the survivors were deemed normal at discharge. *Id.* at 505. Because the percentages stated on page 505 do not add up 100%, I relied on Table 4 data, which showed seven survivors had a normal status at discharge. *See id.* at 505, 506 tbl.4.

382 *See id.*

383 *See* Heather T. Keenan et al., *Neurodevelopmental Consequences of Early Traumatic Brain Injury in 3-Year-Old Children*, 119 PEDIATRICS e616, e619–e620 (2007) (source also referenced in Appendix A, "General" literature, controlled study #5); Matthieu Vinchon et al., *Infantile Traumatic Subdural Hematomas: Outcome after Five Years*, 39 PEDIATRIC NEUROSURGERY 122, 124–25 (2003) (source also referenced in Appendix A, "Neurosurgery" literature, prospective study #4); Linda Ewing-Cobbs et al., *Late Intellectual and Academic Outcomes Following Traumatic Brain Injury Sustained During Early Childhood*, 105 J.

Other studies have focused on the significance of a discrepant clinical history to explain significant traumatic findings. A clear, biomechanically plausible account for how the injuries occurred should be available. When the history is absent, minimal, changing, or mechanistically implausible, suspicion of abusive injury is raised. In 2003 Hettler and Greenes, members of an emergency medicine group from Children's Hospital of Boston, examined the very issue of whether certain historical features are predictive of AHT.[384] Their retrospective review of 163 children, age three or younger, included patients admitted from 1993 to 2000 with acute traumatic intracranial injury.[385] The authors classified cases "as either 'definite abuse' or 'not definite abuse'... [based upon] radiologic, ophthalmologic, and physical examination findings, without regard to the presenting history."[386] Forty-nine out of 163 (30%) were classified as "definite abuse" and 114 out of 163 (70%) were classified as "not definite abuse."[387] Upon statistical analysis Hettler and Greenes found that no history of trauma had a 97% specificity and 92% positive predictive value for AHT.[388] When analyzed in the subgroup of patients with persistent neurologic abnormality at discharge, no history of trauma had a specificity of 100% and positive predictive value of 100% for AHT.[389] Studies by Duhaime et al. and Keenan et al. also confirm the association of discrepant clinical history and AHT.[390]

NEUROSURGERY: PEDIATRICS 287 (2006), http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2615233/pdf/nihms23194.pdf (source also referenced in Appendix A, "General" literature, comparative studies #11); Hymel et al., *Mechanisms*, *supra* note 285, at 924-25, 927 tbl.4; Vinchon et al., *supra* note 280, at 641 tbl.3; Ewing-Cobbs et al., *supra* note 285, at 303–04.

384 Joeli Hettler & David S. Greenes, *Can the Initial History Predict Whether a Child with a Head Injury has been Abused?*, 111 PEDIATRICS 602, 602 (2003) (source also referenced in Appendix A, "General" literature, retrospective article #17).

385 *Id.*

386 *Id.*

387 *Id.* at 603.

388 *Id.* at 602, 605 tbl.4.

389 *Id.* at 604.

390 Duhaime et al., *supra* note 262, at 184; *see* Heather T. Keenan et al., *Child Outcomes and Family Characteristics 1 Year After Severe Inflicted or Noninflicted Traumatic Brain Injury*, 117

d. Fallacy of Circular Reasoning, Alternative Hypotheses, & Data Gaps

i. "Circular Reasoning"?

It is appropriate at this point to address a criticism frequently levied against the medical literature on AHT: the logical fallacy of "circular reasoning."[391] While certainly some of the medical literature suffers from these design flaws, there are several factors not addressed by this critique. First, how does the logical fallacy of "circular reasoning," which essentially states a poor design of the medical studies, explain the associative findings of subdural hemorrhage and retinal hemorrhages found by Ingraham, Caffey, Guthkelch, Silverman, Kempe, and countless other historical authors, who reported these findings even before the diagnosis of Non-Accidental Injury existed?[392] What was their improper "design" in reporting these associative findings? Were these physicians somehow biased towards reporting these findings? Is it that these astute physicians were not rigorous or meticulous enough in their reasoning and evaluations to exclude other important causes such that the association of SDHs and RHs was not valid? Or is it that we are simply going to attribute the multiple reports of these associated findings to chance?[393] Why is it that multiple historical physicians, separated by significant geographical distance, in unrelated, various fields of medical study, and with no social or medical inclination to make these findings, collectively found the same associated findings? In order to dismiss the associative strength of these findings (subdural hemorrhages and retinal hemorrhages), an appropriate response must first be given to all these historical physicians.

PEDIATRICS 317, 317 (2006); *see also* Keenan et al., *supra* note 285, at 637.

391 *See* Tuerkheimer, *supra* note 4, at 13 & n.76 (citing the testimony of a defense expert, neuroradiologist Dr. Patrick Barnes, in *State v. Edmunds*). The assertion is that writers of much of the medical literature on Abusive Head Trauma "select[ed] cases by the presence of the very clinical findings and test results they [sought] to validate as diagnostic." *Id.* at 13 As Dr. Barnes simply stated, "SBS=SDH + RH [inclusion criteria], therefore, SDH + RH=SBS [conclusion]." *Id.*

392 *See supra* Section II.A. (explaining the direct contributions of these and other authors).

393 *Id.*

Second, some circularity is inevitable, because we are unwilling to experimentally shake infants, and even reliably confessed accounts have some doubt. As detailed above, to the greatest extent possible, numerous well-designed studies set out to control circularity in their experimental design. When scientists critically examined those studies for bias secondary to circularity, not only was that bias lacking, but also scientists found results that were consistent with the rest of the clinical literature.[394] Therefore, although the possibility of circularity is present, and to some degree inevitable, we are unlikely to find substantially better evidence than we currently have for the absence of circularity.

Finally, telling evidence arguing against circularity is the absence of *any* large trials demonstrating a lack of association of either SDHs or RHs with AHT. If circularity were truly a valid criticism of the current clinical medical literature, in over twenty years of research on the topic, would there not exist *one* well-designed study that demonstrated a lack of association of either SDHs or RHs with AHT? Where is that study?

ii. Alternative Hypotheses

There have been two recent alternative hypotheses[395] for SDHs and RHs that have been the subject of some controversy—Geddes' "Unified Hypothesis," and Squier & Mack's "dural immature vascular plexus theory."[396] Geddes' Unified Hypothesis purports that "hypoxia [(lack of oxygen)], brain swelling and raised central

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394 *Id.* (detailing studies that accounted for "circularity").

395 There have been a few other hypothesized mechanisms (such as increased intrathoracic pressure) for SDHs. But addressing all of these hypothesized mechanisms is beyond the scope of this article. For further information regarding these hypothesized mechanisms, I would refer the reader to Frasier et al, *supra* note 14, and Rorke-Adams et al., *supra* note 14.

396 J.F. Geddes et al., *Dural Haemorrhage in Non-Traumatic Infant Deaths: Does it Explain the Bleeding in "Shaken Baby Syndrome"?*, 29 NEUROPATHOLOGY & APPLIED NEUROBIOLOGY 14, 14 (2003) (source also referenced in Appendix A, "Pathology" literature, retrospective article #9); Waney Squier & Julie Mack, *The Neuropathology of Infant Subdural Haemorrhage*, 187 FORENSIC SCI. INT'L 6, 12 (2009); Julie Mack et al., *Anatomy and Development of the Meninges: Implications for Subdural Collections and CSF Circulation*, 39 PEDIATRIC RADIOLOGY 200, 200 (2009) (sources also referenced in Appendix A, "Pathology" literature, review articles #6 & 9).

venous pressure cause blood to *leak* from intracranial veins into the subdural space, and that the cause of subdural bleeding in some cases of infant head injury is therefore not traumatic rupture of bridging veins, but a phenomenon of immaturity."[397] The essential components of this hypothesis are that hypoxic (lack of oxygen) injury to the brain results in increased intracranial pressure and brain swelling, which leads to "leaky" intracranial veins and subdural hemorrhage.[398]

The basis for Geddes' hypothesis was a cohort of fifty postmortem cases: seventeen fetuses, three spontaneous abortions, sixteen perinatal (within a week of life), five neonatal (within one month of life) and nine infant (within one year of life) deaths—all which resulted from non-traumatic causes.[399] Geddes et al. found microscopic intradural (within the layers of the dura, but not on the surface of the brain) blood in thirty-six of the fifty cases (72%).[400] However, if one excludes the fetuses and abortions, microscopic intradural blood was found in just thirteen of the thirty (43%) of the perinatal/neonatal/infant cases.[401] Macroscopic SDH (visible on the surface of the brain) was found in only one of the fifty cases (2%), an infant with overwhelming sepsis (infection).[402] Although an ophthalmologist was a co-author of the study, the authors did not examine or comment on retinal hemorrhages in their cohort.[403] Based upon the microscopic intradural findings, Geddes et al. hypothesized that *intradural* blood could "ooze" in the potential subdural space and result in *macroscopic* SDHs, although this did not occur in forty-nine out of fifty patients in their cohort.[404] Furthermore, based upon their data and calculations, Geddes et al. determined the *p*-value of hypoxia and macroscopic SDH to be

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397 Geddes et al, *supra* note 396, at 14 (emphasis added).

398 *See id.* at 19.

399 *Id.* at 15.

400 *Id.*

401 *See id.* at 15, 17 tbl.2.

402 *Id.* at 15.

403 *See generally id.*

404 *See id.* at 15, 19.

0.15.[405] Thus, based upon their own data, the authors could not even conclude that chance had been ruled out.[406] These results cannot be construed as statistically significant.[407] Geddes et al.'s results were three times higher than the highest limit of statistical acceptability (p=0.05).[408] This is truly notable when one compares it to the vast majority of statistical data supporting AHT (as discussed above), where p-levels are in the order of 0.01 to 0.001.[409]

Since the Unified Hypothesis was published in 2003, only one other peer-reviewed, clinical study has been published in the medical literature supporting this hypothesis.[410] In 2007, Cohen and Scheimberg published the pathologic results of a prospective series of twenty-five fetuses (age twenty-six to forty-weeks) and thirty neonates (age one hour to nineteen days) who suffered hypoxic (lack of oxygen)-ischemic (lack of blood) injury (HII).[411] Cohen and Scheimberg found macroscopic SDHs in sixteen out of twenty-five (64%) fetuses, and twenty out of thirty (66%) neonates.[412] As with Geddes' study, no examination or comment was made on the concurrent presence or absence of RHs.[413]

The critiques of this study have been two-fold. One, it is well known that birth trauma is a cause of SDHs (secondary to dural tears involving the sinuses).[414] Thus, there was no explanation or

[405] *See id.* at 17.

[406] *Id.*

[407] *See id.*

[408] *See id.*

[409] *See supra* Section II.B.2.a.

[410] *See* Marta C. Cohen & Irene Scheimberg, *Evidence of Occurrence of Intradural and Subdural Hemorrhage in the Perinatal and Neonatal Period in the Context of Hypoxic Ischemic Encephalopathy: An Observational Study from Two Referral Institutions in the United Kingdom*, 12 PEDIATRIC & DEVELOPMENTAL PATHOLOGY 169 (2009); (source also referenced at Appendix A, "Pathology" literature, prospective article #1); *see also infra* Part III.A (discussing the legal ramifications of the "Unified Hypothesis" in United Kingdom courts).

[411] Cohen & Scheimberg, *supra* note 410, at 169.

[412] *Id.*

[413] *See generally id.*

[414] *See* C. Smith, & J. Bell, *Shaken Baby Syndrome: Evidence and Experts*, 50 DEV. MED. CHILD NEUROLOGY 6, 6 (2008).

accounting for this confounding variable.[415] It is not known how the authors distinguished which patients' SDHs were secondary to birth trauma and which were secondary to hypoxic-ischemic injury (HII).[416] Two, the age of Cohen and Scheimberg's patient cohort was not similar to the age of patients commonly involved in AHT.[417]

When considering Geddes' hypothesis that hypoxia (lack of oxygen) results in SDHs,[418] radiology studies are also helpful. Clinical radiology studies do not support an association of SDH and hypoxia.[419] MRI studies demonstrate that the pattern of hypoxic-ischemic injury (HII) in the brain is characteristically *intraparenchymal* (inside the brain tissue) hemorrhage, along with cortical (brain tissue) necrosis (death).[420] *SDH is not a part of that pattern*.[421] In 1998, Dubowitz et al. published the results of their retrospective review of the MRIs of twenty-two children (age six months to eleven years), who suffered HII (hypoxic-ischemic injury) after near drowning episodes.[422] While a variety of MRI findings were encountered, *none of the patients had a SDH*, and only one had a possible hemorrhage, and that was intraparenchymal.[423] Similar results have been published by Baenziger et al., Sie et al., Rutherford

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[415] *See generally* Cohen & Scheimberg, *supra* note 410.

[416] *See generally id.*

[417] *Compare* Geddes et al., *supra* note 396, at 14 (using fetuses with gestational ages of 18 to 41 weeks and newborn with ages as high as five months) *with* Cohen & Scheimberg, *supra* note 410, at 169 (using fetuses with gestational ages of 26 to 40 weeks and newborns with ages between 1 hour and 19 days).

[418] Geddes et al., *supra* note 396, at 14.

[419] Tim Jaspan, *Current Controversies in the Interpretation of Non-Accidental Head Injury*, 38 PEDIATRIC RADIOLOGY s378, s382 (Supp. 2008) (source also referenced in Appendix A, "Radiology" literature, review #3).

[420] *See generally* Benjamin Y. Huang & Mauricio Castillo, *Hypoxic-Ischemic Brain Injury: Imaging Findings from Birth to Adulthood*, 28 RADIOGRAPHICS 417, 433 (2008).

[421] *See* Jaspan, *supra* note 419, at s382.

[422] *See* David J. Dubowitz et al., *MR of Hypoxic Encephalopathy in Children after Near Drowning: Correlation with Quantitative Proton MR Spectroscopy and Clinical Outcome*, 19 AM J. NEURORADIOLOGY 1617, 1618 (1998) (source also referenced in Appendix A, "Radiology" literature, retrospective article #7).

[423] *Id.* at 1620–22, 1626.

et al., and Barkovich et al.[424]

CT imaging has also failed to demonstrate SDHs in patients with HII.[425] In 2008, Rafaat et al. published their retrospective review of the CT findings in children suffering drowning episodes.[426] Of the 156 children included in their seventeen-year review, none had an intracranial hemorrhage.[427] Additionally, SDH is "conspicuously absent" from standard textbooks of neonatal neurology or MRI when addressing HII in infancy and childhood.[428]

Two recent pathology studies have evaluated the incidence of SDHs in HII cases. In 2007, Byard et al. published the results of a retrospective study of eighty-two fetuses, infants, and toddlers with proven HII and no trauma.[429] The cooperative study was undertaken by multiple forensic in Australia, the United Kingdom, Germany, Denmark, and the United States.[430] The age range of the eighty-two patients was thirty-five weeks gestation to three years.[431] All cases had histologically confirmed HII.[432] "Causes of the hypoxic episodes were ... sudden infant death syndrome ... [(SIDS)] (N = 30), drowning (N = 12), accidental asphyxia (N = 10),

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[424] *See* O. Baenziger et. al., *Early Pattern Recognition in Severe Perinatal Asphyxia: A Prospective MRI Study*, 35 NEURORADIOLOGY 437, 440 (1993); A. James Barkovich et. al., *Perinatal Asphyxia: MR Findings in the First 10 Days*, 16 AM. J. NEURORADIOLOGY 427, 427 (1995); Mary Rutherford et al., *Hypoxic-ischaemic Encephalopathy: Early and Late Magnetic Resonance Imaging Findings in Relation to Outcome*, 75 ARCHIVES DISEASE CHILDHOOD F145, F145, F151 (1996); L.T. Sie et al., *MR Patterns of Hypoxic-Ischemic Brain Damage After Prenatal, Perinatal or Postnatal Asphyxia*, 31 NEUROPEDIATRICS 128, 128 (2000).

[425] *See* Karim T. Rafaat et al., *Cranial Computed Tomographic Findings in a Large Group of Children with Drowning: Diagnostic, Prognostic, and Forensic Implications*, 6 PEDIATRIC CRITICAL CARE MED. 567, 567 (2008) (source also referenced in Appendix A, "Radiology" literature, retrospective article #11).

[426] *Id.*

[427] *Id.* at 567–68.

[428] Jaspan, *supra* note 419, at s382.

[429] Roger W. Byard et al., *Lack of Evidence for a Causal Relationship Between Hypoxic-Ischemic Encephalopathy and Subdural Hemorrhage in Fetal Life, Infancy, and Early Childhood*, 10 PEDIATRIC & DEVELOPMENTAL PATHOLOGY 348, 348 (2007) (source also referenced in Appendix A, "Pathology," literature, retrospective article # 2).

[430] *Id.*

[431] *Id.*

[432] *Id.*

intrauterine/delivery asphyxia (N = 8), congenital disease (N = 6), aspiration of food/gastric contents (N = 4), inflicted asphyxia (N = 3), epilepsy (N = 1), dehydration (N = 1), drug toxicity (N = 1), complications of prematurity (N = 1), and complications of anesthesia (N = 1)."[433] In four instances, no initiating event was determined and "[i]n no case was there macroscopic evidence of subdural hemorrhage."[434]

In 2010, Hurley et al. published the results of a retrospective study of fifty children less than four years old who had suffered non-traumatic cardio-respiratory arrest and died at their institution between January 2001 and May 2007.[435] Specifically, the authors were looking to see whether there was a causal relationship between hypoxic-ischemic events (associated with cardio-respiratory arrest) and SDHs.[436] All children who had evidence of cranial trauma (even those with findings of occult head trauma on post-mortem examination) were excluded.[437] Additionally, other children were also excluded if they had evidence of a bleeding disorder, infection, metabolic, or degenerative neurological conditions.[438] The authors identified fifty children younger than four years of age who met their strict inclusion criteria; forty-eight of those fifty children were less than twenty-four months old.[439]

The average resuscitation time of children in the study was twenty-one minutes.[440] Forty of the fifty children died and had post-mortem examinations.[441] Thirty-nine of the forty post-mortem

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433 *Id.*

434 *Id.*

435 *See* M. Hurley, et al., *Is There a Causal Relationship Between the Hypoxia-Ischaemia Associated with Cardiorespiratory Arrest and Subdural Haematomas? An Observational Study*, 83 BRIT. J. RADIOLOGY 736, 736–37 (2010) (source also referenced in Appendix A, "Pathology" literature, retrospective article #15).

436 *Id.* at 736.

437 *Id.*

438 *Id.* at 736–37.

439 *Id.* at 737.

440 *Id.*

441 *Id.* at 738.

examinations had no macroscopic evidence of SDH.[442] The one child (a 19-day old infant victim of an overlaying incident), who had macroscopic evidence of SDH, had a clot adhering to the dura, which the pathologist felt was consistent with birth-related trauma.[443] Of the five children in the study who had retinal examinations, none had RHs.[444] Thus, the authors concluded that "*cardiopulmonary collapse per se and the attendant hypoxic-ischemic sequelae do not cause SDH.*"[445] The previously mentioned study by Matschke et al. identified similar results.[446]

The more recent version of Geddes' Unified Hypothesis is Squier and Mack's dural immature vascular plexus theory.[447] In this theory, the authors hypothesize that there is a plexus (network) of vessels within the dura mater that is immature and the most likely source for hemorrhage in non-traumatic conditions.[448] Akin to Geddes' Unified Hypothesis, the authors purport that hypoxia is the preeminent factor causing these immature vessels to leak, and subsequently result in SDHs.[449] However, also akin to Geddes' Unified Hypothesis, this theory offers *no scientific data* linking an *intradural* (within the dura) vascular plexus to the significant *subdural* hemorrhages in trauma.[450] Unlike even Geddes' Unified Hypothesis, this theory *has not been studied in any cohort of patients.* Thus, like Geddes' Unified Hypothesis, this theory is untested by the rigors of scientific falsifiability and unsupported by the medical literature. The legal analysis of these two hypotheses will be discussed in the *Daubert* analysis below.

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442 *Id.*

443 *Id.*

444 *See id.* at 737.

445 *Id.* at 743 (emphasis added).

446 Matschke et al., *supra* note 255, at 1594.

447 *See* Squier & Mack, *supra* note 396, at 8.

448 *Id.* at 8–9.

449 *Id.* at 10.

450 *See id.* at 10, 12.

iii. Data Gaps

In every field of medicine, there are areas of incomplete information, where research and further investigation are beneficial. This is true for child abuse pediatrics as well. However, incomplete information does not necessarily equate to insufficient information. As previously outlined, vast amounts of historical reports, research data, and clinical experience have established quality, evidence-based information for the diagnosis of AHT with a reasonable degree of medical certainty.

That being said, some questions remain unanswered. Current areas in question include: 1) what are the exact tolerance and failure limits of the multiple intracranial structures (the dura mater, cortical bridging veins, the unmyelinated infant brain) of the human infant; 2) how do those structures, as well as other intracranial entities (such as cerebrospinal fluid), independently and collectively act to increase or decrease biomechanical forces; 3) what are the exact forces required to induce SDHs and DAI (Diffuse Axonal Injury) in the human infant brain; 4) what are the tolerance and failure limits of the infant cervical and thoracic spine;[451] 5) what is the exact mechanism for RHs and what role do multiple physiologic factors, such as increased intracranial pressure and biochemical (prostaglandins) release, play in their causation; and, 6) what are the incidence and prevalence of rare AHT "mimickers" (osteogenesis

[451] Much has recently been made of the biomechanical research arguing against the validity of AHT/SBS. *See* Bazelon, *supra* note 5. Given the ethical limitations of research in the field, much prior pediatric biomechanical research was scaled data based upon adult values. *See* Jason F. Luck et al., *Tensile Mechanical Properties of the Perinatal and Pediatric PMHS Osteoligamentous Cervical Spine*, 52 STAPP CAR CRASH J. 107, 107–09 (2008). This left what was described by many learned researchers in the field as a "*significant void in pediatric cervical spine biomechanics.*" *Id.* at 107. Although recent biomechanical research upon post-mortem infants is an improvement on that prior data, it is still limited and approximate. *See id.* at 109. In fact, Luck et al. found that "juvenile animal surrogates estimate the stiffness of the human cervical spine fairly well." *Id.* at 107. Along that vein of approximate data, recent animal studies (specifically, seven to ten day-old anesthetized lambs vigorously shaken by humans) have produced the exact same injuries commonly found in AHT/SBS—subdural hemorrhages and retinal hemorrhages. *See* John W. Finnie et al., *Diffuse Neuronal Perikaryal Amyloid Precursor Protein Immunoreactivity in an Ovine Model of Non-Accidental Head Injury (the Shaken Baby Syndrome)*, 17 J. CLINICAL NEUROSCIENCE 237, 237–39 (2010).

imperfecta, glutaric aciduria type 1, etc.) in AHT cases. Ethical and logistical challenges may limit progress to research in the child abuse field.

These questions, and others, have already been identified by experts in the field of AHT as areas of present and future research.[452] Improvements in the biofidelity of anthropomorphic doll models, computer finite modeling of the intracranial and intraocular structures, and the identification of potentially specific biochemical markers of traumatic brain injury are just some of the examples of advancements in AHT research. Efforts to address these unknowns will only further enhance our understanding of AHT.

C. Coming to the Diagnosis of AHT

AHT is "those constellations of injuries that are caused by the directed application

of force to an infant or young child, resulting in physical injury to the head and/or its contents."[453] Commonly observed injuries include scalp injury (e.g., bruises, lacerations/abrasions, swelling), skull fractures, intracranial (inside the skull) hemorrhage (i.e., SDH, subarachnoid hemorrhage, epidural hemorrhage, intraparenchymal hemorrhage), diffuse axonal injury,[454] cerebral edema (brain swelling), encephalopathy, cervical spine fractures, cervical spinal cord injury/hemorrhage, retinal hemorrhages, rib fractures, and long bone fractures. While any of the above injuries can result from, or accompany, AHT, the most common injuries associated with AHT are SDHs and RHs.

Recent legal literature and cases have cited a "diagnostic triad" of SDHs, RHs and encephalopathy as defining AHT.[455] As this

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[452] *See, e.g.*, Brian J. Forbes et. al., *Inflicted Childhood Neurotrauma (Shaken Baby Syndrome): Ophthalmic Findings*, 41 J. PEDIATRIC OPHTHALMOLOGY & STRABISMUS 80, 86 (2004).

[453] *See* Chiesa & Duhaime, *supra* note 13, at 317.

[454] "Diffuse Axonal Injury" refers to damage of the brain to a widespread, not focal, area; it most commonly manifests as lesions of the white matter tracts of the brain. *See* Douglas H. Smith et al., *Diffuse Axonal Injury in Head Trauma*, 18 J. HEAD TRAUMA REHABILITATION 307, 308 (2003).

[455] *See* Tuerkheimer, *supra* note 4, at 4 & n.18, 7 n.39.

review has described, there is a clear, strong, and highly statistically significant association of SDHs and RHs with trauma.[456] However, the mere presence alone of SDHs and RHs does not establish a diagnosis of AHT.

A thorough evaluation, which includes, at a minimum, a complete medical history and physical examination, is required to rule out other causes for the findings. A multidisciplinary approach that involves careful review of psychosocial and investigative details is ideal. Akin to the well-establish medical diagnosis of battered child syndrome, AHT also finds its foundation in "the degree and type of injury [that] is at variance with the history given regarding the occurrence of trauma."[457]

Arriving at the diagnosis is no different than arriving at any other clinical medical diagnosis: it starts with a "chief complaint." In the context of AHT, usually this comprises a presenting symptom or symptoms, such as apnea (stopping breathing), irritability, change in mental status, seizures, lethargy, vomiting or others.[458] With that initial presenting symptom(s), a clinical provider will obtain a comprehensive medical history. This includes a detailed history of the events surrounding the presenting symptom(s), a trauma history, a history of infectious symptoms or exposures, a detailed past medical history (including prior illnesses, surgeries, hospitalizations, and birth history, if applicable), a developmental history, a history of relevant family medical illnesses/disorders, and a comprehensive psychosocial history (including identification of psychosocial stressors, preexisting or concurrent mental health disorders, substance abuse, domestic violence, and prior concerns for child maltreatment/neglect).[459] Typically, this history is obtained by asking the caregiver open-ended, non-suggestive questions, such as: "What happened/did you do next?" or, "How did the infant/child act then/thereafter?" or, "Tell me about your child's

456 Although not discussed in this review, "encephalopathy" is also associated with trauma.

457 *See* Kempe et al., *supra* note 148, at 143.

458 *See* Chiesa & Duhaime, *supra* note 13, at 321.

459 *Id.* at 319–20.

daily activities in the days prior."[460]

Subsequent to the history, the clinical provider conducts, when applicable, a detailed, entire-body physical examination. [461] Special attention is paid to the head, skin, and abdominal, genitourinary, and skeletal systems to assess for signs of trauma.[462] Although the physical examination is an important part of the diagnostic process, historical reports and recent studies have confirmed the absence of any physical findings of trauma on exam in upwards of 31% of AHT cases.[463]

After obtaining a history and performing a physical examination, the clinician considers the various diagnoses that might explain the clinical presentation.[464] This is also known as the "differential" (list of possible causes).[465] The clinician will formulate differentials for all the relevant injuries. For the limited purposes of this article, the most common injuries involved in AHT—SDHs and RHs—have been considered. When presented with the differentials for those injuries (listed in Appendix B and C), the clinician then goes through the complex inferential and deductive process of differential refinement.

Whereas this clinical methodology was once believed to be a linear, Bayesian analysis, it is now understood that the diagnostic process is a dynamic, non-linear, unstructured method of problem-solving.[466] Consequently, and especially in AHT cases, the clinician engages in a multi-disciplinary process of attaining additional

[460] *Id* at 319.

[461] *Id.* at 320. In certain cases, specifically, in certain cases of fatal AHT, a detailed physical examination either is impractical (secondary to the critical care needs of the child) or unwarranted, as further physical examination information will be obtained via autopsy. *See id.* at 323.

[462] *Id.* at 320.

[463] *See* Carole Jenny et al., *Analysis of Missed Cases of Abusive Head Trauma*, 282 JAMA 621, 623 & tbl.2 (1999) (showing physicians failed to detect AHT 31.2% of the time) (source also referenced in Appendix A, "General" literature, retrospective study #19); *see also* Hymel et al., *Head Injury Depth*, *supra* note 285, at 712, 716 tbl.3 (showing abused children might not show scalp or skull injury, but still may have brain injury).

