

Identifying, Investigating, and Litigating Cases Involving Abusive Head Trauma

January 29, 2021

Index of Appendices

Appendix A	PowerPoint Presentation Slides
Appendix B	<i>People v Leo Ackley</i> , 497 Mich 381 (2015)
Appendix C	Guthkelch, <i>Problems of Infant Retino-Dural Hemorrhage with Minimal External Injury</i> , 12 Hous J Health L & Policy 201 (2012)
Appendix D	Findley, et al, <i>Shaken Baby Syndrome, Abusive Head Trauma, and Actual Innocence</i> ,” 12 Hous J Health L & Policy 209 (2012)
Appendix E	Bandak, <i>Shaken Baby Syndrome: A Biomechanics Analysis of Injury Mechanisms</i> , 151 Forensic Sci Int’l 71 (2005)
Appendix F	Gilliland, <i>Interval Duration between Injury and Severe Symptoms in Nonaccidental Head Trauma in Infants and Young Children</i> , 43 J Forensic Sci 723 (1998)
Appendix G	National Association of Medical Examiners Position Paper: <i>Recommendations for the Postmortem Assessment of Suspected Head Trauma in Infant and Young Children</i> (2013)
Appendix H	List of Potential Experts and Additional Resources
Appendix I	Checklist

Appendix A

IDENTIFYING, INVESTIGATING, AND DEFENDING CASES INVOLVING ABUSIVE HEAD TRAUMA

Lillian Diallo (lilliandiallo@sbcglobal.net)
Erin Van Campen (evancampen@sado.org)

1

Shaken Baby Syndrome (SBS)

- An unconfirmed hypothesis
- Diagnosed based occurrence of three symptoms (the triad)
 - ▣ Subdural Hemorrhage
 - ▣ Retinal Hemorrhage
 - ▣ Cerebral Edema

More Info in Appendices C, D

2

The Mechanism of Injury

- SBS theory:
 - ▣ Shaking causes the head to whip back and force
 - ▣ Whipping causes internal impact of brain against either side of skull
 - ▣ Causes the veins along the outside of the brain to tear and bleed into the subdural spaces
 - ▣ Causes swelling
 - ▣ Causes retinal veins to tear and bleed

More Info in Appendix D

3

Abusive Head Trauma (AHT)

- Newer term adopted by American Academy of Pediatrics in 2009
- Includes Shaken Baby Syndrome, blunt force trauma, or combination of the two
- Might also be described as “non-accidental, intentionally inflicted injury”

More Info in Appendices C and D

4

Diagnosis of Exclusion

- SBS and AHT are NOT the only causes of the triad or head injuries in children
- Critical to know what other conditions/causes were considered and how they were excluded
- Divide within the medical community – those who believe they can reliably diagnose SBS/AHT and those who question it

More Info in Appendices C and D

5

Getting it Right

- Dr. Norman Guthkelch, Neurosurgeon
- First conceived of the concept of SBS
- 2012 article expressing concerns about the use of the diagnosis
- “...there has arisen a level of emotion and divisiveness...that has interfered with our commitment to pursue the truth.”

Guthkelch's 2012 Article is Appendix C

6

What's in a Name?

- Terms SBS/AHT assert unique mechanism of injury and intent
- Guthkelch recommends diagnosing specific injuries (i.e. retino-dural hemorrhages of infancy) rather than using the terms SBS/AHT
- "This would allow us to investigate causation without appearing to assume that we already know the answer."

More Info in Appendix C

7

Getting it Right

"Often, "getting it right" simply means saying, clearly and unequivocally, "we don't know."

- Guthkelch

More Info in Appendix C

8

SBS/AHT in the Media

- Substantial NYT Article about nanny case of the '90s
- Substantial Washington Post piece presenting many different perspectives on the controversy
- Both provide great plain English overview of the controversy, perspectives from both sides, case studies, and interviews with experts

Both websites listed in Appendix H

9

SBS/AHT and Innocence

- SBS/AHT diagnoses have played a role in a number of cases where the accused was wrongfully convicted
- The National Registry of Exonerations includes at least 13 people who were originally convicted of child abuse, manslaughter, or murder on the basis of unreliable SBS/AHT diagnoses
 - See www.exonerationregistry.org

More Info in Appendix D

10

Alternative Causes or Contributing Factors

- Short falls
- Medical conditions and illnesses
 - Possibly resulting from difficult labor
 - Stroke
 - Venous thrombosis
 - Metabolic disorders
 - Nutritional deficiencies
- Re-bleeding of chronic subdural collections
 - Hydrocephaly
- Vaccine-induced encephalitis
- Subsequent medical procedures
 - Hypoxia
 - Intubation
 - Craniectomy

More Info in Appendix D

11

People v Leo Ackley

- Granted new trial on ineffective assistance of counsel claim in abusive head trauma case
- Unanimous Michigan Supreme Court Decision by Justice McCormack
- Published June 29, 2015
- 497 Mich 381

Ackley slip opinion is Appendix B

12

People v Leo Ackley

- Ackley Court recognized that there is a “prominent controversy within the medical community regarding the reliability of SBS/AHT diagnoses.”
 - Slip Op p 10, citing Findley article (Appendix D)
- “[T]he expert is the case.”
 - Slip Op p 15
- Critical of “counsel’s failure to engage ‘expert testimony rebutting the state’s expert testimony’ and to become ‘versed in the technical subject matter most critical to the case’”
 - Slip Op p 15

Ackley slip opinion is Appendix B

13

Identifying Cases Involving SBS/AHT

- Indicators your case involves this medical controversy:
 - Diagnosis of SBS/AHT
 - “Non-accidental, intentionally inflicted”
 - Alternative explanation “could not have generated enough force to cause injury”
 - “Child would have been symptomatic immediately following injury”

More Info in Appendices C, D

14

You Need to Consult Experts When...

- There is singularity of the injury
- There is a reported history of a short fall or other accidental injury
- There is a history of medical issues or illness

More Info in Appendix D

15

Investigating SBS/AHT Cases

- Interview all people who spent time with child
- Get all medical records from case, including any radiology and photos
- Get child's medical history from birth, and ideally medical records from birth
- If child is deceased, get autopsy report, all photos, and slides

More Info in Appendices B, D

16

Consulting the Right Experts

- Different experts necessary for different specific issues related to SBS/AHT diagnoses
- Not enough to just consult an expert – post-*Ackley*, defense attorneys must consult the right experts
- Request funds – post-*Ackley*, trial courts need to provide funds for indigent defendants to consult the right experts

More Info in Appendices B, D, H

17

Know what *all* the experts have to say

- Request reports or summaries from all experts who may testify
 - MCR 6.201(A)(3)
- Do the experts' opinions fall within their fields of expertise?
- What evidence did the experts consider?

18

Preparing to Cross-Examine Experts

- Consult with your own experts about each expert report or opinion summary

- Use scientific publications to impeach

- Consider Motions in Limine to preclude experts from testifying
 - *Daubert*
 - Area of Expertise
 - Expert Qualifications

More Info in Appendix D

19

Key Points of Controversy

- Possibility of lucid intervals

- “Force” necessary to cause serious or fatal head injuries

More Info in Appendix D

20

Lucid Intervals

- “The child would have been symptomatic immediately following the injury”

- Implicates timing of injury

- All injuries develop differently

- Hemorrhages are bleeds, necessarily happen over time
 - Can be very quick or can take some time
 - Hemorrhage can clot, stalling symptoms until bleeding resumes

More Info in Appendices D, F

21

Lucid Intervals in Cases of Fatal Injuries

- Post-mortem, there are techniques available to determine the age of an injury
- Cell organization refers to healing that begins immediately following an injury until death
 - The greater the degree of organizing, the older the injury
- Were dura samples stained to study organization?
- Did a neurologist provide an opinion of comparative CTs?

More Info in Appendices D, F, G

22

“Force” Necessary to Cause Injury

- “The force necessary to cause this injury is comparable to a multistory fall or an auto collision”
- Implicates intent, plausibility of alternative causes of injury
- A matter of belief, not medical science
- Are opinions provided by experts in physics?
- Did experts consult with experts in fields where they lacked expertise?

More Info in Appendices D, E

23

It’s a Matter of Physics

- Study of force involves the application of physics
 - Biomechanical engineers study the application of mechanical engineering and physics to quantify the effects of forces on and within the human body
 - Includes identifying mechanism of injury
 - Head acceleration vs force
- Studies have shown that head acceleration generated by adult human shaking is comparable to a one-foot fall

More Info in Appendices D, E

24

Empirical Studies

- While rare, children can suffer serious and fatal head injuries from short falls

- Likelihood of serious or fatal injury depends on a variety of circumstances, such as angle of fall, angle of impact, surface child falls onto

- Children have suffered fatal head injuries resulting from falls shorter than 3 feet

More Info in Appendices B, D

25

Age Matters

- Huge differences between infants and toddlers in terms of mobility

- Potential for accidental injury involving 6-month-old vs 2-year-old

- Did the experts take age into consideration in forming their opinions?

More Info in Appendix D

26

Who You Gonna Call?

- You may need experts in:
 - Forensic pathology
 - Neuropathology
 - Biomechanics
 - Pediatrics
 - Radiology
 - Neuroradiology
 - Childhood development

- You may also want to consult with lawyers who specialize in these cases

More Info in Appendix H

27

FUNDING FOR EXPERTS:

People v Johnny Kennedy

- Recognized the federal constitutional right of indigent defendants to expert assistance at state expense
- Unanimous Michigan Supreme Court Decision by Justice Viviano
- Published June 29, 2018
- 502 Mich 206

28

FUNDING FOR EXPERTS:

- Now available through Wayne County's Office of Public Defense
 - No longer need to seek funding from court
 - Can seek funding *ex parte*
 - If funding is denied, need to make a record of efforts to obtain
- More info available at:
<https://www.waynecounty.com/departments/corpcounsel/forms-for-assigned-counsel.aspx>

29

Resources

- Resource pack includes:
 - Ackley
 - Findley article providing thorough overview
 - Scientific journal articles on specific points like lucid intervals
 - List of potential experts and attorneys you may want to consult, as well as additional resources for initial research

30

Lillian Diallo
lilliandiallo@sbcglobal.net
(313) 965-6633

Erin Van Campen
evancampen@sado.org
(313) 256-9833

Appendix B

Syllabus

Chief Justice:
Robert P. Young, Jr.

Justices:
Stephen J. Markman
Mary Beth Kelly
Brian K. Zahra
Bridget M. McCormack
David F. Viviano
Richard H. Bernstein

This syllabus constitutes no part of the opinion of the Court but has been prepared by the Reporter of Decisions for the convenience of the reader.

Reporter of Decisions:
Corbin R. Davis

PEOPLE v ACKLEY

Docket No. 149479. Argued March 10, 2015. Decided June 29, 2015.

Leo D. Ackley was convicted by a jury in the Calhoun Circuit Court of first-degree felony murder, MCL 750.316(1)(b), and first-degree child abuse, MCL 750.136(b)(2), after his live-in girlfriend's three-year-old daughter died while in his care. At trial, the prosecution called five medical experts who testified that the child had died as the result of a head injury that was caused intentionally, while defense counsel called no experts, despite having been provided court funding for expert assistance and the name of a well-known forensic pathologist who could support the defense theory that the injuries had resulted from an accidental fall. Defendant appealed his convictions as of right, arguing that his lawyer's failure to meaningfully challenge the prosecution's expert testimony violated his Sixth Amendment right to the effective assistance of counsel. The Court of Appeals, BOONSTRA, P.J., and SAWYER and SHAPIRO, JJ., remanded the matter for an evidentiary hearing under *People v Ginther*, 390 Mich 436 (1973), after which the trial court, James C. Kingsley, J., granted defendant's motion for a new trial. The prosecution appealed. The Court of Appeals, OWENS, P.J., and MURRAY and RIORDAN, JJ., reversed in an unpublished opinion per curiam issued April 22, 2014 (Docket No. 318303), holding that the trial court had abused its discretion by granting a new trial because defense counsel's decisions regarding experts were trial strategy and no prejudice had resulted. Defendant appealed. The Supreme Court ordered and heard oral argument on whether to grant the application for leave to appeal or take other peremptory action, limited to the issue whether defendant was denied the effective assistance of counsel based on trial counsel's failure to adequately investigate the possibility of obtaining expert testimony in support of the defense. 497 Mich 910 (2014).

In a unanimous opinion by Justice MCCORMACK, the Supreme Court, in lieu of granting leave to appeal, *held*:

Defendant was denied the effective assistance of counsel by his trial counsel's failure to investigate adequately and to attempt to secure suitable expert assistance in the preparation and presentation of his defense. Expert testimony was critical in this case to explain whether the cause of the child's death was intentional or accidental. Defense counsel's failure to attempt to engage a single expert witness to rebut the prosecution's expert testimony, or to attempt to consult an expert with the scientific training to support the defense theory of the case, fell below an objective standard of reasonableness, and there was a reasonable probability that this error affected the outcome of the trial. Accordingly, defendant was entitled to a new trial.

1. The Court of Appeals erred by concluding that defense counsel's decision to consult only Dr. Brian Hunter in preparation for trial was objectively reasonable. There was no objectively reasonable explanation in the record for counsel's decision to confine his pursuit of expert assistance to Hunter, a self-proclaimed opponent of the very defense theory counsel was to employ at trial, despite Hunter's having referred counsel to at least one other expert who could provide qualified and suitable assistance. Counsel's failure to engage expert testimony rebutting the state's expert testimony and failure to become versed in the technical subject matter constituted a constitutional flaw in the representation, not reasonable strategy. Given the centrality of expert testimony to the prosecution's proofs and the highly contested nature of the underlying medical issue, counsel's single error of failing to consult an expert who could meaningfully assist him constituted ineffective assistance.

2. But for counsel's deficient performance, there was a reasonable probability that the outcome of defendant's trial would have been different. Defendant's conviction turned on the jury's assessment of the prosecution's theory that the child's fatal injuries were the result of intentional abuse, which was advanced through the testimony of five experts. Because defendant's own testimony and that of his lay character witnesses were extremely unlikely to counter this formidable expert testimony, expert assistance in defendant's favor was critical to provide the jury with another viable and impartial perspective on the facts of the case while contradicting the prosecution's theory of how the child died. The prosecution's voluminous expert testimony made the need for an effective response by defense counsel particularly apparent and strong, and it rendered counsel's failure to offer expert testimony particularly glaring and harmful to the defendant. This consequence militated in favor of defendant's claim of relief. Further, the prosecution's nonexpert evidence was highly circumstantial, heavily contested, and far from dispositive of the issue of defendant's guilt. While a battle of the experts might not have ensured defendant's acquittal, counsel's failure to prepare or show up for the battle sufficiently undermined confidence in the outcome of this case to entitle defendant to relief.

Court of Appeals judgment reversed; conviction vacated; case remanded to the trial court for further proceedings.

OPINION

Chief Justice:
Robert P. Young, Jr.

Justices:
Stephen J. Markman
Mary Beth Kelly
Brian K. Zahra
Bridget M. McCormack
David F. Viviano
Richard H. Bernstein

FILED June 29, 2015

STATE OF MICHIGAN

SUPREME COURT

PEOPLE OF THE STATE OF MICHIGAN,

Plaintiff-Appellee,

v

No. 149479

LEO DUWAYNE ACKLEY, a/k/a LEO
DUANE ACKLEY, JR., and LEO
DUWAYNE ACKLEY II,

Defendant-Appellant.

BEFORE THE ENTIRE BENCH

MCCORMACK, J.

The question before us is whether the defendant was denied the effective assistance of counsel by his trial counsel's failure to investigate adequately and to attempt to secure suitable expert assistance in the preparation and presentation of his defense. In this case involving the unexplained and unwitnessed death of a child, expert testimony was critical to explain whether the cause of death was intentional or accidental. Contrary to the determination of the Court of Appeals, we conclude that defense counsel's failure

to attempt to engage a single expert witness to rebut the prosecution's expert testimony, or to attempt to consult an expert with the scientific training to support the defendant's theory of the case, fell below an objective standard of reasonableness, and created a reasonable probability that this error affected the outcome of the defendant's trial. See *Strickland v Washington*, 466 US 668, 694; 104 S Ct 2052; 80 L Ed 2d 674 (1984). Accordingly, we reverse the judgment of the Court of Appeals, vacate the defendant's convictions, and remand for proceedings consistent with this opinion.

I. FACTS AND PROCEDURAL HISTORY

The defendant was convicted by a jury of first-degree felony murder, MCL 750.316(1)(b), and first-degree child abuse, MCL 750.136b(2), after his live-in girlfriend's three-year-old daughter died while in his care. According to the defendant, the child had been napping alone in her room before he discovered her lying unresponsive on the floor next to the bed. The prosecution alleged that the defendant killed the child, either by blunt force trauma or shaking. The defendant denied hurting the child, and said that she must have died as the result of an accidental fall.

Given the lack of eyewitness testimony and any other form of direct evidence, expert testimony was the cornerstone of the prosecution's case. The prosecution called five medical experts to testify at trial about the cause of the child's death: two general pediatricians, a pediatric critical care doctor, a trauma surgeon, and a forensic pathologist.¹ Each testified that the child died as a result of abusive head injury caused

¹ The prosecution also called an expert in emergency medicine, who testified regarding the child's initial triage and treatment in the Battle Creek Health Systems Emergency Department.

either by nonaccidental shaking, blunt force trauma, or a combination of both. The defense, in contrast, called no expert in support of its theory that the child's injuries resulted from an accidental fall, although the court had provided funding for expert assistance.

