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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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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).

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

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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

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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

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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

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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%,

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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

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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. Greener, *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

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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

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[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

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[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

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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

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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

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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

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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.
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> . . . .
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> . . . *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. . . .

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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

Accidents/Falls:

Meta-Analysis/Systematic Reviews/Guidelines:

1) David L. Chadwick et al., *Annual Risk of Death Resulting from Short Falls among Young Children: Less than 1 in 1 Million*, 121 PEDIATRICS 1213 (2008).
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).

Controlled Studies/Trials:

1) M. Belechri et al., *Bunk Versus Conventional Beds: A Comparative Assessment of Fall Injury Risk*, 56 J. EPIDEMIOLOGY & COMMUNITY HEALTH 413 (2002).

Comparative Cohorts /Prospective Case Series:

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).
3) Ann-Christine Duhaime et al., *Head Injury in Very Young Children: Mechanisms, Injury Types and Ophthalmologic Findings in 100 Hospitalized Patients Younger than 2 Years of Age*, 90 PEDIATRICS 179 (1992).
4) David S. Greenes & Sara A. Schutzman, *Clinical Indicators of Intracranial Injury in Head-Injured Infants*, 104 PEDIATRICS 861 (1999).
5) David S. Greenes & Sara A. Schutzman, *Clinical Significance of Scalp Abnormalities in Asymptomatic Head-Injured Infants*, 17 PEDIATRIC EMERGENCY CARE 88 (2001).
6) Karl Johnson et al., *Accidental Head Injuries in Children under 5 Years of Age*, 60 CLINICAL RADIOLOGY 464 (2005).

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).

Retrospective Case Series/Case Reports:

1) Abbey Alkon et al., *Injuries in Child-Care Centers: Rates, Severity, and Etiology*, 94 PEDIATRICS 1043 (1994).
2) David L. Chadwick & Connie Salerno, *Likelihood of the Death of an Infant or Young Child in a Short Fall of Less than 6 Vertical Feet*, 35 J. TRAUMA 968 (1993).
3) Horace B. Gardner, *A Witnessed Short Fall Mimicking Presumed Shaken Baby Syndrome (Inflicted Childhood Neurotrauma)*, 43 PEDIATRIC NEUROSURGERY 433 (2007).
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).
6) John R. Hall et al., *The Mortality of Childhood Falls*, 29 J. TRAUMA 1273 (1989).
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).

15) JA Murray et al., *Pediatric Falls: Is Height a Predictor of Injury and Outcome?*, 66 AM. SURGEON 863 (2000).
16) Prasit Nimityongskul & Lewis D. Anderson, *The Likelihood of Injuries when Children Fall Out of Bed*, 7 J. PEDIATRIC ORTHOPAEDICS 184 (1987).
17) Se-Hyuck Park et al., *Head Injuries from Falls in Preschool Children*, 45 YONSEI MED. J. 229 (2004).
18) Srikumar Pillai et al., *Fall Injuries in the Pediatric Population: Safer and Most Costeffective Management*, 48 J. TRAUMA 1048 (2000).
19) Melanie L. Pitone & Magdy W. Attia, *Patterns of Injury Associated with Routine Childhood Falls*, 22 PEDIATRIC EMERGENCY CARE 470 (2006).
20) John Plunkett, *Fatal Pediatric Head Injuries Caused by Short-Distance Falls*, 22 AM. J. FORENSIC MED. & PATHOLOGY 1 (2001).
21) Gregory D. Reiber, *Fatal Falls in Childhood: How Far Must Children Fall to Sustain Fatal Head Injury? Report of Cases and Review of the Literature*, 14 AM. J. FORENSIC MED. & PATHOLOGY 201 (1993).
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).
23) Jeff E. Schunk et al., *The Utility of Head Computed Tomographic Screening in Pediatric Patients with Normal Neurologic Examination in the Emergency Department*, 12 PEDIATRIC EMERGENCY CARE 160 (1996).
24) Michael Y. Wang et al., *Injuries from Falls in the Pediatric Population: An Analysis of 729 Cases*, 36 J. PEDIATRIC SURGERY 1528 (2001).
25) R.A. Williams, *Injuries in Infants and Small Children Resulting from Witnessed and Corroborated Free Falls*, 31 J. TRAUMA 1350 (1991).
26) Julia Wrigley & Joanna Dreby, *Fatalities and the Organization of Child Care in the United States: 1985 – 2003*, 70 AM. SOCIOLOGICAL REV. 749 (2005).
27) Yusuf Yagmur et al., *Falls From Flat Roofed Houses: A Surgical Experience of 1643 Patients*, 35 INJURY 425 (2004).