[464] *See* Chiesa & Duhaime, *supra* note 13, at 321 (discussing differential diagnoses)

[465] *See id.*

[466] *See infra* notes 523–24.

information.[467] The clinician cooperates with multiple agencies (social services and law enforcement) and multiple medical disciplines (radiology, ophthalmology, neurosurgery, etc.) to obtain additional history and clinical information.[468] Furthermore, the clinician examines existing laboratory and radiologic data, and determines the necessity of additional laboratory and/or radiologic testing.[469] Once having received the additional information, the clinician synthesizes that information with the known pathophysiologic processes of the human body, the evidence-based statistical information on the injuries, and the clinician's own experience in patient care.[470]

For SDHs and RHs, many of the potential disorders on the differential can be eliminated through a detailed history, physical examination, and initial laboratory and radiologic information.[471] *In the vast majority of cases, the common denominator for SDHs and RHs will be trauma.*[472] From there, the clinician must determine whether the clinical information is consistent with either accidental trauma or AHT.[473]

In arriving at that determination, the clinician closely examines the historical information for consistency. Inconsistency can appear in a variety of ways. The history provided for the injury may have internal features to the story, which are inconsistent with themselves. A history may *substantially* evolve or change as it is told to multiple providers. Other examples of inconsistency include: 1) a history that is absent in the presence of severe injuries; 2) a history that is inconsistent with the known developmental capabilities of the child; 3) a history that is inconsistent, pathophysiologically,[474] with

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[467] *See* Chiesa & Duhaime, *supra* note 13, at 320. In fact, a multidisciplinary child protection team approach has become the standard of care in many jurisdictions. *See id.* at 319.

[468] *See id.* at 321.

[469] *See id.* at 322.

[470] *See id.* at 319–20.

[471] *Id.* at 321.

[472] *See id.* at 321, 323

[473] *See id.* at 322.

[474] This presumes that after reasonable medical investigation there is still no other discernible

the injuries; or 4) a history that is inconsistent with the extensive clinical studies and statistical information (described in the section above, and in Appendix A on SDHs and RHs). As has long been validated, both medically and legally, through the diagnosis of battered child syndrome, if a clinician determines the injuries are "at variance with the history given regarding the occurrence of trauma,"[475] then the clinician can diagnose AHT/non-accidental trauma with a reasonable degree of medical certainty.

D. "A Shifted Consensus?"

As mentioned above, recent authors and cases have cited "a shift in mainstream medical opinion" against the validity of AHT as a medical diagnosis.[476] Other proffers have included: "[a]nd as technology and scientific methodology advanced, researchers questioning the basis for SBS reached *a critical mass*."[477] There is but one simple question for these assertions: Where is the evidence/data for these assertions (other than the opinions of known defense experts)?

Rather than respond in like, with unsupported generalizations, this author will simply cite, with supporting, verifiable references, the various international and domestic medical organizations that have publicly acknowledged the validity of AHT as a medical diagnosis:[478]

1)The World Health Organization[479]

2)The Royal College of Paediatrics and Child Health[480]

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medical cause for the injuries.

[475] *See* Kempe et al., *supra* note 148, at 143.

[476] Tuerkheimer, *supra* note 4, at 5 (citing State v. Edmunds 746 N.W.2d 590, 598–99 (Wis. Ct. App. 2008)).

[477] *See* Tuerkheimer, *supra* note 4, at 14 (emphasis added).

[478] Some of the below listed organizations have explicitly acknowledged support through practice guidelines or similar promulgations, while others have implicitly done so by providing clinician or patient education materials on their websites.

[479] *See* Jonathan Dart & Sarah Cumberland, *Fragile Brain, Handle with Care*, 87 BULL. WORLD HEALTH ORG. 331, 331–32 (2009); Fact Sheet No. 150, Child Maltreatment, World Health Org. (Aug. 2010), http://www.who.int/mediacentre/factsheets/fs150/en/index.html.

[480] THE ROYAL COLL. OF PAEDIATRICS & CHILD HEALTH & ROYAL COLL. OF RADIOLOGISTS,

3)The Royal College of Radiologists[481]
4)The Royal College of Ophthalmologists[482]
5)The Canadian Paediatric Society[483]
6)The American Academy of Pediatrics[484]
7)The American Academy of Ophthalmology[485]
8)The American Association for Pediatric Ophthalmology and Strabismus[486]
9)The American College of Radiology[487]
10)The American Academy of Family Physicians[488]
11)The American College of Surgeons[489]
12)The American Association of Neurologic Surgeons[490]

STANDARDS FOR RADIOLOGICAL INVESTIGATIONS OF SUSPECTED NON-ACCIDENTAL INJURY 10 (March 2008), http://www.rcpch.ac.uk/sites/default/files/asset_library/Publications/S/StandardsforRadiologicalInvestigationsD.pdf.

481 *See id.*

482 *See* G. Adams et al., *Update from the Ophthalmology Child Abuse Working Party: Royal College Ophthalmologists*, 18 EYE 795, 795–96 (2004) *available at* www.rcophth.ac.uk/page.asp?section=493&search=.

483 *See Joint Statement on Shaken Baby Syndrome*, CANADIAN PAEDIATRIC SOC'Y, http://www.cps.ca/english/statements/pp/cps01-01.htm (last visited Oct. 23, 2011).

484 Christian et al., *supra* note 6, at 1410.

485 Alex V. Levin et al., *Information Statement: Abusive Head Trauma/Shaken Baby Syndrome*, AM. ACAD. OF OPHTHALMOLOGY (June 2010), http://one.aao.org/ce/practiceguidelines/clinicalstatements_content.aspx?cid=914163d5-5313-4c23-80f1-07167ee62579.

486 *Info for Patients: Shaken Baby Syndrome*, AM. ASS'N FOR PEDIATRIC OPHTHALMOLOGY & STRABISMUS, http://www.aapos.org/terms/conditions/97 (last visited Oct. 23, 2011).

487 *See* James S. Meyer, et al., *ACR Appropriateness Criteria: Suspected Physical Abuse – Child*, AM. COLL. RADIOLOGY http://www.acr.org/SecondaryMainMenuCategories/quality_safety/app_criteria/pdf/ExpertPanelonPediatricImaging/SuspectedPhysicalAbuseChildDoc9.aspx (last reviewed 2009).

488 *See* Liz Horsley, *AAP Guidelines on Evaluating Suspected Child Physical Abuse*, 77 AM. FAM. PHYSICIANS 1461, 1461–64 (2008), *available at* http://www.aafp.org/afp/2008/0515/p1461.html.

489 *See Patient Education*, AM. COLL. OF SURGEONS, http://www.facs.org/patienteducation/patient-resources/nervoussystem.html (last visited Aug. 26, 2011).

490 *Patient Information: Shaken Baby Syndrome*, AM. ASS'N OF NEUROLOGICAL SURGEONS (Nov.

13)The Pediatric Orthopaedic Society of North America[491]
14)The American College of Emergency Physicians[492]
15)The American Academy of Neurology[493]

While it is certainly true that the public promulgations of the various international and domestic medical societies are not representative of each and every member of that society, it is safe to conclude they are representative of the majority of its members. The notable subspecialties that have some discord amongst their members are pathologists (represented by the National Association of Medical Examiners) and biomechanical engineers.

III.THE *DAUBERT* ANALYSIS AND BEYOND

A. The *Daubert* Analysis

A *Daubert*/Trilogy scrutiny of AHT evidence/testimony can only begin at one place: *Daubert*. The *Daubert* court stated that when faced with a proffer of scientific testimony, "the trial judge must determine at the outset, pursuant to Rule 104(a), whether the expert is proposing to testify to (1) scientific knowledge that (2) will assist the trier of fact to understand or determine a fact in issue."[494] These are well-recognized as the reliability and relevance requirements of the trial judge's gate-keeping responsibilities.

In assessing reliability, the *Daubert* court clearly stated there is

2005) http://www.aans.org/Patient%20Information.aspx (follow "Click here to view Conditions and Treatments" hyperlink; then follow "Shaken Baby Syndrome" hyperlink).

[491] *See* *Child Abuse*, PEDIATRIC ORTHOPAEDIC SOC'Y OF N. AM, http://www.posna.org/education/StudyGuide/childAbuse.asp (last visited Oct. 23, 2011); *Fractures Associated with Head Injury*, PEDIATRIC ORTHOPAEDIC SOC'Y OF N. AM., http://www.posna.org/education/StudyGuide/fracturesAssociatedwithHeadInjury.asp (last visited Oct. 23, 2011).

[492] *See* Doraliz Hidalgo & Bernard L. Lopez, *Head Trauma in Children Younger Than 2 Years*, CRITICAL DECISIONS EMERGENCY MED., Apr. 2007, at 16 (presenting instruction for emergency physicians).

[493] *Shaken Baby Syndrome*, AM. ACAD. OF NEUROLOGY, http://www.aan.com/apps/disorders/index.cfm?event=database%3adisorder.view&disorder_id=1060 (last visited Oct. 23, 2011).

[494] Daubert v. Merrell Dow Pharm., Inc., 509 U.S. 579, 592 (1993) (footnotes omitted).

no checklist or specific test. However, in assessing the validity of the methodology underlying the proposed scientific testimony, the court enunciated four factors for the trial judge to consider:

1) whether a theory or technique could be (and had been) tested—also known as "falsifiability" or "testability;"[495]
2) "whether the theory or technique had been subjected to peer review and publication;"[496]
3) whether there was a "known or potential rate of error;"[497] and
4) whether there was "general acceptance" in the relevant scientific community.[498]

These four factors will be the starting point of our analysis.

The first two factors, the falsifiability of AHT and its subjection to peer review, are readily addressable. As has been demonstrated above, AHT has been tested or subjected to the scientific rigors of falsifiability by *multiple* disciplines and *multiple* methods.[499] Pediatricians, specifically those specializing in child abuse and neglect, have, over many years, studied and tested various facets of AHT diagnosis, such as symptom presentation, historical factors, physical examination findings, laboratory and radiologic findings, and outcomes.[500] Radiologists have utilized imaging modalities (CT and MRI) to assess the frequency and specificity of certain intracranial injuries, like SDHs, in traumatic and non-traumatic scenarios.[501] Biomechanical engineers have examined AHT from

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495 *Id.* at 593.

496 *Id.*

497 *Id.* at 594.

498 *Id.* Other factors for a trial court's consideration include whether "the expert's qualifications are sufficient . . . [whether] the method has been put to non-judicial uses . . . 'whether the expert's proposed testimony grows naturally and directly out of research the expert has conducted independent of the litigation' . . . 'whether the expert has unjustifiably extrapolated from accepted premise to unfounded conclusion' . . . [and] 'whether the expert has adequately accounted for alternative explanations.'" David v. Black & Decker (US) Inc., 629 F. Supp. 2d 511, 514 (W.D. Pa. 2009) (citing Magistrini v. One Hour Martinizing Dry Cleaning, 180 F. Supp. 2d 584, 594 (D.N.J. 2002), *aff'd* 68 Fed. Appx. 356 (3d Cir. 2003)) (citation omitted).

499 *See supra* Section II.B.2 ("Statistical Evidence").

500 *See, e.g.*, Hymel et al., *Head Injury Depth*, *supra* note 285, at 712–13.

501 *See, e.g.*, Dubowitz et al., *supra* note 422, at 1617 (using MRI in near drowning episodes);

primarily a "physical forces" perspective, seeking to exact quantifiable answers to the forces required to cause the intracranial and spinal injuries seen in AHT.[502] And, finally, pathologists have comparatively studied the microscopic and macroscopic tissue manifestations of the intracranial, intraocular, and spinal injuries in accidental and AHT cases.[503]

But not only has AHT been studied in multiple disciplines and by multiple methods, it also has been studied by *multiple* researchers from *multiple* nations. As has been discussed above, there exist at least 700 peer-reviewed, clinical medical articles, comprising thousands of pages of medical literature, published by over 1000 different medical authors, from at least twenty-eight different countries.[504] Additionally, AHT has been peer-reviewed and published in the following disciplines: biomechanical engineering, general pediatrics, neonatology, neurology, neurosurgery, nursing, obstetrics, ophthalmology, orthopedics, pathology (forensic pathology), radiology, and rehabilitative medicine.[505] In fact, given its association with significant medical injuries and child fatalities, AHT is the most peer-reviewed and well-published topic in child abuse pediatrics. Thus, it is difficult for one to assert or argue that the diagnosis of AHT has not been subjected to the rigors of scientific falsifiability, stringently peer reviewed, or well published.

The third criterion—the known or potential rate of error—is *Daubert*'s reference to statistical evidence either in support of or against a particular theory.[506] While certain scientific disciplines have a readily computable error rate, certain scientific disciplines do not. In clinical medical studies, the best approximation of an error

Wells et al., *supra* note 286, at 252 (assessment using CT).

[502] *See, e.g.*, Luck et al., *supra* note 451, at 107, 109 (showing use of a physical forces perspective).

[503] *See, e.g.*, Geddes et al., *supra* note 396, at 18–19.

[504] *See supra* text accompanying notes 234–35.

[505] *See infra* Appendix A.

[506] *See In re* Neurontin Mktg., Sales Practices & Prod. Liab., 612 F.Supp. 2d 116, 140 (D. Mass. 2009) ("Statistical evidence significance is one of the factors the Court should examine when determining whether a drug can cause an adverse event."); *see also* Daubert v. Merrell Dow Pharm., Inc., 509 U.S. 579, 594 (1993) ("[I]n the case of a particular scientific technique, the court ordinarily should consider the known or potential rate of error.")

rate is the *p*-value. Remembering the general statistics section above, the *p*-value is the probability that the result obtained is secondary to chance.[507] Although chance is not *per se* error, in clinical medical studies, it is the best approximation, and the cut-off or threshold, for determining what data is reliable.

As discussed above, there are numerous systematic reviews, controlled trials, and well-designed, prospective, and retrospective studies that demonstrate a highly significant statistical association of SDHs and RHs with AHT. For example, recent studies and systemic reviews have calculated the specificity and positive predictive value of severe RHs for abusive head injury to be on the order of 93-97% and 71-96%, respectively.[508] In fact, Vinchon et al. recently determined that the concurrence of these factors—SDH, RH, and the absence of evidence of impact to the head—was 100% specific for abusive injury.[509]

In order to truly appreciate the strength of this statistical evidence, we must, at this point, discuss the concept of "convergent validation."[510] Simply stated, "convergent validation" is the confirmation of a relationship of variables when that relationship is demonstrated by multiple independent measures.[511] The higher

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507 As stated in the general statistics section above, in social sciences and medicine, this "observed significance level" (the *p*-value) is usually set at 5% (or 0.05) for "statistically significant," or 1% (or 0.01) for "moderately high" statistical significance, and 0.1% (or 0.001) for "high or strong" statistical significance. *See supra* Section II(B)(1)(b).

508 *See* S. Maguire et al., *supra* note 364, at 860 (systematic review showing positive predictive value for RH of 71%); Vinchon et al., *supra* note 285, at 380 (recent study showing specificity of 93.2% for RH in AHT and 100% of severe RH in AHT); *see also* Vinchon et al., *supra* note 280, at 642 tbl.4 (recent study showing severe RH specificity of 0.974 and a positive predictive value of 0.961).

509 *See* Vinchon et al., *supra* note 280, at 637.

510 In the 1950s, two eminent psychologists, Campbell and Fiske, sought to provide validation for psychological assessment tools that assessed vague variables such as courteousness, honesty, self-centeredness, imaginativeness, talkativeness, etc. *See* Donald T. Campbell & Donald W. Fiske, *Convergent and Discriminant Validation by the Multitrait-Multimethod Matrix*, 56 PSYCHOL. BULL. 81, 98 tbl.13 (1959). In creating the multitrait-multimethod approach to assessing validity of psychological assessment tools, Campbell and Fiske determined that one of the key components was the concept of "*convergent validation.*" *Id.* at 81.

511 *Id.* at 81.

these independent measures correlate with each other, the greater the validity of the results.[512] With SDHs and RHs, the concept of convergent validation explains the increased statistical strength and validity of their results. Both injuries have been studied by multiple independent measures—general pediatrics studies, radiology studies, and pathology studies—and all independent measures have correlating results. Thus, the medical literature on AHT has also addressed *Daubert's* third criterion.

Finally, with regards to general acceptance within the relevant scientific community criterion, there are several issues that warrant further discussion. First, in the field of AHT, what constitutes the "relevant" scientific community? Is it general pediatricians? Pediatricians who specialize in child abuse and neglect? Pathologists? Ophthalmologists? Second, what constitutes "general acceptance" within that community? Is it a majority of members, or is unanimity or near unanimity required? Third, how is appropriate evidence of general acceptance adduced? Is the opinion testimony of one random member sufficient? Or is something more definitive required, such as opinion results of a majority of members or a policy statement promulgated by a medical society? Finally, what is the appropriate course of action when multiple disciplines are involved, as in AHT (general pediatrics, radiology, ophthalmology, neurosurgery, and occasionally pathology), and each are relevant scientific communities? Can a specialist from one discipline testify to scientific evidence from the other disciplines?

Although many courts, U.S. and international, have concluded that AHT is a generally accepted valid medical diagnosis[513] within

512 *Id.*

513 See People v. Martinez, 74 P.3d 316, 323 (Colo. 2003) ("[W]e assume, as it is not in dispute, that the scientific principles of shaken-impact syndrome and subdural hematomas resulting from extreme accidents are reasonably reliable"); State v. McClary, 541 A.2d 96, 102 (Conn. 1988) (shaken baby syndrome is generally accepted by medical science); State v. Torres, 121 P.3d 429, 437 (Kan. 2005) (testimony by physicians that infant's injuries were shaken baby syndrome, and not consistent with falling off a chair was sufficient for conviction of felony murder); State v. Leibhart, 662 N.W.2d 618 (Neb. 2003) (expert testimony on shaken baby syndrome admissible; passes *Daubert*); Order Denying Motion to Exclude Testimony on AHT/SBS at 5, State v. Mendoza, No. 071908696 (Utah Dist. Ct., June 5, 2009) ("[T]he State's experts made a very compelling ... showing that SBS is both still widely accepted and applicable to the current case"); *see also* R v. Harris, [2005] EWCA

the relevant scientific community, they have offered little guidance on what the relevant scientific community or general acceptance is and how those determinations came to be. With regards to AHT, the relevant scientific community should be those medical providers who, within their discipline, spend a reasonable portion if not majority, of their clinical time and practice in the evaluation and care of children suspected of AHT and abuse, who remain abreast of the most recent peer-reviewed literature on AHT and child abuse, and who either have obtained subspecialty certification, or are eligible for subspecialty certification, in the field of child abuse.[514] The satisfaction of these criteria will aid a court in assuring that the testimony provided is tethered to standards of medical practice, thereby satisfying *Kumho*.[515]

The clinical practice of evaluating and caring for children suspected of AHT and abuse is a crucial element in the determination of the relevant scientific community. There are medical subspecialists (general pediatricians, pathologists, radiologists, ophthalmologists, etc.), and even non-medical persons (biomechanical engineers), who are well versed and well read on the literature surrounding AHT. But, a mere reading knowledge of a particular topic cannot be considered relevant to the scientific community. Experiential knowledge is commensurate, if not superior, to didactic knowledge. As the U.K. High Court stated in a recent appeal of shaken baby syndrome cases:

> *The fact that an expert is in clinical practice at the time he makes his report is of significance.* Clinical practice affords experts the opportunity to maintain and develop their experience. . . . Clinicians learn from each case in which they are engaged. Each case makes them think and as their experience develops so does their understanding. Continuing experience gives them the opportunity to adjust previously held opinions, to alter their views. . . . Such clinical experience . . . may provide a far more reliable source of evidence than that provided by those who have ceased to practice their expertise in a continuing

(Crim) 1980, [267] (Eng.); R v. Henderson; R v. Butler; R v. Oyediran, [2010] EWCA (Crim) 1269, [7] (Eng.).

514 While other criteria, such as academic appointment, research, and publication, are desirable, they are not necessary to declare one as a part of the "relevant" scientific community.

515 *See* Kassirer & Cecil, *supra* note 54, at 1383 (discussing *Kumho*).

> clinical setting and have retired from such practice. Such experts are, usually, engaged only in reviewing the opinions of others. They have lost the opportunity, day by day, to learn and develop from continuing experience.[516]

Thus, those providers who, in their discipline, do not spend a reasonable portion of their practice in the evaluation and care of AHT and child abuse patients cannot be considered the relevant scientific community within the meaning of *Daubert* and *Kumho*.

Courts have historically relied upon opinion testimony to provide evidence of the general acceptance of AHT within the scientific community.[517] Since there is no medical or scientific literature assessing the opinions of physicians on the validity of AHT as a medical diagnosis, a concern with prior opinion testimony on general acceptance is that its foundation may have rested upon the *ipse dixit* of the expert. Consequently, as expert opinions on the general acceptance of AHT occasionally varied from location to location, and from time to time, so have some court decisions.[518]

Although there is no medical or scientific study assessing the opinions of physicians on the validity of AHT, there is still substantive evidence to that effect—the public promulgations of the relevant national and international medical societies. The very *raison d'etre* of national and international medical societies is to represent the professional interests of the individual members within those societies. As such, these national and international medical societies have inherent, formal processes for obtaining individual member input on relevant professional topics, considering that input and the relevant scientific literature, and then formulating policy statements, practice guidelines or other educational materials on those topics.

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516 R v. Henderson; R v. Butler; R v. Oyediran, [2010] EWCA (Crim) 1269, [208] (Eng.) (emphasis added).

517 *See, e.g.*, *Martinez*, 74 P.3d at 323; *McClary*, 541 A.2d at 102; State v. Edmunds, 746 N.W.2d 590, 593 (Wis. Ct. App. 2008); Order Determining Admissibility of Expert Testimony on AHT/SBS at 22-23, Commonwealth v. Davis, No. 04-CR-205 (Ky. Cir. Ct., Apr. 17, 2006); Order Denying Motion to Exclude Testimony on AHT/SBS at 6, State v. Mendoza, No. 071908696 (Utah Dist. Ct., June 5, 2009).

518 *Compare* Order Denying Motion to Exclude Testimony on AHT/SBS at 5-6, State v. Mendoza, No. 071908696 (Utah Dist. Ct., June 5, 2009) (accepting AHT testimony), *with Edmunds*, 746 N.W.2d at 594 (giving a new trial because scientific doubt surrounds AHT diagnoses).

While not representative of each and every member of that society, it is safe to conclude that the promulgations of the national and international medical societies are at least representative of the professional views of a majority of its members.

With that said, it is virtually unanimous among national and international medical societies that AHT is a valid medical diagnosis.[519] Amongst clinical practitioners, from pediatricians to radiologists, from the American Academy of Pediatrics to the World Health Organization, the validity of AHT as a medical diagnosis is unquestioned. Thus, the fourth *Daubert* criterion has also been addressed.

Although the four general considerations enunciated in *Daubert* are satisfied by the AHT literature, the trilogy makes clear that, overall, it is the *methodology* that is of paramount importance, not the conclusions generated or the criterion satisfied. Does the AHT expert have "good grounds"[520] for coming to his/her conclusions? Is there a logical nexus between his/her methodology and the opinions that are generated? Has the expert exercised the "same level of intellectual rigor"[521] that the expert would use outside the courtroom when working in his/her relevant discipline? Or is AHT just junk science that's not "even good enough to be wrong"[522] and thus inadmissible scientific testimony/evidence?

In assessing the methodology in AHT, it is important to remember that arriving at the diagnosis of AHT employs no different methodology than arriving at any other clinical diagnosis. At its core, clinical medical decision-making is grounded in the roots of the scientific method. Extensive study into physician cognition has revealed valuable insights into the clinical diagnostic process (the methodology sought to be evaluated by *Daubert*). Whereas it was once thought that physician clinical reasoning proceeded in a

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519 *See supra* Section II.B.c.1—"A Shifted Consensus?"—where fifteen national and international medical societies are listed as publicly supporting the validity of AHT as a medical diagnosis. As mentioned in that section, the only "relevant" disciplines with some discord are pathologists and biomechanical engineers.

520 Daubert v. Merrell Dow Pharm., Inc., 509 U.S. 579, 590 (1993).

521 Kumho Tire Co. v. Carmichael, 526 U.S. 137, 152 (1999).

522 Breyer, *supra* note 57, at 6.

discretely linear fashion known as Bayesian analysis,[523] recent research has demonstrated the diagnostic process is actually a non-linear, unstructured method of problem-solving that employs both inferential and deductive reasoning.[524]

The physician gathers information on a patient's symptoms and signs and generates hypotheses (also known as a differential diagnosis).[525] Through the attainment of additional clinical information (via various diagnostic tests), the physician goes through an inferential and deductive process of hypothesis refinement until a consistent "working diagnosis" is achieved.[526] Hypothesis refinement utilizes a variety of reasoning strategies—probabilistic, causal and deterministic—to discriminate among the existing diagnoses of the differential diagnosis.[527] While being

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523 *See* JEROME P. KASSIRER & RICHARD I. KOPELMAN, LEARNING CLINICAL REASONING 16 (1991) ("Bayesian analysis assembles a complete set of diagnostic hypotheses that can explain a given set for clinical findings. For each hypothesis, a set of relevant attributes is identified (historical findings, physical findings, complications, predisposing factors, laboratory results) that might help discriminate among the diagnoses. The prior probability of each diagnostic hypothesis is specified numerically, as is the probability that each attribute is found in each disease entity. Then, a calculation is make of the likelihood of each disease entity given the disease prevalence and the probability of each clinical attribute."). Although physician reasoning does not exclusively proceed in a Bayesian fashion, physicians do frequently rely on Bayesian reasoning (combining disease prevalence with their knowledge of frequency of signs and symptoms in a given disease) in the diagnostic process. *See* Henifin, et al., *supra* note 91, at 467.

524 *See* Jerome P. Kassirer & Frank A. Sonnenberg, *The Scientific Basis of Diagnosis*, *in* TEXTBOOK OF INTERNAL MEDICINE 14, 14–15 (William N. Kelley ed., J.B. Libbincott Co. 1989); KASSIRER & KOPELMAN, *supra* note 523, at 3.

525 *See* Kassirer & Sonnenberg, *supra* note 524, at 14; *see also* KASSIRER & KOPELMAN, *supra* note 523, at 16 (defining differential diagnosis).

526 *See* Kassirer & Sonnenberg, *supra* note 524, at 15; *see also* KASSIRER & KOPELMAN, *supra* note 523, at 11 ("Hypothesis refinement is an evolving, sequential process of data gathering and interpretation."). Rather than exclusively relying on statistical data on disease prevalence to generate diagnostic hypotheses, the physician also utilizes "heuristics" (or shortcuts/rules of thumb) to make the task of information gathering manageable and efficient. KASSIRER & KOPELMAN, *supra* note 523, at 4.

527 *See* Kassirer & Sonnenberg, *supra* note 524, at 15; *see also* KASSIRER & KOPELMAN, *supra* note 523, at 11. ("Hypothesis refinement is an evolving sequential process of data gathering and interpretation."). Probabilistic reasoning is Bayesian-type reasoning where prior probabilities of diseases are considered and combined with a physician's knowledge of the frequency of signs and symptoms in a given disease and the probabilities of specific test information. These assist the physician in a probabilistic assessment of the most likely

mindful of the pitfalls of heuristics, the physician ultimately proceeds to hypothesis confirmation when the laws of diagnostic adequacy, coherency, and parsimony are satisfied.[528]

Many courts have held that the "differential diagnosis" methodology is a reliable method of ascertaining medical causation.[529] Courts have stated that the "differential diagnosis is a well-recognized and widely-used technique in the medical community to identify and isolate causes of disease and death."[530] As long as the expert "at least considers alternative causes," then testimony based upon the "differential diagnosis" methodology is admissible.[531]

U.S. courts have previously assessed the methodology underlying AHT and deemed it valid.[532] In more recent cases, U.S. courts have reassessed its sufficiency, and have still deemed it

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hypothesis. Causal reasoning "is a function of the anatomical, physiological and biochemical mechanisms that operate normally in the human body and the pathophysiologic behavior of these mechanisms in disease." *See* KASSIRER & KOPELMAN, *supra* note 523, at 28. Physicians "are accustomed to use *any* reliable data to assess causality, no matter what their source.... Temporal proximity can be a potent factor in causal decision making ...." Kassirer & Cecil, *supra* note 54, at 1384.