The defendant appealed his convictions as of right, arguing that he was entitled to a new trial because his lawyer's failure to meaningfully challenge the prosecution's expert testimony regarding the cause of the child's death violated his Sixth Amendment right to the effective assistance of counsel. The Court of Appeals remanded for an evidentiary hearing pursuant to *People v Ginther*, 390 Mich 436; 212 NW2d 922 (1973). *People v Ackley*, unpublished order of the Court of Appeals, entered May 24, 2013 (Docket No 310350).

At the *Ginther* hearing, the defendant's trial counsel testified that he contacted only one expert to prepare for trial: forensic pathologist Brian Hunter. Dr. Hunter testified that, after reviewing some of the case materials, he advised counsel "right off the bat" that he was "not the best person" for the defense. He also explained to counsel that there was a marked difference of opinion within the medical community about diagnosing injuries that result from falling short distances, on the one hand, and shaken baby syndrome (SBS) or, as it is sometimes termed, abusive head trauma (AHT), on the other hand. Hunter asserted that this divide is "like a religion" because each expert has deeply held beliefs about when each diagnosis is supported, and the defendant should have the benefit of an expert who, "[i]n his or her religion, believes this could be a short-fall death." Hunter emphasized to counsel that he was on the wrong side of this debate to be able to assist the defendant.

Hunter then referred counsel to at least one well-known forensic pathologist,² Dr. Mark Shuman, who had conducted substantial research on short falls. Hunter characterized Dr. Shuman as the “best person” to assess the “complex” short-fall mechanism involved in the defendant’s theory. Hunter could not promise that Dr. Shuman would “buy into every story the defendant is selling,” but he informed counsel that Dr. Shuman was a “man of science . . . he’s the guy that’s going to give you your best shot.”

Counsel testified that he never contacted Shuman, or any other expert in short falls. Nor did he read any medical treatises or other articles about the medical diagnoses at issue. Though recognizing that expert testimony can carry great weight with a jury, he nevertheless stated that while it may have been “prudent” for him to have consulted “the over 400 treatises available” in preparing his cross-examinations of the prosecution’s experts “that wasn’t the strategy.”³ Instead, he requested a second consultation with Hunter, offering the simple (albeit inexplicable) justification that Dr. Shuman “was not going to work out.” Hunter reiterated his concerns with defense counsel’s choice to use him, unambiguously warning counsel again that “you don’t want me as your defense expert.”

² There was conflicting testimony between Hunter and defense counsel about Hunter’s referral(s). According to counsel, Hunter referred him to two experts: Dr. Shuman and Dr. Werner Spitz. According to Hunter, he referred counsel to Dr. Shuman only. In any event, counsel admitted that he never contacted either expert.

³ Defense counsel explained that he preferred to attack the experts exclusively through the “gray area” that Hunter supplied—namely, that there had been no studies as to the actual force necessary to achieve fatal blunt-force head injuries in children.

Counsel testified that he nevertheless continued to rely on only Hunter in his trial preparation, consulting him at least two more times before trial. Specifically, counsel provided Hunter with additional—but incomplete⁴—portions of the case materials so that Hunter could give counsel advice on how to approach the prosecution’s experts. Counsel admitted that Hunter’s advice was his only method of preparing to cross-examine the prosecution’s experts on the viability of their SBS/AHT theory of the child’s cause of death.⁵

Finally, the parties stipulated to the admission of an affidavit from Dr. Werner Spitz, another well-known expert in forensic pathology. After reviewing the autopsy report, postmortem photographs, and the trial transcripts, Dr. Spitz opined that the bruises on the child’s body were consistent with the intubation and CPR she received on the day of her death. He then averred that he would have testified that the child’s head injuries could not be attributed to SBS/AHT but were caused by a likely accidental “mild impact.”

Based on this evidence, the trial court granted the defendant a new trial. It found that counsel’s original failure even to attempt to contact either Dr. Shuman or Dr. Spitz

⁴ Most notably, counsel failed to provide Hunter with certain critical case materials regarding injuries the child had suffered not long before her death, including: (1) a witness statement that the child had fallen off a trampoline, had struck her head, had briefly gone unconscious, and had been complaining of headaches in the days leading up to her death, and (2) the police report of the accident, which indicated that the child had been lethargic, had been vomiting, and had lost control of her bowels the day before she died.

⁵ Counsel explained at the *Ginther* hearing that he was not paid for pretrial preparation.

was objectively unreasonable, and that there was a reasonable probability of a different result at trial had counsel engaged his own medical expert.

The Court of Appeals reversed, concluding that while there was no clear error in the trial court's findings of fact, the trial court had abused its discretion in finding a constitutional violation because counsel's "decision not to consult a second expert constituted trial strategy." *People v Ackley*, unpublished opinion per curiam of the Court of Appeals, issued April 22, 2014 (Docket No. 318303), p 4. The court also held that even if counsel should have contacted an expert other than Hunter, no prejudice resulted in light of all the evidence against the defendant.

The defendant sought leave to appeal in this Court. We heard oral argument on the application, limited to the issue of "whether the defendant was denied the effective assistance of counsel based on trial counsel's failure to adequately investigate the possibility of obtaining expert testimony in support of the defense."⁶

II. STANDARD OF REVIEW

Whether the defendant received the effective assistance of counsel guaranteed him under the United States and Michigan Constitutions is a mixed question of fact and law. *People v Trakhtenberg*, 493 Mich 38, 47; 826 NW2d 136 (2012), citing *People v Armstrong*, 490 Mich 281, 289; 806 NW2d 676 (2011). This Court reviews for clear error the trial court's findings of fact in this regard, and reviews de novo questions of constitutional law. *Trakhtenberg*, 493 Mich at 47.

⁶ *People v Ackley*, 497 Mich 910 (2014).

III. ANALYSIS

Both the Michigan and United States Constitutions require that a criminal defendant be afforded the assistance of counsel in his or her defense. US Const, Am VI; Const 1963, art 1, § 20. To be constitutionally effective, counsel's performance must meet an "objective standard of reasonableness." *Trakhtenberg*, 493 Mich at 52. To show that this standard has not been met, a defendant must "overcome the strong presumption that counsel's performance was born from a sound trial strategy." *Id.*, citing *Strickland v Washington*, 466 US at 689. But "a court cannot insulate the review of counsel's performance by calling it trial strategy"; counsel's strategy must be sound, and the decisions as to it objectively reasonable. *Trakhtenberg*, 493 Mich at 52. Courts must determine whether the "strategic choices [were] made after less than complete investigation," or if a "reasonable decision [made] particular investigations unnecessary." *Strickland*, 466 US at 690-691.

To obtain relief for the denial of the effective assistance of counsel, the defendant must show that counsel's performance fell short of this "objective standard of reasonableness" and that, but for counsel's deficient performance, "there is a reasonable probability that the outcome of [the defendant's trial] would have been different." *Trakhtenberg*, 493 Mich at 51. "A reasonable probability is a probability sufficient to undermine confidence in the outcome." *Strickland*, 466 US at 694.

A. COUNSEL'S PERFORMANCE

Turning first to the performance prong of the *Strickland* analysis, we disagree with the Court of Appeals that counsel's decision to consult only Dr. Hunter in preparation for trial was objectively reasonable. Rather, like the trial court, we conclude that counsel

performed deficiently by failing to investigate and attempt to secure an expert witness who could both testify in support of the defendant's theory that the child's injuries were caused by an accidental fall and prepare counsel to counter the prosecution's expert medical testimony.

As defense counsel was well aware before trial, the prosecution's theory of the case was that the defendant intentionally caused the child's unwitnessed injuries, a premise that it intended to prove with expert testimony. This testimony would require a response, and indeed, the court granted counsel funding to seek expert assistance of his own. Yet counsel contacted only Hunter, who repeatedly made clear that he credited the prosecution's SBS/AHT theory and disagreed with the defense's theory. While conceding that the SBS/AHT diagnosis was not universally accepted within the medical community, Hunter explained to counsel that he "really d[id]n't think [he] could help" the defendant because he was on the wrong side of this debate in his field.

As a solution, he advised counsel to consult Dr. Shuman, who not only was on the defendant's side of the SBS/AHT debate generally, but was significantly more likely to agree with the defendant's claim that the child's death in this case must have been accidental. Hunter even suggested that Dr. Shuman was more qualified because he had studied short falls extensively. Whereas Hunter was part of the group of experts who "don't have a good model" to support the accidental fall theory, Dr. Shuman was "someone who has dug into the physics" and the "proposed models" of a short-fall injury. Hunter also characterized Dr. Shuman as a "man of science" and as "the best expert in these types of situations." Yet counsel ignored this advice. He did not contact Dr.

Shuman or any other forensic pathologist with expertise in short falls, rendering Hunter his expert by default.

Counsel did not have sufficient information to legitimate this “choice.” While an attorney’s selection of an expert witness may be a “paradigmatic example” of trial strategy, that is so only when it is made “*after* thorough investigation of [the] law and facts” in a case. *Hinton v Alabama*, ___ US ___; 134 S Ct 1081, 1088; 188 L Ed 2d 1 (2014) (emphasis added). In this case, the record betrays no objectively reasonable explanation for counsel’s decision to confine his pursuit of expert assistance to Hunter, a self-proclaimed *opponent* of the very defense theory counsel was to employ at trial, despite Hunter’s referral to at least one other expert who could provide qualified and suitable assistance to the defendant. Nor is there any indication that counsel had the requisite familiarity with SBS/AHT or short-fall death theories to justify his settling on consulting only Hunter. To the contrary, counsel admittedly failed to consult any of the readily available journal articles on SBS/AHT and short-fall deaths, and did not otherwise educate himself or conduct any independent investigation of the medical issues at the center of the case, beyond his limited consultations with Hunter. See *Trakhtenberg*, 493 Mich at 54 n 9 (noting that “a defense attorney may be deemed ineffective, in part, for failing to consult an expert when counsel had neither the education nor the experience necessary to evaluate the evidence and make for himself a *reasonable, informed determination* as to whether an expert should be consulted or called to the stand”) (quotation marks and citation omitted); *Lindstadt v Keane*, 239 F3d 191, 202 (CA 2, 2001) (noting that counsel’s lack of familiarity with pertinent sexual abuse studies and failure to conduct any relevant research “hamstrung” his effort to

effectively cross-examine the prosecution's expert witness); *Holsomback v White*, 133 F3d 1382, 1387-1389 (CA 11, 1998) (holding that counsel's failure to conduct an adequate investigation into medical evidence of sexual abuse was ineffective).

We fail to see how counsel's sparse efforts satisfied his "duty to make reasonable investigations or to make a reasonable decision that makes particular investigations unnecessary," *Hinton*, 134 S Ct at 1088, quoting *Strickland*, 466 US at 690-691, especially in light of the prominent controversy within the medical community regarding the reliability of SBS/AHT diagnoses. See *State v Edmunds*, 308 Wis 2d 374, 391-392; 746 NW2d 590 (2008) (holding that the "significant dispute" and "shift in the mainstream medical community" regarding SBS/AHT diagnoses since the defendant's trial established a reasonable probability that a different result would be reached in a new trial, entitling the defendant to relief); Findley et al., *Shaken Baby Syndrome, Abusive Head Trauma, and Actual Innocence: Getting It Right*, 12 Hous J Health L & Policy 209, 212 (2012) (explaining that, in SBS/AHT cases, "it is critical to assess the reliability of the diagnoses under the standards of evidence-based medicine"). In this case involving such "substantial contradiction in a given area of expertise," counsel's failure to engage "expert testimony rebutting the state's expert testimony" and to become "versed in [the] technical subject matter" most critical to the case resulted in two things: a defense theory without objective, expert testimonial support, and a defense counsel insufficiently equipped to challenge the prosecution's experts because he possessed only Dr. Hunter's reluctant and admittedly ill-suited input as his guide. *Knott v Mabry*, 671 F2d 1208, 1212-1213 (CA 8, 1982). This "constitute[d] a constitutional flaw in the representation" of the defendant, not reasonable strategy. *Id.* at 1213.

In concluding otherwise, the Court of Appeals stressed that counsel is not required to shop for experts until finding one who will offer favorable testimony. We do not dispute that general proposition, but we fail to see its relevance here. In this case, counsel did no consultation at all beyond settling on the very first expert he encountered, despite the importance of expert medical testimony in the case and despite that expert's specific recommendation to contact a different and more suitable expert.

Nor can we agree with the Court of Appeals that Dr. Hunter's comments regarding Dr. Shuman's impartiality rendered it "reasonable for [counsel] to conclude that consulting a second expert would not be useful." *Ackley*, unpub op at 4. Hunter's warning that Dr. Shuman "would not buy into every story" or blindly accept the defendant's theory is consistent with scientific integrity, is desirable, and is, indeed, advantageous in the context of expert testimony. But more importantly, Hunter's core message on this very point was that counsel *should* engage Dr. Shuman, a qualified expert better suited to support the defendant's theory. And without having done any research on SBS/AHT or short-fall injuries, or having made any contact with Dr. Shuman, counsel "was ill equipped to assess his credibility or persuasiveness as a witness', or to evaluate and weigh the risks of putting him on the stand." *Towns v Smith*, 395 F3d 251, 260 (CA 6, 2005) (citation omitted). "To make a reasoned judgment about whether evidence is worth presenting, one must know what it says." *Couch v Booker*, 632 F3d 241, 246 (CA 6, 2011). Finally, as Dr. Spitz's affidavit plainly demonstrates, Dr. Hunter's advice to consult another expert was well founded.

Accordingly, we conclude that counsel's efforts to investigate and attempt to secure suitable expert assistance in preparing and presenting defendant's case fell below

an objective standard of reasonableness. While the Court of Appeals may be correct that counsel's deficiencies in this regard did not infect all of his conduct throughout the trial, see *Ackley*, unpub op at 6, the rest of his advocacy could not cure this crucial error. As the Supreme Court has said, "a single, serious error may support a claim of ineffective assistance of counsel." *Kimmelman v Morrison*, 477 US 365, 383; 106 S Ct 2574; 91 L Ed 2d 305 (1986). Given the centrality of expert testimony to the prosecution's proofs and the highly contested nature of the underlying medical issue, counsel committed exactly that kind of error by failing to consult an expert who could meaningfully assist him in advancing his theory of defense and in countering the prosecution's theory of guilt.

B. PREJUDICE

We further conclude that, but for counsel's deficient performance, "there is a reasonable probability that the outcome of [the defendant's trial] would have been different." *Trakhtenberg*, 493 Mich at 51; *Strickland*, 466 US at 694. As set forth above, the defendant's conviction turned on the jury's assessment of the prosecution's cause-of-death theory, which was advanced through the testimony of five experts, each of whom concluded that the child's injuries were the result of some form of intentional abuse. The defendant's own testimony and that of his lay character witnesses were extremely unlikely to counter this formidable expert testimony. Therefore, the absence of expert assistance in the defendant's favor was critical. It prevented counsel from testing the soundness of the prosecution's experts' conclusions with his own expert testimony and with effective cross-examination. And again, as Dr. Spitz's affidavit shows, such expert

assistance *was* available and would have provided the jury with another viable and impartial perspective on the facts of the case while contradicting the prosecution's theory of how the child died.

The Court of Appeals nonetheless found the prejudice from counsel's deficient performance insufficient to warrant relief, given both the strength of the other, nonexpert evidence of the defendant's guilt, and the sheer multitude of expert testimony the prosecution had marshaled in support of its position. We disagree times two.

First, we fail to see particular strength in the prosecution's nonexpert evidence, which was highly circumstantial, heavily contested, and far from dispositive of the issue of defendant's guilt. There was no explanation for the child's injuries beyond the theories presented by the experts, and the prosecution produced no witnesses who testified that the defendant was ever abusive. In fact, some testimony supported the opposite conclusion; according to the child's mother, the defendant's disciplinary tactics were no different from her own, there was no indication that either of her daughters feared the defendant, there were alternative explanations for some of the child's bruises and physical symptoms,⁷ and the child willingly spent time with the defendant and called him "daddy."⁸ And while the prosecution claimed that the child began to exhibit health

⁷ The child's mother attributed these bruises to the child's diet and physical activity, and the prosecution's forensic pathologist stated that the child was mildly anemic and that her bruising had no pattern indicating an object or a hand.

⁸ The Court of Appeals also cited the "peculiar" nature of the defendant's actions on the day of the incident as an indication of his guilt. Specifically, the panel found significant the defendant's failure to seek help from his neighbors after discovering the child on the floor, his attempt to revive her by pouring cold water over her, his decision to retrieve the family dog before fleeing the family's home, and his decision to first go to his mother's

issues around the time that the defendant entered her life, there was witness testimony to contradict this assertion, and the source and timing of these issues did not coincide with the defendant's move into the family's home or with his assumption of childcare duties.⁹ In short, our review of this nonexpert evidence makes plain why the prosecution chose to build its case primarily through the testimony of five experts, but it does little to weaken our conclusion that defense counsel's failure to meaningfully engage and respond to this expert testimony created a reasonable probability of a different outcome at trial.