Biomechanical Studies:

1) Faris A. Bandak, *Shaken Baby Syndrome: A Biomechanics Analysis of Injury Mechanisms*, 151 FORENSIC SCI. INT'L 71 (2005).
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).
4) Brittany Coats & Susan S. Margulies, *Potential for Head Injuries in Infants from Low-Height Falls*, 2 J. NEUROSURGERY: PEDIATRICS 321 (2008).
5) Brittany Coats et al., *Parametric Study of Head Impact in the Infant*, 51 STAPP CAR CRASH J. 1 (2007).
6) Z. Cooper & F. Albermani, *Mechanical Response of Infant Brain to Manually Inflicted Shaking*, 224 J. ENGINEERING MED. 1 (2010).
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).
9) C. Z. Cory et al., *The Potential and Limitations of Utilising Head Impact Injury Models to Assess the Likelihood of Significant Head Injury in Infants After a Fall*, 123 FORENSIC SCI. INT'L 89 (2001).
10) Ann-Christine Duhaime et al., *The Shaken Baby Syndrome: A Clinical, Pathological and Biomechanical Study*, 66 J. NEUROSURGERY 409 (1987).
11) T.A. Gennarelli & L.E. Thibault, *Biomechanics of Acute Subdural Hematoma*, 22 J. TRAUMA 680 (1982).
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4) Yutaka Sato, *Imaging of Nonaccidental Head Injury*, 39 PEDIATRIC RADIOLOGY 230 (Supp. 2009).

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

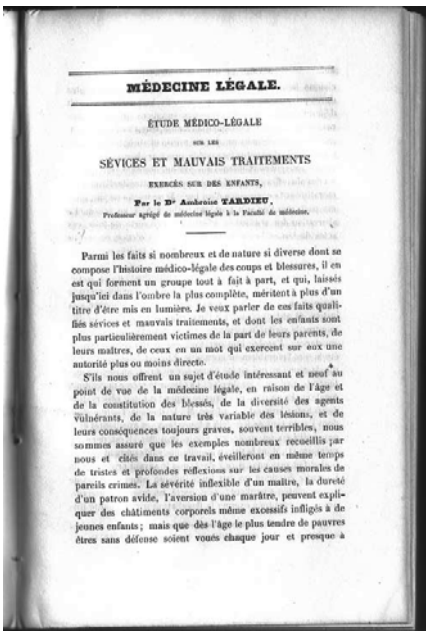
Papilledema/Increased Intracranial Pressure

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:AugusteAmbroseTardieu.jpg.



MÉDECINE LÉGALE.

ÉTUDE MÉDICO-LÉGALE

SUR LES

SÉVICES ET MAUVAIS TRAITEMENTS

EXERCÉS SUR DES ENFANTS,

Par le Dr Ambroise TARDIEU,

Professeur agrégé de médecine légale à la Faculté de médecine.

Parmi les faits si nombreux et de nature si diverse dont se compose l'histoire médico-légale des coups et blessures, il en est qui forment un groupe tout à fait à part, et qui, laissés jusqu'ici dans l'ombre la plus complète, méritent à plus d'un titre d'être mis en lumière. Je veux parler de ces faits qualifiés sévices et mauvais traitements, et dont les enfants sont plus particulièrement victimes de la part de leurs parents, de leurs maîtres, de ceux en un mot qui exercent sur eux une autorité plus ou moins directe.