528 "Adequacy occurs when a "diagnostic hypothesis ... encompasses all surviving hypotheses and ... accounts for all the patient's findings, whether abnormal or normal." KASSIRER & KOPELMAN, *supra* note 523, at 32. Coherency occurs "when a patient's findings are consistent with the altered pathophysiology of the hypothesized disease state." *Id.* Parsimony is "the simplest possible explanation all of the [patient's] findings." *Id.*

529 *See* Best v. Lowe's Home Ctrs. Inc., 563 F.3d 171, 179, 183–84 (6th Cir. 2009) (stating a differential diagnosis can be adequate grounds for a causation opinion under *Daubert*); Hyman & Armstrong, P.S.C. v. Gunderson, 279 S.W.3d 93, 107, 109 (Ky. 2008); Westberry v. Gislaved Gummi AB, 178 F.3d 257, 263 (4th Cir. 1999). *But see*, Moore v. Ashland Chem. Inc., 151 F.3d 269, 279 (5th Cir. 1998) (denying admissibility of expert testimony based upon the differential diagnosis); *Moore* 151 F.3d at 288 (dissent).

530 *See Gunderson*, 279 S.W.3d at 107 (citing Globetti v. Sandoz Pharms. Corp., 111 F.Supp.2d 1174 (N.D. Ala. 2000)).

531 *In re* Paoli R.R. Yard PCB Litig., 35 F.3d 717, 759 (3d Cir. 1994) (noting there is "a requirement that experts at least consider alternative causes" and that this concept is "at the core of differential diagnosis."); *see* Heller v. Shaw Industries, Inc., 167 F.3d 146, 156 (3d Cir. 1999) (stating that before allowing differential diagnosis reasoning as grounds for causation, a medical expert must rule out "obvious alternative causes," but not, "categorically, all other possible causes" of an injury).

532 *See* State v. McClary, 541 A.2d 96, 102 (1988) (noting shaken baby syndrome is generally accepted by medical science).

valid.[533] But, the assessment of the validity of the methodology underlying AHT is not peculiar to U.S. courts.

In the United Kingdom, AHT has been a topic of significant medico-legal concern recently. The U.K. High Court recently heard four appeals on alleged "battered babies" cases.[534] In *R v. Harris* (a consolidation of the four appeals) the U.K. High Court examined the issue of whether newly-developed "medical research . . . [had created] 'fresh evidence' which . . . [cast] doubt on the safety of each conviction."[535] The High Court stated:

> At the heart of these appeals . . . was a challenge to the accepted hypothesis concerning "shaken baby syndrome" (SBS); or, as we believe it should be more properly called, non-accidental head injury (NAHI). The accepted hypothesis depends on findings of a triad of intracranial injuries consisting of encephalopathy (defined as disease of the brain affecting the brain's function); subdural haemorrhages (SDH); and retinal haemorrhages (RH).[536]

In evaluating the sufficiency of the "triad," the High Court received testimony from over twenty international experts in the field of AHT—"ten medical expert witnesses called on behalf of the appellants and eleven called on behalf the Crown . . . [and] written evidence of four further witnesses."[537] As a part of its examination of the "newly-developed research," the High Court studied Dr. Geddes' Unified Hypothesis:

> Between 2000 and 2004 a team of distinguished doctors led by Dr Jennian Geddes, a neuropathologist with a speciality in work with children, produced three papers setting out the results of their research into the triad. In the third paper "Geddes III", the team put forward a new hypothesis, "the unified hypothesis," which challenged the supposed infallibility of the triad. . . .

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533 *See* United States v. Vallo, 238 F.3d 1242, 1245 (10th Cir. 2001); People v. Dunaway, 88 P.3d 619, 633–34 (Colo. 2004); People v. Martinez, 74 P.3d 316, 323, 324–25 (Colo. 2003); State v. Leibhart, 662 N.W.2d 618, 627–28 (Neb. 2003); State v. Glenn, 900 So.2d 26, 34–35 (La. Ct. App. 2005); Order Denying Motion to Exclude Testimony on AHT/SBS at 5–6, State v. Mendoza, No. 071908696 (Utah Dist. Ct., June 5, 2009).

534 R v. Harris, [2005] EWCA (Crim) 1980, [4]–[5].

535 *Id.* at [3].

536 *Id.* at [56].

537 *Id.* at [5].

> When Geddes III was published it was, and still is, very controversial.... However, *early on in the hearing it became apparent that substantial parts of the basis of the unified hypothesis could no longer stand.* Dr Geddes, at the beginning of her cross-examination, accepted that the unified hypothesis was never advanced with a view to being proved in court.... Further, she accepted that the hypothesis might not be quite correct; or as she put it: "I think we might not have the theory quite right. I think possibly the emphasis on hypoxia—no, I think possibly we are looking more at raised pressure being the critical event."[538]

In concluding that Geddes' Unified Hypothesis could no longer be considered credible, the High Court stated:

> As a result of critical papers published in the medical journals, as we have already stated, Dr Geddes when cross-examined frankly admitted that the unified hypothesis could no longer credibly be put forward. In cross-examination she accepted that she could no longer support the hypothesis that brain swelling was the cause of subdural haemorrhages and retinal haemorrhages. She did, however, state that she believed that raised intracranial pressure (ICP) might prove to be an independent cause of both lesions. When asked by Mr Horwell if she had published a paper on this hypothesis she said that she had not and that her research was still incomplete.... "In our judgment, it follows that the unified hypothesis can no longer be regarded as a credible or alternative cause of the triad of injuries....
>
> ... These four appeals raise different medical issues and do not necessarily fail because the unified hypothesis has not been validated. But it does mean that the triad, itself a hypothesis, has not been undermined in the way envisaged by the authors of Geddes III.[539]

The High Court then conducted "sufficiency of evidence" reviews on the four cases.[540] Based upon an appellate standard of review of "whether the evidence, if given at the trial, might reasonably have affected the decision of the trial jury,[541]" the High Court determined that, in two cases, the "fresh" evidence "might reasonably have affected the jury's decision to convict"[542] and set aside those

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538 *Id.* at [57]-[58] (emphasis added).

539 *Id.* at [68]-[69] (emphasis added).

540 *See id.* at [102]-[103].

541 *Id.* at [101].

542 *Id.* at [153].

convictions.[543] In the two other cases, the High Court sustained or modified the convictions.[544]

B. Other Legal Challenges to AHT

Although a comprehensive examination of all the challenges surrounding AHT testimony and evidence is beyond the scope of this article, a couple of more recent challenges shall be addressed briefly.[545] One, akin to Geddes' Unified Hypothesis, is an assertion of an alternative explanation for the injuries seen in AHT. It is the abovementioned "dural immature vascular plexus" theory by Squier and Mack.[546] This theory is but another example of a more general, overarching challenge to the medical evidence base underlying AHT. By proffering another valid scientific explanation for the injuries in AHT, the contention is that there will then be doubt regarding the "non-accidental," "abusive," and "traumatic" nature of the injuries.

In the dural immature vascular plexus theory, the authors hypothesize that there is a plexus (network) of vessels within the dura mater that is immature and a likely source for "hemorrhage in non-traumatic conditions."[547] Secondary to the immaturity of these vessels, in situations of hypoxia, these vessels "leak," and subsequently result in SDHs.[548] Akin to Geddes' Unified Hypothesis, hypoxic-ischemic injury is the preeminent factor

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[543] *Id.* at [153], [266].

[544] *Id.* at [185], [219].

[545] Other challenges to admissibility of AHT testimony have included 403 challenges (that a medical diagnosis of child abuse is confusing to a jury in relation to the legal definition of child abuse, within a particular state, and consequently, the prejudicial value outweighs the probative value) and challenges to the admissibility of testimony on the amount of force required to cause the injuries. *See* People v. Martinez, 74 P.3d 316, 321-22 (Colo. 2003). For a comprehensive review of the evidentiary challenges in AHT testimony, see John E.B. Myers, MYERS ON EVIDENCE IN CHILD, DOMESTIC, AND ELDER ABUSE CASES (Aspen Publishers, vol. 1 2005) and John E. B. Myers, MYERS ON EVIDENCE IN CHILD, DOMESTIC AND ELDER ABUSE CASES (Aspen Publishers, supp. 2007).

[546] *See supra* Section II(B)(2)("Statistical Evidence").

[547] Mack et al., *supra* note 396, at 208.

[548] Squier & Mack, *supra* note 396, at 10.

leading to "hemorrhage in non-traumatic conditions."[549]

This most recent alternative hypothesis for the causation of SDHs and RHs does not survive Trilogy scrutiny. Unlike even Geddes' Unifed Hypothesis, this theory offers *no scientific data* linking an intradural (within the dura) vascular plexus to the significant subdural hemorrhages seen in AHT.[550] Although published as a review article in a peer-reviewed medical journal, it has not been the subject of *any* scientific study, in *any* cohort of patients. Consequently, it has not been tested by the scientific rigors of falsifiability, and has adduced no evidence-based medical literature. Furthermore, by adhering to Geddes' medically and legally discredited theory of hypoxic-ischemic injury as the "unifying" cause for SDHs and RHs, this theory remains outside mainstream medical opinion. Thus, any scientific testimony based upon this theory would be based solely upon the *ipse dixit* of the expert, and inadmissible under *Joiner* and *Kumho*.

Because the theory attempts to perpetuate Geddes' discredited Unified Hypothesis, two recent United Kingdom court opinions have questioned the scientific objectivity of one of its authors, Dr. Squier. In a U.K. family court opinion, the court stated:

> Both Dr. Cohen and Dr. Squier subscribe to the Geddes III hypothesis in one form or another. Put at its simplest, each are of the view that hypoxia in children can lead to subdural haemorrhages and retinal haemorrhages in the absence of trauma.
>
> . . . .
>
> . . . *They go against the mainstream of current thinking* and the analysis of the Court of Appeal in *R v. Harris*. . . .
>
> . . . .
>
> *Dr. Cohen and Dr. Squier support Geddes III, even though Dr. Geddes herself in Harris withdrew from her own unified hypothesis*. . . .
>
> In considering the evidence of Dr. Cohen and Dr Squier, I remind myself that four years have passed since Dr. Geddes accepted that her unified hypothesis could no longer credibly be put forward. . . .

549 *See id.*; Mack et al., *supra* at 396, at 208.

550 *See supra* Part (d)(ii) Alternative Hypotheses.

> I have to consider whether or not these experts have "developed a scientific prejudice" or whether they are in the vanguard of research and learning.[551]

The court then concluded:

> I do not doubt the commitment of Dr. Squier and Dr. Cohen to the advancement of the understanding of Shaken Baby Syndrome. As already indicated, I make no criticism and, indeed, it would be wrong to do so, of the fact that neither of them hold mainstream views. There is a significant fundamental difference between academic theories and hypotheses, on the one hand, and the rigorous forensic analysis which is required in care proceedings . . . .
>
> *Dr. Squier and Dr. Cohen, I find with regret, have each fallen into that category of expert identified by Butler-Sloss P. in Re LU & LB, namely the expert who has developed a scientific prejudice. As a consequence, I accept the submission of the Local Authority that Dr. Squier has permitted her convictions to lead her analysis. . . .* [E]ach of the significant factual errors made by her served to support her hypothesis of choking and hypoxia.
>
> *The overwhelming preponderance of evidence in this case is to the effect that, as of today, medical opinion is that hypoxia does not lead to subdural haemorrhages and retinal haemorrhages* . . . .[552]

When Dr. Squier provided testimony in a recent criminal appellate matter, the U.K. High Court stated:

> Dr Squier's stance, in oral evidence before us, casts significant doubt upon the reliability of the rest of her evidence and her approach to this case. It demonstrates, to our satisfaction, that she was prepared to maintain an unsubstantiated and insupportable theory in an attempt to bolster this appeal.
>
> . . . .
>
> In the light of our view as to the quality of Dr Squier's evidence before us we conclude it is not capable of undermining the safety of the verdict. For those reasons, we reject the application to call fresh evidence.[553]

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551 A Local Auth. v. S, [2009] EWHC (Fam) 2115 [63], [199], [201]-[203] (Eng.) (emphasis added).

552 *Id.* at [284]-[286] (emphasis added) (heading omitted).

553 R v. Henderson; R v. Butler; R v. Oyediran, [2010] EWCA (Crim) 1269 [188], [190] (Eng.)

The other recent challenge to the admissibility of AHT testimony asserts that a physician's diagnosis of "abusive" or "inflicted" injury is an "improper comment on the *mens rea*" element of an offense and consequently, an improper "invasion of the province of the jury."[554] In medicine, physicians routinely diagnose intentional acts of patients that result in medical problems. For example, in eating disorders such as bulemia (binge and purge type) and anorexia nervosa, the patient's intentional acts of either purging food recently eaten (bulemia) or not eating food (anorexia) so as to not gain weight are key diagnostic features of those disorders. Many other medical diagnoses—self-cutting behavior, trichotillomania (hair pulling), and illicit substance abuse, to name a few—exist where primary care physicians, in the routine course of clinical medical practice, diagnose intentional acts of patients as key components of medical disease. Additionally, pathologists (specifically forensic pathologists and medical examiners) are routinely called upon to determine intent in the manner and cause of death. And, psychiatrists are sometimes requested to determine an individual's capacity to satisfy the *mens rea* elements of criminal offenses. The practice of child abuse pediatrics is no different than these other practices of medicine.

Courts have long held that, as long as a physician does not testify to the ultimate question of the defendant's guilt or innocence, a physician may opine that injuries are "nonaccidental," "inflicted," or "abusive."[555] In *Estelle v. Maguire* the U.S. Supreme Court recognized the admissibility of medical testimony on the issue of intent when it considered the admissibility of 404(b) evidence in

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(emphasis added).

554 State v. Smallwood, 955 P.2d 1209, 1220-21 (Kan. 1998).

555 *See* State v. Smith, 877 So. 2d 1123, 1127-29 (La. Ct. App. 2004) (fatal shaking and impact case; doctor testified child's injuries were abusive); State v. Smallwood, 955 P.2d 1209, 1221 (1998) (infant died of inflicted head injury; pathologist opined the child died of abuse: "by stating that, based upon her medical experience, Kaine died as a result of child abuse, either shaking or a blow to the skull, Dr. Gould was not testifying as to the ultimate question of Smallwood's guilt or innocence. Expert testimony in the form of an opinion is not objectionable because it embraces the ultimate issue or issues to be decided by the trier of fact.").

order to prove "battered child syndrome."[556] The Supreme Court wrote:

> The demonstration of battered child syndrome "simply indicates that a child found with [serious, repeated injuries] has not suffered those injuries by accidental means." Thus, evidence demonstrating battered child syndrome helps to prove that the child died at the hands of another and not by falling off a couch for example, it also tends to establish that the "other," whoever it may be, inflicted the injuries intentionally.[557]

As with battered child syndrome, the non-accidental or abusive determination in AHT finds its diagnostic underpinning in "the degree and type of injury [that] is at variance with the history given regarding the occurrence of the trauma."[558] Recently, in *State v. Torres*, the Supreme Court of Kansas concluded that a physician's opinion that an infant's death was a "textbook case" of "shaken baby or shaken impact syndrome" did not invade the province of a jury so long as the expert did not testify as to "the ultimate question of the defendant's guilt or innocence."[559] Thus, these most recent challenges to the admissibility of AHT testimony lack legal and medical foundation.

C. Beyond Daubert: The Marriage of Medical and Legal Perspectives

Given the abundance of medical literature in support of AHT—the significant statistical strength of much of that literature, the recognition by many U.S. and U.K. courts of the validity of that literature and of the diagnosis of AHT—one must seek explanation for the variability in some court decisions. Why have some courts concluded that there is a "significant and legitimate debate in the medical community" on AHT,[560] while others have not?[561] Why

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556 Estelle v. McGuire, 502 U.S. 62, 68 (1991).

557 *Id.* (citation omitted).

558 *See* Kempe et al., *supra* note 148, at 143; Tuerkheimer, *supra* note 4, at 31.

559 *See* State v. Torres, 121 P.3d 429, 446–47 (Kan. 2005).

560 State v. Edmunds, 746 N.W.2d 590, 596 (Wis. Ct. App. 2008).

561 *See* R v. Henderson; R v. Butler; R v. Oyediran, EWCA (Crim) 1269 at [188]–[190]; Order

have some concluded that the diagnosis of AHT is "based on inconclusive research,"[562] while the vast majority have not?[563] Several reasons exist.

First, as mentioned above, the adduction of evidence on what is general acceptance within the relevant scientific community has in many cases, unfortunately, been upon the *ipse dixit* of the expert. In *State v. Edmunds*, the Court determined, based upon "expert medical testimony," that "a significant and legitimate debate in the medical community has developed in the past ten years" on AHT.[564] However, those "experts" provided no substantive medical literature affirming that "significant and legitimate debate."[565] Highlighting the shortcomings of such evidence, one expert witness in a U.K. AHT case stated:

> Al-Sarraj told the court that there are 40–44 neuropathologists in the country of whom a maximum of 10 or 12 are forensic neuropathologists. To his knowledge, the only neuropathologist in the UK believing that hypoxia can cause subdural haemorrhages is Dr. Waney Squier. In addition, he said there are two or three other people who share her opinion who are working in different, but related, specialities, of whom Dr. Cohen and Dr. Scheimberg (Dr. Cohen's co-author) are presumably two. Dr. Al-Sarraj said:
>
> > *"They come in all the defence cases, so you do not realise that they are in such a minority."*[566]

Second, the pecuniary interest in providing expert testimony cannot be underestimated. It has posed and continues to pose a significant risk to the presentation of unbiased medical information. Third, in addition to pecuniary interest, as discussed above, personal prejudices can also affect scientific analysis. This can result

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Denying Motion to Exclude Testimony on AHT/SBS at 1–4, State v. Mendoza, No. 071908696 (Utah Dist. Ct., June 5, 2009).

562 Order Determining Admissibility of Expert Testimony on AHT/SBS at 22, Commonwealth v. Davis, No. 04-CR-205 (Ky. Cir. Ct., Apr. 17, 2006).

563 *See, e.g.*, State v. Leibhart 662 N.W.2d 618, 627–28 (Neb. 2003); Order Denying Motion to Exclude Testimony on AHT/SBS at 5–6, State v. Mendoza, No. 071908696 (Utah Dist. Ct., June 5, 2009).

564 *See Edmunds*, 746 N.W.2d at 596.

565 *See id.*

566 A Local Auth. v. S, [2009] EWHC (Fam) 2115 [199] (Eng.) (emphasis added).

in the adherence to disproven theories and the presentation of skewed information. Finally, the increasing complexity of scientific and medical information has placed onerous burdens on the single, gate-keeping trial judge. Given the lack of dispositive medical guidance from a unified, unbiased, multi-disciplinary, medical body, courts have been left to fend for themselves, relying upon whatever seemingly reliable medical information is presented. Naturally, variability in some decisions has ensued.

If the marriage of the legal and medical perspectives is to survive, especially with regards to AHT, then the medical and legal fields must remain faithful to their obligations, and seek to strengthen their union. Courts must remember Justice Breyer's admonition— "seek decisions that fall within the boundaries of scientifically sound knowledge"[567] and keep out science that "isn't even good enough to be wrong."[568] This article has provided evidence-based medical literature supporting the scientific soundness of AHT and the lack of such evidence for theories such as Geddes' Unified Hypothesis and Squier and Mack's dural immature vascular plexus theory. Concurrent with that obligation, courts must recognize when there is a *legitimate and responsible* disagreement among medical experts, and allow the jury to resolve that dispute among the experts. Finally, when confronted with the complexities of medical and scientific information, courts should seek assistance from impartial court-appointed scientific experts to explain the medical and scientific information.

For medicine's part, the national medical societies of the relevant disciplines should coordinate with Federal Judicial Center (FJC) and National Academy of Sciences, Committee on Science, Technology and Law, to establish a registry of potential independent medical experts on AHT. Along those lines, the relevant national medical societies should promulgate policies limiting expert medical testimony fees, and support state and federal legislation towards that effect. Finally, the judiciary, via the FJC, and the relevant medical disciplines, specifically child abuse

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567 Breyer, *supra* note 57, at 4.

568 *Id.* at 6.

pediatricians, should engage in reciprocal educational efforts on the responsibilities and limitations of expert testimony in AHT.

IV. CONCLUSION

What has been presented for the reader is:

i) a brief examination of the extensive clinical medical literature on the topic of AHT;

ii) evidence-based clinical medical studies on SDHs and RHs that demonstrate highly significant statistical associations of those injuries with AHT;

iii) verifiable references to fifteen national and international medical societies who have publicly endorsed the validity of AHT;

iv) medical and legal rationales refuting alternative hypotheses (such as Geddes' Unified Hypothesis and Squier and Mack's Dural Immature Vascular Plexus Theory) for the injuries common to AHT; and

v) national and international case law examining, and ultimately confirming, the validity of the medical evidence in support of AHT.

These reasons, and years of clinical experience, are the foundation for the opinions given by the vast majority of medical professionals called to evaluate suspected AHT. The diagnosis of AHT, long recognized as a valid diagnosis, occurs within the same professional culture of science and practice (methodology) that leads to the diagnosis and treatment of millions of pediatric patients in the U.S. every year. Many of these diagnoses are matters of life and death, and sometimes these diagnoses lead to the courtroom. For the legal profession to treat this aspect of pediatric medicine as separate from the rest of medicine is unjustifiable. It is understandable that lawyers will look for opportunities to create doubt in the minds of jurors. However, the only way to appropriately improve the chances for justice in the courts with respect to AHT is to assure that an unbiased, financially-unmotivated, medical expert testifies to the current state of medical evidence.

APPENDIX A

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2) S.A. Schutzman et al., *Evaluation and Management of Children Younger Than Two Years Old with Apparently Minor Head Trauma: Proposed Guidelines*, 107 PEDIATRICS 983 (2001).

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1) Christine T. Chiaviello et al., *Stairway-Related Injuries in Children*, 94 PEDIATRICS 679 (1994).
2) Christine T. Chiaviello et al., *Infant Walker-Related Injuries: A Prospective Study of Severity and Incidence*, 93 PEDIATRICS 974 (1994).
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4) David S. Greenes & Sara A. Schutzman, *Clinical Indicators of Intracranial Injury in Head-Injured Infants*, 104 PEDIATRICS 861 (1999).
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7) Kimberly S. Quayle et al., *Diagnostic Testing for Acute Head Injury in Children: When are Head Computed Tomography and Skull Radiographs Indicated?*, 99 PEDIATRICS e.11 (1997).

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4) David S. Greenes & Sara A. Schutzman, *Occult Intracranial Injury in Infants*, 32 ANNALS EMERGENCY MED., 680 (1998).
5) Karen D. Gruskin, Sara A. Schutzman, *Head Trauma in Children Younger Than 2 Years: Are There Predictors for Complications?*, 153 ARCHIVES PEDIATRIC & ADOLESCENT MED. 15 (1999).
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7) Raye Helfer et al., *Injuries Resulting When Small Children Fall Out of Bed*, 60 PEDIATRICS 533 (1977).
8) Maija Holsti et al., *Pediatric Closed Head Injuries Treated in an Observation Unit*, 21 PEDIATRIC EMERGENCY CARE 639 (2005).
9) Anthony Kim et al., *Analysis of Pediatric Head Injury from Falls*, 8 NEUROSURGICAL FOCUS e3 (2000).
10) Harvey Kravitz et al., *Accidental Falls from Elevated Surfaces in Infants from Birth to One Year of Age*, 44 PEDIATRICS 869 (1969).
11) S. Levene & G. Bonfield, *Accidents on Hospital Wards*, 66 ARCHIVES DISEASE CHILDHOOD 1047 (1991).
12) Thomas Lyons & Kim Oates, *Falling out of Bed: A Relatively Benign Occurrence*, 92 PEDIATRICS 125 (1993).
13) Diane Macgregor, *Injuries Associated with Falls from Beds*, 6 INJURY PREVENTION 291 (2000).
14) L. Morrison et al., *Infant-furniture Related Injuries among Preschool Children in New Zealand, 1987–1996*, 38 J. PAEDIATRICS & CHILD HEALTH 587 (2002).

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22) Simon P. Ros & Frank Cetta, *Are Skull Radiographs Useful in the Evaluation of Asymptomatic Infants Following Minor Head Injury?*, 8 PEDIATRIC EMERGENCY CARE 328 (1992).
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2) Gina E. Bertocci et al., *Influence of Fall Height and Impact Surface on Biomechanics of Feet-First Free Falls in Children*, 35 INJURY 417 (2004).
3) D. Chadwick, *The Potential and Limitations of Utilising Head Impact Injury Models to Assess the Likelihood of Significant Head Injury in Infants After a Fall*, 139 FORENSIC SCI. INT'L 269 (2004).
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7) C. Z. Cory & M. D. Jones, *Development of a Simulation System for Performing In Situ Surface Tests to Assess the Potential Severity of Head Impacts from Alleged Short Falls*, 163 FORENSIC SCI. INT'L 102 (2006).
8) C. Z. Cory & B. M. Jones, *Can Shaking Alone Cause Fatal Brain Injury? A Biomechanical Assessment of the Duhaime Shaken Baby Syndrome Model*, 43 MED., SCI. & LAW 317 (2003).
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28) Brielle M. Paolini et al., *Pediatric Head Injury Prediction: Investigating the Distance Between the Skull and the Brain Using Medical Imaging*, 45 BIOMEDICAL SCI. & INSTRUMENTATION 161 (2009).
29) Michael T. Prange et al., *Anthropomorphic Simulations of Falls, Shakes, and Inflicted Impacts on Infants*, 99 J. NEUROSURGERY 143 (2003).
30) Michael T. Prange & Susan S. Margulies, *Regional, Directional, and Age-Dependent Properties of the Brain Undergoing Large Deformation*, 124 J. BIOMECHANICAL ENGINEERING 244 (2002).
31) Michael T. Prange et al., *Mechanical Properties and Anthropometry of the Human Infant Head*, 48 STAPP. CAR CRASH J. 279 (2004).
32) Ramesh Raghupathi et al., *Traumatic Axonal Injury is Exacerbated following Repetitive Closed Head Injury in the Neonatal Pig*, 21 J. NEUROTRAUMA 307 (2004).
33) Ramesh Raghupathi & Susan S. Margulies, *Traumatic Axonal Injury after Closed Head Injury in the Neonatal Pig*, 19 J. NEUROTRAUMA 843 (2002).
34) Nagarajan Rangarajan et al., *Finite Element Model of Ocular Injury in Abusive Head Trauma*, 13 J. AM. ASS'N PEDIATRIC OPHTHALMOLOGY & STRABISMUS 364 (2009).
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36) Betty S. Spivack, *Chapter Five: Biomechanics of Abusive Head Trauma*, 5 J. AGGRESSION, MALTREATMENT & TRAUMA 55 (2001).

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4) Mark S. Dias et al., *Preventing Abusive Head Trauma Among Infants and Young Children: A Hospital-Based, Parent Education Program*, 115 PEDIATRICS e470 (2005).
5) Heather T. Keenan et al., *Neurodevelopmental Consequences of Early Traumatic Brain Injury in 3-Year-Old Children*, 119 PEDIATRICS e616 (2007).
6) John Pollina, Jr. et al., *Cranial Birth Injuries in Term Newborn Infants*, 35 PEDIATRIC NEUROSURGERY 113 (2001).
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Comparative Cohorts/Prospective Case Series:

1) Robin L. Altman et al., *Abusive Head Injury as a Cause of Apparent Life-Threatening Events in Infancy*, 157 ARCHIVES PEDIATRICS & ADOLESCENT MED. 1011 (2003).
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4) Kirsten Bechtel et al., *Relationship of Serum S100B Levels and Intracranial Injury in Children with Closed Head Trauma*, 124 PEDIATRICS e697 (2009).
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APPENDIX B

DIFFERENTIAL DIAGNOSIS OF SUBDURAL HEMORRHAGES:

Trauma
- Inflicted/Abusive
- Accidental
- Birth

Metabolic Diseases
- Glutaric Aciduria Type 1
- Menke's Disease
- Hemophagocytic Lymphohistiocytosis
- Nutritional deficiencies

Genetic Syndromes
- Osteogenesis Imperfecta
- Ehlers-Danlos Syndrome Type II
- Hereditary Hemorrhagic Telangectasia

Coagulopathies (Clotting Disorders)
- Hemophilia
- Hemorrhagic Disease of the Newborn

Tumors
- Lymphoblastic Leukemia
- Neuroblastoma

Infections
- HSV meningoencephalitis
- Bacterial meningitis

APPENDIX C

DIFFERENTIAL DIAGNOSIS OF RETINAL HEMORRHAGES:

Trauma
- Inflicted/Abusive
- Accidental
- Birth

Metabolic Diseases
- Glutaric Aciduria Type 1
- Hemophagocytic Lymphohistiocytosis
- Nutritional deficiencies

Genetic Syndromes
- Osteogenesis Imperfecta
- Ehlers-Danlos Syndrome Type II

Anemia

Coagulopathies (Clotting Disorders)
- Hemophilia
- Hemorrhagic Disease of the Newborn

Carbon Monoxide Poisoning

Vasculitis

Hypoxia/Hypo or Hypertension

Papilledema/Increased Intracranial Pressure

Tumors
- Lymphoblastic Leukemia
- Cerebral Aneurysm
- Hemangioma

Infections
- HSV meningoencephalitis
- Bacterial meningitis

FIGURES

Fig. 1. Image of Auguste Ambroise Tardieu (1818-1879). PD-1923. Image originally from Goupil et Cie. http://www.biusante.parisdescartes.fr/histmed/image?CIPC0155, *available at* http://en.wikipedia.org/wiki/File:AugusteAmbroiseTardieu.jpg.