Nor do we agree with the Court of Appeals that the sheer volume of the prosecution's expert testimony rendered any such efforts by defense counsel futile. This reasoning presumptively prioritizes quantity over quality, and takes no account of the comparative persuasiveness of the "child abuse" and "accidental fall" theories at issue in

house rather than the hospital. We do not disagree that the defendant's behavior was relevant and, furthermore, that a jury might consider it evidence of guilt. The probability that the jury would do so, however, might be said to make it even more critical that counsel counter the expert-endorsed theory of his client's guilt with an expert-endorsed theory of his client's innocence. Had counsel provided a different lens through which to view his client's behavior, those same "peculiar" actions by the defendant might have instead been perceived as the missteps of a panicked, but nonetheless innocent, caretaker.

⁹ For example, the Court of Appeals cited the child's hair loss as one physical manifestation of abuse, but according to her mother, her hair began thinning before the defendant moved in with the family. In any event, doctors diagnosed it as an infection, not a stress-related issue. The child's regression in toilet training was also emphasized as evidence of abuse. Yet a report from the child's pediatrician attributed her developmental progress, including the fact that she had even *begun* her toilet training, to the defendant's care. Unfortunately, defense counsel never called the child's pediatrician to testify, though these facts could have refuted the prosecution's allegations that the defendant had been physically abusing the child over a sustained period. Counsel's only "explanation" for this omission was that this credible counter-evidence was not needed because it did not fit in with his "trial strategy" of attributing the child's blunt force trauma to a fall from the bed.

the case. It also places the defendant in a near-impossible position, whereby the prejudice caused by his counsel's error is effectively used to foreclose his claim of relief based upon that very error. The prosecution's voluminous expert testimony made the need for an effective response by defense counsel particularly apparent and strong, and it rendered counsel's failure to offer expert testimony particularly glaring and harmful to the defendant. Because of counsel's omissions and the resulting absence of suitable expert assistance, the prosecution's expert testimony appeared uncontested and overwhelming. Contrary to the Court of Appeals, we believe this consequence militates in favor of, rather than against, the defendant's claim of relief.

The Court of Appeals' analysis thus vastly underestimated the value of expert assistance to the defense and the impact of its absence, ignoring the fact that in a SBS/AHT case such as this, where there is "no victim who can provide an account, no eyewitness, no corroborative physical evidence and no apparent motive to kill," the expert "is the case" Tuerkheimer, *The Next Innocence Project: Shaken Baby Syndrome and the Criminal Courts*, 87 Wash U L Rev 1, 27 (emphasis added). Here, expert testimony was not only integral to the prosecution's ability to supply a narrative of the defendant's guilt, it was likewise integral to the defendant's ability to counter that narrative and supply his own. Had an impartial, scientifically trained expert corroborated the defendant's theory, the defendant's account of the child's death would not have existed in a vacuum of his own self-interest. While we cannot say that a battle of the experts would have ensured the defendant's acquittal, counsel's failure to prepare or show up for the battle sufficiently "undermine[s our] confidence in the outcome" of this case to entitle the defendant to relief. *Strickland*, 466 US at 694.

IV. CONCLUSION

For the reasons set forth above, we conclude that the defendant is entitled to a new trial because of his counsel's constitutionally ineffective failure to investigate adequately and to attempt to secure appropriate expert assistance in the preparation and presentation of his defense. Accordingly, we reverse the judgment of the Court of Appeals, vacate the defendant's convictions, and remand to the Calhoun County Circuit Court for further proceedings consistent with this opinion.

Bridget M. McCormack
Robert P. Young, Jr.
Stephen J. Markman
Mary Beth Kelly
Brian K. Zahra
David F. Viviano
Richard H. Bernstein

Appendix C

PROBLEMS OF INFANT RETINO-DURAL HEMORRHAGE WITH MINIMAL EXTERNAL INJURY

A.N. Guthkelch*

This contribution is offered as a reflection—after 40 years' consideration—on a problem of potential child abuse which has caused a great deal of controversy since it was first described.¹ While controversy is a normal and necessary part of scientific discourse, there has arisen a level of emotion and divisiveness on shaken baby syndrome/abusive head trauma that has interfered with our commitment to pursue the truth.

What follows is a Serious Call—I borrow the phrase from the title of a religious treatise by the 18th century Protestant clergyman, William Law—to members of the medical and legal professions *to consider these problems with restraint*. It is, in short, a call for civility in scientific discourse.

* A. Norman Guthkelch is a retired professor of neurosurgery. He was a faculty member at the medical schools of, among others, the University of Hull, the University of Pittsburgh, and the University of Arizona, and a visiting professor at Harvard Medical College. He was honored for his contributions to the field of shaken baby syndrome/abusive head trauma at the Fourth National Conference on Shaken Baby Syndrome (2002) and the First European Conference on Shaken Baby Syndrome (2003).

1 A.N. Guthkelch, *Infantile Subdural Haematoma and its Relationship to Whiplash Injuries*, 2 *British Med. J.* 430-31 (1971).

**PROBLEM #1. IS THE NAME 'SHAKEN BABY SYNDROME'
JUSTIFIED BY THE FACTS?**

There is a serious epistemological difficulty here: one that seems not to have been clearly recognized. Of the several hundred syndromes in the medical literature, almost all are named either after their discoverer (e.g., Adie's Syndrome) or for a prominent clinical feature (e.g., Stiff Man Syndrome). In contrast, the appellation shaken baby syndrome (SBS) asserts a unique etiology (shaking). It also implies intent since it is difficult to 'accidentally' shake a baby. A newer term, abusive head trauma (AHT), implies both mechanism (trauma) and intent (abusive).

Since subdural and retinal hemorrhages (with or without cerebral edema) may also be observed in accidental or natural settings, I suggest that the elements of the classic triad of retinal hemorrhage, subdural hemorrhage and cerebral edema would be better defined in terms of their medical features. Since subdural hemorrhages in infancy originate in the dura, perhaps "retino-dural hemorrhage of infancy" would be an acceptable name for the primary findings. Other medical findings, e.g., cerebral edema, can be added to the title as appropriate. This would allow us to investigate causation without appearing to assume that we already know the answer.

**PROBLEM #2. CAN SHAKING CAUSE RETINO-DURAL
HEMORRHAGE IN INFANCY WITH MINIMAL EXTERNAL INJURY?
AND, IF SO, CAN ONE REASONABLY INFER SHAKING (OR OTHER
FORMS OF ABUSE) FROM THESE FINDINGS?**

If shaking is responsible for significant damage to the central nervous system and its coverings, one must ask why the forces generated by humans or laboratory machines shaking a dummy have so often proved insufficient to cause the disruption of these tissues. Uscinski (2004), quoting Ommaya (1968), reasoned that since there is "an injury threshold for neural tissue," and this cannot be attained by shaking alone, there must be an extra feature, such as impact, to

explain the findings.² This is consistent with the work of Duhaime and others.³ But this argument leaves out of account the biophysical differences between the mature and the infant brain, recently stressed by Squier and Mack.⁴ These include microscopic or submicroscopic damage, not only directly to the brain itself but also to the control of its blood vessels via the meningeal nerve-tissue.⁵ Such effects may be mediated by reflex mechanisms, causing infant susceptibility to hemorrhage from minor trauma or naturally occurring events.⁶ Such consequences would be difficult, if not impossible, to replicate in a laboratory model.

Since minor trauma may cause disproportionate harm to infants, it is appropriate to advise parents and caretakers not to shake babies, just as it is wise to advise them not to drop babies or to place them in positions from which they could fall or in which siblings or objects could fall on them. Such precautions recognize that babies are developmentally vulnerable, and that some may be more vulnerable than others.

It does not follow, however, that one can infer shaking (or any other form of abuse) from a finding of retino-dural hemorrhage in infancy. Tuerkheimer has pointed out the danger of assuming criminal intent simply because the classic triad of retino-dural hemorrhage and encephalopathy is present and no one can think of any other explanation.⁷ While society is rightly shocked by any assault on its weakest members and demands retribution, there seem to have been instances in which both medical science and the law have gone too far in hypothesizing and criminalizing alleged acts of violence in which the only evidence has been the presence of the

2 Ronald Uscinski, *Shaken Baby Syndrome*, 9 J. Am. Physicians Surgeons 76 (2004) (quoting A.K. Ommaya, Whiplash Injury and Brain Damage, 204 J. Am. Med. Assoc. 75 (1968)).

3 A.C. 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 184 (1992).

4 Waney Squier & Julie Mack, *The Neuropathology of Infant Subdural Haemorrhage*, 187 Forensic Sci. Int'l 6 (2009).

5 *Id.* at 8-10.

6 *Id.*

7 Deborah Tuerkheimer, *Science-Dependent Prosecution and the Problem of Epistemic Contingency: A Study of Shaken Baby Syndrome*, 3 Ala. L. Rev. 523 (2011).

classic triad or even just one or two of its elements. Often, there seems to have been inadequate inquiry into the possibility that the picture resulted from natural causes. In reviewing cases where the alleged assailant has continued to proclaim his/her innocence, I have been struck by the high proportion of those in which there was a significant history of previous illness or of abnormalities of structure and function of the nervous system, suggesting that the problem was natural or congenital, rather than abusive. Yet these matters were hardly, if at all, considered in the medical reports.

PROBLEM #3. CAN WE SAFELY ASSUME THAT THERE IS A CONSTANT RELATIONSHIP BETWEEN THE FORCE OF SHAKING AND/OR IMPACT AND THE RESULTING DAMAGE TO BODY TISSUES, PARTICULARLY THOSE OF THE NERVOUS SYSTEM AND ITS COVERINGS?

To raise this question does not suggest that *any* violence against a child is acceptable; it is not. But it *is* relevant in considering the extent to which a medical witness can quantify the degree of force required to cause specific medical findings, especially if the question invites a comparison to a high-speed motor vehicle crash or a fall from a high building or asks for an opinion on intent. Any medical expert who answers in the negative questions such as "Given the injuries that you have described in this case, doctor, have you any doubt that they were inflicted with intent to kill, or at least in total disregard of that possibility?" is exceeding his or her authority. New work by Squier and Mack on the neuropathology of the infant brain and its coverings emphasizes the complex relationships between the brain, the dura, and the thin-walled bridging veins that lead from the cerebral cortex to the dural venous sinuses.⁸ Given these complexities, we should not expect to find an exact or constant relationship between the existence or extent of retino-dural hemorrhage and the amount of force involved, let alone the state of mind of the perpetrator. Nor should we assume that these findings are caused by trauma, rather than natural causes.

⁸ *Supra* note 4 at 7.

In my 1953 article, I reviewed 24 cases of infantile subdural effusions, primarily subdural hematoma.⁹ In the article, I emphasized that infantile subdural effusions are not rare and that “the frequency with which [they] are found is proportional to the intensity with which they are sought.”¹⁰ Most of these cases occurred in the first few months of life (11 before age 3 months, 5 between 3 and 6 months, and 2 thereafter).¹¹ Seventy-five percent were associated with abnormal or difficult labor, and two cases were associated with a head injury two weeks prior.¹² Subdural effusions were also found in association with meningitis (5 cases) and venous sinus thrombosis, which may complicate any infective focus (1 case).¹³ Of those with known histories, there were two sets of twins and two premature babies.¹⁴ In 75% of the cases, the hematoma was surrounded by a membrane that was adherent to the dura, and in almost all instances the fluid obtained from subdural tap was xanthochromic, with a variable quantity of fresh blood, confirming that this process had been ongoing for some time, in many cases possibly from birth.¹⁵

In my 1971 article, I described two of my own cases involving potential shaking—one in which the mother had shaken the child to clear his throat during a fit of coughing since she feared he might choke, and another in which the mother admitted she and her husband “might have” shaken the baby when he cried at night.¹⁶ I also mentioned an earlier case in which Caffey described a mother who grabbed the arm of a baby who was rolling off a table to prevent his fall, jerking him in the process.¹⁷ In none of these cases was there any apparent malicious intent or evidence that the shaking was

9 A. N. Guthkelch, *Subdural Effusions in Infancy: 24 Cases*, 1 *British Med. J.* 233–39 (1953).

10 *Id.* at 233.

11 *Id.* at 236.

12 *Id.* at 234.

13 *Id.* at 236.

14 *Id.* at 234.

15 *Id.* at 235.

16 *Supra* note 1 at 431.

17 *Id.* at 430.

severe. While these events may have triggered a subdural hemorrhage or rehemorrhage, it is unwarranted to go from this possibility to the assumption that unexplained subdural hemorrhages, with or without retinal hemorrhage or encephalopathy, are caused by violent shaking or other forms of abuse.

Today, advanced radiological imaging is finding more and more subdural hemorrhages, with one study indicating that 46% of asymptomatic infants have subdural hemorrhages following normal births.¹⁸ This percentage is likely higher in symptomatic infants, following difficult births, or on pathology. While most birth-related subdural hemorrhages appear to resolve without symptoms, the babies who later become symptomatic may be the ones in which the birth hemorrhages reflected more serious underlying damage or became chronic, developing membranes that were subject to rebleeding. In such cases, the focus should not be on finding a "culprit" - or blaming the last person with the baby - but rather on the early identification of babies with pre-existing conditions and the development of treatment options.

PROBLEM 4. WHY IS IT IMPORTANT TO GET IT RIGHT, AND HOW SHOULD WE PROCEED?

When we make incorrect medical diagnoses, our advice and treatment is likely to be suboptimal or even harmful. This is particularly true in cases involving retino-dural hemorrhage of infancy, where a wrong diagnosis may send innocent parents and caretakers to prison. Since the reported conviction rate in fatal child abuse cases is 88%, this is not a trivial concern.¹⁹ A recent study further notes that five separate reports have found rates of AHT in the U.S. that are approximately double those in Canada, the U.K. and New Zealand, a striking difference that is not obviously attributable to differences in the actual incidence of abuse and may reflect a lack

18 V. Rooks et al., *Prevalence and Evolution of Intracranial Hemorrhage in Asymptomatic Term Infants*, 29 Am. J. Neuroradiology 1085 (2008).

19 D. Albert, et al, *Insuring Appropriate Expert Testimony for Cases Involving the "Shaken Baby,"* 308 JAMA 39 (2012).

of objectivity in diagnosis.²⁰ My own experience in reviewing a dozen cases in which the alleged perpetrator continued to assert his/her innocence “through thick and thin” suggests that a less tendentious assessment of the data gained in difficult – and often imperfectly documented – cases explains some of this disparity.

“Getting it right” requires that we distinguish between hypotheses and knowledge. SBS and AHT are hypotheses that have been advanced to explain findings that are not yet fully understood. There is nothing wrong in advancing such hypotheses; this is how medicine and science progress. It *is* wrong, however, to fail to advise parents and courts when these are simply hypotheses, not proven medical or scientific facts, or to attack those who point out problems with these hypotheses or who advance alternatives. Often, “getting it right” simply means saying, clearly and unequivocally, “we don’t know.”

In evaluating individual cases, it is also important not to rely too heavily on statistics, alluring as it might seem to do so. Statistics are helpful when we are dealing with relationships between well-defined populations, but this stage has not been achieved in the study of SBS/AHT. Instead, cases involving retino-dural hemorrhage of infancy encompass varying age groups, genetic characteristics, underlying conditions, and potential causes, including birth injuries, dehydration, metabolic disorders, illness and seizure disorders.

We must also consider the impact of even a relatively small percentage of misdiagnoses. Even if we knew that 90% of infant retino-dural hemorrhages were caused by abuse, the assumption of abuse could result in false accusations or convictions involving a large number of innocent parents or caretakers (up to 1 in 10 of the accused). This figure will increase if many or most retino-dural hemorrhages of infancy are attributable to accidents or natural causes, including birth injuries.

Given the importance of “getting it right,” the evidence base for SBS/AHT should be carefully and independently evaluated by scientists who are not involved in this controversy. Since the issue is not what the majority of doctors (or lawyers) think but rather what is

20 T. Fujiwara et al., *Using International Classification of Diseases, 10th Edition, Codes to Estimate Abusive Head Injuries in Children*, 43 Am. J. Preventative Med. 218 (2012).

supported by reliable scientific evidence, the evidence should be reviewed by individuals who have no personal stake in the matter, and who have a firm grounding in basic scientific principles, including the difference between hypotheses and evidence. It will not be easy to select such a group, but the effort will be justified in terms of justice and happiness.

In closing, may I be so bold as to suggest that as members of learned professions, we should never forget two dicta attributed to the great Canadian physician, William Osler, and his colleague Thomas McCrae? The first is this: "As is our pathology, so is our practice." The second is equally succinct: "More is missed by not looking than not knowing." Today, we need to develop a better understanding of the pathology of the infant brain and its coverings, and we need to look much more rigorously at the evidence. Only then will we be able to confidently correlate the medical findings with clinical symptoms and causes.

As C.P. Scott, the revered editor of the Manchester Guardian newspaper for most of the first half of the 20th century, used to say: "comment is free, facts are sacred." Over the past forty years, there has been much comment on retino-dural hemorrhage in infancy, but we have not yet determined all the facts. For that, we need new research on the pathology rather than a vain repetition of hypotheses. In obtaining a better understanding of the pathology, such research will assist in the earlier diagnosis and treatment of sick or injured children; it will also serve the cause of justice.