S'ils nous offrent un sujet d'étude intéressant et neuf au point de vue de la médecine légale, en raison de l'âge et de la constitution des blessés, de la diversité des agents vulnérants, de la nature très variable des lésions, et de leurs conséquences toujours graves, souvent terribles, nous sommes assuré que les exemples nombreux recueillis par nous et cités dans ce travail, éveilleront en même temps de tristes et profondes réflexions sur les causes morales de pareils crimes. La sévérité inflexible d'un maître, la dureté d'un patron avide, l'aversion d'une marâtre, peuvent expliquer des châtiments corporels même excessifs infligés à de jeunes enfants; mais que dès l'âge le plus tendre de pauvres êtres sans défense soient voués chaque jour et presque à

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'HYGIÈNE PUBLIQUE ET DE MÉDECINE LÉGALE 361–98 (1860))

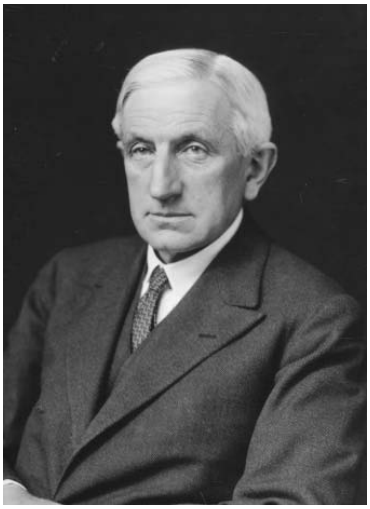


Fig. 3. Image of Wilfred Batten Lewis Trotter (1872–1939). Reproduced with permission © Godfrey Argent Studio.

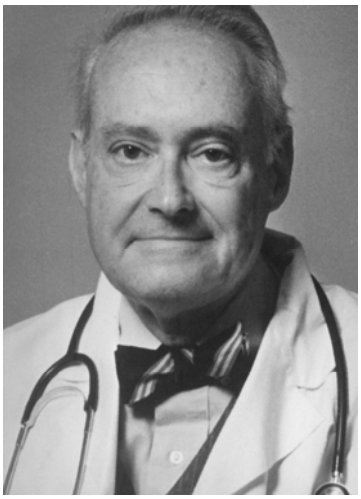


Fig. 4. Dr. C. Henry Kempe.
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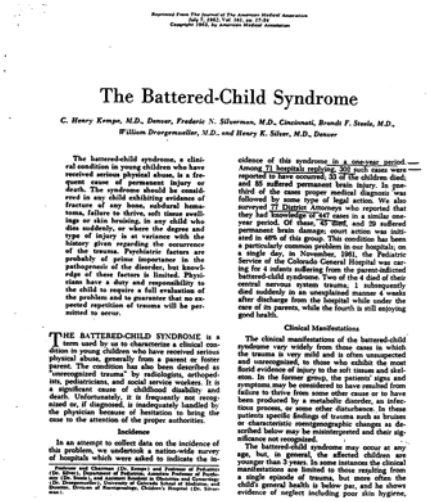

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July 7, 1962, Vol. 181, pp. 17-24
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The Battered-Child Syndrome

C. Henry Kempe, M.D., Denver, Frederic N. Silverman, M.D., Cincinnati, Brandt F. Steele, M.D., William Droegemueller, M.D., and Henry K. Silver, M.D., Denver

The battered-child syndrome, a clinical condition in young children who have received serious physical abuse, is a frequent cause of permanent injury or death. The syndrome 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 the trauma. Psychiatric factors are probably of prime importance in the pathogenesis of the disorder, but knowledge of these factors is limited. Physicians have a duty and responsibility to the child to require a full evaluation of the problem and to guarantee that no expected repetition of trauma will be permitted to occur.