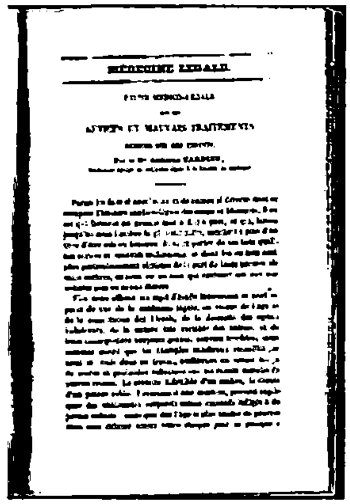


Fig. 2. First page of Ambroise Tardieu's *Etude medico- legale sur les sevices et mauvais traitements exerces sur des enfants* (*Forensic study on cruelty and ill treatment of children*), 1860. Reprinted from Ambroise Tardieu, Etude Medico-Legale sur les Sevices et MauvaisTraitements Exerces sur des Enfants, 13 ANNALES D'HYGIENE PUBLIQUE ET DE MEDECINE LEGALE 361-98 (1860))

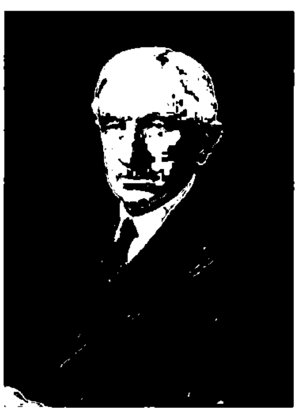
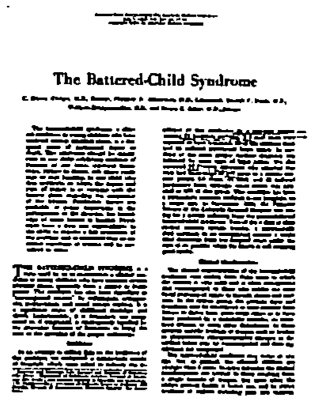


Fig. 3. Image of Wilfred Batten Lewis Trotter (1872-1939). Reproduced with permission © Godfrey Argent Studio.

Fig. 4. Dr. C. Henry Kempe.
Reprinted with permission of The Kempe Foundation for the Prevention and Treatment of Child Abuse and Neglect.



The Battered-Child Syndrome

Fig 5. First page of The Battered-Child Syndrome. JAMA Vol.181 July 7, 1962, pp.17-24. Copyright © 1962 American Medical Association. All rights reserved. Reprinted with permission from JAMA.

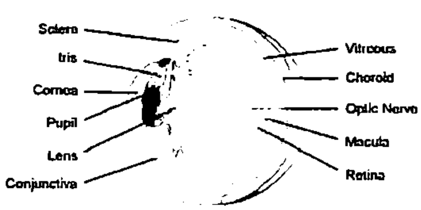


Fig. 6. Human Eye
Reprinted courtesy of http://ihsanatomy4.wikispaces.com

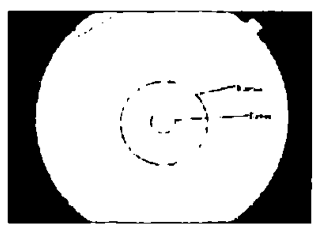


Fig. 7. Normal Retina, demonstrating the area of the retina called the posterior pole: fovea and macula (within circles), optic nerve (bright whitish appearing circle on left-hand side) and its head manifesting as a circular disc (optic disc), and retinal vessels emanating from the optic nerve. Reprinted from *Eye Disease Anatomy*, Ref#: EDA06, NAT'L EYE INST., http://www.nei.nih.gov/phot/eyedis/index.asp (circles added by author).

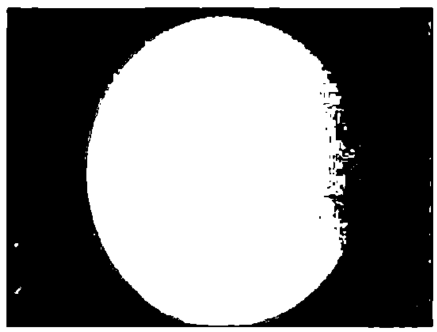


Fig. 8. Mild nonspecific retinal hemorrhages confined to the posterior pole. (Courtesy of Alex V. Levin, MD. MHSc, Wills Eye Institute, Philadelphia)

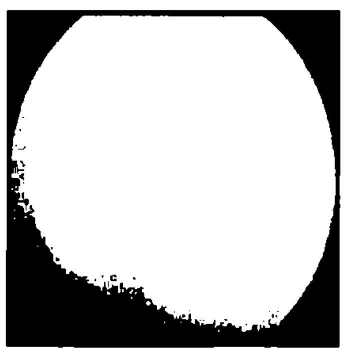


Fig. 9. Severe retinal hemorrhages, too numerous to count, such that there is virtually no visible normal retina. (Courtesy of Alex V. Levin, MD. MHSc, Wills Eye Institute, Philadelphia)

Fig. 10. Macular traumatic retinoschisis. (Courtesy of Alex V. Levin, MD. MHSc, Wills Eye Institute, Philadelphia)

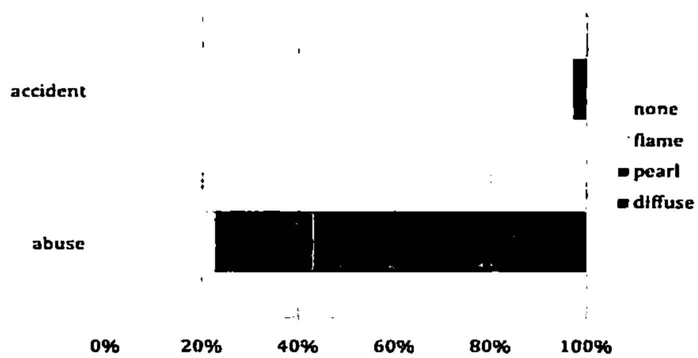


Fig. 11. "Retinal hemorrhage in the AT and IHI groups. Although most cases of abuse were associated with severe hemorrhage, seven had no hemorrhage, and three had only mild hemorrhages." Matthieu Vinchon et al., *Confessed Abuse Versus Witnessed Accidents in Infants: Comparison of Clinical, Radiological, and Ophthalmological Data in Corroborated Cases*, 26 Child's Nervous Sys. 637, 641 fig.3 (2009). Conversely, no or mild RHs were found in 34 cases of AT, *id.* at 639, 641 fig.3, and only "one had severe hemorrhage caused by direct facial impact." *Id.* at 641 fig.3. (Figure reprinted with permission of publisher.)

PLEASE STAY TUNED FOR AN ACADEMIC RESPONSE TO *A* DAUBERT *ANALYSIS OF ABUSIVE HEAD TRAUMA/SHAKEN BABY SYNDROME* IN VOLUME 12 OF THIS JOURNAL.

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Letter to the Editor

Shaken baby syndrome: A flawed biomechanical analysis

Keywords: Injury; Infant; Shaken; Baby; Rotational; Acceleration/deceleration; Syndrome; Neck

To the Editor,

We are gravely concerned that the conclusions reached by Bandak [1] may be invalid due to apparent numerical errors in his estimation of forces experienced in an infant neck during vigorous shaking. More specifically, we have repeated the author's calculations and we find values of neck forces that are actually more than 10 times *lower* than those presented in Bandak's Table 3.

Using the free body diagram of the infant head and neck (Fig. 3), Bandak identified the two components of neck force during rotation of the head—the tangential force F_t and the normal force F_n. Bandak described the basic equations for neck forces during a simplified shaking event, but did not present detailed methods for calculating the upper neck loads. We define them here for completeness:

$$F_t = m_{head}a_t = m_{head}r\frac{d^2\theta}{dt^2} \tag{1}$$

$$F_n = m_{head}\frac{v^2}{r} = m_{head}r\left(\frac{d\theta}{dt}\right)^2 \tag{2}$$

where r is the length of the neck in meters, m_{head} the mass of the head in kilograms, a_t the tangential linear head acceleration in meters per second squared, $d^2\theta/dt^2$ the angular acceleration of the head in radians per second squared, v the linear velocity of the head in meters per second, and $d\theta/dt$ the angular velocity of the head in radians per second.[1] As Bandak pointed out, when F_n reaches its maximum value, F_t is at a minimum, so it would be incorrect to sum or otherwise combine peak F_n and F_t to estimate peak neck forces. Yet, using the same angular acceleration and velocity values Bandak reported from the literature, we calculate forces 10 times lower than those presented in Bandak's Table 3.

For example, to calculate neck forces for the most severe shaking event reported in Bandak's Table 3, we used the largest angular acceleration and angular velocity values, the longest neck length and the heaviest head mass provided in Table 3 (15,000 rad/s^2, 150 rad/s, 6.35 cm, and 1.59 kg, respectively). Substituting these values into Eqs. (1) and (2) above, we find that normal force F_n exceeds the tangential force F_t, and is calculated as follows:

$$F_{n,high} = m_{head}(r)\left(\frac{d\theta}{dt}\right)^2 = (1.59\,\text{kg})\left(6.35\,\text{cm}\times\frac{1\,\text{m}}{100\,\text{cm}}\right)\left(150\frac{\text{rad}}{\text{s}}\right)^2 = 2272\,\text{N}$$

However, Bandak reported $F_{n,high}$ at 35,931 N in Table 3, a value 15.8 times higher than the correct value. Similarly, to calculate forces for the least severe shaking event discussed by Bandak, we used the minimum values of each parameter range provided by Bandak's Table 3 and calculated the lower range of the normal force as:

$$F_{n,low} = m_{head}(r)\left(\frac{d\theta}{dt}\right)^2 = (0.68\,\text{kg})\left(3.81\,\text{cm}\times\frac{1\,\text{m}}{100\,\text{cm}}\right)\left(50\frac{\text{rad}}{\text{s}}\right)^2 = 65\,\text{N}$$

The corresponding value reported by Bandak in Table 3 is 1027 N.

We repeated the force calculations for all values in Bandak's Table 3 and our attempts to reproduce these neck force calculations consistently yield values that are at least 10 times *lower* than those reported for shaking in Table 3 and Fig. 4 of Bandak's paper. While in some cases the error appears to be a failure to include the neck length, there is no single, simple explanation responsible for the errors that appear in *every* value in Table 3. Also, Prange and Myers [2] analysis of the same data yielded neck forces similar to what we have calculated here.

DOIs of original articles: 10.1016/j.forsciint.2005.12.017, 10.1016/j.forsciint.2006.01.001.

[1] It is important to note that the equations for tangential and normal acceleration in Bandak's methods and repeated in this letter do not account for chest acceleration, and it is not known if the actual neck forces would be higher or lower if chest acceleration were considered.

doi:10.1016/j.forsciint.2005.12.018

EXHIBIT 7

Based upon his flawed calculations, Bandak erroneously concluded that the neck forces in even the least severe shaking event far exceed the published injury tolerance of the infant neck. However, when accurately calculated, the range of neck forces is considerably lower, and includes values that are far *below* the threshold for injury. In light of the numerical errors in Bandak's neck force estimations, we question the resolute tenor of Bandak's conclusions that neck injuries would occur in all shaking events. Rather, we propose that a more appropriate conclusion is that the possibility exists for neck injury to occur during a severe shaking event without impact.

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Susan Margulies*
University of Pennsylvania, Department of Bioengineering, PA 19104-6392, USA

Michael Prange
Exponent, Inc., 3401 Market Street, Suite 300, Philadelphia, PA 19104, USA

Barry S. Myers
Duke University, Department of Biomedical Engineering, Durham, NC 27708-0281, USA

Matthew R. Maltese
The Children's Hospital of Philadelphia, Philadelphia, PA 19104, USA

Songbai Ji
University of Pennsylvania, Department of Bioengineering, Philadelphia, PA 19104-6392, USA

Xinguo Ning
University of Pennsylvania, Department of Bioengineering, Philadelphia, PA 19104–6392, USA

Jacob Fisher
Exponent, Inc., 3401 Market Street, Suite 300, Philadelphia, PA 19104, USA

Kristy Arbogast
Cindy Christian
University of Pennsylvania, Department of Pediatrics, Philadelphia, PA 19104, USA

*Corresponding author. Tel.: +1 215 898 0882; fax: +1 215 573 2071
E-mail address: margulies@seas.upenn.edu (S. Margulies)

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EDITOR'S CHOICE

Which clinical features distinguish inflicted from non-inflicted brain injury? A systematic review

S Maguire,[1] N Pickerd,[1] D Farewell,[2] M Mann,[3] V Tempest,[1] A M Kemp[1]

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[1] Child Health, School of Medicine, Cardiff University, Cardiff, UK; [2] Department of Primary Care and Public Health, School of Medicine, Cardiff University, Cardiff, UK; [3] Support Unit for Research Evidence, Cardiff University, Cardiff, UK

Correspondence to:
S Maguire, Child Health, School of Medicine, Cardiff University, Heath Park, Cardiff CF14 4XN, UK; sabinemaguire@yahoo.co.uk

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ABSTRACT

Aim: A systematic review of the scientific literature to define clinical indicators distinguishing inflicted (iBI) from non-inflicted brain injury (niBI).

Methods: An all language literature search of 20 electronic databases, websites, references and bibliographies from 1970–2008 was carried out. Relevant studies were independently reviewed by two trained reviewers, with a third review where required. Inclusion criteria included primary comparative studies of iBI and niBI in children aged <18 years, with high surety of diagnosis describing key clinical features. Multilevel logistic regression analysis was conducted, determining the positive predictive value (PPV) and odds ratios (OR) with p values for retinal haemorrhage, rib/long bone/skull fractures, apnoea, seizures and bruising to head/neck.

Results: 8151 studies were identified. 320 were reviewed and 14 included, representing 1655 children, 779 with iBI. Gender was not a discriminatory feature. In a child with intracranial injury, apnoea (PPV 93%, OR 17.06, $p<0.001$) and retinal haemorrhage (PPV 71%, OR 3.504, $p = 0.03$) were the features most predictive of iBI. Rib fractures (PPV 73%, OR 3.03, $p = 0.13$) had a similar PPV to retinal haemorrhages, but there were less data for analysis. Seizures and long bone fractures were not discriminatory, and skull fracture and head/neck bruising were more associated with niBI, although not significantly so.

Conclusions: This systematic review shows that apnoea and retinal haemorrhage have a high odds ratio for association with iBI. This review identifies key features that should be recorded in the assessment of children where iBI is suspected and may help clinicians to define the likelihood of iBI.

What is already known on this topic

- Considerable scepticism is being expressed as to how to distinguish inflicted (iBI) from non-inflicted brain injury (niBI).
- Varying weight has been given to specific clinical features of iBI in previous publications.
- Apnoea, retinal haemorrhages and rib fractures are felt to be important but there is no comprehensive statistical estimate of predictive power regarding iBI.

What this study adds

- The finding of apnoea and/or retinal haemorrhage in a child with a brain injury is more strongly associated with iBI than niBI.
- Children where iBI may be a differential diagnosis should undergo fundoscopy by an ophthalmologist and if <2 years old should have a skeletal survey.
- Infants less than 6 months of age with a brain injury are more likely to have sustained an iBI than older infants.

In 1974, radiologist John Caffey published his paper "The whiplash shaken infant syndrome: manual shaking by the extremities with whiplash induced intracranial and intraocular bleedings, linked with residual permanent brain damage and mental retardation".[1] The title captured a proposed mechanism for the most serious form of physical child abuse and referred to the association of intracranial and retinal haemorrhages. The article described the diagnostic challenges of identifying a condition where there are often no external signs of trauma to the head, skull fractures or a history of trauma of any kind, and associated injuries may be subtle or occult, particularly in the case of co-existing fractures. Caffey recognised that these children were often subjected to repeated traumatic episodes. Despite advances in investigative neurology and widespread awareness of this condition, inflicted brain injury (iBI) is commonly under-recognised[2,3] and remains a diagnostic challenge.

Children with iBI often present with significant intracranial injury in the context of a history of a minor fall, trivial injury or no reported trauma. Clinical symptoms and signs vary from the most non-specific, for example vomiting or irritability, to those that clearly point to a central neurological insult such as reduced consciousness.[4] Some may present with other inflicted injuries, such as bruising or fractures, as the primary indicator. For some children with a non-specific clinical presentation, the possibility of an iBI may not be considered.[3,4] As in most cases of physical child abuse there is no diagnostic test for iBI, the diagnosis is made on a balance of probability and after careful exclusion of other possible causes of the clinical presentation which include accidental injury and medical conditions such as rare metabolic conditions (glutaric aciduria[5]), coagulation disorders,[6–8] infective encephalopathies, etc. It has been recognised that up to 50% of children with iBI had presented previously with signs of physical abuse that were missed.[2,3]

Although well recognised as a condition that is prevalent in infants and babies, the estimated

EXHIBIT 8

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[illegible]

Databases

- All EBM Reviews — ACP Journal Club (ACP), Cochrane Database of Systematic Reviews (COCH), Database of Abstracts of Reviews of Effects (DARE), Cochrane Central Register of Controlled Trials, Health Technology Assessment, National Health Service Economic Evaluation, Cochrane Methodology Register
- ASSIA (Applied Social Sciences Index and Abstracts)
- ChildData
- CINAHL (Cumulative Index to Nursing and Allied Health Literature)
- EMBASE
- MEDLINE
- MEDLINE In-Process & Other Non-Indexed Citations
- SCOPUS
- Open SIGLE (System for Information on Grey Literature in Europe)*
- Social Care Online
- TRIP Plus
- Web of Knowledge — ISI Proceedings
- Web of Knowledge — ISI Science Citation Index
- Web of Knowledge — ISI Social Science Citation Index

Websites

- The Child Brain Injury Trust (CBIT), www.cbituk.org
- Child Welfare Information Gateway (formerly National Clearing House on Child Abuse and Neglect), http://www.childwelfare.gov/
- International Society for Prevention of Child Abuse and Neglect (ISPCAN), http://www.ispcan.org/
- The National Center on Shaken Baby Syndrome, http://www.dontshake.com/

*Up to 2005 when the database ceased indexing.

incidence of inflicted head injury is 21–24 per 100 000 infants under the age of 1 year.[10 11] Cases may present regularly to specialist neurology units but present infrequently to general paediatric units. Clinicians in all settings need to be aware of the clinical indicators for this condition to make evidence based decisions as to when they should consider investigating an infant where iBI is a possible cause and to inform a final diagnosis. Many are asked to present their professional or expert opinion in the family or criminal courts as part of the child protection process and they must be able to cite relevant evidence to back up their statements. Although there is a considerable amount of published scientific literature in this field, there have been few attempts to systematically review and critically appraise the world literature. We have attempted to repair this deficit and have addressed the question: "What are the clinical features that distinguish inflicted from non-inflicted brain injury?". The neuro-radiological features of iBI/niBI are the subject of a separate review

METHODS

We conducted an all language literature search from 1970–2008, searching 20 databases, websites, references and bibliographies, using over 100 keyword combinations (box 1, table 1). This yielded 320 studies for review by reviewers, drawn from paediatricians, paediatric neurologists, neuro-radiologists and ophthalmologists, designated and named doctors/nurses in child protection. All reviewers underwent standardised critical appraisal training, based on the NHS Centre for Reviews and Dissemination critical appraisal standards,[12] supported by a dedicated electronic critical appraisal module All studies underwent two independent reviews and a third if there was disagreement (fig 1).

Inclusion/exclusion criteria and quality standards

We defined the term "brain injury" as extra-axial haemorrhage (subdural, extradural, subarachnoid) and/or injury to the brain (hypoxic ischaemic injury, parenchymal injury, contusion, diffuse axonal injury, cerebral oedema). Inclusion/exclusion criteria are listed in box 2. Inflicted brain injury (iBI), the item of interest, refers to children who had sustained brain injury from physical child abuse. Those with non-inflicted brain injury (niBI) had other causative mechanisms including accidental trauma, and medical causes of intracranial lesions.

Given the nature of this research field, our optimal study type was population based studies that compared the clinical features of iBI and niBI with consecutive case ascertainment. As only studies with both niBI and iBI cases could be used for any analysis of predictive power of the features identified, many oft cited (non-comparative) studies were not eligible for inclusion. The included studies detailed the clinical features of interest but did not rely solely on these for the determination of diagnosis (iBI vs niBI). Only studies after 1970 were considered for inclusion, as radiological techniques prior to this would not be relevant to current practice. Included studies had a high surety of diagnosis of iBI[13] and confirmation of cause in niBI. To determine a high surety of abuse, we implemented our previously published "ranking of abuse", that is, children stated as "abused" in the study had had that outcome confirmed by multi-agency child protection teams, legal decision, witnessed abuse or perpetrator admission (rank 1–2)[14] (table 2). Studies where the decision of abuse had relied solely on clinical features were excluded to minimise selection bias and circularity. Where studies included a category "aetiology indeterminate", these cases were excluded from the final analysis.

Statistical analysis

The analysis was limited by the items that the authors chose to report and we were able to analyse the following features:

- apnoea
- retinal haemorrhages
- rib fractures
- long bone fractures
- bruising to the head and/or neck
- seizures
- skull fractures

We were dependent on the primary authors' definitions of the above terms. "Apnoea" reflects either recorded or reported apnoea in the two studies that recorded this item.[15 16] "Seizures" were not always defined, but some authors did record that some children presented in status epilepticus.[16] "Long bone fractures" included any fracture to the femur, tibia, fibula, radius, ulna or humerus Likewise, "skull" or "rib fractures" included any type or location of fracture, respectively.

We included only data from studies where a key feature was explicitly mentioned by the authors Further, even when a feature was included in the study, not all children in each group were examined for the feature in question For example, not all children in the niBI group had a fundoscopy examination to look for retinal haemorrhages We have elected to adopt an

Table 1 Keywords and search strategy

Keywords		
Set 1		
battered child	child protection	non-accidental injury
shaken baby	children	non-accidental trauma
battered baby	inflicted brain injury	physical abuse
battered infant or shaken infant	inflicted cerebral injury	shaking baby syndrome
child	inflicted traumatic head injury	shaking impact syndrome
child abuse	inflicted traumatic brain	soft tissue injury
child maltreatment	intentional abuse	
Set 2		
abusive head trauma	growing skull fracture	intraventricular hematoma
bleeding into brain	haematoma	laceration
blow to the head	haemorrhagic retinopathy	laminar necrosis
brain	head injuries	leptomeningeal cyst
brain damage	head trauma	multiple skull fracture
brain haemorrhages	hematoma	neurologic injury in child abuse
brain hemorrhages	hemorrhagic retinopathy.af.	neuropathology
brain injuries	hydrocephalus	non-accidental head injury
brain swelling	hygroma	parafalcine
brainstem	hypoxic-ischaemic injury	parenchymal contusion, laceration
central nervous system	hypoxic-ischemic injury	retinal haemorrhage
cerebral	impact injury	retinal hemorrhage
cerebral atrophy	infarction	sciwora
cerebral edema	inflicted brain injury	shaking impact syndrome
cerebral injuries	inflicted cerebral injury	shearing injury
cervical lumbar	inflicted traumatic brain injury	skull fractures
cervical spine injury	inflicted traumatic head injury	spinal cord injury
cervical spine	interhemispheric	subarachnoid hematoma
neuropathology	intracerebral bleeding	subdural hematoma
contusion	intracerebral haemorrhage	subdural haematoma
contusional tear	intracerebral hemorrhage	subdural hygroma
cranial injury	intracranial haemorrhage	thoracic lumbar sacral
craniocerebral trauma	intracranial hemorrhage	traumatic effusions
craniocervical	intracranial injuries	ventricular haemorrhage
diagnostic triad	intraparenchymal tear	ventricular hemorrhage
diffuse axonal injury	intraparenchymal haemorrhage	whiplash impact syndrome
eggshell fracture	intraparenchymal hemorrhage	whiplash injury
encephalomalacia		whiplash shaken infant
encephalopathy		
extracranial CNS injury		
extradural haemorrhage		
extradural hemorrhage		

Search strategy	
1 Child/	54. (retinal hemorrhage or retinal haemorrhage).af.
2. (child: or infant: or toddler:).af.	55. skull fracture:.af.
3. 1 or 2	56. (spinal cord injury adj3 radiologic abnormality).af.
4. non-accidental injur:.af.	57. spinal cord injur:.af.
5. non-accidental trauma.af.	58. (subdural haematoma or hematoma).af.
6. (non-accidental: and injur:).af	59. (subarachnoid hematoma or subarachnoid haematoma).af.
7. soft tissue injur:.af.	60. (subdural haemorrhage or subdural hemorrhage).af.
8. physical abuse.af.	61. (ventricular haemorrhage or ventricular hemorrhage).af.
9 (inflicted brain injur: or inflicted cerebral injur:).af	62. whiplash impact syndrome.af.
10. (inflicted traumatic head injur: or inflicted traumatic brain injur:).af.	63. whiplash injur:.af.
11. (or/4-10) and 3	64. whiplash shaken infant.af.
12. (child abuse or child maltreatment or child protection).af.	65. infarction.af.
13. (battered child or shaken baby or battered baby).af.	66. (hypoxic-ischemic injur: or hypoxic-ischaemic injur:).af.
14. (battered infant or shaken infant).af.	67. (contusion: or contusional tear).af.
15. Shak: Baby Syndrome.af.	68. (hematoma or haematoma).af.
16. shak: impact syndrome.af.	69. laceration:.af.
17. Caffey-Kempe syndrome.af.	70. shearing injur:.af.
18. battered child syndrome.af.	71. traumatic effusion:.af.

Continued

Table 1 Continued

Search strategy	
19. *"Child Abuse"/di [Diagnosis]	72. subdural hygroma.af.
20. infant traumatic stress syndrome.af.	73. hygroma.af.
21. parent-infant traumatic stress syndrome.af.	74. interhemispheric.af.
22. or/12–21	75. parafalcine.af.
23. 11 or 22	76. (brain or brainstem).af.
24. abusive head trauma.af.	77. cerebral.af.
25. bleeding into brain.af.	78. intraparenchymal.af.
26. blow to the head.af.	79. sciwora.mp.
27. brain damage.af.	80. spinal cord injury without radiologic abnormality.af.
28. (brain haemorrhage: or brain hemorrhage:).af	81. cervical lumbar.af.
29. (brain swelling or cerebral edema).af.	82. thoracic lumbar sacral.af.
30. cerebral injur:.af.	83. leptomeningeal cyst.af.
31. cervical spine injur:.af.	84. growing skull fracture.af.
32. cervical spine neuropathology.af.	85. hydrocephalus.af.
33. cranial injur:.af.	86. laminar necrosis.af.
34. craniocerebral trauma.af.	87. encephalomalacia.af.
35. diffuse axonal injur:.af.	88. cerebral atrophy.af.
36. extracranial CNS injur:.af.	89. craniocervical.af.
37. extracranial Central Nervous System injur:.af.	90. encephalopathy.af.
38. central nervous system injur:.af.	91. (intraparenchymal hemorrhag: or intraparenchymal haemorrhag:).af.
39. (extradural haematoma or hematoma).af.	92. Haemorrhagic retinopathy.af.
40. extradural haemorrhage.af.	93. hemorrhagic retinopathy.af.
41. haemorrhagic retinopathy.af.	94. (Haemorrhagic retinopathy adj3 retinal haemorrhages).af.
42. (head injur: or head trauma).af.	95. Extradural haemorrhage.mp. or Extradural hemorrhage.af.
43. impact injur:.af.	96. (extradural haemorrhag: or extradural hemorrhag:).af.
44. intracerebral bleeding.af.	97. (extradural spinal haemorrhag: or extradural spinal hemorrhag:).af.
45. (intracerebral haemorrhage or intracerebral hemorrhage).af.	98. (Haemorrhagic retinopathy adj3 retinal haemorrhag:).af.
46. (intracranial haemorrhage or intracranial hemorrhage).af.	99. (Hemorrhagic retinopathy adj3 retinal hemorrhag:).af.
47. intracranial injur:.af	100 (retinal hemorrhag: or retinal haemorrhag:).af.
48. (intraventricular hematoma or intraventricular haematoma).af.	101 or/24–100
49. (multiple skull fractur: or eggshell fractur:).af.	102. 23 and 101
50. (neurological injur: adj3 child abuse).af	
51. neuropathology.af.	
52. non-accidental head injur:.af.	
53. (parenchymal contusion or laceration).af.	

extremely conservative imputation approach to account for this missing information.[11] For example, if we are analysing a feature that we suspect may be associated with iBI, for instance, retinal haemorrhages: in the group of children with iBI, if only 6/10 iBI children underwent fundoscopy, we made the assumption that the four children who were not examined would not have had the feature.