Appendix D

SHAKEN BABY SYNDROME, ABUSIVE HEAD TRAUMA, AND ACTUAL INNOCENCE: GETTING IT RIGHT

Keith A. Findley, Patrick D. Barnes, David A. Moran, and Waney Squier*

In the past decade, the existence of shaken baby syndrome (SBS) has been called into serious question by biomechanical studies, the medical and legal literature, and the media. As a result of these questions, SBS has been renamed abusive head trauma (AHT). This is, however, primarily a terminological shift: like SBS, AHT refers to the two-part hypothesis that one can reliably diagnose shaking or abuse from three internal findings (subdural hemorrhage, retinal hemorrhage, and encephalopathy) and that one can identify the perpetrator based on the onset of symptoms. Over the past decade, we have learned that this hypothesis fits poorly with the anatomy and physiology of the infant brain, that there are many natural and accidental causes for these findings, and that the onset of symptoms does not reliably indicate timing.

* Keith A. Findley is a Clinical Professor of Law, University of Wisconsin Law School, Co-Director of the Wisconsin Innocence Project, and President of the Innocence Network. Patrick D. Barnes is Chief, Section of Pediatric Neuroradiology and Co-Medical Director, MRI/CT Center, Lucile Salter Packard Children's Hospital, and Professor of Radiology, Stanford University Medical Center. David A. Moran is a Clinical Professor of Law, University of Michigan Law School and Co-Director, Michigan Innocence Clinic. Waney Squier is a Consultant Neuropathologist at the Oxford University John Radcliffe Hospital and Honorary Clinical Lecturer at the University of Oxford.

We wish to thank Geoffrey Kirkwood for his outstanding research assistance and thoughtful editorial suggestions.

In the last volume of this journal, Dr. Sandeep Narang marshaled the arguments and evidence that he believes support the diagnostic specificity of the medical signs that are used to diagnose SBS/AHT. Dr. Narang does not dispute the alternative diagnoses but nonetheless argues that, in the absence of a proven alternative, the SBS/AHT hypothesis is sufficiently reliable to support criminal convictions. The cited studies do not, however, support this position since they assume the validity of the hypothesis without examining it and classify cases accordingly, often without considering alternative diagnoses. To address this problem, Dr. Narang argues that, in diagnosing SBS/AHT, we should rely on the judgment of child abuse pediatricians and other clinicians who endorse the hypothesis. Reliance on groups that endorse a particular hypothesis is, however, antithetical to evidence-based medicine and Daubert, which require an objective assessment of the scientific evidence.

In the past decades, thousands of parents and caretakers have been accused—and many convicted—of abusing children based on a hypothesis that is not scientifically supported. While we must do everything in our power to protect children, we must refrain from invoking abuse as a default diagnosis for medical findings that are complex, poorly understood, and have a wide range of causes, some doubtlessly yet unknown. To this end, we are calling for collaboration between the medical and legal communities for the sole purpose of “getting it right.”

TABLE OF CONTENTS

I. INTRODUCTION	212
II. FROM SBS TO AHT: A DIAGNOSIS IN FLUX	218
A. A PLETHORA OF TERMS	218
1. Shaking	218
2. Shaken baby syndrome (SBS)	219
3. Shaken impact syndrome	219
4. Abusive head trauma	219
5. Blunt force trauma	220
6. Semantic confusion and the courts	220
B. A BRIEF HISTORY OF SBS / AHT	223
1. The origins	223
2. The warnings	227

3. 2001: a developing schism.....	229
4. A decade of debate.....	233
a. 2002 NIH conference.....	233
b. Biomechanics.....	236
c. SBS and evidence-based medicine.....	237
d. Alternative diagnoses.....	238
e. The position papers revisited.....	240
f. Increasing divergence.....	242
g. The triad: where are we now?.....	244
C. ONGOING DEBATES.....	245
1. Short falls.....	245
2. Timing (“lucid intervals”).....	249
3. Retinal hemorrhages.....	251
4. Bruises, fractures and other findings.....	254
5. Confessions.....	256
6. New hypotheses.....	261
III. THE MEDICAL EVIDENCE: OLD AND NEW.....	263
A. LITERATURE SUPPORTING THE AHT DIAGNOSIS.....	263
1. The methodology.....	264
2. The evidence.....	265
a. Duhaime (1992).....	267
b. Ewing-Cobbs (1998).....	268
c. Feldman (2001).....	268
d. Wells (2002).....	269
e. Bechtel (2004).....	269
f. Hobbs (2005).....	270
g. Vinchon (2005).....	270
h. Matschke (2009).....	271
i. Vinchon (2010).....	272
j. Other studies.....	273
3. The Flaws.....	273
a. Circularity.....	274
b. Rule-out diagnoses.....	276
c. Clinical judgment.....	280
d. Observer bias.....	281
e. Reversing the burden of proof.....	285

f. Interpretive errors	286
i. P-values.....	286
ii. The Prosecutor's Fallacy.....	287
iii. Improper classifications	290
B. THE SKEPTICS: NEW RESEARCH, OLD ANATOMY	291
1. Studies that identify the lack of support for the traditional SBS hypothesis.....	291
2. Studies that identify problems with the SBS/AHT hypothesis	292
3. A shifting paradigm.....	296
IV. MEDICAL AND LEGAL STANDARDS OF RELIABILITY	298
A. MEDICAL DIAGNOSIS: ART OR SCIENCE?	299
B. DAUBERT: IS SBS/AHT READY FOR THE COURTROOM?	302
C. THE COSTS OF MISDIAGNOSIS	306
V. THE PATH FORWARD	306
A. RESEARCH	307
B. WORKING TOGETHER.....	308
C. ACKNOWLEDGING THE COMPLEXITIES.....	309
D. WORKING UNDER CONDITIONS OF UNCERTAINTY.....	312

I. INTRODUCTION

For decades, shaken baby syndrome (SBS) was an accepted medical and legal diagnosis. As the shaking mechanism came into serious question, SBS was renamed abusive head trauma (AHT). Regardless of terminology, SBS/AHT refers to the two-part medicolegal hypothesis that, in the absence of a confirmed alternative explanation, one can reliably diagnose shaking or abuse from three internal findings—subdural hemorrhage, retinal hemorrhage, and encephalopathy (brain abnormalities and/or neurological symptoms), and that one can identify the perpetrator based on the onset of symptoms. Because the consequences of an SBS/AHT diagnosis can devastate children and families, it is critical to assess the reliability of the diagnosis under the standards of evidence-based medicine¹ and *Daubert v. Merrell Dow Pharmaceuticals, Inc.*² Dr.

¹ See, e.g., Connie Schardt & Jill Mayer, Tutorial for an *Introduction to Evidence-Based Practice*,

Sandeep Narang's article in this journal identifies the research basis for the SBS/AHT hypothesis and the applicable medicolegal standards.³ However, in concluding that the SBS/AHT hypothesis meets the standards of evidence-based medicine and *Daubert*, the article neglects the underlying flaws in the supporting research and the shift in our understanding of the science over the past decade.

For all the heat in the debates about the validity of SBS/AHT, there is in reality a growing, if frequently unexpressed, consensus on the nature of the problem and the flaws in the hypothesis. Today, there is general agreement that child abuse was historically under-recognized and that abuse can produce subdural hemorrhage, retinal hemorrhage, and brain damage—the “triad” of medical findings that has traditionally been used to confirm shaking or other forms of abuse.⁴ There is also general agreement that violently shaking a child is unacceptable and could cause serious injury or even death.⁵ At the same time, there is now widespread, if not universal, agreement that the presence of the triad alone—or its individual components—is not enough to diagnose abuse. In the United Kingdom, the Crown Prosecution Service Guidelines of March 2011 endorsed this view,⁶

U.N.C. HEALTH SCI. LIBR. (2010), <http://www.hsl.unc.edu/services/tutorials/ebm/index.htm>; Gordon H. Guyatt et al., *Users' Guides to the Medical Literature XXV, Evidence-Based Medicine: Principles for Applying the Users' Guides to Patient Care*, 284 J. AM. MED. ASS'N. 1290 (2000).

² 509 U.S. 579 (1993).

³ Sandeep Narang, *A Daubert Analysis of Abusive Head Trauma/Shaken Baby Syndrome*, 11 HOUS. J. HEALTH L. & POL'Y 505, 506-07, 539-60 (2011).

⁴ See, e.g., *id.* at 523, 569-29, 570.

⁵ See, e.g., Emily Bazelon, Mary Case, Christopher Greeley, Ronald H. Uscinski, Waney Squier, Round Table Discussion: Anatomy of an AHT Diagnosis, Investigation and Prosecution, 2011 New York City Abusive Head Trauma/Shaken Baby Syndrome Training Conference (Sept. 23, 2011) (notes on file with authors) (all participants agreed that violent shaking is dangerous and may injure or kill an infant); Kay Rauth-Farley, et. al., *Current Perspectives on Abusive Head Trauma*, in ABUSIVE HEAD TRAUMA IN INFANTS AND CHILDREN; A MEDICAL, LEGAL, AND FORENSIC REFERENCE 1, 1 (G.W. Med. Publ'g 2006) (“It is widely accepted that shaking a young child or infant is dangerous”).

⁶ *Non Accidental Head Injury Cases (NAHI, formerly referred to as Shaken Baby Syndrome [SBS]) - Prosecution Approach*, CROWN PROSECUTION SERVICE (March 24, 2011), http://www.cps.gov.uk/legal/1_to_o/non_accidental_head_injury_cases/ (“it is unlikely that a charge for a homicide (or attempted murder or assault) offense could be justified where the only evidence available is the triad of pathological features.”).

while in the U.S., the diagnostic specificity of the “triad” was recently described as a “myth” by a leading proponent of the SBS/AHT hypothesis.⁷ As we develop more fully below, there is also a growing consensus that certain features of the diagnosis were inaccurate, including some that were frequently used to obtain criminal convictions. For example, it is no longer generally accepted that short falls can never cause the triad, that there can be no period of lucidity between injury and collapse (a key element in identifying the perpetrator), or that massive force—typically described as the equivalent of a multi-story fall or car accident—is required.⁸

As Dr. Narang points out, the list of alternative causes for the triad or its components is now so broad that it cannot be addressed in a single article.⁹ One of the child abuse textbooks recommended by Dr. Narang lists the differential diagnosis (alternative causes or “mimics”) as: prenatal and perinatal conditions, including birth trauma; congenital malformations; genetic conditions; metabolic disorders; coagulation disorders; infectious disease; vasculitis and autoimmune conditions; oncology; toxins and poisons; nutritional deficiencies; complications from medical-surgical procedures, including lumbar puncture; falls; motor vehicle crashes; and playground injuries.¹⁰ In all likelihood, other causes are still

⁷ Carole Jenny, Presentation on *The Mechanics: Distinguishing AHT/SBS from Accidents and Other Medical Conditions*, slide 33, 2011 New York City Abusive Head Trauma/Shaken Baby Syndrome Training Conference (Sept. 23, 2011), (powerpoint available at http://www.queensda.org/SBS_Conference/SBC2011.html).

⁸ See *infra* notes 125, 130-131, 145 and accompanying text.

⁹ Narang, *supra* note 3, at 507, note 13 (“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”). See also *id.* at Appendix B (differential diagnosis for subdural hemorrhage includes inflicted trauma, accidental trauma, birth trauma, metabolic disease, nutritional deficiencies, genetic syndromes, clotting disorders, tumors and infection) and Appendix C (differential diagnosis for retinal hemorrhage include all of the diagnoses for subdural hemorrhage as well as anemia, carbon monoxide poisoning, vasculitis, hypoxia, hypotension, hypertension, papilledema, and increased intracranial pressure); Julian T. Hoff et al., *Brain Edema*, 22 NEUROSURG.NEUROSURGICAL FOCUS, MAY 2007, at 1 (causes of brain edema include trauma, stroke and tumors).

¹⁰ Andrew P. Sirotnak, *Medical Disorders that Mimic Abusive Head Trauma*, in ABUSIVE HEAD TRAUMA IN INFANTS AND CHILDREN: A MEDICAL, LEGAL, AND FORENSIC REFERENCE 191-226 (G.W. Med. Publ'g 2006); M. Denise Dowd, *Epidemiology of Traumatic Brain Injury*:

undiscovered.¹¹ Like Dr. Narang, we refer the reader to the literature for a discussion of the alternative causes.¹²

Given this emerging consensus, our disagreement with Dr. Narang is narrow but critical. Since biomechanical studies have consistently concluded that shaking does not generate enough force to produce the types of traumatic damage associated with SBS/AHT, particularly in the absence of neck damage, Dr. Narang does not defend shaking as a mechanism or argue that there are no diagnostic alternatives. Instead, as is typical in the current debates about these issues, he contends that the less-specific diagnosis of AHT is supported by current medical science when subdural and retinal hemorrhage are identified and other known causes ruled out.¹³

Changing the name of the syndrome from SBS to AHT does not, however, resolve the disagreement. In describing AHT, Dr. Narang does not offer new evidence but instead relies on the assumptions that provided the basis for the SBS hypothesis.¹⁴ This hypothesis assumed that each element of the triad was, virtually by definition, traumatic, *i.e.*, that subdural and retinal hemorrhages were caused by the traumatic rupture of bridging veins and retinal blood vessels and that encephalopathy was caused by the traumatic rupture of axons

Recognizing Unintentional Head Injuries in Children, in *ABUSIVE HEAD TRAUMA IN INFANTS AND CHILDREN: A MEDICAL, LEGAL, AND FORENSIC REFERENCE* 11-14 (G.W. Med. Publ'g 2006).

¹¹ We are, for example, just beginning to identify the many variations of the human genome, the thousands of metabolites and enzymes that must function properly to sustain life, and the unique anatomic and physiological characteristics of the infant brain.

¹² In 2011, two of the co-authors of this article—Dr. Barnes and Dr. Squier—addressed the differential diagnoses in major invited reviews of the medical evidence on SBS/AHT in the fields of pediatric neuroradiology and pediatric neuropathology, their own specialties. Patrick D. Barnes, *Imaging of Nonaccidental Injury and the Mimics: Issues and Controversies in the Era of Evidence-Based Medicine*, 49 *RADIOLOGIC CLINICS N. AM.* 205 (2011); Waney Squier, *The “Shaken Baby” Syndrome: Pathology and Mechanisms*, 122 *ACTA NEUROPATHOLOGICA* 519 (2011). For a more complete discussion of the literature, we refer the readers to these reviews and to the articles cited by Dr. Narang.

¹³ Narang, *supra* note 3, at 570-73.

¹⁴ In describing AHT causation, Dr. Narang relies upon the classic SBS hypothesis, with no reference to the more recent literature (discussed below). *See, e.g., id.* at 541 (“In inertial [*i.e.* shaking] 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”); *id.* at 553-54 (“[S]everal lines of research and analysis point towards acceleration-deceleration forces at the vitreo-retinal interface...as the causative mechanism for severe [retinal hemorrhages]”).

(the nerve fibers that connect the cells throughout the brain). We now know, however, that the triad does not necessarily or generally reflect the traumatic rupture of bridging veins or retinal blood vessels; that the encephalopathy virtually always reflects hypoxia-ischemia (lack of oxygen) rather than the traumatic tearing of axons; and that the triad can also result from natural disease processes and accidents.¹⁵ Consequently, it is no longer valid to reason backwards from the triad to a diagnosis of trauma or abuse.

The AHT label also raises new problems. Without an identified mechanism, it is not possible for biomechanical engineers to reconstruct or for doctors, judges or juries to critically evaluate the proposed mechanism or mechanisms. The AHT label does not, moreover, address the more recent criticisms of SBS/AHT, which have shifted from biomechanics to the unique characteristics of the developing brain. Finally, like the SBS label, the AHT label subsumes the answer to the question “what causes the triad or its elements” within its very name, making it difficult to discuss the issues objectively.

Since the existing evidence does not meet the standards of evidence-based medicine and we cannot ethically experiment with babies, Dr. Narang suggests that we rely on the “clinical judgment” of the doctors, particularly child abuse pediatricians, who endorse the SBS/AHT hypothesis and defer to the literature that assumes the accuracy of their judgments.¹⁶ As a practical matter, this would shield the SBS/AHT hypothesis from the scientific scrutiny envisioned by evidence-based medicine and *Daubert* and eliminate any claim that the hypothesis has been scientifically validated. We suggest that this approach also violates the medical and legal

¹⁵ See, e.g., *infra* notes 68-71, 74, 105, 107, 109.

¹⁶ Narang *supra* note 3, at 580-82 (arguing that the relevant scientific community be limited to those who have obtained subspecialty certification or are eligible for subspecialty certification in the field of child abuse pediatrics). This certification program, which was created by leading advocates of the SBS/AHT hypothesis, incorporates the traditional SBS/AHT hypothesis into its curriculum. See Am. Bd. of Pediatrics Subboard Child Abuse Pediatrics, *Content Outline: Child Abuse Pediatrics: Subspecialty In-Training, Certification and Maintenance of Certification Examinations* (last revised Nov. 2010), <https://www.abp.org/abpwebsite/takeexam/subspecialtycertifyingexam/contentpdfs/chab.pdf>; Robert W. Block & Vincent J. Palusci, *Child Abuse Pediatrics: A New Pediatric Subspecialty*, 148 J. PEDIATRICS 711(2006).

precepts of “first do no harm” and “innocent until proven guilty.”