THE BATTERED-CHILD SYNDROME is a term used by us to characterize a clinical condition in young children who have received serious physical abuse, generally from a parent or foster parent. The condition has also been described as "unrecognized trauma" by radiologists, orthopedists, pediatricians, and social service workers. It is a significant cause of childhood disability and death. Unfortunately, it is frequently not recognized or, if diagnosed, is inadequately handled by the physician because of hesitation to bring the case to the attention of the proper authorities.

Incidence

In an attempt to collect data on the incidence of this problem, we undertook a nation-wide survey of hospitals which were asked to indicate the incidence of this syndrome in a one-year period. — Among 71 hospitals replying, 302 such cases were reported to have occurred. 33 of the children died, and 85 suffered permanent brain injury. In one-third of the cases proper medical diagnosis was followed by some type of legal action. We also surveyed 77 District Attorneys who reported that they had knowledge of 447 cases in a similar one-year period. Of these, 47 died, and 29 suffered permanent brain damage; court action was initiated in 49% of this group. This condition has been a particularly common problem in our hospitals; on a single day, in November, 1961, the Pediatric Service of the Colorado General Hospital was caring for 4 infants suffering from the parent-inflicted battered-child syndrome. Two of the 4 died of their central nervous system trauma; 1 subsequently died suddenly in an unexplained manner 4 weeks after discharge from the hospital while under the care of its parents, while the fourth is still enjoying good health.

Clinical Manifestations

The clinical manifestations of the battered-child syndrome vary widely from those cases in which the trauma is very mild and is often unsuspected and unrecognized, to those who exhibit the most florid evidence of injury to the soft tissues and skeleton. In the former group, the patients' signs and symptoms may be considered to have resulted from failure to thrive from some other cause or to have been produced by a metabolic disorder, an infectious process, or some other disturbance. In these patients specific findings of trauma such as bruises or characteristic roentgenographic changes as described below may be misinterpreted and their significance not recognized.

The battered-child syndrome may occur at any age, but, in general, the affected children are younger than 3 years. In some instances the clinical manifestations are limited to those resulting from a single episode of trauma, but more often the child's general health is below par, and he shows evidence of neglect including poor skin hygiene,

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.

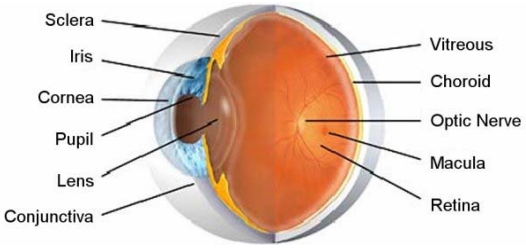

Fig. 6. Human Eye
Reprinted courtesy of http://lhsanatomy4.wikispaces.com

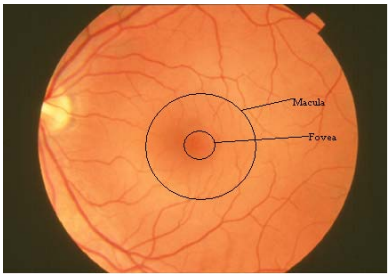

Fig. 7. Normal Retina, demonstrating the area of the retina called the posterior pole: fovea and macula (within circles), optic nerve (bright whitish appearing circle on left-hand side) and its head manifesting as a circular disc (optic disc), and retinal vessels emanating from the optic nerve. Reprinted from *Eye Disease Anatomy*, Ref#: EDA06, NAT'L EYE INST., http://www.nei.nih.gov/phot/eyedis/index.asp (circles added by author).