By contrast, in the group of children with niBI, if an investigation for this feature was not performed, for example, if only 2/10 niBI children underwent fundoscopy, we made the assumption that the eight children who were not examined would have had the feature. Only in the case of skull fractures, a feature whose presence we suspect to be associated with niBI, was the opposite imputation performed. We believe that by approaching the missing data in this way, we err on the side of caution, and risk underestimating, rather than overestimating the strength of the discrimination provided by this feature. We have specifically employed this technique to avoid reinforcing any prior assumptions, but rather to determine a valid estimate of the significance of each of the features under analysis.

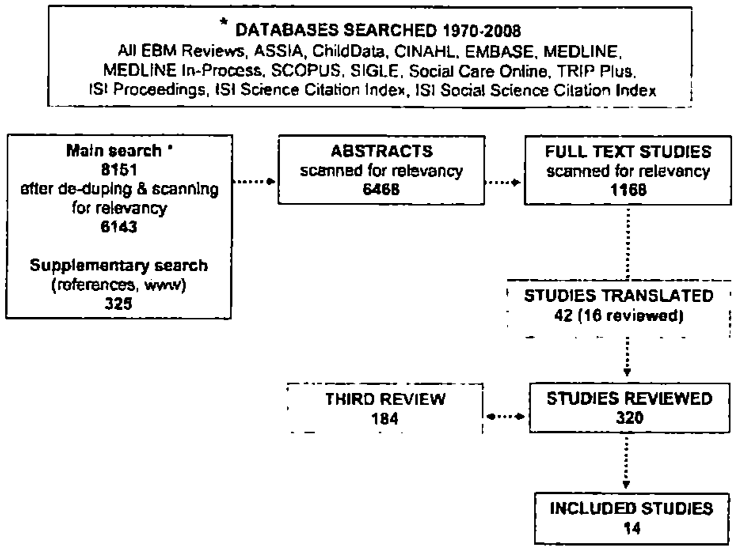
These imputations being given, we then conducted a multilevel logistic regression analysis,[12] allowing not only the prevalence of abuse to vary between studies, but also the odds ratios (OR) for the features in question. By allowing the odds ratio to differ between studies, we again aim to minimise the risk of circularity, where an individual study may have overly relied upon a particular feature in order to arrive at the diagnosis of abuse. For each feature, we report the estimated odds ratio for the feature in discriminating between iBI and niBI, together with a 95% confidence interval (95% CI). We also provide a p value to determine if this odds ratio is significantly different from 1, and a positive predictive value (PPV), the estimated probability of abuse given the presence of this feature in a child with brain injury, together with a 95% CI.

RESULTS

Of 320 reviewed studies, 14 met the inclusion criteria.[illegible] All were published after 1995. Six were cross-sectional studies,[illegible] five case series,[illegible] two case–control studies[illegible] and one a longitudinal cohort study.[21] Eight studies involved prospective enrolment of cases.[illegible] The six retrospective studies achieved full case ascertainment from hospital inpatient coding and/or radiology databases. Full study details are given in online appendix A.

Of the 306 studies excluded, the most common reasons for exclusion were the lack of comparative data or a rank of abuse of less than 1 or 2. A small number (11 studies) were excluded

Figure 1 Main phases and number of studies identified at each stage.



Inclusion criteria
- children aged 0 to <18 years
- observational comparative study (cross-sectional/case–control/case series/longitudinal cohort)
- children with inflicted brain injury diagnosed on CT/MR: intracranial haemorrhage (extra-axial with or without additional brain injury – ± intra-parenchymal haemorrhage, ± diffuse axonal injury ± hypoxic ischaemic injury ± cerebral contusion ± cerebral oedema)
- ranking of abuse 1 or 2 for inflicted brain injury (iBI)
- non-inflicted brain injury (niBI): non-inflicted aetiology confirmed
- children who were alive at presentation
- all language studies
- relevant clinical details given for each group

Exclusion criteria
- studies about complications, management or prognosis of iBI/niBI
- consensus statements or personal practice studies
- non-comparative studies
- studies addressing exclusively post mortem neuro-pathological findings
- studies with mixed adult and child data, where the children's data cannot be extracted
- methodologically flawed studies (eg, significant bias, where iBI was not adequately confirmed or where inadequate clinical details were given)
- studies that only addressed head injury where there was no intracranial abnormality
- studies with a low surety of diagnosis of inflicted injury (rank 3–5 abuse)

due to study design (expert opinion/review article, etc) and a further 10 included mixed adult and child data. Many studies were excluded on more than one basis (eg, rank of abuse/non-comparative/lack of detail on features for analysis).

Studies represented combined data on 1655 children, 779 of whom had suffered iBI and 876 niBI. Eleven of 14 studies included children less than 3 years of age.[illegible] Seven studies ascertained all children who were hospitalised with traumatic head injuries,[illegible] six studies included all children with subdural haemorrhage of any aetiology (trauma, coagulopathy, metabolic disorder or post infection, etc)[illegible] and one study included children with subdural or extradural haemorrhage.[illegible] The majority (seven) were conducted in the USA,[illegible] five were European[illegible] (three UK[illegible]) and one each was from Australia[27] and Hong Kong.[18] In nine studies, children were included solely on the basis of age and confirmation of cause. However, five had specific exclusion criteria (table 3).

Gender was recorded in 960 cases and was not a distinguishing feature. More boys than girls sustained brain injuries, regardless of aetiology (PPV of iBI if the child is a boy 0.397 (95% CI 0.277 to 0.550), OR 0.697, $p>0.2$)

It was not possible to undertake a formal analysis of age as a discriminating feature as each study reported it differently. Eight studies confirmed that children with iBI were significantly younger than those who sustained niBI.[illegible] Of the

Table 2 Abuse ranking

Ranking	Criteria used to define abuse
1	Abuse confirmed at case conference or civil, family or criminal court proceedings or admitted by the perpetrator, or independently witnessed
2	Abuse confirmed by stated criteria including multi-disciplinary assessment
3	Diagnosis of abuse defined by stated criteria
4	Abuse stated as occurring, but no supporting detail given as to how it was determined
5	Abuse stated simply as "suspected", no details on whether it was confirmed or not

Table 3 Additional exclusion criteria of included studies

Author/year	Additional exclusion criteria
Ewing-Cobbs L, Kramer L, Prasad M, *et al* (1998)[illegible]	Prior traumatic brain injury, metabolic abnormality, preterm (<32 weeks)
Hettler J, Greenes DS (2003)[illegible]	Coagulopathy, neonatal neurological abnormality, structural abnormality, surgery or previous intracranial haemorrhage
Fung ELW, Sung RYT, Nelson EAS, *et al* (2002)[illegible]	Subdural haemorrhage due to infection or surgery
Pierre-Kahn V, Roche O, Dureau P, *et al* (2003)[illegible]	Cardiopulmonary resuscitation, coagulation abnormality, severe dehydration or delayed eye examination
Ettaro L, Berger RP, Songer T (2004)[illegible]	Concussion, cerebral lacerations or contusions

eight studies addressing children less than 3 years of age, where mean ages were available,[15 16 19 20 22 24 25 27] three studies found no significant difference between iBI and niBI.[illegible] Five studies[illegible] stated that children with iBI were significantly younger than those with niBI. Four of these studies[illegible] gave the mean ages of children with iBI as less than or equal to 6 months, while the niBI groups ranged between 7.5 and 12.4 months.[illegible]

Combined feature analysis
It was not possible to identify which children had specific combinations of features, as this level of detail was not given.

Apnoea
Apnoea appeared to be a highly discriminatory finding, with a PPV of 93% (97.5% CI 0.733 to 0.986) and an OR of 17.062 (97.5% CI 5.018 to 58.014, $p<0.001$). However, it was one of the least recorded clinical items, only being analysed by two authors (405 children), perhaps reflecting that its significance has only been recognised more recently.[illegible]

Retinal haemorrhage
Although 1263 children were included in studies that considered this feature, only 998 had fundoscopy (670 iBI and 328 niBI). Many children presenting with witnessed trauma did not have formal fundoscopy, whereas the majority of children with iBI did. Therefore, to account for studies where this was not explicit,[illegible] we used the conservative approach described above. Retinal haemorrhages were strongly associated with iBI, with a PPV of 71% (97.5% CI 0.465 to 0.868) and an OR of 3.504 (97.5% CI 1.088 to 11.280, $p=0.03$). A child with an intracranial injury who has co-existent retinal haemorrhages is significantly more likely to have iBI than niBI.

Rib fractures
Rib fractures were considered in studies representing 1002 children, and data were available on 903 children. Rib fractures were associated with inflicted injury, with a PPV of 73% (97.5% CI 0.500 to 0.882) and an OR of 3.027 (97.5% CI 0.716 to 12.799). The wide confidence interval reflects the fact that relatively few children had data recorded on this item. Our conservative analysis therefore results in lower significance.

Seizures
Seizures were considered in studies involving 760 cases, and results were documented in 758. Seizures were more associated with iBI than niBI, but not significantly so, with a PPV of 66% (97.5% CI 0.454 to 0.821) and an OR of 2.924 (97.5% CI 0.731 to 11.694, $p=0.13$).

Long bone fractures
One of the difficulties in analysing fractures as recorded in studies, is knowing whether the author is counting individual fractures, or individual children who have sustained fractures. In studies where this was not explicit,[illegible] we made the assumption that the number of children with fractures was recorded, a conservative approach. Studies considering long bone fractures represent 1020 children, of whom 921 underwent skeletal imaging. Overall, the PPV of long bone fractures for iBI was 59% (97.5% CI 0.48 to 0.69), OR 1.722 (97.5% CI 0.824 to 3.601, $p=0.14$). Therefore a long bone fracture in association with an intracranial injury was a weak predictor for iBI, but not significantly so.

Skull fractures and bruising to the head and neck
Skull fractures were considered in studies representing 1014 children, and documented as present or absent in 916 children. Skull fractures were more strongly associated with niBI than iBI, with a PPV for iBI of 44% (97.5% CI 0.223 to 0.678), OR 0.852 (97.5% CI 0.316 to 2.301, $p>0.3$). Likewise, bruising to the head and neck was more common in the niBI children, but it was the least recorded item, weakening its significance. Only 212 cases had this feature considered, and overall the PPV of head and/or neck bruising for iBI was 37% (97.5% CI 0.035 to 0.906), OR 0.811 (97.5% CI 0.070 to 9.410, $p>0.2$). No specific mention was made of the use of 3D reconstruction techniques to define such fractures; the authors simply recorded their presence or absence when looked for.

DISCUSSION
Making the clinical distinction between iBI and niBI in children is always going to be challenging. By producing a multilevel logistic regression of specific clinical features on over 1600 children, we have shown that there is scientific evidence to support the distinction between iBI and niBI, and we are able to offer positive predictive values and odds ratios for individual clinical indicators in children hospitalised with brain injury. The published literature does not allow an analysis of combined features.

This review is the largest of its kind, and offers for the first time a valid statistical probability of iBI when certain key features are present (eg, retinal haemorrhages). Although in some instances, for example, rib fractures, the sample size limits our statistical power to detect the full effect of this feature, as with any systematic review, we are of course limited by the amount of high quality data available in the published literature. However, even with this limited data set, the presence of rib fractures (PPV 73%, OR 3.027) is still indicates an association with iBI rather than niBI, and emphasises how important it is to conduct appropriate imaging to determine if such fractures are present.

It appears that the younger the child, the greater the likelihood that an intracranial injury is due to abuse

However, children as old as 10 years with severe iBI have also been described in isolated case reports.[3-5]

Apnoea appears to be a critical distinguishing feature (PPV for abuse 93%, OR 17.06, $p<0.001$). There is increasing evidence that hypoxic ischaemic injury and its attendant complications are an integral part of the cascade in iBI.[4] The presence or absence of apnoea must be recorded in all cases of head injury in children.

Retinal haemorrhages are a valuable discriminator, reinforcing the need for fundoscopy on any child with unexplained intracranial injury, or suspected iBI. Indirect ophthalmoscopy should be conducted by an ophthalmologist, as studies have shown that other clinicians are either unable to conduct an accurate examination, or when they do, may miss up to 13% of retinal haemorrhages present.[5]

Fractures in physical abuse are often occult.[5] Therefore, in line with the Royal College of Paediatrics and Child Health/Royal College of Radiologists standards for the radiological investigation of suspected physical abuse, any child aged less than 2 years in whom an inflicted injury is considered, should have a full skeletal survey, including oblique views of the ribs.[5] Seizures were not a valuable discriminatory finding. Perhaps an analysis of specific patterns of seizures (eg, status epilepticus, complex, focal) would be more informative.

It was not possible to analyse the value of history given as a predictor, as a number of studies had used absence of a history of trauma or inconsistent explanation as part of their definition of abuse.[20 21] Only one large study set out to estimate the value of the initial history in predicting whether the head injury was inflicted or non-inflicted.[22] Study design ensured that the history did not contribute to the decision as to whether the child belonged to the iBI or niBI group. The study concluded that having no history of trauma had a high specificity (0.97) and high PPV (0.92) for inflicted injury.

Although many studies addressed encephalopathic features, the differing classifications that were used (altered consciousness, coma, irritability, Glasgow Coma Score (GCS)) precluded any combined analysis of this information. It is recognised that performing a GCS on young infants is imprecise; a study[3] that noted an increased relative risk of this item for iBI (RR 1.7, 95% CI 0.9–3.2) did not attach weight to this feature.

We did not address social features which are more culturally specific, and less suited to meta-analysis. There is also the risk of introducing bias by undue reliance on social features, as shown by Jenny,[3] who noted that white infants from two parent families were more likely to have their iBI missed than ethnic minority children or those living with one parent.

This review did not address biomechanical features. It is recognised that iBI can occur as a consequence of shaking,[1 40] impact,[41] shaking and impact,[42 43] water intoxication,[44 45] suffocation[46] or strangulation.[47] In our review skull fracture and bruising to the head/neck were more strongly associated with niBI than iBI, suggesting blunt trauma as a common mechanism of niBI. Clinical evidence of impact may only be found on post mortem,[48] so conclusions as to whether an impact injury has occurred or not in iBI cannot reliably be drawn from the clinical features alone.

It is recognised that subdural haemorrhage (SDH) can occur in infancy from other causes, and consideration must be given to possible organic disease, such as metabolic disorders (eg, glutaric aciduria),[6] albeit this has characteristic MRI findings. While coagulopathy may cause intracerebral haemorrhage,[7-9] it may also be secondary to an iBI,[47] highlighting the need for thorough investigations in this group. It is recognised that SDH in particular, may occur as a consequence of birth, particularly following instrumental delivery,[49-50] although a neonatal SDH without skull fracture has also been described where the mother was assaulted ante-natally.[51]

While this review has highlighted certain clinical features that in association with intracranial injury have a high positive predictive value and odds ratio for iBI, such as retinal haemorrhage, apnoea or rib fractures, none of these features are exclusive to iBI. It is essential that the diagnosis of iBI is only made after taking into consideration full details on history (birth, medical, family, developmental, social, details of any reported trauma) and a detailed examination, and having actively excluded other relevant organic diseases. It is vital that future work should endeavour to collect data on all the features above, with detailed recording of precise abnormalities found, for example, on ophthalmology, if we are to refine these statistical correlations.

Baroness Kennedy has made it abundantly clear that clinicians must be able to cite scientific evidence to support their clinical opinions when providing evidence to the courts,[52] and it is challenging for any clinician to keep up to date with all the current evidence (in the year 2007 alone 242 studies relating to iBI were published). Therefore, we feel that this unique, rigorously conducted systematic review will be a valuable resource for clinicians and other professionals in the field, providing, as it does, odds ratios of abuse for seven easily documented clinical features.

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Estimating the Probability of Abusive Head Trauma: A Pooled Analysis

Sabine Ann Maguire, Alison Mary Kemp, Rebecca Caroline Lumb and Daniel Mark Farewell

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EXHIBIT 9

..imating the Probability of Abusive Head Trauma: A Pooled Analysis

WHAT'S KNOWN ON THIS SUBJECT: In recent debate in medicolegal circles, clinical basis for a diagnosis of abusive head trauma (AHT) has been questioned. Studies have been underpowered to address the key clinical questions. Estimates of the association of single clinical variables with AHT are recognized

WHAT THIS STUDY ADDS: This study provides an estimate of probability of AHT based on 6 clinical features. In a child younger than 3 who has an intracranial injury and ≥3 of these features, the positive predictive value of AHT is >85% (odds ratio: >100).

CONTEXT AND OBJECTIVE: To determine which combinations of clinical features assist in distinguishing abusive head trauma (AHT) from nonabusive head trauma.

METHODS: Individual patient data from 6 comparative studies of children younger than 3 years with intracra...al injury were analyzed to determine the association between AHT and combinations of apnea; retinal hemorrhage, rib, skull, and long-bone fractures; seizures; and head and/or neck bruising. An aggregate analysis of data from these studies used multiple imputation of combined clinical features using a bespoke hotdeck imputation strategy, which accounted for uncertainty arising from missing information.

RESULTS: Analyzing 1053 children (348 had AHT), excluding nonsignificant variables (gender, age, skull fractures), for a child with an intracranial injury and 1 or 2 of the 6 features, the positive predictive value (PPV) of AHT varies from 4% to 97% according to the different combinations. Although rarely recorded, apnea is significantly associated with AHT (odds ratio [OR]: 6.89 [confidence interval: 2.08–22.86]). When rib fracture or retinal hemorrhage was present with any 1 of the other features, the OR for AHT is >100 (PPV > 85%). Any combination of 3 or more of the 6 significant features yielded an OR of >100 (PPV for AHT > 85%).

CONCLUSIONS: Probabilities of AHT can be estimated on the basis of different combinations of clinical features. The model could be further developed in a prospective large-scale study, with an expanded clinical data set, to contribute to a more refined tool to inform clinical decisions about the likelihood of AHT. *Pediatrics* 2011;128:e550–e564

AUTHORS: Sabine Ann Maguire, MRCPI, MRCPCh, FRCPI,[a] Alison Mary Kemp, MRCP, DCH, FRCPCH, FRCP,[a] Rebecca Caroline Lumb, BA,[a] and Daniel Mark Farewell, MMath, PhD[b]

Departments of [a]Child Health and [b]Primary Care and Public Health, School of Medicine, Cardiff University, Cardiff, United Kingdom

KEY WORDS

abusive head trauma, child abuse, diagnostic tool, pooled estimates of probability of AHT

ABBREVIATIONS

AHT—abusive head trauma
ICI—intracranial injury
nAHT—nonabusive head trauma
PPV—positive predictive value
OR—odds ratio
RH—retinal hemorrhage
CI—confidence interval

Dr Maguire had the initial study idea and was principle investigator for primary systematic review, drafted and revised the article, and gave final approval for publication. Ms Lumb entered all anonymized data into the database, formatted the original article, communicated with all primary authors and edited the article. Dr Farewell designed and conducted all statistical analyses wrote the statistical component of article, and contributed to overall editing; and Prof Kemp was program director for primary systematic review and contributed to the conception of analysis, drafts of the article, and subsequent editing.

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Address correspondence to Sabine Ann Maguire, MRCPI, MRCPCh, FRCPI Wales College of Medicine, Cardiff University UHW, Heath Site, Cardiff CF14 4XN, United Kingdom. E-mail sabinemaguire@yahoo.co.uk

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Abusive head trauma (AHT) remains the commonest form of fatal child abuse and predominantly affects infants.[1] These children classically present with an intracranial injury (ICI) that is evident on neuroimaging or at post mortem, where there is no explanation of trauma, or a history that is inconsistent with the severity of injury or the developmental level of the child. The challenge for the clinician is to determine, firstly, which children should be investigated to exclude AHT; secondly, when concerning features are found, how confident one can be that AHT is the cause rather than nonabusive head trauma (nAHT) or an organic disease. After a full clinical assessment, the multidisciplinary team must piece together all available (eg, clinical, social, forensic) information and determine the likelihood of an abusive etiology. These decisions are difficult, and the pressures to make the correct diagnosis are considerable. A child with missed AHT may later present with a fatal injury[2]; on the other hand, an incorrect diagnosis of AHT will have devastating consequences for the child and family.

When cases of suspected child abuse enter the legal child protection process, the medical evidence contributes heavily to decisions about the future risks to the child, and child care proceedings, regardless of whether the evidence informs a criminal prosecution. In a recent law review article, Turkheimer questioned the diagnosis of AHT on the basis of combinations of clinical features.[3] This stimulated controversial argument in The New York Times: Tuerkheimer suggested that experts are questioning the scientific basis for shaken baby syndrome (sic),[4] which was roundly refuted by responses from M. Barr and J. Leventhal,[5] among others, thus highlighting the need for an agreed scientific basis to contribute to this diagnosis. In their role as expert witnesses in court, clinicians in the United Kingdom have also come under pressure to support their opinion with scientific evidence, as highlighted by Baroness Kennedy: "A doctor can be convinced based on his or her experience that a defendant is guilty, but unless there is compelling evidence, supported scientifically, he or she should not express that view in criminal proceedings."[6] As such, an evidence-based calculation that assimilates the clinical data and contributes to determining the probability of AHT would be a valuable adjunct for the professionals concerned.

Although many studies have evaluated the diagnostic utility of individual features in identifying children with AHT, only a small number have explored how combined features predict the condition[7,8]; given that AHT is relatively uncommon in any given population,[9] individual studies are frequently underpowered to draw statistically valid conclusions for multiple combinations of features.[10] Previously, we conducted a meta-analysis of high quality studies that derives the positive predictive values (PPV) and odds ratios (ORs) for AHT for individual features.[11] However, in a clinical scenario, the diagnosis of AHT is based on an interpretation of the combined findings. In this study we aim to combine individual patient data from 6 large population-based comparative studies of AHT and nAHT to estimate the probability of AHT, given different combinations of features identifiable during the initial evaluation of such cases. In so doing, it may provide a statistical estimate to assist clinicians.

METHODS

We conducted a systematic review of the clinical features of AHT and nAHT[11] (apnea, retinal hemorrhages [RHs], rib, skull, and long-bone fractures; seizures; and head and/or neck bruising) and identified 14 high quality comparative studies. We contacted the authors of the most recent larger studies that encompassed the majority of the clinical indicators of AHT and nAHT in children younger than 3.[11] Of the 9 authors we contacted, 2 did not respond, 1 was unable to help, and the authors of the remaining 6 studies, all published since 2003,[9,12–16] generously provided anonymized, individual-level patient information. We have used these data to perform an individual-patient data pooled-analysis to relate combinations of clinical features to AHT or nAHT.

All studies included children younger than 3 who were admitted to hospital with an ICI, defined as any combination of subdural hemorrhage, subarachnoid hemorrhage, extradural hemorrhage, intraparenchymal injury, cerebral contusion, diffuse axonal injury, hypoxic ischemic injury and/or associated cerebral edema. The inclusion criteria with respect to ICI type varied between studies; cases of isolated skull fracture with no ICI were excluded. In 2 instances,[15,16] the authors provided us with additional data over and above that which they had published in the referenced studies. All extra cases had been ascertained in the same manner as that described in the published study.

We defined the different features as follows: RHs included any pattern of RH that was clearly documented on ophthalmological examination. We confirmed an absence of RH when the eyes had been examined and no hemorrhages found; otherwise the data were deemed "missing." "Long-bone fractures" included metaphyseal or diaphyseal fractures, both old and new, affecting any of the long bones. We classified a fracture as "absent" when there were no fractures reported on a skeletal survey; if no skeletal survey was performed, fracture data were re-

TABLE 1 Abuse Ranking

Ranking	Criteria Used to Define Abuse
1	Abuse confirmed at case conference or civil family or criminal court proceedings or admitted by perpetrator or independently witnessed
2	Abuse confirmed by stated criteria, including multidisciplinary assessment
3	Diagnosis of abuse defined by stated criteria
4	Abuse stated as occurring, but no supporting detail given as to how it was determined
5	Abuse stated simply as "suspected", no details on whether it was confirmed

corded as "missing." "Seizure" included documented seizures, either single or, in some cases, status epilepticus. "Apnea" and "head/neck bruising" were simply recorded as "present," "absent," or "missing."

We defined AHT (terminology as recommended in the recent American Academy of Pediatrics' recommendations[17]) as ICI where abuse had been confirmed as a cause. We only included confirmed cases of AHT, ie, those ranked 1 or 2 for abuse, according to our previously published 'rank of abuse' (Table 1). This was to ensure that the decision was based on a thorough child protection assessment and multidisciplinary decision or court proceedings to minimize circularity by dependence purely on the clinical features alone to determine if abuse had taken place.[11] All the nonabused children had confirmed causes (traumatic or organic); any cases deemed indeterminate or simply "suspected abuse" in the original data set were excluded from the analysis.

For full details of the statistical methods employed, see the Appendix. In summary, we performed an aggregate analysis of the 6 studies, taking into account the uncertainty arising from missing information on certain clinical features. To do so, we employed multiple imputation, using a bespoke hot-deck imputation strategy informed by examination of the raw data. Wherever possible, we imputed data from another child in the same or a similar study, particularly when several items were missing. If fewer than 5 children could be found to match the child who had missing information, we refrained from imputing from the matched cases because this can lead to underestimation of the uncertainty surrounding the missing features. Instead, we completed our imputed data by filling in any remaining missing items from the margins of the observed distribution of that feature, within their etiology group. We generated 10 imputed data sets for analysis.

To each of the imputed data sets, we fitted a multilevel logistic regression model. Logistic regression focuses on the probability of AHT, given information on the presence or absence of certain features. A multilevel version was used to account for the fact that the different studies were drawn from different populations and, in particular, had different prevalences of AHT. Results from the 10 imputed data sets were combined according to established procedures, and expressed as ORs, with 95% confidence intervals (CIs) and PPVs. We emphasize that a large OR need not correspond to a large PPV because a PPV takes into account the underlying prevalence of AHT in this specific population.

We fitted 3 multilevel logistic regression models. Initially, we examined the relationship between AHT, age, and gender, adjusting for variability in the prevalence of AHT between studies. We then added all 7 clinical features to the model, further adjusting for between-study variation in the dependence of etiology (AHT or nAHT) on each clinical feature. To improve statistical efficiency, we then refitted this model, omitting nonsignificant features. Finally, we assessed the predictive accuracy of the model. To do this, we used fivefold cross-validation: this process divides the data into 5 parts, using 4 to refit the multilevel model, and the remaining part to test its predictive accuracy. This process is then repeated, with each of the 5 parts being used to test the model in turn.

RESULTS

The 6 included studies[9,12–16] represent data on 1053 children (348 sustained AHT, 705 sustained nAHT). The study designs were similar; they were all population-based and included children younger than 3 who were admitted to regional hospitals with ICI diagnosed on neuroimaging. Four studies included all children with traumatic brain injury,[12–14,16] and 2 included all children who were admitted with subdural hemorrhage.[9,15] Studies confirmed etiology of nAHT cases but applied different exclusion criteria. Two of these studies were prospective,[12,16] with the remainder retrospective[9,13–15] but with detailed consecutive case ascertainment.

Analysis

Because demographic information was available for all children, it was possible to examine the diagnostic utility of age and gender without resorting to multiple imputation. No significant gender differences were observed, although it was clear that more boys than girls sustained brain injury, regardless of etiology (Table 2). All 3 of the youngest age groups (0–5 months, 6–11 months, 12–23 months) had higher prevalence of AHT than the eldest group (24–36 months), and the differences between the age groups were significant (Table 3). The odds in the 2 youngest groups were ~4 times the baseline odds. Consistent with study ascertainment, we also observed substantial between-study variability in the prevalence of abuse.