While child abuse that results in neurological damage or death is horrific, particularly when committed by parents and caretakers who literally hold in their hands the lives of their infants, we have learned from the daycare cases of the 1980s and 1990s that the strong emotions that accompany allegations of child abuse can increase the likelihood of false convictions.¹⁷ In a 1990 symposium on pretrial publicity, Judge Abner Mivka, a highly respected member of the U.S. Court of Appeals for the District of Columbia, observed:

I do not think you can get a fair child abuse trial before a jury anywhere in the country. I really don't... I don't care how sophisticated or smart jurors are, when they hear that a child has been abused, a piece of their mind closes up, and this goes for the judge, the juror, and all of us.¹⁸

Given these dangers, it is critical to carefully assess the quality of the evidence used to diagnose child abuse and to make clear the extent to which the diagnosis rests on hypotheses or personal opinion rather than scientific knowledge. This is particularly important when judges and jurors are being asked to render judgments on unresolved and highly controversial issues in complex areas of medicine.

In Part II, we briefly review the changes in the SBS/AHT hypothesis over the past decade and identify the issues that are currently the subject of debate. The shifts can be captured in a sentence: since 2000, we have learned that much of what we thought we knew was wrong. In Part III, we examine the quality of the research that Dr. Narang cites to support the SBS/AHT hypothesis as well as the research that casts doubt on this hypothesis. In Part IV, we apply the applicable medical and legal standards to this research. In Part V, we suggest a path forward to help us better differentiate between child abuse and the wide array of accidental and natural

¹⁷ See, e.g., DOROTHY RABINOWITZ, *NO CRUELER TYRANNIES: ACCUSATION, FALSE WITNESS, AND OTHER TERRORS OF OUR TIMES* (1st ed. Free Press 2003) (reporting on daycare, Wenatchee and other child sex abuse scandals of the 1980s and 1990s); Maggie Jones, *Who Was Abused?*, N. Y. TIMES, Sept. 19, 2004, <http://query.com/gst/fullpage.html?res=9F03EFD61330F93AA2575AC0A9629C8B63&scp=1&sq=maggie+jones+who+was+abused&st=cse&pagewanted=1> (reporting on Bakersfield scandals); *Summary of the Cleveland Inquiry*, 297 BRIT. MED. J. 190 (1988).

¹⁸ Forum, *Panel One: What Empirical Research Tells Us, and What We Need to Know About Juries and the Quest for Impartiality*, 40 AM. U. L. REV. 547, 564-65 (1991).

causes that may produce the same or similar findings. It is our hope that Dr. Narang and others will join us in this endeavor to “get it right.”

II. FROM SBS TO AHT: A DIAGNOSIS IN FLUX

Our increased understanding of the infant brain and the biomechanics of injury is reflected in an evolving terminology that acknowledges the flaws in the original SBS hypothesis.¹⁹ Despite widespread acknowledgement of these flaws, the new terminology, AHT retains the automatic diagnosis of abuse for the medical findings previously attributed to shaking and rests on the same assumptions as SBS, many of which have been discredited or disproven.²⁰ After clarifying the terminology, we discuss the shifts in the literature that resulted in the new terminology. We then identify the areas of current agreement and debate.

A. A Plethora of Terms

In addressing the changes in the SBS/AHT hypothesis, it is important to distinguish between five terms and diagnoses: “shaking,” “shaken baby syndrome,” “shaken impact syndrome,” “abusive head trauma,” and “blunt force trauma.” Much of the disagreement in this area reflects the confusion of these terms and conflation of the underlying concepts.

1. *Shaking.*

“Shaking” refers to the physical act of shaking a child, irrespective of injury. Shaking to punish or in frustration is always inappropriate. In infants with large heads and weak necks—or even in older children—violent shaking may lead to disastrous consequences, particularly in a child with predisposing factors.

¹⁹ See e.g., *infra*, notes 55, 68-70, 94-95.

²⁰ See, e.g., *infra* notes 55, 68-71, 74, 94-95.

2. *Shaken baby syndrome.*

“Shaken baby syndrome” (SBS) refers to the hypothesis that violent shaking may be reliably diagnosed based on the triad of subdural hemorrhage, retinal hemorrhage, and encephalopathy (brain damage) if the caretakers do not describe a major trauma (typically described as equivalent to a motor vehicle accident or fall from a multistory building) and no alternative medical explanation is identified. Under this hypothesis, the rapid acceleration and deceleration of shaking causes movement of the brain within the skull, resulting in the traumatic rupture of bridging veins, retinal blood vessels, and nerve fibers throughout the brain (diffuse axonal injury). This hypothesis came into question when biomechanical studies consistently concluded that shaking generated far less force than impact, did not meet established injury thresholds, and would be expected to injure the neck before causing bridging vein rupture or diffuse axonal injury.

3. *Shaken impact syndrome.*

“Shaken impact syndrome” was advanced to address the biomechanical criticisms of shaking as a causal mechanism for the triad. Under this hypothesis, subdural hemorrhage, retinal hemorrhage, and encephalopathy were attributed to shaking followed by impact, such as tossing or slamming the child onto a hard or soft surface. If there were no bruises or other signs of impact, it was hypothesized that the child was thrown onto a soft surface, such as a mattress or pillow.

4. *Abusive head trauma.*

As shaking came under increasing scrutiny, a plethora of new terms arose that did not invoke shaking as a mechanism.²¹ At

²¹ These terms include “intentional traumatic brain injury (iTBI),” “nonaccidental injury (NAI),” “nonaccidental head injury (NAHI),” “nonaccidental trauma (NAT),” “inflicted neurotrauma” and “abusive head trauma (AHT)”. See Narang, *supra* note 3, at 505 (Abusive Head Trauma (AHT) has been known over the years by multiple terms, including Whiplash Shaken Baby Syndrome, Shaken Impact Syndrome, Inflicted Childhood Neurotrauma and

present, the most popular replacement term—and the term used by Dr. Narang—is abusive head trauma, or AHT. AHT refers to any deliberately inflicted injury to the head, regardless of mechanism. In 2009, the American Academy of Pediatrics recommended that pediatricians use this term instead of SBS but endorsed shaking as a plausible mechanism based on confession evidence.²² AHT also includes hitting the child on the head, crushing the child, throwing the child onto a hard or soft surface, or any other conceivable manner of harming the head. Under the AHT hypothesis, such acts may be inferred from the triad of findings previously attributed to shaking, with or without other evidence of trauma, at least in the absence of another acceptable explanation. Used in this sense, AHT is most often used by pediatricians.

5. *Blunt force trauma.*

Blunt force trauma to the head refers to any impact that does not penetrate the scalp, including accidents (e.g., falls onto the floor or other surfaces) and abuse (e.g., hitting the child on the head or throwing the child on the floor). This term does not imply intent and is used in cases with skull fractures and bruises as well as in cases that rely primarily or exclusively on the triad. This term is most often used by forensic pathologists.

6. *Semantics and the courts.*

As reflected in Dr. Narang's article, the trend in recent years has been to move away from terms involving shaking towards generalized terms such as AHT, which avoids the criticisms of shaking by relying upon an undetermined mechanism. Without a defined mechanism, however, it is difficult for parents or caretakers to defend themselves. How does one defend against an unknown mechanism, particularly one that leaves no clues as to its cause? In effect, by changing the name, supporters of the AHT hypothesis continue to rely on traditional SBS assumptions—specifically, the

Non-Accidental Trauma; to the lay public, it is most commonly recognized as Shaken Baby Syndrome (SBS).)

²² Cindy W. Christian, et al., *Abusive Head Trauma in Infants and Children*, 123 PEDIATRICS 1409, 1409-11 (2009).

assumption that the triad findings are caused largely or entirely by trauma—while discarding the shaking mechanism, producing what may be viewed as a medicolegal “bait and switch.”

When combined with unfamiliar medical concepts, these terminological shifts can result in considerable confusion, even at the level of the U.S. Supreme Court. This confusion is exemplified by the U.S. Supreme Court decision in *Cavazos v. Smith*.²³ In *Smith*, a California grandmother with no history of abuse or neglect was convicted of causing the death of her 7-week-old grandson by violent shaking.²⁴ This was not a classic SBS/AHT case since the child had minimal subdural/subarachnoid hemorrhage with no retinal hemorrhage or brain swelling—there were no fractures, no sprains, and no other indicia of trauma other than a “tiny” abrasion and corresponding bruise, which the prosecution’s medical expert agreed did not produce brain trauma.²⁵ The state’s experts testified nonetheless that the death was consistent with violent shaking that caused the brain or brainstem—not just the bridging veins and axons—to tear in vital areas, however, the Ninth Circuit overturned the conviction, stating that there was “no physical evidence of . . . tearing or shearing, and no other evidence supporting death by violent shaking.”²⁶ A 6-3 majority of the Supreme Court reversed the Ninth Circuit, stating that the Ninth Circuit’s assertion that “there was no evidence in the brain itself of the cause of death” was “simply false” and there “was ‘evidence in the brain itself.’”²⁷ In support of this claim, the majority cited evidence of subdural, subarachnoid, optic nerve and interhemispheric bleeding.²⁸ However, these findings are *outside* the brain and are associated with a multitude of nontraumatic causes.²⁹ The majority went on to say that “[t]hese affirmative indications of trauma formed the basis of the

²³ 132 S. Ct. 2 (2011) (per curiam).

²⁴ *Id.* at 3-5

²⁵ *Id.* at 9 (Ginsburg, Breyer & Sotomayor, JJ. dissenting).

²⁶ *Id.* at 5-6 (quoting *Smith v. Mitchell*, 437 F.3d 884, 890 (9th Cir. 2006)).

²⁷ *Id.* at 7. (emphasis in original).

²⁸ *Id.*

²⁹ See, e.g., *infra* notes 105, 107, 109, 154, 155; Narang, *supra* note 3, at Appendices B and C; Sirotnak, *supra* note 10, at 193-214.

experts' opinion that [the child] died from shaking so severe that his brainstem tore."³⁰ The autopsy did not, however, find any tears in the brainstem, which was not examined microscopically since the pathologists felt they "'wouldn't have seen anything anyway.'"³¹ In short, the Supreme Court was willing to send Ms. Smith—a grandmother described as "warm hearted, sensitive, and gentle"—back to prison to serve a sentence of 15 years to life based on an injury no one could find.³² Ultimately, given the doubts about guilt, the majority suggested that clemency might be appropriate. Governor Brown granted clemency on April 6, 2012.³³

To understand how we got to the point where invisible injuries are acceptable as proof beyond a reasonable doubt of murder, one must understand the history of SBS/AHT.

³⁰ *Id.*

³¹ *Id.* at *9 (Ginsburg, Breyer & Sotomayor, JJ. dissenting) (quoting Tr. 803, 1299).

³² *Id.* at *10-*11. This case was not so much an endorsement of the SBS hypothesis as an expression of the deference the law gives to evidence accepted by a jury, including medical opinions—even speculative and unproven ones—in criminal cases. The majority emphasized that it was bound by legal principles requiring deference to jury verdicts, especially in federal habeas corpus review of state court convictions. *Id.* at *6-*7 (per curiam). To the extent the Court commented on the science, it suggested there was indeed considerable reason to doubt the medical opinions and conviction. *Id.* at *4-*6. The dissent pointed out expressly that changes in the medical literature since the child's death in 1996 cast considerable doubt on the conviction and the SBS theories underlying it. *Id.* at *10-*11 (Ginsburg, Breyer & Sotomayor, JJ. dissenting). Even the majority acknowledged, "[d]oubts about whether Smith is in fact guilty are understandable," and lamented that "the inevitable consequence of this settled law [of deference to juries] is that judges will sometimes encounter convictions they believe to be mistaken, but they must nonetheless uphold." *Id.* at *4, *7.

³³ Carol J. Williams, *Brown Commutes Sentence of Woman Convicted of Killing Grandson*, L. A. TIMES, Apr. 7, 2012, <http://articles.latimes.com/2012/apr/07/local/la-me-shaken-baby-clemency-20120407>. In a review of the medical evidence prior to the grant of clemency, a pathologist at the Los Angeles County coroner's office described eight "diagnostic problems" with the coroner's original ruling that the child had died from violent shaking or a blow to the head. He wrote that the "conservative approach would be to acknowledge these unknowns. The cause of death should be diagnosed as undetermined." See also Joseph Shapiro & A.C. Thompson, *New Evidence in High-Profile Shaken Baby Case*, NAT'L PUB. RADIO, Mar. 29, 2012, <http://www.npr.org/2012/03/29/149576627/new-evidence-in-high-profile-shaken-baby-case>.

B. A Brief History of SBS/AHT

1. *The Origins.*

For time immemorial, seemingly healthy infants have collapsed or died without any known medical explanation.³⁴ In the early 1970s, Dr. Guthkelch (a British neurosurgeon) and Dr. Caffey (an American pediatric radiologist) suggested that shaking might explain the unexpected collapse or death of a subset of infants who presented with subdural hemorrhage but typically had no external signs of injury.³⁵ While shaking was at that time viewed as benign—in one of Dr. Guthkelch's examples, the parent was attempting to save a child from choking—Dr. Guthkelch was concerned that the whiplash effect of shaking could produce subdural hematomas in infants, especially given their weak neck muscles and relatively large heads.³⁶ In 1974, Dr. Caffey described a two-part sequence in which shaking causes an infant's head to strike its chest and back in "rapid, repeated, to-and-fro, alternating, acceleration-deceleration flexions."³⁷ Like Dr. Guthkelch, Dr. Caffey was concerned that parents and caretakers did not realize the dangers of shaking, and he recommended a nationwide education campaign to warn of the potential consequences of any action in which the heads of infants were jerked and jolted.³⁸

Over the years, the shaking/whiplash hypothesis evolved into the medicolegal hypothesis of "shaken baby syndrome."³⁹ This

³⁴ See, e.g., D. L. Russell-Jones, *Sudden Infant Death in History and Literature*, 60 ARCHIVES OF DISEASE IN CHILDHOOD 278 (1985).

³⁵ See A. N. Guthkelch, *Infantile Subdural Haematoma and its Relationship to Whiplash Injuries*, 2 BR. MED. J. 430 (1971); see also John 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, 401 (1974).

³⁶ See Guthkelch, *supra* note 35, at 431. As Dr. Guthkelch recently told NPR, at that time in Northern England, parents sometimes punished their children by shaking them, which was considered socially acceptable. See also Joseph Shapiro, *Rethinking Shaken Baby Syndrome*, NAT'L PUB. RADIO, June 29, 2011, <http://www.npr.org/2011/06/29/137471992/rethinking-shaken-baby-syndrome>.

³⁷ Caffey, *supra* note 35, at 401.

³⁸ *Id.* at 402-403.

³⁹ See generally Brian Holmgren, *Prosecuting the Shaken Infant Case*, in THE SHAKEN BABY SYNDROME: A MULTIDISCIPLINARY APPROACH 275 (Stephen Lazowitz & Vincent J. Palusci eds.,

hypothesis held that shaking may cause a “triad” of medical findings—subdural hemorrhage, retinal hemorrhage and encephalopathy (brain damage)—and that in the absence of other known explanations, it may be safely inferred from these findings that the child has been shaken.⁴⁰ While this conclusion was sometimes supported by other signs of physical injury, such as bruises or fractures, there were often no signs of trauma.⁴¹ In other cases, only one or two elements of the triad were present.⁴²

In the absence of other signs of trauma, the diagnosis was based on the belief that the triad elements were in and of themselves traumatic in origin.⁴³ Specifically, subdural hemorrhages were attributed to the traumatic rupture of the bridging veins that convey blood from the brain to the large veins (or sinuses) in the fibrous dura lining the skull.⁴⁴ Retinal hemorrhages were similarly attributed to the traumatic rupture of retinal blood vessels, while encephalopathy (brain damage) was attributed to the traumatic rupture of the axons (nerve fibers) that connect the nerve cells throughout the brain.⁴⁵ Because the brain damage was often bilateral and widespread, it was assumed the force needed to cause these findings was comparable to, or greater than, that found in multistory falls or motor vehicle

2001) (outlining the prosecution of SBS in criminal cases).

⁴⁰ See *id.* at 306 (Stephen Lazoritz & Vincent J. Palusci eds., 2001) (“retinal hemorrhages, bilateral subdural hematoma, and diffuse axonal injury are highly specific for SBS as a mechanism”).

⁴¹ *Id.*

⁴² See, e.g., Cavazos, 123 S.Ct. at 3 (affirming conviction in case involving “minimal subdural and subarachnoid hemorrhaging” but no retinal hemorrhages or brain swelling); Hess v. Tilton, CIV S-07-0909 WBSEFB, 2009 WL 577661 (E.D. Cal. Mar. 5, 2009) (affirming conviction in case involving brain swelling and retinal hemorrhages but no subdural hemorrhage), report and recommendation adopted, CIVS070909WBSEFBP, 2009 WL 800156 (E.D. Cal. Mar. 25, 2009).

⁴³ See Mary E. Case et al., *Position Paper on Fatal Abusive Head Injuries in Infants and Young Children*, 22 AM. J. FORENSIC MED. PATHOL. 112 (2001).

⁴⁴ See *id.* at 114-15.