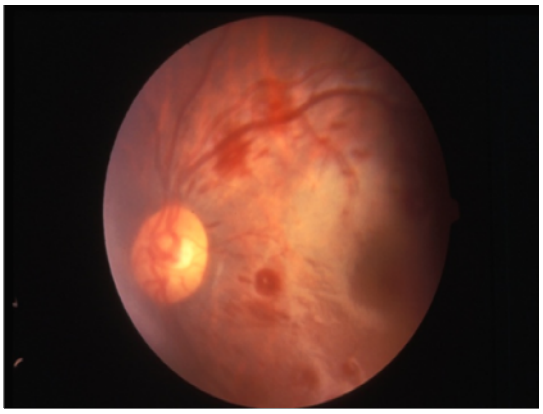

Fig. 8. Mild nonspecific retinal hemorrhages confined to the posterior pole. (Courtesy of Alex V. Levin, MD. MHSc, Wills Eye Institute, Philadelphia)

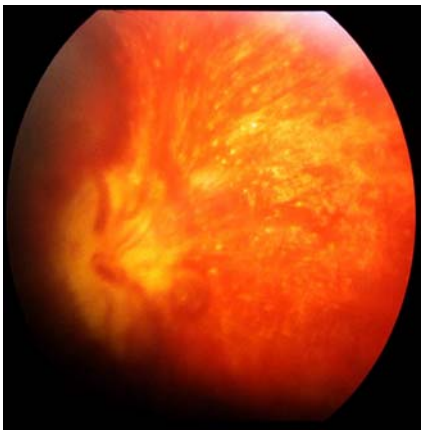

Fig. 9. Severe retinal hemorrhages, too numerous to count, such that there is virtually no visible normal retina. (Courtesy of Alex V. Levin, MD. MHSc, Wills Eye Institute, Philadelphia)

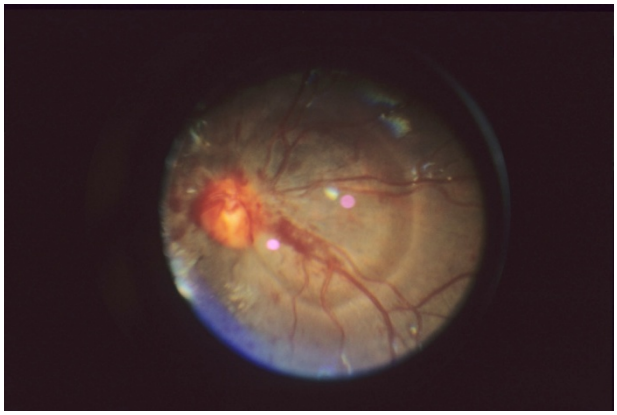

Fig. 10. Macular traumatic retinoschisis. (Courtesy of Alex V. Levin, MD. MHSc, Wills Eye Institute, Philadelphia)

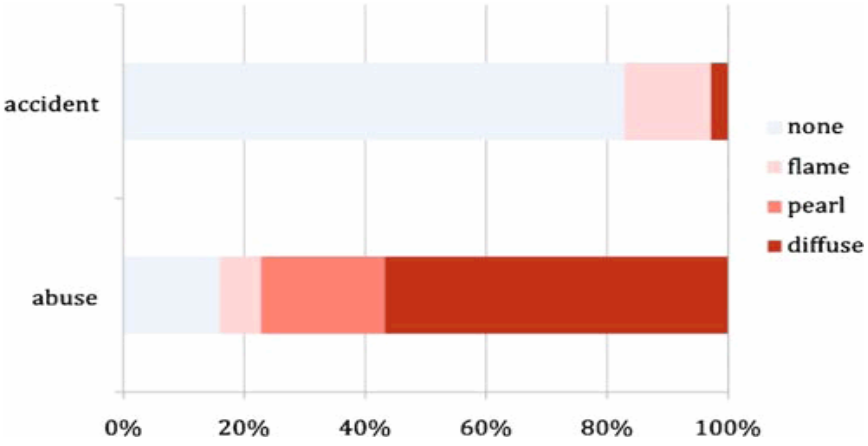

Fig. 11. "Retinal hemorrhage in the AT and IHI groups. Although most cases of abuse were associated with severe hemorrhage, seven had no hemorrhage, and three had only mild hemorrhages." Matthieu Vinchon et al., *Confessed Abuse Versus Witnessed Accidentsin Infants: Comparison of Clinical, Radiological, 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.