TABLE 2 Univariate Summaries According to Study

Features	Bechtel et al[12]		Ettaro et al[13]		Hettler and Greenes[14]		Hobbs et al[9]		Kemp et al[15]		Vinchon et al[16]	
	n	%	n	%	n	%	n	%	n	%	n	%
Head trauma												
nAHT	50	78	316	84	216	69	31	48	67	40	25	36
AHT	14	22	61	16	95	31	34	52	100	60	44	64
Age group, mo												
0–5	31	48	121	32	202	65	34	52	51	31	45	65
6–11	20	31	77	20	68	22	18	28	33	20	14	20
12–23	10	16	79	21	41	13	4	6	53	32	10	14
24–35	3	5	100	27	0	0	9	14	30	18	0	0
Gender												
Female	31	49	155	41	106	34	29	45	58	35	23	33
Male	33	52	222	59	205	66	36	55	109	65	46	67
Skull fracture												
Absent	28	44	154	41	149	48	0	0	142	85	57	83
Present	36	56	220	58	162	52	0	0	25	15	12	17
Data missing	0	0	3	1	0	0	65	100	0	0	0	0
Rib fracture												
Absent	16	25	358	95	306	98	0	0	141	84	52	75
Present	3	5	14	4	5	2	0	0	26	16	17	25
Data missing	45	70	5	1	0	0	65	100	0	0	0	0
Long-bone fracture												
Absent	17	27	343	91	294	95	0	0	138	83	57	83
Present	2	3	29	8	17	5	0	0	29	17	12	17
Data missing	45	70	5	1	0	0	65	100	0	0	0	0
RH												
Absent	45	70	328	87	225	72	35	54	98	57	33	48
Present	16	25	44	12	86	28	30	46	56	34	36	52
Data missing	3	5	5	1	0	0	0	0	15	8	0	0
Head and/or neck bruising												
Absent	30	47	358	95	0	0	0	0	107	64	35	51
Present	28	44	19	5	0	0	0	0	60	35	34	49
Data missing	6	9	0	0	311	100	65	100	0	0	0	0
Apnea												
Absent	0	0	0	0	0	0	57	88	116	69	51	74
Present	0	0	0	0	0	0	8	12	36	22	18	26
Data missing	64	100	377	100	311	100	0	0	15	9	0	0
Seizures												
Absent	46	72	0	0	235	76	46	71	84	50	33	48
Present	8	12	0	0	76	24	19	29	66	40	36	52
Data missing	10	16	377	100	0	0	0	0	17	10	0	0

TABLE 3 Regression Analysis of Demographic Information: Global Test of Significance of Age

	Regression Coefficient	Lower Confidence Limit	Upper Confidence Limit	OR	Lower Confidence Limit	Upper Confidence Limit	P	Random Effects SD
Intercept	−1.943	−3.050	−0.835	0.143	0.047	0.434	.001	1.135
0–5 mo	1.500	0.428	2.572	4.480	1.534	13.088	<.001	1.074
6–11 mo	1.399	0.731	2.066	4.051	2.078	7.897	—	<0.001
12–23 mo	0.752	−0.067	1.571	2.121	0.935	4.813	—	0.483
Male	0.280	−0.021	0.580	1.322	0.979	1.787	.069	<0.001

Multiply Imputed Multilevel Logistic Regression

For each of the 10 imputed data sets, we used multilevel logistic regression to determine the diagnostic value of all features identified during initial investigations in combination. We report effect sizes on the logistic scale and in terms of ORs, giving 95% CIs for these estimates, and PPVs, together with P values and estimates of the SDs of the random effects (Table 4). In this model, the reference category is a girl aged between 24 and 36 months, but now, additionally, they are known to have no other clinical features present (ie, ICI alone).

Nonsignificant Features

Once other clinical features were known, age was no longer significantly associated with etiology at the 5% level. In other words, given information about the presence or absence of the 7 clinical features, knowing the child's

TABLE 4 Full Regression Model of Demographic and Clinical Features Expressed as ORs With 95% CIs, P Values, and Estimates of the SDs of the Random Effects

	Regression Coefficient	Lower Confidence Limit	Upper Confidence Limit	OR	Lower Confidence Limit	Upper Confidence Limit	P	Random Effects SD
Intercept	−4.002	−5.505	−2.500	0.018	0.004	0.082	<0.001	1.238
0–5 mo	1.138	−0.339	2.615	3.120	0.712	13.667	0.068	1.371
6–11 mo	1.284	0.277	2.291	3.612	1.320	9.884	—	<0.001
12–23 mo	0.267	−1.093	1.627	1.306	0.335	5.088	—	0.910
Male	0.149	−0.435	0.732	1.160	0.647	2.079	0.616	0.194
Skull fractures	−0.749	−1.928	0.430	0.473	0.145	1.537	0.213	1.142
Rib fractures	3.755	1.963	5.548	42.746	7.119	256.668	<0.001	0.446
Long-bone fractures	2.635	1.179	4.092	13.949	3.250	59.865	<0.001	0.971
RHs	3.450	2.824	4.076	31.497	16.840	58.912	<0.001	0.159
Head and/or neck bruising	1.513	0.160	2.865	4.539	1.174	17.546	0.027	1.115
Apnea	1.938	0.742	3.135	6.948	2.100	22.985	0.001	0.617
Seizures	1.624	0.675	2.574	5.075	1.963	13.117	0.001	0.762

age as well did not significantly alter the probability that the child had been abused. Gender remained uninformative where AHT was concerned. Skull fracture(s) were the only clinical feature(s) with an OR of <1 (ie, more suggestive of nAHT than AHT), although this association did not reach significance.

Because we found no statistical evidence that including these terms in a model for AHT was important, we refitted the model, dropping the nonsignificant features. The reduced, multiply-imputed multilevel analysis gave rise to Table 5, on which we base our substantive conclusions.

ICI Alone

When a child younger than 3 had an ICI, with none of the clinical features noted above, the estimated probability of AHT was ~4% (Table 5). Although we did account for study-specific associations between AHT and age, AHT and gender, and AHT and skull fractures, we reiterate that age did not help to determine the likelihood of abuse when all other clinical features were known.

Results When Each Feature Is Solely Present in a Child With ICI

Fractures

For rib fractures there was the strongest evidence of AHT, with an OR of ~45 (Figs 1 and 2). Note, however, the wide CIs that surround this estimate, owing to the very small numbers of rib fractures present in both etiology groups. Long-bone fractures are similarly indicative of AHT (OR: 13.75), slightly less strongly than rib fractures. Because the prevalence of long-bone fractures was marginally higher than that of rib fractures, the CIs surrounding this estimate are narrower.

RH, Head and/or Neck Bruising, Apnea, and Seizures

RHs were 1 of the more frequently recorded items, and had an estimated OR of ~35. Therefore, whereas a child with an ICI alone had a probability of AHT of 4%, this rose to 58% if ICI and only RHs were present. Head and/or neck bruising had a weaker, marginally significant relationship with AHT. A combination of ICI and bruising to the head and/or neck increased the probability of AHT from 4% to 15%. It is worth emphasizing, however, that this left 85% of children with this pattern of injury who did not have AHT.

TABLE 5 Reduced Regression Model

	Regression Coefficient	Lower Confidence Limit	Upper Confidence Limit	OR	Lower Confidence Limit	Upper Confidence Limit	P	Random Effects SD
Intercept	−3.213	−4.358	−2.067	0.040	0.013	0.127	<0.001	1.117
0–5 mo								1.210
6–11 mo								0.614
12–23 mo								1.069
Male								0.160
Skull fracture								1.240
Rib fractures	3.800	2.036	5.565	44.720	7.659	261.096	<0.001	0.311
Long-bone fractures	2.621	1.235	4.006	13.747	3.440	54.937	<0.001	0.930
RHs	3.519	2.882	4.157	33.764	17.855	63.846	<0.001	0.211
Head and/or neck bruising	1.451	0.072	2.830	4.268	1.075	16.951	0.038	1.175
Apnea	1.931	0.732	3.129	6.893	2.079	22.858	0.001	0.623
Seizures	1.624	0.695	2.553	5.072	2.003	12.843	0.001	0.737

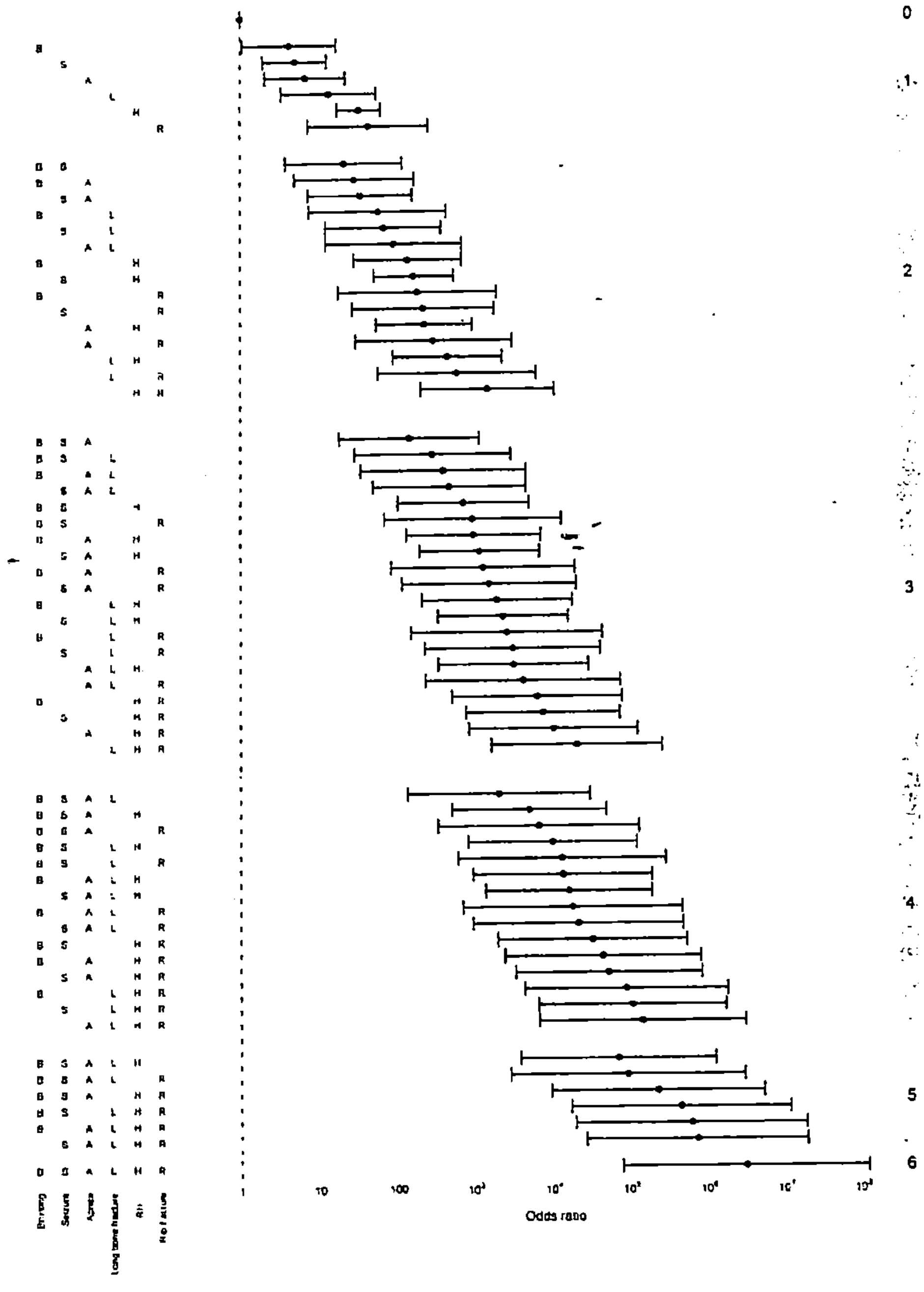
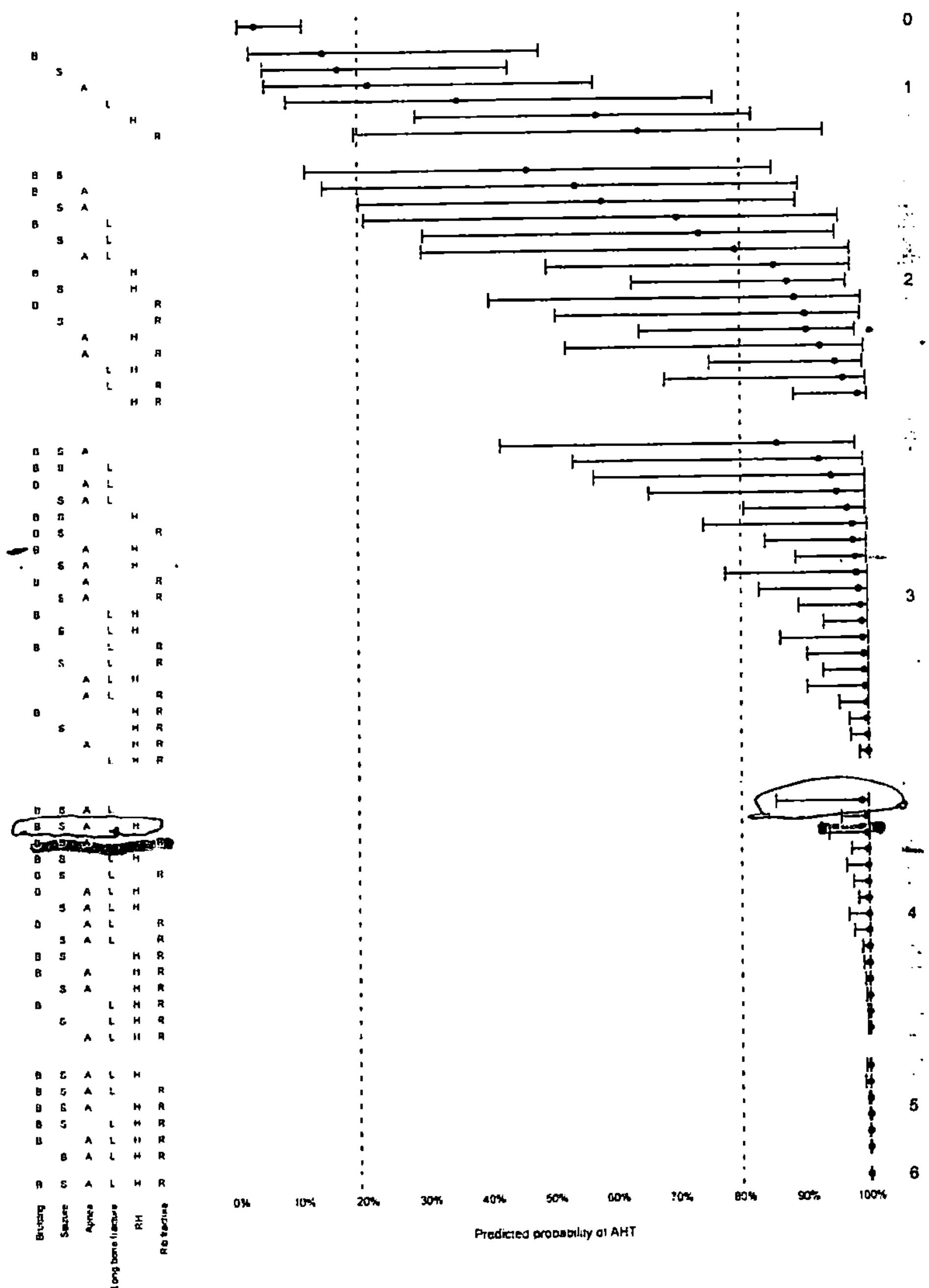


FIGURE
OR for AHT in children younger than 3 years who have an ICI (log scale).



PPV of AHT in children younger than 3 years who have an ICI.

Either apnea or seizures alone, in association with ICI, significantly increased the likelihood of AHT, both at a level comparable to that of head and/or neck bruising alone (an OR of 5). Apnea was the slightly stronger of the 2, although this finding is subject to a degree of uncertainty because of the large amount of missing data (recorded in only 301 of 1053 cases). It is hoped that this feature will be more commonly recorded in future studies because of increasing recognition of its association with AHT.

Results When Multiple Features Are Present

A unique advantage of our study is the ability to consider the presence or absence of multiple clinical features in combination. Thus far, we have used the data to focus on situations where we knew that only a single clinical feature was present; however, we now turn to the more frequent scenario where more than 1 feature may be present (Figs 1 and 2). It is immediately apparent that when only 1 or 2 features were present, the ability to distinguish AHT from nAHT depends heavily on the specific feature(s) in question. For instance, if a child with ICI had head and/or neck bruising in combination with apnea, the estimated probability of AHT was 54% (OR: 29); however, if the child had apnea combined with RHs, this estimated probability rose to 90%, and the OR was almost 10 times higher. Likewise, if the child presented with apnea and seizures but no other features, the estimated probability of AHT was only 58% (OR: 35); however, if a child had seizures and rib fractures, the probability of AHT rose to 90%, and the OR was 227.

Once 3 or more of the significant features were present, ORs were >100, and PPVs for AHT were uniformly above 85%, irrespective of the specific features. In Fig 2 we show precise estimates for each of the 64 possible combinations.

Accuracy of the Model: Model Checking

In the absence of an additional, independent source of data against which to test our findings, we used cross-validation to assess the accuracy of our predictions. Details of the model checking are provided in the Appendix. We chose high cut-off limits, such that we deemed a predicted probability to be correct if the PPV for AHT or nAHT was >80%, and the predicted etiology was true (see Fig 2). Any PPV less than this was regarded as indeterminate. On this basis, our predicted etiology was correct 80% of the time, indeterminate 15% of the time, and incorrect in 5% of cases. This highlights that no set of clinical features was unique to AHT or nAHT, and such features must be considered in the context of all other medical and social aspects of the case in question.

DISCUSSION

Our analysis of more than 1000 infants and young children with ICI of confirmed etiology represents the largest published analysis of combined clinical features to estimate the probability of AHT. We have shown that in a child younger than 3 with an ICI and 1 or 2 of the key clinical features, the probability of AHT varied depending on the number and specific features present, with RHs and rib fractures being the most discriminating. Three or more of the key features were highly predictive of AHT. This analysis offers the potential to underpin a clinical opinion with a valid scientific estimate of probability.

In the study we draw on the raw data from the most recent large-scale comparative epidemiologic publications, and we are indebted to the authors of the primary studies included. This is the first detailed multivariate analysis to be produced and, in common with all studies in this difficult field, there are limitations; however, we believe that this work makes a timely and valuable contribution to the clinical field, in particular at a time when the validity of diagnosing AHT from combinations of clinical features is being questioned.[3]

Although information on the presence of apnea was missing in a large number of cases, the majority of these (377+311) came from 2 large studies that had otherwise very complete recording of features. Where data (eg, apnea) was missing, we addressed this by using similar individuals within the data set to impute missing data, an approach that is statistically valid provided the data are missing at random.[18] In statistical terms, clinical decisions about what investigations to perform define the mechanism leading to missing data. Because such decisions are usually taken sequentially and on the basis of the results of the previous investigations (that is, the observed data), the "missing at random" assumption is reasonable in this context. All of the features, other than apnea, were investigated for in the majority of the studies.

The difference between the included studies highlights the varied approach to the clinical assessment of these children. The overall study findings reiterate the importance of a comprehensive and standardized investigation of all young children with an ICI of uncertain etiology, recording the presence or absence of seizures, apneic episodes, bruising, and findings from fundoscopy and skeletal survey to derive an initial clinical probability of AHT. The cases included in this analysis were investigated in the late 1990s and early 2000s. Undoubtedly, future studies are likely to include magnetic resonance imaging, more rigorous skeletal survey protocols and full ophthalmological data, each of which potentially reveals a greater understanding of the individual diagnostic indicators of AHT. However, the features included in our analysis are those that all frontline clinicians are likely to identify before referring the child into a specialist center for more detailed assessment, and, as such, the results of this study could be used to support such additional detailed assessments.

This model has been developed from recent high quality, large scale comparative studies currently available from the international scientific literature. However, we believe that it has the potential for future development, into a sophisticated tool to aid the clinical decision process when assessing a young child with unexplained head trauma. A large-scale prospective study is urgently needed to collect a more extensive standardized clinical data set from a cross-section of young children with ICI and apply a multivariate analysis. A proposed study of this nature would need to be conducted on an international basis to collect sufficient case numbers over a reasonable time scale and to incorporate more detail about the nature of the currently recognized clinical indicators such as seizure type and duration, the precise location and pattern of RHs,[19,20] and detailed neuroradiological features of ICI found in each group.[21] Other features may also be contributory eg, history on presentation,[14] vomiting,[22] coexistent injury,[1,23] conscious level on arrival,[16] and burns[1]; with increasing features included in the analysis, larger numbers will be required specifically to ensure that enough cases with each possible combination (features present or absent) are included to enable valid statistical analysis of every scenario.

Diagnostic studies in this field are open to criticism of circularity because of their dependence on a constellation of clinical features, as opposed to a single gold-standard diagnostic test, which does not exist. Ultimately, in any individual case, a child either has, or has not, suffered AHT and, consequently, a diagnosis of AHT either is, or is not, correct. However, except in cases of independently witnessed injury, a diagnosis must rest on a probabilistic assessment of how likely it is that AHT took place. It is not possible to restrict research in this field to independently witnessed abuse, which represents a tiny proportion of cases. We have attempted to minimize the risk of circularity by only analyzing cases where abuse was confirmed by a comprehensive evaluation of all the medical and social features, after a multidisciplinary assessment of the full case details and, in many cases, by "finding of fact" in care or criminal legal proceedings or perpetrator admissions. Likewise, nAHT cases were only included when the etiology was confirmed. To adequately power studies of what is, in statistical terms, a small population, some authors frequently combine presumptive and 'suspected' abuse into 1 category.[13] Others[10] add indeterminate cases to the noninflicted cases or combine suspected abuse with confirmed abuse. We have rigidly excluded such cases from our analysis and the extremely large data set and internal cross validation offers reassurance that our estimates are valid. In addition, the strength of this work lies in a regression analysis of 6 significant variables in 1053 children, generating 64 possible combinations, which represents a wider range of possible combinations than could be achieved from any single study. The findings from this comparative study mitigate against the circularity argument to some extent. We have demonstrated that there is a difference between the predictive probability of different combinations of features, strongly influenced by the specific features in question. However, even for strongly influential features, such as RHs, not all cases were because of AHT, as shown by a PPV of 58% for those children with an ICI and RHs and no other clinical features. These data includes the most challenging clinical scenario, namely the likelihood of abuse when a child has ICI but none of the other distinguishing clinical features. For a child younger than 3 with an ICI alone, the estimated probability that the brain injury is an AHT is 4% in this data set.

Although the use of this analysis to determine the probability of AHT in a given case will never replace the diagnostic skills of the clinician, it has the potential to contribute to decision-making. This could assist frontline professionals when deciding whether to refer a child for specialist clinical and multiagency investigation of possible AHT and contribute to decision-making at various points along the referral and assessment process. It could also assist clinicians offering medical testimony in civil or criminal proceedings, in demonstrating why certain combinations of features are more or less predictive of an abusive etiology. Contrary to the view expressed by Tuerkheimer[3] that "the scientific underpinnings of SBS have crumbled over the past decade," this large-scale analysis confirms the association of AHT with specific combinations of clinical features and, furthermore, it has enormous potential as a prototype for a larger-scale prospective study to include more sophisticated details of clinical indicators, which would be valuable to clinicians and other professionals working in this field.

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APPENDIX: DETAILS OF STATISTICAL METHODS

We began our analysis by visualizing the raw data with a parallel-coordinates plot (Fig A1). Each child is represented in this plot by a connected line that joins the recorded values of the variables of interest. Variables are transformed to share a common scale, and their values can be read off the vertical axes The plot is then divided by injury etiology and by the source study, and lines are jittered and colored to alleviate problems with overplotting. Densely colored areas of the plots represent more children than the light, sparse areas. For example, the predominant pattern of features seen in the AHT group of children in the Vinchon et al[1] study is the youngest age group, boys, without skull, rib, or long-bone fractures, with retinal haemorrhages, no information on bruising and apnea but with seizures.

Inspection of the raw data informed our hotdeck multiple-imputation strategy. The Kemp et al[7] study had complete information on all children and, thus, required no imputation. Hobbs et al[3] had only a small amount of missing data, and (where at least 5 matches could be found) this was imputed from either itself or from the Kemp et al study.[7] Ettaro et al[4] had a small amount of missing fracture data, and neither apnea nor seizures were recorded for these children. The fracture data we imputed from within the same study matched on all available features. This being done, we then found children in the Vinchon et al study[1] who matched the Ettaro et al sample[4] on all features common to both studies, using this to impute information on seizures. A similar strategy was used to bring information on bruising from the Ettaro et al study[4] into the Vinchon et al[1] cases. At this stage, both Ettaro et al[4] and Vinchon et al[1] had complete information except for apnea (always excepting any instances in which insufficient matching children could be found).

After a within-Bechtel et al[5] imputation to fill in the sporadically missing items, data from (the imputed versions of) the Ettaro et al[4] and Vinchon et al[1] studies were used to update the Bechtel et al[5] data. At this stage Bechtel et al,[5] Ettaro et al,[4] and Vinchon et al[1] were all used to form a sampling frame from which to impute into Hettler and Greenes,[6] who chose not to record many items but, importantly, recorded information on the presence or absence of apnea. At this point Hettler and Greenes,[6] Hobbs et al,[3] and Kemp et al[7] had (in principle) complete information, whereas Bechtel et al,[5] Ettaro et al,[4] and Vinchon et al[1] lacked data only on apnea. By sampling information on apnea from similar children in the former set into the latter, we completed the hotdeck stage of imputation.

In practice, there were several instances in this process where fewer than 5 matching children could be found. The choice of a minimum of 5 matched individuals is somewhat arbitrary, but it is reasonably likely that 4 or fewer children may have exactly the same observed information on the missing item(s), especially if the prevalence of the relevant feature is particularly low or particularly high. Thus, imputing from such a sample could erroneously suggest that the missing item(s) were, in fact, known. To circumvent this problem, the final stage of our imputation was to sample from the margin of the feature concerned conditional on the etiology of the neurologic injury.

Figure A2 displays the 10 imputed data sets, made slightly transparent so that similarities and differences between imputations may be seen. Note that results of the 2 subdural-only studies (Hobbs et al[3] and Kemp et al[7]) do not show the propensity for skull fractures in the nAHT group seen (or imputed) in the other 4 studies. This may be because of a number of organic causes of subdural hemorrhage in this group, whereas the nAHT groups in the other studies were trauma-related. An interesting feature of the Hettler and Greenes[6] imputations is that rib and long-bone fractures are never imputed to be present, in contrast with skull fractures and bruising. Another obvious difference between the 2 etiology groups is the number of isolated features in the nAHT groups as opposed to the high prevalence of multiple recorded features in the AHT groups.

Following the modelling described in the article, we gave detailed consideration to the predictive performance of our chosen model. Figure A3 shows the raw data again but this time colored according to the predicted probability of AHT (based on the final, reduced model). Because there was substantial missing information, these predictions were averaged across the 10 imputed data sets. A green color suggests AHT with a probability less than 20%; orange denotes ambiguous cases with predicted probabilities of AHT that are between 20% and 80%; and a red color corresponds to high probabilities (>80%) of AHT.

We see that our modelling is reasonably successful at discriminating between the 2 groups of interest. The predominance of green and orange in the left-hand plots and the frequency of red lines in the right-hand plots suggest that, on the whole, the model is fitting quite well. However, of (at least) equal importance is the degree to which the model can suggest incorrect etiologies; it is possible to have a rib fracture in the nAHT group; conversely, some children with no obvious injury (particularly evident in the Hobbs et al[3] study) can have AHT.

As mentioned in "Methods," we recognize the risk of overestimating the risk of abuse by relying solely on within-

Clinical and Radiographic Characteristics Associated With Abusive and Nonabusive Head Trauma: A Systematic Review

AUTHORS: Shalea J. Piteau, MD, MSc,[a] Michelle G.K. Ward, MD,[b] Nick J. Barrowman, PhD,[c] and Amy C. Plint, MD, MSc[b,d]

[a]*Department of Pediatrics, Queen's University, Kingston, Ontario, Canada; Departments of* [b]*Pediatrics and* [d]*Emergency Medicine, University of Ottawa, Ottawa, Ontario, Canada; and* [c]*Clinical Research Unit, Children's Hospital of Eastern Ontario Research Institute, Ottawa, Ontario, Canada*

KEY WORDS
abusive head trauma, head injury, children, abuse, systematic review

ABBREVIATIONS
AHT—abusive head trauma
CI—confidence interval
nAHT—nonabusive head trauma
OR—odds ratio

Drs Piteau and Plint conceived and designed the study; Drs Piteau and Ward screened literature search results and identified articles for retrieval, reviewed full papers for inclusion, and extracted data for meta-analysis; Dr Barrowman analyzed the data; and all authors interpreted the data, critically revised the draft, and gave final approval of the version to be published. Dr Piteau is the guarantor for the study.

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Address correspondence to Shalea Piteau, MD, MSc, Department of Pediatrics, Kingston General Hospital, Queen's University, 76 Stuart St, Kingston, Ontario, Canada K7L 2V7. E-mail: 7sjp2@queensu.ca

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BACKGROUND AND OBJECTIVE: To systematically review the literature to determine which clinical and radiographic characteristics are associated with abusive head trauma (AHT) and nonabusive head trauma (nAHT) in children.