⁴⁵ See *id.* at 113-14, 117-118 (describing shear injury with tearing of axonal processes); 116 (presence of retinal hemorrhages highly correlates with rotational head injury; potential mechanisms include increased intracranial pressure, direct trauma to retina, and traction caused by the vitreous pulling away from the retina).

accidents.⁴⁶ Thus, if the history provided by the caretakers did not include a major accident, the history was considered to be inconsistent with the findings, and abuse was considered to be the only plausible explanation.⁴⁷ In children who had no external signs of trauma, it was further hypothesized that the abuse must have consisted of violent shaking.⁴⁸

A corollary of the SBS hypothesis—and one that was particularly important for the legal system—was that the injury could be timed and the perpetrator identified based solely on the medical findings.⁴⁹

⁴⁶ See *id.* at 120 (“fatal accidental shearing or diffuse brain injuries require such extremes of rotational force that they occur only in obvious incidents such as motor vehicle accidents. Besides vehicular accidents, other fatal accidental childhood head injuries tend to involve crushing or penetrating trauma, which is readily evident. These injuries tend to be the result of falling from considerable heights (greater than 10 feet) or having some object penetrate the head”); compare Alex Levin et al., Clinical Statement, *Abusive Head Trauma/Shaken Baby Syndrome*, AM. ACADEMY OPHTHALMOLOGY, (MAY 2010), available at http://one.aao.org/ce/practiceguidelines/clinicalstatements_content.aspx?cid=914163d5-5313-4c23-80f1-07167ee62579 (retinal hemorrhages typical of AHT/SBS are uncommon in severe accidental head trauma such as falls from a second-story level or a motor vehicle collision).

⁴⁷ For example, Edward J. Imwinkelried, *Shaken Baby Syndrome: A Genuine Battle of the Scientific (and Non-Scientific) Experts*, 46 CRIM. L. BULL. 156 (2010) and cases cited therein note that “the most common analogies [used by prosecution experts] are to the amount generated by high speed automobile accidents and a fall from a several-story building. The experts analogize to these “real-life accident scenarios” in order to give the trier of fact a sense of the ‘massive, violent’ force required to produce this kind of brain injury”; cited cases include *Mitchell v. State*, No. CACR 07-472, 2008 WL 316166 (Ark. Ct. App. Feb. 6, 2008) (examining pediatrician equated the force necessary to produce the triad with that of a high-speed automobile accident); *People v. Dunaway*, 88 P.3d 619, 631, 632 (Colo. 2004) (prosecution expert stated that subdural hemorrhages occur in “such things as falling from a several story building or being in a high speed motorcycle accident or a child say is on a bicycle hit by a car...when we see subdurals in accidental injury, it’s from a major trauma. It requires massive force”); *In re Matter of Child*, 880 N.Y.S. 2d 760 Fam. Ct. 2008) (prosecution expert stated that SBS findings “simulate being in a car crash at ‘around 35 to 40 miles per hour’”). Such testimony is similar to the sample closing arguments provided to prosecutors. See, e.g., Brian K. Holmgren, *supra* note 39 at 325 (the evidence tells us that the amount of force visited on little Bobby was the equivalent of a fall from several stories onto a hard surface or an unrestrained motor vehicle collision at a speed of 50-60 m.p.h.; force equivalent to at least 100-200G’s). It does not, however, reflect the actual forces of manual shaking, which are less than a fall from a sofa or from the chest level of an adult. See *infra*, note 95.

⁴⁸ Imwinkelried, *supra* note 47.

⁴⁹ See Deborah Tuerkheimer, *The Next Innocence Project: Shaken Baby Syndrome and the Criminal Courts*, 87 WASH. UNIV. L. REV. 1, 5, 18 (2011) (noting “(u)nequivocal testimony regarding

Since the damage caused by the traumatic rupture of nerve fibers throughout the brain would be devastating with immediate loss of function (as in concussion), there could be no period of relative normality ("lucid interval") following the injury.⁵⁰ It was therefore widely accepted that the last person with the baby must have been responsible.⁵¹ In effect, SBS quickly became a criminal category of *res ipsa loquitur* cases, *i.e.*, cases in which "the thing speaks for itself." This eliminated the need for any additional evidence, including motive or history of abuse, and resulted in quick, easy and virtually routine convictions of parents and caretakers based solely on the medical testimony of prosecution experts.⁵²

Given the underlying assumptions of the SBS hypothesis, the suggestion that birth injuries, short falls, or natural causes could result in the triad, or that a child might have a lucid interval after such an injury, was viewed as heretical. How could birth injuries produce findings that did not become apparent for days, weeks or months after birth? How could short falls produce traumatic findings akin to—or worse than—those seen in major motor vehicle accidents and multistory falls? How could a natural disease process rupture veins and axons, causing diffuse traumatic brain injury? And how could there be a lucid interval after bridging veins had been ruptured and axons torn throughout the brain? Not surprisingly, those who suggested such possibilities were often disparaged or vilified.⁵³ Unfortunately, those attacks continue to this

timing—*i.e.*, that symptoms necessarily would appear instantaneously upon the infliction of injury—proves the perpetrator's identity"); *see also Case, supra*, note 43 at 118 (suggesting that children with nonaccidental head injuries show an immediate decrease in their level of consciousness at injury).

⁵⁰ *See Tuerkheimer*, note 51 at 18.

⁵¹ *Id.* (noting that parents and caretakers have been accused of shaking the child in their care because they were present immediately before the child's loss of consciousness).

⁵² *See, e.g., Imwinkelried, supra* note 47 ("it seems clear that during the past two decades, prosecution expert testimony about shaken baby syndrome has contributed to thousands of convictions").

⁵³ Those who question the scientific basis for SBS/AHT are routinely accused of incompetence, greed, indifference to child abuse and, more recently, of possibly having histrionic/borderline personality disorders. *See, e.g., Christopher Spencer Greeley*, Assoc. Professor of Pediatrics, Univ. of Tex. Health Sci. Ctr. at Houston, Presentation at New York City Abusive Head Trauma/Shaken Baby Syndrome Training Conference: Dissent or Denialism?: A Scholarly Misadventure with the Medical Literature (and the Media), (Sept.

day.⁵⁴

2. *The warnings.*

Despite its popularity, there were early warning signs that the SBS hypothesis might be flawed.⁵⁵ The first serious warning arose in 1987, when Dr. Duhaime, a young neurosurgeon, and several biomechanical engineers attempted to validate the SBS hypothesis by measuring the force of shaking and comparing it to accepted head injury thresholds.⁵⁶ While crude, these early experiments indicated that the force generated by shaking an infant was well below established head injury criteria and was only approximately one-fiftieth the force generated by impact.⁵⁷ This study concluded:

[T]he shaken baby syndrome, at least in its most severe acute form, is

23, 2011), available at http://www.queensda.org/SBS_Conference/Denialism&TheMedicalLiterature,0911, NYC, Handout.pdf (suggesting that researchers who question SBS/AHT theory use “sleaze tactics” and may have “histrionic/borderline” personality disorders); see also Brian Holmgren, Keynote Address at Eleventh International Conference on Shaken Baby Syndrome/Abusive Head Trauma: *To Tell the Truth—Examining Defense Witness Testimony in Abusive Head Trauma Cases* (Sept. 13, 2010) (showing excerpts of testimony from defense experts juxtaposed with an image of Pinocchio with a growing nose at a keynote presentation teaching doctors and prosecutors how to discredit defense witnesses; this presentation concluded with a sing-along to the tune of “If I only had a brain” led by a prominent child abuse pediatrician, joined by prosecutors and doctors, mocking those who propose diagnostic alternatives to SBS/AHT) brochure at http://www.dontshake.org/pdf/Program_Atlanta2010_8-18-10%20v2.pdf (presentation notes and lyrics on file with authors); Robert M. Reece et al., *The Evidence Base for Shaken Baby Syndrome: Response to Editorial from 106 Doctors*, 328 BRIT. MED. J. 1316, 1316 (2004) (arguing that SBS skeptics have a “worrisome and persistent bias against the diagnosis of child abuse in general”). Personal and professional attacks of this nature have made scientific debate difficult.

⁵⁴ While Dr. Narang does not endorse these attacks, he does suggest, without offering evidence, that those who point out flaws in the SBS diagnosis or identify alternative causes are motivated by monetary gain. Narang, *supra* note 3, at 592 (“[T]he 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”). In our experience, the marginal income for defense experts is generally small relative to the workload and the hostility encountered in the courtroom and professional settings. Because the funding is often inadequate, defense experts often provide pro bono reports and/or testimony based on the research in their own specialties.

⁵⁵ See, e.g., Ann-Christine Duhaime et al., *The Shaken Baby Syndrome: A Clinical, Pathological, and Biomechanical Study*, 66 J. NEUROSURG. 409 (1987).

⁵⁶ *Id.*

⁵⁷ See *id.* at 413.

not usually caused by shaking alone. Although shaking may, in fact, be a part of the process, it is more likely that such infants suffer blunt impact. . . . Unless a child has predisposing factors such as subdural hygromas, brain atrophy, or collagen-vascular disease, fatal cases of the shaken baby syndrome are not likely to occur from the shaking that occurs during play, feeding, or in a swing, or even from the more vigorous shaking given by a caretaker as a means of discipline.⁵⁸

Dr. Duhaime later suggested that the triad was likely caused by shaking followed by impact, possibly on a soft padded surface.⁵⁹

Further warnings arose during the 1997 Louise Woodward trial, popularly known as the "Boston nanny case."⁶⁰ In *Woodward*, Dr. Patrick Barnes, a pediatric neuroradiologist then at Harvard and one of the co-authors of this article, testified for the prosecution.⁶¹ In the same case, several credible and well-established experts presented, perhaps for the first time, serious alternatives to the SBS hypothesis. At the trial, Dr. Jan Leestma, the author of *Forensic Neuropathology*, Dr. Michael Baden, a well-known forensic pathologist, and Dr. Ronald Uscinski, a Georgetown neurosurgeon, testified that the child had a chronic (old) subdural hemorrhage that rebled.⁶² At the time, this was viewed as a "courtroom diagnosis," and its proponents were attacked by supporters of the SBS hypothesis.⁶³ Today, however, rebleeding from a chronic subdural hemorrhage is widely accepted,

⁵⁸ *Id.* at 414.

⁵⁹ See, e.g., A. C. 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, 183 (1992) (in "Shaken Impact Syndrome," head injury is caused by rapid angular deceleration to the brain through impact after a shaking episode; if the head strikes a soft padded surface, contact forces will be dissipated over a broad area and external or focal injuries may be undetectable).

⁶⁰ See *Commonwealth v. Woodward*, 694 N.E.2d 1277, 1281 (1998); see also Carey Goldberg, *Massachusetts High Court Backs Freeing Au Pair in Baby's Death*, N. Y. TIMES (June 17, 1998) available at <http://www.nytimes.com/1998/06/17/us/massachusetts-high-court-backs-freeing-au-pair-in-baby-s-death.html?ref=louisewoodward>.

⁶¹ Like many others, Dr. Barnes has revisited these issues since 1997, with particular emphasis on the teachings of evidence-based medicine and the correlation between the neuroradiology and neuropathology of the infant brain.

⁶² The Woodward case also involved a skull fracture, making timing difficult. See Special Report, *Timetable of Woodward Case*, BBC NEWS (Nov. 10, 1997), available at http://news.bbc.co.uk/2/hi/special_report/1998/woodward/29232.stm.

⁶³ David L. Chadwick et al., *Shaken Baby Syndrome—A Forensic Pediatric Response*, 101 PEDIATRICS 321, 321 (1998).

even by supporters of the SBS/AHT hypothesis.⁶⁴

Following the *Woodward* case, a number of forensic pathologists questioned the validity of the SBS diagnosis, with one leading forensic pathologist urging his colleagues to refrain from the type of “dramatic, unscientific” remarks that were permeating courtroom testimony, such as the standard phrase: “the equivalent of a fall from a two-story building.”⁶⁵

3. 2001: a developing schism.

The public airing of the issues in the *Woodward* case led to a renewed interest in SBS among researchers. In 2001, Dr. Geddes, a British neuropathologist, and her colleagues published careful studies of the brains of infants who had reportedly died from abuse.⁶⁶ The results of these studies were unexpected.⁶⁷ In the first study (“Geddes I”),⁶⁸ the researchers found that the brain pathology was predominantly hypoxic or ischemic (*i.e.*, due to lack of an oxygenated blood supply) rather than traumatic in nature. Unlike the traumatic hemorrhages found in adults and older children, moreover, the subdural hemorrhages in allegedly abused infants were typically thin and trivial in quantity—containing far less blood than would be

⁶⁴ See, e.g., Marguerite M Caré, *Neuroradiology, in ABUSIVE HEAD TRAUMA IN INFANTS AND CHILDREN, A MEDICAL, LEGAL, AND FORENSIC REFERENCE* 73, 81 (G.W. Med. Publ'g 2006) (septations or membranes that develop within chronic hematomas may predispose infants to repeated episodes of bleeding within these collections; such rebleeding can occur with little or no trauma).

⁶⁵ Cyril H. Wecht, *Shaken Baby Syndrome, Letter to the Editor*, 20 AM. J. FORENSIC MED. PATHOL. 301 (1999); see also John Plunkett, *Shaken Baby Syndrome and the Death of Matthew Eappen, A Forensic Pathologist's Response*, 20 AM. J. FORENSIC MED. PATHOL. 17, 20 (1999). As discussed below, forensic pathologists have always been more skeptical of the SBS hypothesis than other specialties, particularly pediatricians.

⁶⁶ David I. Graham, *Editorial: Paediatric Head Injury*, 124 BRAIN 1261, 1261 (2001) (Geddes and her colleagues conducted a “meticulous clinicopathological correlation in 53 cases of non-accidental paediatric head injury”).

⁶⁷ Dr. Geddes has described her surprise that the microscopic examinations failed to find the widespread and severe traumatic brain damage assumed to be present in shaken infants. Jennian Geddes, *Questioning Traditional Assumptions*, BARTS AND THE LONDON CHRONICLE, Spring 2006, available at http://www.qmul.ac.uk/alumni/publications/blc/blc_spring06.pdf.

⁶⁸ J. F. Geddes et al., *Neuropathology of Inflicted Head Injury in Children, I. Patterns of Brain Damage*, 124 BRAIN 1290, 1294 (2001).

expected from ruptured bridging veins, as hypothesized in SBS. While some infants showed evidence of localized axonal injury to the craniocervical junction or cervical cord, the majority did not, casting further doubt on the SBS hypothesis. In the second study ("Geddes II"), Dr. Geddes and her colleagues described the scientific evidence supporting a traumatic origin for the brain damage in allegedly abused children as "scanty." In many respects, the findings in these children were virtually indistinguishable from the findings in infants who had died natural deaths.⁶⁹

While far from dispositive, the implications of Geddes I and II were devastating: if Dr. Geddes and her colleagues were correct, the SBS hypothesis, which rested on the notion that the triad was caused by the traumatic tearing of veins and axons, was likely wrong. While traumatically torn axons are by definition caused by trauma, there are many non-traumatic causes for hypoxic axonal injury. The brain may, for example, be deprived of oxygen because the heart or lungs are not functioning properly or because the child is suffering from widespread infection (sepsis). This research raised, for the first time, the possibility that the brain findings that had been attributed to traumatically torn axons from violent shaking might reflect hypoxia-ischemia from any medical condition that affected the flow of oxygen to the brain. Dr. Geddes' research also raised problems with timing: if the brain damage was secondary to the deprivation of oxygenated blood from any source, the ensuing brain swelling could develop quickly or slowly, over a period of hours to days, with collapse occurring whenever the brain's basic needs were no longer met by the dwindling supply of oxygenated blood. Although Geddes I and II were heavily criticized at the time, it is now widely accepted that the brain swelling seen in allegedly shaken infants is hypoxic-ischemic rather than traumatic in nature.⁷⁰

⁶⁹ J.F. Geddes et al., *Neuropathology of Inflicted Head Injury in Children, II. Microscopic Brain Injury in Infants*, 124 *BRAIN* 1299, 1299,1305 (2001).

⁷⁰ See, e.g., Mark S. Dias, *The Case for Shaking*, in *CHILD ABUSE AND NEGLECT, DIAGNOSIS, TREATMENT AND EVIDENCE* 362, 368 (Carole Jenny, ed., 2011) (it is increasingly clear from neuroimaging studies and post-mortem analyses that the widespread cerebral and axonal damage in AHT cases is ischemic rather than directly traumatic); Neil Stoodley, *Non-Accidental Head Injury in Children: Gathering the Evidence*, 360 *THE LANCET* 272 (2002) (noting the growing evidence that hypoxic-ischaemic damage is of greater importance than traumatic axonal or shearing injury in the pathophysiology of nonaccidental head injury).

Biomechanical objections to the SBS hypothesis also returned to the forefront in 2001. In April, Professor Werner Goldsmith, a professor of biomechanical engineering at the University of California at Berkeley, raised the biomechanical concerns with the National Institutes of Health (NIH). In his presentation, Professor Goldsmith noted that while the vast majority of pediatric head injuries were accidental, others resulted from abuse or physiological (natural) causes, unaccompanied by mechanical trauma.⁷¹ Given the difficulty of determining causation, he urged the development of more sophisticated biomechanical models and more reliable head injury criteria for infants. He also urged biological specialists, medical professionals and biomechanicians to collaborate in investigating the properties of the immature infant brain and surrounding blood vessels that might make them more susceptible to trauma.⁷² Such a program, Professor Goldsmith suggested, would “enormously reduce the number of cases now brought into criminal courts, and the concomitant costs, estimated to be in the multiple millions of dollars, as well as avoid the true trauma, emotionally, financially, and temporally, of individuals falsely accused of abuse when the occurrence was accidental.”⁷³

In the same year, Dr. John Plunkett, a forensic pathologist, published an article on fatal short falls from playground equipment.⁷⁴ While most of the children were older than typical SBS infants, his report included a videotaped fall of a toddler from a plastic indoor play gym that resulted in the triad findings and death after a short lucid interval. This videotape provided seemingly indisputable proof that the triad could result from falls of less than three feet and that

⁷¹ Werner Goldsmith, Presentation, *Biomechanics of Traumatic Brain Injury in Infants and Children*, NAT. INSTITUTES OF HEALTH (April 2001) (on file with authors). As Professor Goldsmith recognized, “head injury” includes any insult to the brain, whether from accidental, abusive or natural causes. This terminology often causes confusion in the literature.