METHODS: We searched MEDLINE, EMBASE, PubMed, conference proceedings, and reference lists to identify relevant studies. Two reviewers independently selected studies that compared clinical and/or radiographic characteristics including historical features, physical exam and imaging findings, and presenting signs or symptoms in hospitalized children ≤6 years old with AHT and nAHT.

RESULTS: Twenty-four studies were included. Meta-analysis was complicated by inconsistencies in the reporting of characteristics and high statistical heterogeneity. Notwithstanding these limitations, there were 19 clinical and radiographic variables that could be meta-analyzed and odds ratios were determined for each variable. In examining only studies deemed to be high quality, we found that subdural hemorrhage(s), cerebral ischemia, retinal hemorrhage(s), skull fracture(s) plus intracranial injury, metaphyseal fracture(s), long bone fracture(s), rib fracture(s), seizure(s), apnea, and no adequate history given were significantly associated with AHT. Epidural hemorrhage(s), scalp swelling, and isolated skull fracture(s) were significantly associated with nAHT. Subarachnoid hemorrhage(s), diffuse axonal injury, cerebral edema, head and neck bruising, any bruising, and vomiting were not significantly associated with either type of trauma.

CONCLUSIONS: Clinical and radiographic characteristics associated with AHT and nAHT were identified, despite limitations in the literature. This systematic review also highlights the need for consistent criteria in identifying and reporting clinical and radiographic characteristics associated with AHT and nAHT. *Pediatrics* 2012;130:1–9

EXHIBIT 10

Abusive head trauma (AHT) is the most common cause of traumatic death in children younger than 1 year[1] and is the most common cause of death due to child abuse.[1] Survivors of AHT are left with significant morbidity, with 45% having permanent neurologic sequelae compared with 5% of nonabuse cases.[2]

While the annual incidence of AHT is estimated at ~24 to 34 per 100 000 children younger than 1 year,[3–5] this is likely an underestimate because many cases are not brought to medical attention and others are not recognized as abuse. AHT in children can be difficult to diagnose because the symptoms are nonspecific, the history is often inaccurate, and the victim is often preverbal. In 1 study, physicians missed the diagnosis on initial presentation in one-third of cases and this resulted in repeated trauma, increased morbidity, and death.[6] As there is no gold standard diagnostic test for child abuse, the diagnosis relies on clinical and radiographic features as well as supporting social and child welfare information. AHT may be recognized in part by historical features (eg, no history of trauma, a low-impact trauma history, a changing or inconsistent history, or a history incompatible with the injuries or incompatible with the child's developmental stage), certain types of intracranial injuries (eg, diffuse subdural hemorrhages), and associated injuries (eg, retinal hemorrhages, metaphyseal fractures, and patterned bruising).[6–12]

We planned a systematic review to summarize the best available evidence comparing the clinical and radiographic characteristics of AHT and nonabusive head trauma (nAHT) in hospitalized children. By identifying these features we hope to help front-line clinicians in the difficult task of distinguishing between AHT and nAHT trauma. Such information may improve outcomes for the victims of abuse and their siblings through the early identification of child abuse.

METHODS

We followed a protocol for this review, in which all eligibility criteria, outcomes, and analyses were specified a priori (available from authors on request).

Search Strategy

The main search strategy (Supplemental Information 1) was developed by an experienced librarian and included MEDLINE (1950 to November, week 2, 2010) and EMBASE (1980 to week 45, 2010); both searches were executed through the Ovid interface. Other search strategies included searches of relevant conference proceedings (American Academy of Pediatrics, Canadian Pediatric Society, Pediatric Academic Societies, Society for Academic Emergency Medicine, Canadian Association of Emergency Physicians, American College of Emergency Physicians) for 1999–2009, contact with the primary author of relevant studies, and review of the reference lists of included studies and relevant reviews. There was no restriction on year, language, or publication status. Studies identified as eligible were used as seed articles for a PubMed-related article search conducted on November 18, 2010 (Supplemental Information 1). The first 50 related articles (ranked by relevance) not previously identified by the subject search were retained and screened.

Study Selection

Studies were included if they compared historical features, physical examination or imaging findings, or presenting signs or symptoms of AHT and nAHT in hospitalized children ≤6 years old. Head trauma was defined as skull fracture on computed tomography, MRI, or plain radiography or at least 1 of the following on computed tomography or MRI: subdural hemorrhage, epidural hemorrhage, subarachnoid hemorrhage, intraparenchymal hemorrhage, brain contusion, diffuse axonal injury, cerebral edema, and hydrocephalus. Exclusion criteria included duplicate publications, review articles, opinion pieces, consensus statements, studies evaluating abusive injury exclusive of head trauma, noncomparative studies evaluating AHT, studies evaluating nonhospitalized children, studies examining only clinical outcomes, management or postmortem investigations, or studies judged to be methodologically weak owing to significant bias. Studies were judged to have significant bias if the same variables were applied in both the diagnosis and the characterization of abuse; for example, the pattern of head trauma was the primary factor used to define abuse and then was analyzed as a variable of abuse.

Two reviewers (S.J.P. and M.G.K.W.) independently reviewed the titles and abstracts generated by the search to identify potentially relevant articles. The 2 reviewers then independently assessed the full article by using a standardized form with eligibility criteria established a priori. Disagreements were resolved by consensus or by a third reviewer (A.C.P.) as necessary. The third reviewer (A.C.P.) additionally reviewed the eligibility of all studies deemed eligible by both reviewers (S.J.P. and M.G.K.W.), and disagreements were resolved by consensus. Authors were asked for clarification when it was unclear whether all children in the study met our definition of head trauma.

Assessment of Methodological Quality

Two independent reviewers (S.J.P. and M.G.K.W.) assessed study quality and disagreements were resolved by consensus or by the third reviewer (A.C.P.). Reviewers were not blinded to author or other publication details. Study quality was assessed according to the ability to accurately categorize the etiology of head trauma as abusive or nonabusive based on a priori criteria. In the absence of a gold standard diagnostic test for child abuse, the quality of the a priori criteria was assessed based on its ability to be free of bias and circular logic to arrive at a diagnosis of child abuse. Specifically, we applied the previously published 5-point scale, "ranking criteria for the definition of abuse," to categorize studies as high (score of 1 or 2) or low quality (score of 3–5) (Table 1).[7,13–17]

Data Extraction

Two reviewers (S.J.P. and M.G.K.W.) independently extracted data to a standardized form and entered it into Microsoft Excel (Microsoft Corp, Redmond, WA). Only consensus data were used in the review. Extracted data included: (1) characteristics of the study (design, year of publication, etc), (2) description of study populations (age, number of participants, etc), (3) standards used to determine abusive and nonabusive nature of injury, (4) type of head trauma (subdural hemorrhage, epidural hemorrhage, subarachnoid hemorrhage, intraparenchymal hemorrhage, skull fracture plus intracranial injury, isolated skull fracture, cerebral ischemia, cerebral edema, and diffuse axonal injury), and (5) other clinical variables. Other clinical variables included historical features (no adequate history, history of low-impact trauma, inconsistent history, the injury was blamed on home resuscitative efforts or on siblings, or delay in seeking care after an injury), presenting signs and symptoms (seizures at or within 24 hours of presentation, vomiting, apnea at presentation, lethargy, abnormal neurologic status such as cranial nerve palsy or paresis), other physical findings (such as scalp swelling, retinal hemorrhage(s), rib fracture(s), long bone fracture(s), metaphyseal fracture(s), any bruising, head and neck bruising, other organ injury, cardio-respiratory compromise, and Glasgow Coma Scale score on admission), and operative interventions. Where studies included a category of head trauma of indeterminate etiology, these cases were excluded. Where studies included children with injuries that did not meet our definition of head trauma along with those that did meet our definition, we extracted data for eligible children only, and if data could not be extracted for only these children, the study was excluded.

TABLE 1 Ranking of Criteria Used to Define Abuse[17]

Ranking	Criteria Used to Define Abuse
1	Abuse confirmed at case conference or civil, family, or criminal court proceedings or admitted by perpetrator
2	Abuse confirmed by stated criteria including multidisciplinary assessment
3	Diagnosis of abuse defined by stated criteria
4	Abuse stated as occurring, but no supporting detail given as to how it was determined
5	Abuse stated simply as "suspected"; no details on whether it was confirmed

Data Analysis

Data were expressed as pooled odds ratios (ORs) with 95% confidence intervals (CIs). For ORs >1, the variable is associated with AHT and for ORs <1, the variable is associated with nAHT. Data were pooled by using the Dersimonian-Laird random effect method by using the empirical OR with inverse variance weighting except when at least 1 cell in a 2×2 table was 0, in which case 0.5 was added to all the cells in the table before computing the empirical OR and variance. Heterogeneity was examined by using the I^2 statistic. Significance was determined by a P value $<.05$. Analyses were conducted by using custom programs written by using R version 2.10.[18]

Due to the inconsistency in the specific clinical and radiographic variables reported between studies and in the manner of reporting these variables between studies, we limited our meta-analysis to 19 variables.[19] These variables were chosen because they were considered clinically relevant, were reported in at least 2 studies, and could be dichotomized into present and absent. These variables were subdural hemorrhage(s), epidural hemorrhage(s), subarachnoid hemorrhage(s), retinal hemorrhage(s), skull fracture(s) co-occurring with intracranial injuries, isolated skull fracture(s), cerebral ischemia, cerebral edema, diffuse axonal injury, scalp swelling (defined as either soft tissue swelling or subgaleal hematoma), metaphyseal fracture(s), long bone fracture(s), rib fracture(s), any bruising, head and neck bruising (distinguished from other signs of external head trauma), apnea at presentation, seizure(s) at presentation or within 24 hours of presentation, vomiting, and no adequate history (such as no history of trauma).

Clinical and radiographic variables were analyzed in 2 separate groups: (1) all studies that reported a given variable and (2) high-quality studies only (rank 1 or 2). Given that the most common age group affected by abuse is children <3 years old, we had a priori planned a subgroup analysis by age (children ≤ 3 and ≥ 3 years of age). This analysis was, however, not possible given the manner in which data were presented within included studies. When possible, the mean age of children in the 2 cohorts was compared. A Wilcoxon signed rank test tested the hypothesis that the abused children were younger than those not abused, accounting for the pairing within studies of children identified as abused and not abused. We expanded our a priori analysis plan by examining this hypothesis for studies that included only children up to 24 and 36 months old, respectively.

During data extraction, it became clear that studies could be grouped into 3 categories by the manner in which they reported clinical and radiographic characteristics, and this resulted in the need to expand our a priori analysis plan to consider missing data. For example, in reporting retinal hemorrhages, some studies clearly stated that all children in each cohort had fundoscopic exams,[15,20,21] some studies reported this variable for only the subset of children in each cohort who had fundoscopic exams,[10,22–24] while others (mainly retrospective cohort studies) reported the number of retinal hemorhages as if all children in each cohort had a fundoscopic exam, but in detailed review, it was clear that this was highly unlikely.[2,11,24–28] We determined that of the 19 variables reported in this review, retinal hemorrhages and rib, long bone, and metaphyseal fractures were most likely to be affected by informative missing data,[29] and these variables required a sensitivity analysis. These variables were chosen as specific investigations (skeletal survey or bone scan) or a specialized physical exam skill (fundoscopic examination) would be needed to rule out these findings and that these investigations and exams were much more likely to be completed for children in whom abusive trauma was suspected. For the sensitivity analysis, we considered all studies, as well as high-quality studies alone, and examined (1) those studies in which all children had the relevant investigation or examination and (2) those studies in which either all children had the relevant investigation or examination done and/or studies in which the proportion of the children having the investigation or examination done was clearly stated. We chose not to impute missing values (eg, we did not assume that unexamined children in the nonabuse cohort had the finding typically associated with abuse) given that in many of the retrospective studies we could not determine how many children had the investigation or examination done.

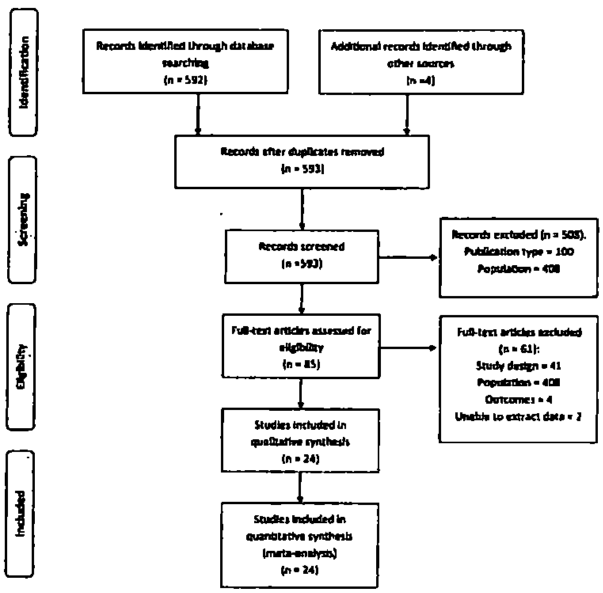

FIGURE 1
Study flow.

RESULTS

Description of Studies

Figure 1 shows the flow of studies through the selection process. Five hundred ninety-six studies were identified; 85 were deemed possibly relevant on screening and full text publications were obtained, and of these, 24 met inclusion criteria (Fig 1).[2,8–12,15–21,26–28,30–38] Two included studies[37,38] were identified by reviewing reference lists of relevant reviews.[17] One study[2] appeared to report data for 40 children that were also included in a second[36] publication; only variables with no overlap were included. Table 2 describes the included studies. Twenty-one studies were deemed high quality (rank 1 or 2), and 3 studies were deemed low quality (rank 3, 4, or 5).

Clinical and Radiographic Variables

There was a wide range of clinical content in the included studies with 82 variables reported overall. Studies reported 1 to 35 variables, with a mean of 15 variables. Subdural hemorrhage was the most commonly reported variable, being reported in 15 studies. The following characteristics were reported in at least 10 studies: retinal hemorrhages, subarachnoid hemorrhage, epidural hemorrhage, seizure, and the injury mechanisms of motor vehicle accidents, falls, and admitted/witnessed assault.

Inconsistency in the definition and reporting of clinical and radiographic variables as well as high statistical heterogeneity presented challenges to meta-analysis. For example, subdural hemorrhage was reported as total, unilateral, bilateral, mixed density, interhemispheric, or subdural hemorrhage plus other injuries.[2,8–12,16,21–23,25–28,31–35,37,38] Skull fracture was reported as simple, complex, multiple, bilateral, depressed, nonparietal, diastatic fractures >3 mm, or fractures crossing suture lines.[8,9,11,12,15,22,24–26,28,31–38] By selecting variables that were clinically relevant, reported in at least 2 studies, and that could be dichotomized into present or absent, we were able to individually meta-analyze 19 clinical and radiographic variables. Table 3 provides a summary of the results of the meta-analysis for each

TABLE 2 Characteristics of Included Studies ($N = 24$)

First Author	Year of Publication	Country	Age, mo	Sample Size[a]	Type of Study	Study Quality Rank
Bechtel[9]	2004	US	0–24	82	Prospective	1 and 2
Billmire[24]	1985	US	0–12	70	Retrospective	1 and 2
Datta[35]	2005	UK	0–24	52	Retrospective	1 and 2
Duhaime[12]	1992	US	0–24	68	Prospective	1 and 2
Ettaro[23]	2004	US	0–36	377	Retrospective	1 and 2
Ewing-Cobbs[2]	1998	US	0–72	40	Prospective	1 and 2
Ewing-Cobbs[33]	2000	US	0–72	60	Prospective	1 and 2
Feldman[27]	2001	US	0–36	54	Prospective	1 and 2
Hettler[10]	2003	US	0–36	163	Retrospective	1 and 2
Hobbs[34]	1984	UK	0–24	89	Retrospective	1 and 2
Hoskote[37]	2002	UK	0–24	16	Retrospective	4
Hymel[8]	2007	US	0–36	41	Prospective	1 and 3
Ichord[31]	2007	US	0–36	52	Prospective	1 and 2
Keenan[36]	2004	US	0–24	152	Prospective	1 or 2
Kelly[21]	2004	New Zealand	0–24	64	Retrospective	4
McKinney[30]	2008	US	0–36	32	Retrospective	1 and 2
Myhre[32]	2007	Norway	0–36	52	Retrospective	2
Reece[11]	2000	US	0–36	179	Retrospective	1 and 2
Ruppel[18]	2001	US	0–72	13	Prospective	5
Shugerman[27]	1996	US	0–36	87	Retrospective	2
Tzioumi[23]	1998	Australia	0–24	36	Retrospective	1 and 2
Vavilala[25]	2007	US	0–60	10	Prospective	1 and 2
Vinchon[21]	2005	France	0–24	150	Prospective	1 and 2
Vinchon[15]	2009	France	0–24	84	Prospective	1

High-quality studies = 1–2; low-quality studies = 3–6 as based on the criteria used to determine the etiology of head injury.
[a] Sample size reported is that of children in each included study that met our definition of head trauma and for whom data relevant to the review could be extracted. The Duhaime,[12] McKinney,[30] Reece,[11] and Billmire[26] studies included children who did not meet our definition of head trauma and the sample size reported is for only those children that meet our definition.

individual characteristic, and Supplemental Information 2 shows forest plots for these characteristics. The degree of statistical heterogeneity varied markedly between the variables and ranged from low to high (Table 3). Of note, a high OR does not directly correlate with a high positive predictive value for a given feature since this depends on the prevalence of abuse in the individual studies.

Variables Associated With AHT

When examining all studies, subdural hemorrhage(s), cerebral ischemia, cerebral edema, retinal hemorrhage(s), skull fracture(s) co-occurring with intracranial injury, metaphyseal fracture(s), long bone fractures(s), rib fracture(s), any bruising, seizure(s) at or within 24 hours of presentation, apnea at presentation, and no adequate history were individually significantly associated with AHT (Table 3). Including only high-quality studies, cerebral edema and any bruising were no longer significantly associated with AHT.

Variables Associated With nAHT

When examining all studies, epidural hemorrhage(s), isolated skull fracture (s), scalp swelling, and head and neck bruising were significantly associated with nAHT (Table 3). When analysis was limited to high-quality studies, head and neck bruising was no longer significantly associated with nAHT.

Variables Not Significantly Associated With AHT or nAHT

When examining all studies and only high-quality studies, subarachnoid hemorrhage(s), diffuse axonal injury, and vomiting were not significantly associated with AHT or nAHT. Head and neck bruising, any bruising, and cerebral edema were not significantly associated with AHT or nAHT in only high-quality studies.

Sensitivity Analysis

We performed a sensitivity analysis for 4 variables: retinal hemorrhages and long bone, metaphyseal, and rib fractures (Supplemental Information 3 and 4). While retinal hemorrhages remained significantly associated with AHT, long bone and rib fractures did not, and there were no studies examining metaphyseal fractures to include in the analysis. The small number of studies also limited the analyses for rib and long bone fractures.

Age

Fourteen studies contributed data to the age analysis. Seven studies did not report the mean age of the individual cohorts,[12,25,26,30,32,34,38] 3 studies reported the mean age for both cohorts but we extracted data only for children meeting our eligibility criteria and we could not extract the corresponding age data,[11,12,24] and 1 article reported mean age[35] but this overlapped with data reported in another article.[2] In all except 1 study,[8] the mean age of children identified as abused was lower than the mean age of children identified as not abused ($P < .0004$). The range of mean ages reported was 2.1 to 22 months in the abuse cohort and 5.64 to 43 months in the nonabuse cohort. Among the abuse cohort, the mean age was <12 months in 12 (86%) studies and <6 months in 6 (43%) studies. Among the nonabuse cohort, the mean age was <12 months in 7 (50%) studies and <6 months in 1 (7%) study. When the analysis was restricted to the 11 studies that include children <36 months old and to the 6 studies that include children <24 months old, the mean age of the children identified as abused remained lower than the mean age of children identified as not abused ($P = .003$ and $P = .03$, respectively).

DISCUSSION

Principal Findings of the Review

Distinguishing between AHT and nAHT can be challenging. Negative consequences

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TABLE 3 Clinical and Radiographic Characteristics Associated With ABT and nABT

Characteristic	No. of Studies	OR (95% CI); OR >1 Favors Abuse; OR <1 Favors Nonabuse	I^2, %	P
Subdural hemorrhage(s)				
All studies	15	8.92 (6.77–11.74)	0	<.001
High-quality studies	14	8.80 (6.75–11.73)	0	<.001
Epidural hemorrhage(s)				
All studies	10	0.15 (0.08–0.29)	0	<.001
High-quality studies	9	0.13 (0.06–0.26)	0	<.001
Subarachnoid hemorrhage(s)				
All studies	13	1.42 (0.67–3.0)	78	.36
High-quality studies	11	1.31 (0.58–3.0)	80	.52
Cerebral edema				
All studies	9	2.17 (1.06–4.45)	54	.03
High-quality studies	7	2.05 (0.82–5.10)	65	.12
Cerebral ischemia				
High-quality studies	4	4.79 (1.84–2.46)	29	.001
Diffuse axonal injury				
All studies	6	1.47 (0.26–8.32)	61	.66
High-quality studies	4	0.83 (0.06–2.46)	72	.89
Skull fracture(s) + intracranial injury				
High-quality studies	4	7.76 (1.06–57.08)	88	.04
Isolated skull fracture(s)				
High-quality studies	2	0.01 (0.003–0.04)	0	<.001
Long bone fracture(s)				
All studies	8	4.234 (2.50–7.19)	0	<.001
High-quality studies	6	4.344 (2.52–7.49)	0	<.001
Metaphyseal fracture(s)				
All studies	2	11.76 (2.18–63.41)	0	.004
High-quality studies	1	15.06 (1.93–117.72)	Single estimate	<.001
Rib fracture(s)				
All studies	7	6.89 (4.04–7.19)	1	<.001
High-quality studies	6	8.84 (4.42–21.80)	0	<.001
Retinal hemorrhage(s)				
All studies	14	27.12 (15.70–46.84)	24	<.001
High-quality studies	12	28.24 (15.37–51.80)	35	<.001
Any bruising				
All studies	5	4.77 (1.62–14.05)	15	.005
High-quality studies	3	5.35 (0.91–31.41)	0	.06
Head and neck bruising				
All studies	3	0.42 (0.19–0.94)	26	.03
High-quality	2	0.65 (0.27–1.57)	0	.34
Scalp swelling				
High-quality studies	7	0.12 (0.05–0.32)	65	<.001
Apnea				
All studies	4	5.31 (2.34–12.05)	0	<.001
High-quality studies	3	4.89 (2.08–11.49)	0	<.001
Seizure(s)				
All studies	10	7.25 (3.04–17.27)	77	<.001
High-quality studies	9	11.24 (7.30–17.29)	0	<.001
Vomiting				
High-quality studies	3	0.89 (0.02–4.88)	83	.89
No adequate history				
All studies	9	48.94 (12.91–170.63)	69	<.001
High-quality studies	8	52.72 (12.79–217.33)	72	<.001

including re-injury and death have been shown to occur when the diagnosis has been missed.[6] There can also be negative consequences to the child and family with an incorrect diagnosis of abusive injury. For these reasons, accurate diagnoses must be made. From our systematic review we were able to highlight some clinical and radiographic characteristics associated with AHT and nAHT in children.

We found that each of subdural hemorrhage(s), cerebral ischemia, skull fracture(s) in conjunction with intracranial injury, retinal hemorrhage(s), long bone fracture(s), rib fracture(s), metaphyseal fracture(s), seizure(s) at presentation or within 24 hours, apnea at presentation, and no adequate history were individually significantly associated with AHT when only high-quality studies were meta-analyzed. Epidural hemorrhage(s), scalp swelling, and isolated skull fracture(s) were each significantly associated with nAHT when high-quality studies were meta-analyzed. Although we recognize that patterns of injuries are of key importance in making a diagnosis of abuse or nonabuse, limitations in the data, as well as the manner in which it was presented, prevented us from examining whether variables were associated with abuse when found in combination.

Comparison With Other Studies

In 1962, Kempe described "battered child syndrome" as the combination of subdural hemorrhage, skeletal injuries, and bruises.[39] Since then, many studies have supported the association of subdural hemorrhage with child abuse,[6,40,41] the association of epidural hemorrhage with nAHT, and the association of subarachnoid hemorrhage with both mechanisms of injury.[2,27,35] Our study provides evidence that a younger age is associated with AHT, and this may support the hypothesized temporal association between AHT and crying patterns in infants within the first 3 months of life.[42] This finding may also reflect the physiologic and anatomic differences in younger children that may alter the susceptibility to head injury with a given mechanism or force.[43–45]

Maguire et al[13] recently published a systematic review addressing a similar question as our review. Similar to our study, they found that apnea, retinal hemorrhage, rib fractures, long bone fractures, skull fractures associated with intracranial injury, and seizures were associated with abuse.[13] The review also found head and neck bruising was associated with nAHT. Some important differences exist, however, between these 2 reviews, including the definition of head trauma, articles included, characteristics analyzed, statistical methodology, and data representation. Specifically, our review included examination of historical features and the type of intracranial injury. We believe that the type of intracranial injury associated with injury mechanism is critical, as these may be the initial data available to front-line clinicians who must make a preliminary determination around injury mechanism. We included only studies of admitted patients to focus on more seriously injured patients, and we limited our study population to a younger age to focus on the primary age group at risk for AHT. We included more studies in our systematic review, although 3 studies in Maguire et al's 2009 review did not meet our inclusion criteria. We also chose to examine statistical heterogeneity between studies and to examine all studies, as well as only high-quality studies, to address the robustness of our data. Interestingly, when low-quality studies were excluded from analysis, only 3 variables were no longer significantly associated with either AHT or nAHT suggesting our conclusions are fairly robust. We dealt with informative missing data in a different manner. The Maguire et al 2009 review imputed informative missing data when only a proportion of the children in each cohort had the investigations. We chose not to use this approach for our sensitivity analysis since we could not determine the proportion of children in each cohort that had the given investigation or exam performed. The small number of studies accurately reporting missing data limited our sensitivity analysis.

Two other reviews of characteristics associated with AHT and nAHT have been recently published. One, by Kemp et al, reviewed neuroradiologic features in 21 studies of children hospitalized with head trauma. This study's finding that subdural hemorrhage and hypoxic ischemic injury were significantly associated with AHT[46] was in agreement with our results. The authors also found that cerebral edema was associated with AHT. The second study, a patient-level meta-analysis from 6 cohort studies, examined the positive predictive values of a combination of 6 clinical features (head and neck bruising, rib fracture, skull fractures, long bone fractures, retinal hemorrhage, seizures, and apnea) in conjunction with intracranial injury.[47] It reported that any combination of ≥ 3 features yielded a positive predictive value for AHT of 85%. Given the limitations of the articles included in our review, an analysis of combined features was not possible in our study.

Limitations of the Review

The meta-analysis for this review was made difficult by inconsistencies in the criteria used to determine the etiology for head trauma, inconsistencies in defining and reporting clinical and radiographic variables, and a moderate to high degree of statistical heterogeneity between studies. As there are no standardized criteria for the definition of abuse, most authors developed their own criteria, and many of these are fraught with circular reasoning. The diagnosis of AHT relies on historical features, clinical findings, and radiologic interpretations, and it is these same criteria that are used to categorize head trauma as abusive or nonabusive. We attempted to address this limitation by using a published scale to rank the quality of the criteria used for defining abuse[7,13,16,17] and examined our results for all eligible studies, as well as for those using high-quality criteria. However, for features that have been traditionally associated with abuse (such as subdural hemorrhage and retinal hemorrhage), this ranking scale does not compensate well for circularity and thus our results must be interpreted cautiously.

Significant variability in the definition and description of clinical and radiographic variables in the included studies made meta-analysis difficult. This variability highlights the need for the standardization of definitions and descriptions of characteristics to accurately compare data between studies or combine data for meta-analysis. Ultimately, such standardization could advance the development of accurate diagnostic criteria for AHT. The high degree of heterogeneity among studies may reflect this lack of standardization, somewhat weakening the clinical interpretability of the review findings.

All studies included in this review were, as expected, observational studies. Selection, informational, and confounding bias are limitations to these studies. The retrospective studies were limited with respect to reporting missing data. Many studies reported data around characteristics (eg, retinal hemorrhages) as if all children in both cohorts had undergone the necessary investigation or examination (eg, fundoscopy) to look for the characteristic in question; however, a detailed review of the data revealed this to be unlikely. While we performed a sensitivity analysis to examine this issue, our results need to be interpreted cautiously. The retrospective studies in this review were also limited by recall bias; for example, the low-quality studies relied on medical record coding for the diagnosis of abuse, without reviewing supporting detail as to how abuse was determined. By limiting the review to studies of admitted children, only serious head injuries would have been included, but children who died before presentation would have been excluded. As well, children with less severe abusive injuries may be less likely to be brought to the hospital after an abusive event. Thus, a selection bias may occur for children with more severe injuries and symptoms in the abusive cohort being brought to the hospital compared with those in the nonabusive cohort.