⁷² Professor Goldsmith specifically urged research on the rate of blood absorption and effusion of ruptured blood vessels, which is the subject of the Squier & Mack papers (discussed below).

⁷³ *Id.*

⁷⁴ John Plunkett, *Fatal Pediatric Head Injuries Caused by Short-Distance Falls*, 22 AM. J. FORENSIC MED. PATHOL. 1 (2001).

lucid intervals could occur.⁷⁵

By this time, however, the SBS hypothesis had taken on a life of its own. By 2001, shaking as the primary or exclusive cause of the triad had been taught in the medical schools for decades, not as a hypothesis but as scientific fact. Prosecutions were well-publicized, and an effective advocacy group was training social workers and prosecutors to identify, prosecute and win cases against parents and caretakers who had allegedly shaken their children.⁷⁶ Doctors affiliated with this group also produced SBS position papers for the major medical associations. In 2001, the Board of Directors of the National Association of Medical Examiners—the professional association for forensic pathologists—published an article entitled “Position Paper on Fatal Abusive Head Injuries in Infants and Young Children,” which incorporated the SBS hypothesis.⁷⁷ Although this paper did not pass peer review and was not endorsed by the membership,⁷⁸ it was published in the NAME journal, accompanied

⁷⁵ *Id.* at 4. In this case the child’s feet were 28 inches above the floor when she fell; medical records showed a large subdural hemorrhage, bilateral retinal hemorrhages and extensive edema. In the past year, two other videotaped fatal short falls resulting in death have been reported. One was of an infant who fell from a Kroger shopping cart onto concrete in Macon, Georgia, caught on surveillance video (John Stevens, *Three-Month-Old Boy Dies After Falling Out of Shopping Cart as Mother Walked Back to Car*, DAILY MAIL, September 22, 2011, at www.dailymail.co.uk/news/article-2040559). The other was a fall onto a mat at an indoor mall playground shown by the Queens District Attorney’s Office at the 2011 New York City Abusive Head Trauma/Shaken Baby Syndrome Training Conference (Sept. 22, 2011), available at http://www.queensda.org/SBS_Conference/2011_SBS_Conf.pdf.

⁷⁶ The National Center on Shaken Baby Syndrome (NCSBS) began offering SBS prevention programs in 1990 and incorporated as a legal entity in 2000. According to its website, the NCSBS reaches thousands of medical, legal, child protection and law enforcement professionals every year. The National Center on Shaken Baby Syndrome, <http://dontshake.org/> (last visited Aug. 17, 2012).

⁷⁷ Case, *supra* note 43.

⁷⁸ E-mail from Dr. DiMaio, Editor of the *American Journal of Forensic Medicine and Pathology*, to Dr. Plunkett (March 6, 2003) (on file with the author) (“[T]he position paper: was reviewed by peer reviewers and determined not to be a position paper but an ordinary article expressing the opinion of the authors . . . The paper [does] not meet the criteria of a position paper Calling a tail a leg does not make it one.”); Email from Vincent DiMaio to NAME-L@Listserve.cc.emory.edu (Feb. 7, 2002) (on file with the author) (“As editor of the AJFMP, I had serious misgiving about publishing this paper, not because of its contents but in that it is described as a position paper If one bothers to read the box in the lower left corner of the first page of the article, one will see that the paper was rejected as a position paper by the three reviewers As an aside, the paper in its original form was rejected by

by a somewhat ambiguous and little-heeded editorial caveat.⁷⁹ In the same year, the Committee on Child Abuse and Neglect of the American Academy of Pediatrics (AAP) published a similar paper, entitled “Shaken Baby Syndrome: Rotational Cranial Injuries—Technical Report.”⁸⁰ The AAP paper recommended a presumption of child abuse whenever a child younger than one year suffers an intracranial injury. While the NAME paper is no longer in effect and the AAP paper has been substantially modified,⁸¹ these papers gave an imprimatur of scientific and medical endorsement to the SBS hypothesis that was accepted, largely uncritically, by the medical and legal communities.

4. *A decade of debate.*

The decade following the Geddes and Plunkett papers and the NAME/AAP position papers was filled with raucous debate, sometimes more rhetorical than substantive. However, a few key points emerged.

a. 2002 NIH conference.

In 2002, NIH held a conference to address the disputed issues.⁸² By this time, the terminology was shifting away from shaken baby

4 of 5 reviewers Shaken baby syndrome is controversial in that a number of individuals doubt its existence . . .”).

⁷⁹ Case, *supra* note 43, at 112 (“Editor’s note: The Board of Directors of the National Association of Medical Examiners charged the authors of this article with writing a position paper on the shaken baby syndrome. This article was the result. The manuscript was reviewed by three reviewers on the Board of Editors of the American Journal of Forensic Medicine and Pathology. They believed that while it was worthy of publication, it should not be published as a position paper because of the controversial nature of the subject. The Board of Directors responded to this opinion by stating that position papers always deal with controversial subjects”).

⁸⁰ Comm. on Child Abuse and Neglect, Am. Acad. of Pediatrics, *Shaken Baby Syndrome: Rotational Cranial Injuries—Technical Report*, 108 PEDIATRICS 206 (2001).

⁸¹ As addressed below, *infra* Part II. B.4.e. the NAME paper was withdrawn in 2006; the AAP paper was modified in 2009.

⁸² See AM. ACAD. OF PEDIATRICS, INFLECTED CHILDHOOD NEUROTRAUMA: PROCEEDINGS OF A CONFERENCE SPONSORED BY DEP’T OF HEALTH & HUMAN SERVS., NAT’L INST. OF HEALTH, NAT’L INST. OF CHILD HEALTH & HUMAN DEV., OFFICE OF RARE DISEASE, & NAT’L CTR. FOR MED. REHAB. RESEARCH (Robert M. Reece & Carol E. Nicholson eds., 2003). These conference proceedings are one of the two treatises referenced by Narang, *supra* note 3, at 538–39.

syndrome to more generalized terms, such as inflicted neurotrauma and abusive head trauma. Although the conference was limited to supporters of the SBS/AHT hypothesis, the lack of evidentiary support for SBS was repeatedly acknowledged, beginning in a preface to the conference proceedings by Dr. Carol Nicholson, a Program Director at NIH:

The debate over “shaken baby syndrome” continues to rage in our country. Because there is very little scientific experimental or descriptive work, the pathophysiology remains obscure, and the relationship to mechanics even cloudier. . . . What we need is science—research and evidence that just isn’t there right now. The evidence that does exist has not been subjected to evidence-based scrutiny in a multidisciplinary scientific forum.⁸³

Dr. Robert Reece, a Clinical Professor of Pediatrics, made similar points in his preface:

There have been numerous conferences on this subject over the past several years, but to date, none of these has made the analysis of evidence-based literature the mission of the conference. What literature is there that is based on well-designed studies? How many of the more than 600 peer-reviewed articles in the medical literature can withstand the scrutiny of evidence-based analysis?⁸⁴

Dr. Reece emphasized that much of the literature was based on clinical phenomena rather than “bench research” and that the contributions of basic scientists doing research on the physiology and pathophysiology of the central nervous system were essential to understanding these issues.⁸⁵ He also made clear that much of what was being considered at the conference was based on “a preponderance of the evidence” rather than “evidence beyond a reasonable doubt”—the standard required in criminal cases.⁸⁶

Other conference participants addressed the new literature. Although SBS theory had previously held that short falls were benign, Dr. Feldman advised that in a few cases short falls “may be

⁸³ *Id.* at IX (noting that the escalating emotional and forensic advocacy was proving destructive).

⁸⁴ *Id.* at VIII.

⁸⁵ *Id.*

⁸⁶ *Id.*

fatal or have residual effects.”⁸⁷ Dr. Sege noted that while some might argue that additional research, which he characterized as a “massive undertaking,” would simply confirm the current SBS/AHT understandings, “[s]adly, the history of medicine is littered with things known to be true at the time that weren’t.”⁸⁸ Dr. Christian mounted a spirited defense of SBS/AHT theory, claiming that “[h]omicide is the leading cause of injury death in infancy,” but agreed with Dr. Sege that “[t]he literature is replete with case reports of medical diseases that have been misdiagnosed as child abuse.”⁸⁹

The conference participants generally agreed that, despite its volume, the SBS/AHT literature suffered from serious gaps. Dr. Hymel noted that the peer-reviewed SBS/AHT medical literature “largely represents Class 3 scientific evidence from retrospective case series” and “contains little if any firsthand clinical information from admitted perpetrators of inflicted childhood neurotrauma, and no data regarding the *reliability* and/or *validity* of the acute clinical information provided by admitted perpetrators of inflicted neurotrauma.”⁹⁰ Dr. Duhaime warned that SBS/AHT presented a complex puzzle that had been incompletely modeled and that a great deal of work needed to be done using tissues, animals, mathematical models and human observations, superimposed on age-dependent changes and physiological thresholds.⁹¹ Dr. Jenny identified the methodological difficulties with the existing literature:

One resounding criticism in this body of literature poses a methodological dilemma when attempting to study mode of presentation of inflicted head trauma. This dilemma is the problem of circularity of reasoning. That is, we use certain predetermined,

⁸⁷ *Id.* at 33.

⁸⁸ AM. ACAD. OF PEDIATRICS, *supra* note 82, at 41.

⁸⁹ *Id.* at 43.

⁹⁰ *Id.* at 67. As discussed below, under the standards of evidence-based medicine, the available evidence is ranked in four categories, starting with randomized controlled trials (Class 1), which are the most comprehensive and the most reliable, and ending with case studies (Class 4), which may provide valuable but limited insights. Class 3 evidence includes case-control studies and non-consecutive studies with inconsistently applied reference standards. See Bob Phillips, et. al., *Levels of Evidence*, U. OXFORD CENTRE FOR EVIDENCE-BASED MED. (Mar. 2009), <http://www.cebm.net/index.aspx?o=4590>.

⁹¹ AM. ACAD. OF PEDIATRICS, *supra* note 82, at 253.

generally accepted criteria to determine if a child's injuries are inflicted or unintentional, such as delay in seeking care and presence of retinal hemorrhages. Then, when we describe the mode of presentation, those criteria are found to occur most frequently in abused children. A most sticky methodological question is, "What is the gold standard in determining if a child is abused prior to assigning that child to a study cell?" Careful definitions of standards for determining abuse are needed.⁹²

Dr. Dias, a conference organizer, agreed that there was "some degree of a circularity in reasoning; if one defines a particular injury or pattern of injuries a priori as inflicted, then by definition one will rarely, if ever, ascribe these injuries to...an unintentional mechanism."⁹³

b. Biomechanics.

In general, the biomechanical literature continued to conclude that shaking was an unlikely cause of the triad. For example, a 2002 biomechanical review concluded that a three-foot fall produces forces approximately ten times greater than shaking; that spontaneous rebleeds may explain the onset of symptoms in children with chronic subdural hemorrhage; that severe shaking would be expected to damage the cervical cord and spine before producing intracranial injuries; and that the levels of force required for shaking to produce retinal bleeding and damage to the eye are biomechanically improbable.⁹⁴ These findings were similar to those in a joint study conducted by Dr. Jenny, a leading SBS proponent, and Aprica, a Japanese baby products company that had created a more biofidelic model of the human infant.⁹⁵ Other research was in accord: while

⁹² *Id.* at 51-52. Dr. Jenny identified the studies of Duhaime (1987); Ewing-Cobbs (1998); Reece (2000); and Feldman (2001) as "methodologically superior." *Id.* at 51. Three of these are discussed below.

⁹³ *Id.* at 100.

⁹⁴ A.K. Ommaya et al., *Biomechanics and Neuropathology of Adult and Paediatric Head Injury*, 16 BR. J. NEUROSURGERY 220, 226, 232-33 (2002).

⁹⁵ These studies confirmed that the maximum linear acceleration produced by shaking was less than one-third that produced by rolling off a sofa and less than one-tenth that of a fall from chest level when being held by an adult. Violent shaking and slamming on a thin carpet over a wood floor was comparable to the chest level fall, while slamming onto a mat without shaking produced a force approximately fifty percent greater than the fall from chest level. C. Jenny et al., *Development of a Biofidelic 2.5 kg Infant Dummy and Its Application*

impact reaches known injury thresholds, shaking does not produce the force required to rupture bridging veins and axons and would cause extensive cervical spine injury or failure (*i.e.*, neck injury) before causing such effects.⁹⁶ By then, after thirty years, there were still no witnessed accounts of the shaking of a previously well child resulting in the triad, casting further doubt on the mechanism.⁹⁷

c. SBS and evidence-based medicine.

The weaknesses in the literature were not passing unnoticed in the outside world. In a 2003 article published in the NAME journal, Dr. Mark Donohoe, a general practitioner in Australia, examined the research support for SBS through 1998 and concluded what others—including the NIH conference participants—had been saying privately for years: the research basis for shaken baby syndrome was remarkably weak.⁹⁸ Dr. Donohoe described the evidence for SBS as “analogous to an inverted pyramid, with a small database (most of it poor-quality original research, retrospective in nature, and without appropriate control groups) spreading to a broad body of somewhat divergent opinions. One may need reminding that repeated opinions based on poor-quality data cannot improve the quality of

to Assessing Infant Head Trauma During Violent Shaking, in INJURY BIOMECHANICS RESEARCH: THIRTIETH INTERNATIONAL WORKSHOP 129, 137, 140 (2002).

⁹⁶ See, e.g., Michael T. Prange et al., *Anthropomorphic Simulations of Falls, Shakes, and Inflicted Impacts in Infants*, 99 J. NEUROSURG. 143, 143 (2003); Ommaya, *supra* note 94, at 233; see also Ronald H. Uscinski, *Shaken Baby Syndrome: Fundamental Questions* 16 BRIT. J. NEUROSURGERY 217, 218 (2002) (biomechanical research has raised questions about whether shaking is the true cause of intracranial injuries in alleged SBS cases); Ronald H. Uscinski, *Shaken Baby Syndrome: An Odyssey*, 46 NEUROLOGIA MEDICO-CHIRURGICA 57, 59 (2006) (SBS-type accelerations should damage the cervical spinal cord and brainstem before head injury is observed).

⁹⁷ There are also no reported cases of video recordings capturing violent shaking resulting in the triad. While several caregivers have been caught on videotape shaking infants in their care, to our knowledge none of these children exhibited any of the triad findings, or any injury at all.

⁹⁸ Mark Donohoe, *Evidence-Based Medicine and Shaken Baby Syndrome Part I: Literature Review, 1966-1998*, 24 AM. J. FORENSIC MED. PATHOLOGY 239, 241 (2003). Dr. Narang criticizes Dr. Donohoe’s review article and his review of the SBS literature. Narang, *supra* note 3, at 533-35. As discussed *infra*, Part III.B.1., that criticism mistakes the nature of Dr. Donohoe’s inquiry.

evidence."⁹⁹ He concluded that "the commonly held opinion that the finding of SDH [subdural hemorrhage] and RH [retinal hemorrhage] in an infant was strong evidence of SBS was unsustainable, at least from the medical literature."¹⁰⁰

d. Alternative diagnoses.

Given the biomechanical findings, impact took on new significance as the most likely cause of the triad. But this raised new issues. First, if the triad was caused by impact, why did so few children have external signs of impact, such as fractures or bruises? Second, how much force is required to cause injury from impact? And third, can we reliably distinguish between accidental and inflicted impact—and if so, how? These issues were sometimes addressed by simply redefining the "triad"—which had previously been viewed as diagnostic of shaking—as evidence of impact, with or without shaking.¹⁰¹ At the same time, clinicians quite rightly began to look closely for other possible signs of impact or abuse, ranging from small bruises or discolorations to fractures or other bony abnormalities that might help determine causation.¹⁰²

While some researchers and clinicians struggled to differentiate between accidental and inflicted impact, others began to consider—or more precisely re-consider—the role of natural conditions or birth trauma as causal or contributing factors for the triad. As Dr. Guthkelch noted in 1953, subdural effusions are often associated with difficult labor, illness, and/or venous thrombosis, a form of

⁹⁹ Donohoe, *supra* note 98, at 241.

¹⁰⁰ *Id.*

¹⁰¹ See, e.g., Derek A. Bruce and Robert A. Zimmerman, *Shaken Impact Syndrome*, 18(8) PEDIATRIC ANNALS 482, 492-4 (1989) (in light of the Duhaime study, which is the only attempt to examine the forces that can be produced by shaking, the authors concluded that severe acute brain trauma cannot be produced by shaking alone and that the mechanism of injury is more appropriately described as "shaking impact," with impact possibly occurring on sofa or mattress) (emphasis in original).