CONCLUSIONS

Clinical and radiographic variables associated with AHT and nAHT in children were identified, despite important limitations in the existing literature. This systematic review highlights the need for consistent criteria in identifying and reporting the historical, physical, and radiographic variables associated with abusive and nonabusive head injuries. A future multicenter prospective trial evaluating AHT and nAHT by using standard criteria for both the categorization of etiology and for the examination and reporting of characteristics of head trauma would allow for more definitive findings.

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Nonaccidental head injury in children. Historical vignette.

Al-Holou WN[1], O'Hara EA, Cohen-Gadol AA, Maher CO.

Author information

Abstract

Our current understanding of nonaccidental head injury in children is the result of decades of effort and the tireless work of numerous physicians. In 1860 Auguste Ambroise Tardieu, a French forensics expert, recognized important patterns of injury in children and identified nonaccidental trauma as the cause of these injuries. His work was ignored. In the years that followed, physicians continued to report these patterns of injury but were unable to identify the etiology. A fundamental misunderstanding of the usual cause of subdural hematoma (SDH) contributed to the confusion at that time. Early in the 20th century, neurosurgeons such as Wilfred Trotter recognized that SDHs were traumatic in origin. However, even Trotter's efforts to expose faults in the theories that SDHs primarily resulted from inflammatory or infectious processes were not accepted immediately. Eventually, the pattern of injuries in children was again recognized both by neurosurgeons, who began to identify an association between trauma-induced SDHs and retinal hemorrhages, and by radiologists, who began to note SDHs in conjunction with osseous lesions. Not until the 1950s and 1960s, however, did physicians begin to routinely identify nonaccidental trauma as the cause of these injuries. Following the recognition of child abuse, a pattern of injuries in conjunction with shaking was identified and is currently known as shaken baby syndrome. Since its identification, our understanding of this syndrome has been modified as a result of new medical research, legal challenges, and popular media forces.

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Variation in the Diagnosis of Child Abuse in Severely Injured Infants

Matthew Trokel, Anthony Waddimba, John Griffith and Robert Sege

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EXHIBIT 12

ARTICLE

Variation in the Diagnosis of Child Abuse in Severely Injured Infants

Matthew Trokel, MD[a], Anthony Waddimba, MB, ChB[b], John Griffith, PhD[c], Robert Sege, MD, PhD[d]

[a]Department of Pediatrics, Boston Medical Center, Boston, Massachusetts; [b]Department of Public Health, Boston University, Boston, Massachusetts; [c]Biostatistics Research Center and [d]Pediatric and Adolescent Health Research Center, Floating Hospital for Children, Tufts–New England Medical Center, Boston, Massachusetts

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ABSTRACT

OBJECTIVE. Diagnosis of child abuse is difficult and may reflect patient, practitioner, and system factors. Previous studies have demonstrated potential lethal consequences if cases of abuse are missed and suggested a role for continuing medical education in improving the accuracy of diagnosis of suspected abuse. Although the majority of injured American children are treated at general hospitals, most published studies of severe injury resulting from child abuse have been conducted at children's hospitals. The objective of this study was to evaluate the role of hospital type in observed variations in the frequency of diagnosis of child physical abuse among children with high-risk injuries.

METHODS. Hospital discharge data were evaluated, and adjusted rates of abuse diagnosis were reported according to hospital type. A regression model estimated the number of cases of abuse that would have been diagnosed if all hospitals identified abuse as frequently as observed at pediatric specialty hospitals. This study consisted of children who were <1 year old and admitted to US hospitals in 1997 for treatment of traumatic brain injury or femur fracture, excluding penetrating trauma or motor-vehicle–related injury. A total of 2253 weighted cases were analyzed.

RESULTS. The proportion of patients with a medical diagnosis of child abuse varied widely between hospital types: 29% of the cases were diagnosed as abuse at children's hospitals compared with 13% at general hospitals. An estimated 178 infants (39% of total) with these specific injuries would have been identified as abused had they been treated at children's rather than general hospitals.

CONCLUSIONS. Hospital type was associated with large variations in the frequency of diagnosis of child abuse. This variation was not related to observed differences in the patients or their injuries and may result from systematic underdiagnosis in general hospitals. This result has implications for quality-improvement programs at general hospitals, where the majority of injured children in the United States receive emergent medical care.

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Key Words

medical error, hospital discharge data, child abuse, femur fracture, traumatic brain injury, medical error

Abbreviations

TBI—traumatic brain injury
HCUP—Healthcare Cost and Utilization Project
ICD-9-CM—*International Classification of Diseases, Ninth Revision, Clinical Modification*
NACHRI—National Association of Children's Hospitals and Related Institutions
ISS—injury severity score
CI—confidence interval
OR—odds ratio

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Address correspondence to Robert Sege, MD, PhD, NEMC, Box 351, 750 Washington St, Boston, MA 02111. E-mail: rsege@tufts-nemc.org

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WITH NEARLY 3 million referrals to child protective services and >900 000 children found to be victims of child maltreatment in the United States in 2001, abuse continues to have a large impact on the health of children. Approximately 1300 children died as a result of abuse in 2001.[1] Unfortunately, the evaluation and diagnosis of child abuse remains uneven.

Although there is no highly specific test or physical finding that is pathognomonic for child abuse, in premobile children, 2 injuries, femur fracture and traumatic brain injury (TBI), are considered highly suspicious indicators of abuse etiology.[2-4] Previous studies have found that the diagnosis of abuse has been missed in a substantial portion of abused infants with high-risk injuries.[5,6]

The process of suspecting and reporting child abuse is complex; it depends on both physician and patient factors. A survey-based study identified increased suspicion of abuse to be associated with younger children, more severely injured children, single-parent families, and poorly educated mothers.[7] The physician's age, postgraduate education, and tolerance for discipline have been shown to affect suspecting and reporting abuse.[8,9] Two recent retrospective chart reviews from single institutions found large variations in the suspicion and diagnosis of child abuse in hospitalized children with head trauma and fractures based on patient factors, including race, age, and injury severity.[10,11] These studies were conducted at children's specialty hospitals. We are not aware of any published study that evaluated the variation of diagnosis of child abuse between different hospital types across the United States.

The study presented in this report used a large national database of hospital discharge data to evaluate the contribution of hospital type to the variation in diagnosis of child abuse. The investigation focused on patients with injuries frequently caused by child abuse, infants with either femur fracture or TBI that did not result from either penetrating trauma or motor-vehicle accidents. Discharge data concerning children hospitalized in the United States were obtained from the Healthcare Cost and Utilization Project (HCUP) Kids' Inpatient Database 1997, published by the Agency for Healthcare Research and Quality.[12]

METHODS

The Kids' Inpatient Database 1997 was developed as part of the Healthcare Cost and Utilization Project (HCUP) to facilitate the study of a wide range of pediatric disorders.[11] The data set includes information concerning aspects of hospital care and patient outcomes of children ≤18 years old from a sample of hospitals across the United States in 1997. Pediatric discharges were sampled from the following 22 states: Arizona, California, Colorado, Connecticut, Florida, Georgia, Hawaii, Iowa, Illinois, Kansas, Maryland, Massachusetts, Missouri, New Jersey, New York, Oregon, Pennsylvania, South Carolina, Tennessee, Utah, Washington, and Wisconsin.

The sample of discharge information was collected from 2521 hospitals and included 1.9 million discharge records. These hospitals included general community hospitals, academic medical centers, and pediatric hospitals. A systematic random sample was drawn from these hospitals consisting of 10% of births and 80% of other pediatric discharges. Once weighted to account for sampling based on HCUP methodology,[12] the national estimates represented >6.5 million discharges. Patient-level data (age, gender, insurance status, diagnosis, and external cause of injury codes based on *International Classification of Diseases, Ninth Revision, Clinical Modification* [ICD-9-CM] codes) and hospital-level factors (teaching status, National Association of Children's Hospitals and Related Institutions [NACHRI] hospital type, and hospital bed size) were analyzed in this study.

Subjects

From the original data set, we extracted 2253 weighted cases of patients <1 year old with TBI and femur fracture (ICD-9-CM codes: 820.00–821.39) (Fig 1). TBI included all intracranial injury (ICD-9-CM codes: 800.10–800.49, 800.60–800.99, 801.10–801.49, 801.60–801.99, 803.10–803.49, 803.60–803.99, 804.10–804.49, 804.60–804.99, 851.00–854.19, and 950.00–953.9). Children were excluded if they were not admitted through the emergency department (to avoid counting hospital transfers or referrals for specialty care); if they were injured by a motor vehicle, a gunshot, or a knife stabbing; if they did not have any E code assigned; or if they did not have sufficient data available to calculate an injury severity score (ISS).

Measures

Patients were grouped into 3 mutually exclusive groups on the basis of the type of hospital that provided care for

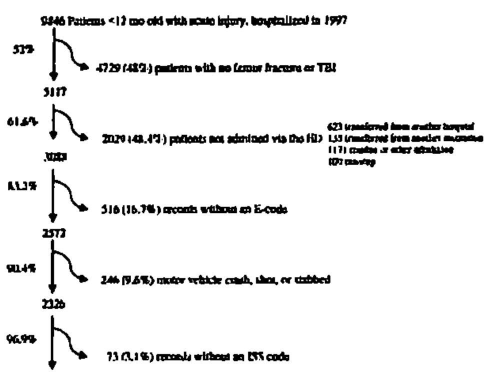

FIGURE 1
Patient inclusion/exclusion flowchart.

the patient: (1) a general hospital; (2) a general hospital with a children's unit; or (3) a children's hospital. The hospital grouping was assigned by the NACHRI.

The analyzed patient-level factors included age, gender, insurance status, injury type, ISS, mechanism of injury, and disposition. Insurance status was categorized as government assistance (Medicare, Medicaid, self-pay, CHAMPUS, and other government insurance) and all other insurers (BlueCross, commercial, preferred provider organizations, health maintenance organizations, and prepaid health plans). Injury type was initially divided into femur fracture and TBI. TBI was further divided into 3 mutually exclusive categories based on the presence or absence of skull fracture: subdural or epidural hemorrhage with skull fracture (ICD-9-CM codes: 800.20–800.30, 800.70–800.80, and 852.20–852.40), subdural hemorrhage without skull fracture (ICD-9-CM codes: 852.20–852.40), and all other intracranial injury. The ISS was calculated from the ICD-9-CM codes by using ICDMAP software (Tri-analytics, Inc, Bel Air, MD). Mechanism of injury was based on ICD-9 E codes and divided into 4 mutually exclusive groups: child abuse (E codes: 904.0–904.9 and 967.0–967.9); fall (E codes: 880.0–888.9 and 987.0–987.9); struck–not child abuse (E codes: 916.0–921.9, 960.0, and 968.1–968.2); and other. Disposition was divided into routine discharge, discharge to another health care facility, or other. Race was not designated in nearly one third of the cohort and, therefore, was not used in the analysis.

Hospital-level factors that were analyzed included teaching status, urban/rural status, and hospital bed size. Designations for teaching hospitals and urban status were derived from the American Hospital Association Annual Survey of Hospitals by the HCUP. Any hospital with an American Medical Association–approved residency program was categorized as a teaching institution; hospitals in a metropolitan statistical area were considered urban. Hospital bed size was grouped by the Agency for Healthcare Research and Policy into 3 groups nested within teaching status and hospital location (small, medium, and large) as described in the technical documentation available from the agency.

Data Management and Analysis

The cohort was divided according to hospital type. The rates of child abuse diagnosis for all injury types were reported within each group. Child abuse diagnosis rates were compared on the basis of specific injury types. Logistic-regression modeling was used to further evaluate the relationship of the diagnosis of child abuse to hospital type. Child abuse was the response variable, and hospital type was the explanatory variable. An adjusted model was then created, adjusting for both patient- and hospital-level factors including age, gender, insurance type, ISS, injury type, hospital bed size, and hospital teaching status. Injury type was classified into 4 mutually exclusive variables: subdural or epidural hemorrhage without skull fracture; subdural or epidural hemorrhage with skull fracture; femur fracture without any subdural/epidural hemorrhage; and the reference group, which included patients with all other intracranial injuries without femur fractures. The residuals for the 2 continuous variables, age and ISS, were assessed for linearity. Age was found to have an improved fit with a quadratic term. The disposition variable was added to the adjusted model to assess for bias in transfer at time of discharge.

To calculate the number of potential child abuse cases missed in general hospitals, a logistic-regression model was constructed in which child abuse was the response variable and age, gender, ISS, insurance status, and injury types were explanatory. No hospital-specific factors were included in the model. This model was applied only to the reference group: patients treated at children's hospitals. A regression equation was created by using the coefficients produced from this reference model. When applied to the remaining cohort, the equation generated the probability of being diagnosed with child abuse while adjusting for the previously mentioned patient factors based on the performance of physicians at children's hospitals. The actual child abuse status was then subtracted from the predicted status to calculate the discrepancy for each patient in both general hospitals and children's units in general hospitals. The mean discrepancy rates with 95% confidence intervals (CIs) were reported for children seen at general hospitals with and without children's units. The total number of patients seen at each type of hospital was multiplied by these mean discrepancy proportions to yield the estimated number of children who would have been categorized as abused had they been cared for in a children's hospital.

RESULTS

The largest group of patients (49%) was admitted to general hospitals, one fourth was admitted to general hospitals with children's units, and the remaining one fourth was admitted to a children's hospital (Table 1). Children who were treated at children's hospitals tended to be younger, more severely injured, and more likely to have private health insurance than those cared for at general hospitals. General hospitals cared for a smaller proportion of children with TBI but a greater proportion of femur fractures than the other 2 hospital types.

The rate of identification of child abuse diagnosis varied widely between the hospital types. Child abuse was identified more often when there was more pediatric specialty care available. Children's hospitals had the highest rates of identified child abuse, with the diagnosis being made more than twice as often as in general hospitals (29% vs 13%; $P < .05$). General hospitals with a children's unit had more abuse identified than general hospitals without a children's unit but less than a chil-

TABLE 1 Patient and Hospital Characteristics According to Hospital Type

	General Hospital	Children's Unit in General Hospital	Children's Hospital
Total, *N* (%)	1086 (100)	589 (100)	578 (100)
Patient and injury characteristics			
Male	617 (57)	311 (53)	365 (63)
Age, median (IQR), d[a]	172 (78–260)	137 (61–234)	153 (76–247)
Government insurance	682 (63)	327 (55)	285 (49)
Injury severity, median (IQR)[a]	4.0 (4–9)	9.0 (4–16)	9.0 (4–13)
Injury type			
Sub-/epidural hemorrhage with skull fracture	75 (7)	81 (14)	71 (12)
Subdural hemorrhage without skull fracture	71 (7)	90 (15)	92 (16)
Other intracranial injury	623 (57)	339 (58)	309 (53)
Femur fracture	315 (29)	78 (13)	111 (19)
Mechanism of injury			
Child abuse	143 (13)	113 (19)	170 (29)
Fall	744 (69)	392 (67)	344 (60)
Struck, not child abuse	144 (13)	41 (7)	41 (7)
Other	53 (5)	43 (7)	23 (4)
Hospital characteristics			
Urban	946 (87)	587 (99)	578 (100)
Teaching	553 (51)	486 (83)	422 (73)
Bed size			
Small	209 (19)	3 (0.5)	311 (54)
Medium	401 (37)	157 (27)	141 (24)
Large	476 (44)	429 (73)	126 (22)

[a] IQR indicates interquartile range (25th–75th percentile).

dren's hospital. Falls, as a proffered cause of injury, remained fairly constant between groups, ranging from 60% in children's hospitals to 69% in general hospitals ($P < .05$).

Nearly all the hospitals in this cohort were in urban settings. Half of the general hospitals were teaching facilities, as opposed to nearly three fourths of the children's hospitals. Most hospitals were distributed fairly evenly across the United States. The majority of general hospitals were reported as large, whereas more than half of the children's hospitals were categorized as small.

The proportion of children who were diagnosed with child abuse varied between hospital types after stratifying by injury (Table 2). Patients with all types of intracranial lesions were identified as victims of child abuse least frequently in general hospitals and most frequently in children's hospitals. This pattern persisted with lesions more suspicious of abuse. In children with a subdural or epidural hemorrhage with skull fractures, ~10% were identified in general hospitals as abused, and nearly one third were identified in children's hospitals as abused. Infants with subdural hemorrhage without skull fractures were most likely to be diagnosed as having been abused. These included nearly one half of the infants with this condition admitted to general hospitals and three fourths of infants admitted to children's hospitals. Children with femur fractures were identified as abuse victims in a similar pattern as intracranial injury, least frequently in general hospitals and most frequently in children's hospitals. In all injury groups, general hospitals with a children's unit identified abuse more frequently than general hospitals without a children's unit but less frequently than children's hospitals.

A reduced regression model showed that hospital type was associated with increased odds of receiving an abuse diagnosis. Infants admitted to children's hospitals had a 2.7-fold (95% CI: 2.1 to 3.5) increased odds of being diagnosed with child abuse compared with infants admitted to general hospitals. This difference persisted after controlling for patient and hospital factors (odds

TABLE 2 Proportion of Infants Diagnosed With Child Abuse for Specific Injuries According to Hospital Type

	General Hospital		Children's Hospital	
	Total Cases, *N*	Child Abuse, *n* (%)	Total Cases, *N*	Child Abuse, *n* (%)
Sub-/epidural with skull fracture	75	8 (11)	71	25 (35)
Subdural without fracture	71	31 (44)	92	67 (73)
Other intracranial injury	623	45 (7)	309	41 (13)
Femur fracture	315	57 (18)	111	41 (37)

TABLE 3 National Estimates for Child Abuse According to Hospital Type

	General Hospital	Children's Unit in General Hospital	Children's Hospital
Total, *N*	1086	589	578
Child abuse, *n*	143	113	170
No. "missed"	114 (84–143)	64 (38–89)	Reference
Total predicted abuse, *n* (95% CI)	257 (227 to 286)	177 (151 to 202)	170

ratio [OR]: 2.7; 95% CI: 2.0 to 3.6). Hospital teaching status did not significantly confound or interact with hospital type. Infants admitted to general hospitals with a children's unit had increased odds (OR: 1.6; 95% CI: 1.2 to 2.2) of receiving a child abuse diagnosis compared with those at general hospitals without a children's unit, but this trend was no longer significant after adjustment (OR: 1.3; 95% CI: 0.9 to 1.7). When we controlled for patients who were discharged from the hospital to another facility, we found that the OR comparing child abuse diagnosis in children's hospitals with that observed in general hospitals increased slightly, from 2.7 to 3.2. There was no change in ORs for general hospitals with a children's unit when controlling for transferred patients.

An estimate was obtained of the number of children who would have been diagnosed as abused if the rate of diagnosis of abuse observed seen at children's hospitals was applied to all hospitals (Table 3). The *c* statistic of the reference model of children diagnosed with abuse at children's hospitals was 0.851. The mean discrepancy for children not diagnosed as abused at general hospitals without children's units was −10.5% (95% CI: −13.2 to −7.8), showing that an additional 10.5% (114) of these children had characteristics similar to the abused children seen at children's hospitals. The total number of children diagnosed with abuse at general hospitals without a children's unit would be 257 (23.6%). General hospitals with a children's unit had a mean discrepancy rate of −10.8% (95% CI: −15.1 to −6.5), yielding an additional 64 abuse cases. The total number of missed cases of abuse with these 2 specific injury types was 178, which corresponds to nearly 40% of all total cases of abuse among infants under 1 year of age with these 2 specific injury types.

DISCUSSION

In this population-based study of 2253 weighted cases of infants who were hospitalized with either a femur fracture or TBI, we explored the proportion of patients diagnosed with child abuse among different hospital types. Infants seen in children's hospitals tended to be younger, were more severely injured, and were more likely to be covered by private insurance than children who were cared for at general hospitals. Infants admitted to general hospitals for severe injuries were diagnosed as abused only half as frequently as those admitted to children's specialty hospitals. This pattern persisted even for the injury most frequently diagnosed as abuse-related: subdural hemorrhage without skull fracture. Using the practice pattern of children's hospitals as the reference group, an additional 178 cases of abuse would have been diagnosed across the United States in 1997 in this cohort of severely injured infants with these 2 specific injury patterns.

Our study found a large variation in the frequency of diagnosis of abuse based on NACHRI hospital type. Hospitals that focused exclusively on pediatric patients were more likely to detect and report child abuse; this discrepancy was not explained by the patient's age, injury severity, or insurance status. This pattern was noted in all injury patterns evaluated. There are several potential explanations for this difference. It is possible that injured children were preferentially evaluated and admitted to children's hospitals simply because they were suspected to have been abused. Additionally, more severely injured children may be preferentially evaluated for abuse in children's hospitals because children's hospitals care for more severely injured children than general hospitals. ICD-9 E codes may not be an accurate method of determining population-based estimates for any diagnosis or condition. To help reduce these biases, we excluded all patients who were transferred between facilities, thereby reducing the possibility that more complex cases would be cared for at specialty centers. We also controlled for patients who were discharged from the hospital to another facility and did not find considerable changes in the ORs of the final model. Although children's hospitals cared for more severely injured children (as measured by the abbreviated injury score), the discrepancy in frequency of diagnosis of abuse remained after injury severity was accounted for in the statistical analysis. Nonchildren's hospitals had a lower rate of E coding and therefore had a higher proportion of cases excluded from the study.

The definitive diagnosis of child abuse is difficult to ascertain; this study used the clinical diagnosis of child abuse as defined by each participating institution. This clinical diagnosis may well be incorrect, and this study may be subject to misclassification bias in that nonabused patients may have been categorized as abused and truly abused patients may never have been considered to be abuse victims. Previous studies have identified a tendency to underdiagnose child abuse[3,4]; therefore, it may be reasonable to assume that more abused patients were misclassified as not abused than nonabused patients were labeled as abused. It is encouraging to note that previous studies suggest that the true rate of child abuse more closely approximates the rate detected at children's hospitals. Hettler and Greenes[13] found that 30% of children 0 to 3 years old with acute traumatic intracranial injury were victims of abuse. Reece and Sege[2] described 50% of subdural hemorrhages resulting from abuse, and Billmire and Myers[14] reported that 92% of intracranial bleeds without skull fractures in children under 1 year of age were the result of abuse. Femur fractures have also been described to be the result of abuse in rates similar to that of children's hospitals.[15,16] The most likely explanation for the observed difference in the likelihood of reaching a diagnosis of child abuse seems to be a systematic underdiagnosis of child abuse at general hospitals.

Several studies have highlighted the danger of missing an abusive etiology among injured children. Child abuse is often a chronic condition that recurs until the victim and perpetrator are no longer in contact. One of the late cardinal signs of abuse is multiple wounds or fractures in various stages of healing. Several case series of child abuse show that 18% to 80% of abused children show some signs of prior abuse.[3,17-19] These abused children most likely saw a physician at some point before receiving the diagnosis of abuse but were not diagnosed appropriately. Jenny et al[5] reported that 31% of abused patients admitted with acute head injuries had been recently evaluated by physicians for complaints consistent with acute head injury and were incorrectly diagnosed. The most common erroneous diagnoses were viral gastroenteritis followed by accidental head injury and ruled-out sepsis.

Dalton et al[6] reported that in a cohort of children <3 years old with femur fractures, 20% of the fractures resulting from abuse were diagnosed at a later date, well after the initial hospitalization. Other case reports of misdiagnosed child abuse have been published.[20,21]

Although the process of identifying and reporting suspected child abuse is complex, recent reports have highlighted the importance of specific continuing medical education. In a cohort of primary care pediatricians, Flaherty et al[7] identified both patient and physician factors that are associated with increased frequency of suspecting abuse as a mechanism of injury. The only practitioner-level factor associated with increased frequency of suspicion of abuse was recent physician education about child abuse. In another report, Flaherty et al[22] described a 10-fold increase in physicians reporting child abuse when they had been exposed to abuse-specific continuing medical education. A study of military physicians showed that, although pediatricians had a similar amount of child maltreatment training in residency to emergency physicians, pediatricians reported a significantly greater amount of continuing medical education in abuse than other specialties after residency.[8] The only factor associated with increased reporting of child abuse in that cohort was the presence of postgraduate abuse training. If this pattern were true among civilian doctors, one might reasonably suppose that one factor in the underdiagnosis of child abuse in general hospitals might be the result of lower levels of abuse-specific continuing medical education.

The quantitative burden of the underdiagnosis of child abuse is likely to be substantial, because the majority of children are cared for at general hospitals with or without pediatric units. Even among this cohort of severely injured infants, general hospitals cared for the largest proportion of patients. Hospital emergency departments are complex environments and are likely to require quality-improvement programs to ensure that not only are clinical staff trained in the recognition of abuse but also that systems are in place to support the identification, medical and social evaluation, and management of abused infants.

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FAILURES OF EMERGENCY RELIEF

"Following the Asian tsunami on 26 December 2004 in which 224,000 people died, 2005 has been a bad year for the poorest and most vulnerable and especially those living in areas of armed conflicts. Two recent reports contain a number of valuable suggestions for the better management of global emergencies.[1,2] Oxfam's statement (18 October 2003) presents an outstanding response to the problem.[1] It covers many issues, perhaps the most important being the role of existing extreme poverty in compounding the effects of natural events. This view is also echoed by Jan Egeland, the Emergency Relief Coordinator for the United Nations (UN), as follows: 'Twenty million lives are at risk in forgotten and neglected crises in Western, Central, Eastern and Southern Africa. These are the silent tsunamis of our time.'[3] Poverty prevention is the key, with one study showing that a disaster in a country with a high level of development kills an average of 44 people compared with 300 in a poorly developed country.[4] This reality is placed in perspective by the latest earthquake in Pakistan where the Government is unable to spend more than 1% of its gross domestic product (GDP) on health because it uses between 75% and 80% to repay debt and service defense."

1. Oxfam. Governments have failed to effectively respond to year of disasters [press release]. Boston, MA: Oxfam; October 18, 2005. Available at: www.oxfam.org.uk/press/releases/disasters181005.htm. Accessed October 26, 2005
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J Roy Soc Med. 2005;98:528–529

original paper by Frace et al. The newer codes will be part of a publication that will be authored by Dr Simon Hambidge and colleagues. The authors apologize for any inconvenience this may have caused the readers or Dr Hambidge and his colleagues in the VSD studies.

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Trokel M, Waddimba A, Griffith J, Sege R. Variation in the Diagnosis of Child Abuse in Severely Injured Infants. PEDIATRICS 2006;117:722–728.

An error appeared in the article by Trokel et al, titled "Variation in the Diagnosis of Child Abuse in Severely Injured Infants" published in the March 2006 issue of *Pediatrics* (doi:10.1542/peds.2004-2731). The name of the author Anthony Waddimba was misspelled. The error has been corrected online.

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Section on Ophthalmology, American Academy of Pediatrics; American Academy of Ophthalmology; American Association for Pediatrics Ophthalmology and Strabismus. Screening Examination of Premature Infants for Retinopathy of Prematurity. PEDIATRICS 2006;117:572–576.

An error appeared in the Policy Statement by Section on Ophthalmology, American Academy of Pediatrics; American Academy of Ophthalmology; and American Association for Pediatric Ophthalmology and Strabismus, titled "Screening Examination of Premature Infants for Retinopathy of Prematurity" that was published in the February 2006 issue of *Pediatrics* (doi: 10.1542/peds.2005-2749). On page 573, Recommendation 1 states: "Infants with a birth weight of less than 1500 g or gestational age of 32 weeks or less (as defined by the attending neonatologist) and selected infants with a birth weight between 1500 and 2000 g or gestational age of more than 32 weeks with an unstable clinical course, including those requiring cardiorespiratory support and who are believed by their attending pediatrician or neonatologist to be at high risk, should have retinal screening examinations performed after pupillary dilation using binocular indirect ophthalmoscopy to detect ROP." The gestational age criterion should be 30 weeks, rather than 32 weeks, so that the corrected recommendation should read "Infants with a birth weight of less than 1500 g or gestational age of 30 weeks or less (as defined by the attending neonatologist) and selected infants with a birth weight between 1500 and 2000 g or gestational age of more than 30 weeks with an unstable clinical course, including those requiring cardiorespiratory support and who are believed by their attending pediatrician or neonatologist to be at high risk, should have retinal screening examinations performed after pupillary dilation using binocular indirect ophthalmoscopy to detect ROP."

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