¹⁰² See, e.g., S. Maguire et al, *Are there patterns of bruising in childhood which are diagnostic or suggestive of abuse? A systematic review*, 90 ARCHIVES DISEASE CHILDHOOD 182, 182, 184 (2005) (reviewing studies that describe bruising in non-abused and abused children; studies on abused children are frequently methodologically weak with quality research urgently needed). The problems encountered in defining children as abused are discussed in Sections III.A.3.a.-III.A.3.f., *infra*.

childhood stroke often associated with infection and/or dehydration.¹⁰³ Metabolic disorders, nutritional deficiencies and infection have also long been recognized as causes of subdural hemorrhage.¹⁰⁴

During this period, the child abuse literature increasingly recognized alternative causes for subdural hemorrhages and other elements of the triad. In 2002, Drs. Jenny, Hymel and Block—all prominent child abuse pediatricians—published an article identifying a wide range of nontraumatic etiologies for subdural hemorrhages and describing minor accidental injuries confirmed by medical personnel that resulted in intracranial hemorrhage.¹⁰⁵ The article further recognized that older subdural collections can re-bleed spontaneously or from minor impact, and that no prospective, comparative studies had measured the frequency or consequences of re-bleeding in young children with chronic subdural collections.¹⁰⁶

In 2003, Dr. Geddes suggested that the subdural and retinal hemorrhages seen in natural deaths and alleged SBS cases may reflect a cascade of events, including raised intracranial pressure, central venous and systemic arterial hypertension, immaturity and hypoxia-related vascular fragility—a suggestion that became known as the “Unified Hypothesis” or Geddes III.¹⁰⁷

By 2006, it was widely recognized by supporters of the SBS/AHT hypothesis that there are many “mimics” of SBS/AHT, including accidental causes and a variety of illnesses and medical conditions,

¹⁰³ A. N. Guthkelch, *Subdural Effusions in Infancy: 24 Cases*, 1 BRIT. MED. J. 233-239 (1953) (abnormal or difficult labor present in 75% of cases; children often present with seizures, vomiting and/or irritability; some are ill and/or have history of short fall; in one, a thrombosed sagittal sinus was identified at autopsy).

¹⁰⁴ Narang, *supra* note 3, at 526, n. 138.

¹⁰⁵ See Kent P. Hymel, et al., *Intracranial Hemorrhage & Rebleeding in Suspected Victims of Abusive Head Trauma: Addressing the Forensic Controversies*, 7 CHILD MALTREATMENT 329, 333-337 (2002) (causes for subdural hemorrhage include prenatal, perinatal, and pregnancy-related conditions; birth trauma; metabolic or genetic disease; congenital malformations; oncologic disease; autoimmune disorders; clotting disorders; infectious disease; poisons, toxins or drugs; and other miscellaneous conditions).

¹⁰⁶ *Id.* at 342, 344.

¹⁰⁷ 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, 19 (2003).

ranging from birth trauma to childhood stroke.¹⁰⁸ Since then, other studies have continued to add to our knowledge. For example, a study by Dr. Rooks and her colleagues found that approximately 46% of asymptomatic newborns had thin subdural hemorrhages, confirming that subdural hemorrhages are not necessarily symptomatic and do not necessarily (or even generally) cause long lasting problems.¹⁰⁹ Another study found a clear correlation between intradural/subdural hemorrhage and the degree of hypoxia in neonates.¹¹⁰ Today, every month seems to bring forth new articles and commentary, adding to the available information but also increasing the confusion. Like Dr. Narang, we do not attempt to review all of these studies but rather address key new articles by subject, noting only that the list of possible causes for findings previously viewed as diagnostic of abuse continues to expand.

e. The position papers revisited.

By 2006, it was evident that the literature on pediatric head injury no longer supported the assumptions underlying the SBS hypothesis and that the major medical associations would have to revise their position papers. This process has resulted in considerable confusion within the medical profession and very little guidance on the proper approach to diagnosis.

In October 2006, the NAME Board of Directors withdrew its

¹⁰⁸ By 2006, the alternative causes or “mimics” included prenatal and perinatal conditions; congenital malformations; genetic conditions; metabolic disorders; coagulation disorders, including venous sinus thrombosis (a form of childhood stroke); infectious disease; vasculitis; autoimmune conditions; oncology; toxins and poisons; nutritional deficiencies; and complications from medical-surgical procedures. See Sirotnak, *supra* note 10; Dowd, *supra* note 10.

¹⁰⁹ V. J. Rooks et al., *Prevalence & Evolution of Intracranial Hemorrhage in Asymptomatic Term Infants*, 29 AM. J. NEURORADIOLOGY 1082, 1085 (2008). While most of these subdural hemorrhages disappeared within the first month, one had evidence of new subdural bleeding at two weeks, with subdural fluid collections still evident at four weeks. With a larger study population, more variations might be expected.

¹¹⁰ Marta C. Cohen & Irene Scheimberg, *Evidence of Occurrence of Intradural & Subdural Hemorrhage in the Perinatal & Neonatal Period in the Context of Hypoxic Ischemic Encephalopathy: An Observational Study from Two Referral Institutions in the United Kingdom*, 12 PEDIATRIC & DEV. PATHOLOGY 169 (2009) (finding a clear correlation between intradural/subdural hemorrhage and the degree of hypoxia in neonates, with bleeding in the parietal dura developing with more severe or prolonged hypoxia).

“Position Paper on Fatal Abusive Head Injuries in Infants and Young Children.”¹¹¹ Although no explanation was offered, the NAME conference of the same date included presentations entitled “Use of the Triad of Scant Subdural Hemorrhage, Brain Swelling, and Retinal Hemorrhages to Diagnose Non-Accidental Injury is Not Scientifically Valid” and “‘Where’s the Shaking?’ Dragons, Elves, the Shaking Baby Syndrome and Other Mythical Entities.”¹¹² No subsequent NAME paper has been approved, leaving it to individual forensic pathologists to reach their own interpretations on causality without guidance from their association. Not surprisingly, this has produced inconsistent conclusions. Today, based on similar or even identical medical findings, some forensic pathologists still endorse shaking as the causal mechanism, while others diagnose blunt force trauma (*i.e.*, impact, accidental or abusive) and yet others consider a wide range of possibilities, including natural causes. In Professor Tuerkheimer’s words, such variances produce “fluky justice.”¹¹³

In 2009, the AAP replaced its technical report on Shaken Baby Syndrome with a policy statement entitled “Abusive Head Trauma in Infants and Children.”¹¹⁴ The authors stated that though the term shaken baby syndrome is often used by physicians and the public,

advances in the understanding of the mechanisms and clinical spectrum of injury associated with abusive head trauma compel us to modify our terminology to keep pace with our understanding of pathological mechanisms. Although shaking an infant has the potential to cause neurologic injury, blunt impact or a combination of shaking and blunt impact can cause injury as well.¹¹⁵

The policy statement advised that while the term shaken baby syndrome “has its place in the popular vernacular,” pediatricians

¹¹¹ E-mail from Gregory G. Davis, Bd. of Directors, NAME, to John Plunkett, MD, and R. Wright (Oct. 17, 2006) (on file with authors). The 2001 NAME position paper had originally been scheduled to sunset in 2006; however, the Board extended it to 2008. In October 2006, the Board rescinded the renewal.

¹¹² Scientific Program, 40th Annual Meeting, Nat’l Ass’n of Medical Examiners, San Antonio, TX (Oct. 13-18, 2006) (on file with authors).

¹¹³ Deborah Tuerkheimer, *Science-Dependent Prosecution & the Problem of Epistemic Contingency: A Study of Shaken Baby Syndrome*, 62 ALA. L. REV. 513, 523-532 (2011).

¹¹⁴ Christian, *supra* note 19.

¹¹⁵ *Id.* at 1409.

should use the term “abusive head trauma” in their medical charts.¹¹⁶ While the policy statement noted that medical diseases can mimic AHT and that pediatricians have a responsibility to consider alternative hypotheses, it did not identify the alternatives or offer any assistance in distinguishing between accidental, nonaccidental and natural causes, leaving this up to individual pediatricians.¹¹⁷

f. Increasing divergence.

Given the disagreements between various organizations and the lack of consensus within organizations, it is increasingly difficult to gauge the extent to which doctors in general agree—or even have the knowledge needed to reach an informed decision—on whether abuse may be determined based on specific medical findings, or what those findings might be. In general, prosecutors and child abuse pediatricians continue to strongly endorse the SBS/AHT hypothesis, resulting in hundreds of successful prosecutions every year. At the same time, there is considerable discontent, particularly among forensic pathologists and neuropathologists. For example, in a recent email, a forensic pathologist testifying on behalf of the prosecution in a criminal case advised the prosecutor that “I don’t know what the breakdown is, but I would not be surprised to learn that it is close to 50/50 among neuropathologists, neurologists, and forensic pathologists as to whether any given case represents non-accidental trauma.”¹¹⁸ While this figure may be high, it seems clear that the

¹¹⁶ *Id.* at 1410.

¹¹⁷ *Id.* at 1409-10.

¹¹⁸ E-mail from Mark Peters, MD, to Sharyl Eisenstein, Assistant State’s Attorney, McHenry County, IL (Sept. 15, 2011) (on file with authors) (regarding Sophia Avila Case #08-073, which resulted in conviction, Oct. 14, 2011). In the same e-mail, Dr. Peters noted that infants can have a lucid interval of several days after head trauma and that a number of medical conditions can cause cerebral hemorrhage, retinal hemorrhage and bone fractures. These conditions should be ruled out before concluding that the injuries are the result of inflicted trauma. “Unfortunately, many or most, cannot be evaluated after death, and the pediatricians taking care of these children before death are not performing these tests for whatever reason. I am beginning to get the impression that when pediatricians see these kinds of cases, they see shaken baby or other non-accidental trauma right from the beginning (as evidenced in the dictated reports), and do not perform tests to rule out these other conditions.” *Id.*

consensus described by Dr. Narang is changing, and that there continues to be very little objective guidance on how to distinguish between accidental, nonaccidental and natural causes of findings previously viewed as diagnostic of shaking.

In 2012, the prediction of the dissenters in *Smith* that “it is unlikely that the prosecution’s experts would today testify as adamantly as they did in 1997” may be coming to pass.¹¹⁹ In February 2012, in an Arizona post-conviction relief case, Dr. Norman Guthkelch, one of the first to hypothesize SBS, provided a declaration stating that the term “Shaken Baby Syndrome is an undesirable phrase and that there was not a vestige of proof when the name was suggested that shaking, and nothing else, caused the triad. Dr. Guthkelch went on to say that a number of other conditions—natural and non-accidental—may lead to the triad, including metabolic disorders, blood clotting disorders, and birth injury, to name a few. In the case at issue, he stated unequivocally that there was insufficient evidence to support a finding of homicide.¹²⁰ In the same case, Dr. A. L. Mosley, the medical examiner who conducted the autopsy and who previously testified that the cause of death was “Shaken/Impact Syndrome,” stated that given the changes in the literature since 2000, there is no longer consensus in the medical community that the findings in his autopsy report are reliable proof of SBS or child abuse, and that if he were to testify today, he would testify that the child’s death was likely due to a natural disease process, not SBS.¹²¹ The charges against Mr. Witt were dismissed with prejudice on October 29, 2012.¹²²

Based on our own experiences, it appears that when subdural and/or retinal hemorrhages are present, child abuse pediatricians tend to diagnose child abuse (SBS/AHT), while forensic pathologists tend to diagnose blunt force trauma, with the manner of death

¹¹⁹ *Cavazos v. Smith*, 132 S. Ct. 2, 10, 181 L. Ed. 2d 311 (2011).

¹²⁰ Declaration of A. Norman Guthkelch, M.D., *State of Arizona v. Drayton Shawn Witt*, Feb. 3, 2012.

¹²¹ Defendant’s Memorandum in Support of Petition for Post-Conviction Relief at 4, *State v. Witt*, No. CR2000-017311 (Ariz. Super. Ct. 2012)

¹²² *State of Arizona v. Drayton Shawn Witt*, Minute Entry, CR 2000-017311 (Superior Court of Arizona, Maricopa County, Oct. 29, 2012).

categorized as accident, homicide or undetermined depending upon the circumstances of the case and the beliefs of the pathologist. While both groups recognize the overlap with natural causes, there is no commonly accepted protocol for investigating alternative causes and very little coordination with the relevant subspecialties.¹²³ As the debate has turned increasingly harsh, moreover, clinicians outside the child abuse arena are often reluctant to participate in what may turn into a free-for-all in the courtroom and beyond.¹²⁴ Given this vacuum, many diagnoses and convictions continue to be based on the presumption that the triad or its components confirm abuse if the parents or caretakers cannot substantiate a known alternative.

g. The triad: where are we now?

In 1996, it was generally accepted that, in the absence of a major motor vehicle accident or fall from a multistory building, the triad was caused primarily or exclusively by shaking.¹²⁵ In 2001, we learned that the diffuse axonal injury attributed to shaking reflected hypoxia ischemia (lack of oxygen) rather than trauma, and that similar findings were found in infants who died natural deaths.¹²⁶ By 2006, the “mimics” of SBS/AHT had expanded to include accidental trauma, birth trauma; congenital, genetic and metabolic disorders, infection, nutritional deficiencies, and a host of other conditions.¹²⁷ And in 2011, just five years later, a leading supporter of SBS theory stated publicly that “[n]o trained pediatrician thinks that subdural hemorrhage, retinal hemorrhage and encephalopathy equals abuse. The ‘triad’ is a myth!”¹²⁸ As this suggests, we are dealing with an

¹²³ The relevant subspecialties include pediatricians, child abuse experts, biomechanics experts, ophthalmologists, neuropathologists, neurosurgeons, neurologists and forensic pathologists.

¹²⁴ The longstanding and coordinated attacks on those who disagree with the SBS hypothesis provide a strong deterrent for anyone who considers voicing a dissenting opinion. See notes 38, 274 and accompanying text.

¹²⁵ Daniel G. Orenstein, *Shaken to the Core: Emerging Scientific Opinion and Post-Conviction Relief in Cases of Shaken Baby Syndrome*, 42 ARIZ. ST. L. J 1305, 1317 (2011).

¹²⁶ See *supra* notes 66-70 and accompanying text.

¹²⁷ See, e.g., Sirotnak, *supra* note 10, at 191-214; Narang, *supra* note 3, at 541 (noting that the differential diagnosis for subdural hemorrhages is extensive).

¹²⁸ Jenny, *supra* note 7, slide 33, at 11.

area that is far more complex and nuanced than previously recognized. We are, moreover, at the beginning, not the end, of our quest for evidence—a quest that requires much greater knowledge of the anatomy and physiology of the infant brain than is currently available. As we struggle to expand our knowledge, we need to engage in a careful and searching analysis of what went wrong while renewing our commitment to “getting it right.”

C. Ongoing Debates

The debate over the validity of the SBS/AHT hypothesis has generated numerous subsidiary questions, including:

1. Can short falls cause the triad, or is extreme force required?
2. Can there be a “lucid interval”?
3. What do retinal hemorrhages tell us about causation?
4. When do fractures, bruises, or other features support an SBS/AHT diagnosis?
5. Do confessions confirm SBS/AHT?
6. How do we handle new hypotheses?

While these questions continue to produce vigorous and often acrimonious debate in the literature and the courtroom, there is sometimes surprising—and often under-recognized—consensus on key points.

1. Short falls.

While it has long been recognized that short falls do not typically result in serious injury to young children,¹²⁹ it was understood for

¹²⁹ See, e.g., Harvey Kravitz et al., *Accidental Falls from Elevated Surfaces in Infants from Birth to One Year of Age*, 44 PEDIATRICS 869, 872–73 (1969) (reporting on 536 accidental falls with 15 hospitalizations; results included 2 skull fractures and 1 subdural hematoma, with no deaths); Helfer et al., *Injuries Resulting When Small Children Fall Out of Bed*, 60 PEDIATRICS 533, 534 (1977) (85 in-hospital falls of children ages 5 and under resulted in one skull fracture and no deaths); S. Levene & G. Bonfield, *Accidents on Hospital Wards*, 66 ARCHIVES DISEASE CHILDHOOD 1047, 1047-48 (1991) (781 hospital accidents in one year period

decades, if not centuries, that children sometimes suffered serious injury or death after falling short distances¹³⁰ and that the outcome of any given fall would be affected by a variety of biomechanical and physiological factors.¹³¹ As mainstream medicine absorbed the SBS/AHT hypothesis, however, a new skepticism took hold that short falls could generate the force necessary to produce the triad. Since SBS/AHT theory held that such findings would require the force of a motor vehicle accident or multistory fall, the injuries attributed by parents and caretakers to short falls were automatically ascribed to abuse, typically violent shaking. New research has restored some of the traditional nuance as videotaped and witnessed short falls have confirmed that short falls can be fatal¹³² and biomechanical studies have confirmed that the force of impact (including short falls) is much greater than the force of shaking.¹³³ The current consensus is that short falls (typically defined as falls of

involving children under age 16 resulted in 2 limb fractures and 2 skull fractures, one from fall from bed and one from fall from chair; no deaths); Thomas J. Lyons & R. Kim Oates, *Falling Out of Bed: A Relatively Benign Occurrence*, 92 *PEDIATRICS* 125 (1993) (records of children who fell out of hospital beds or cribs showed one skull fracture and one fractured clavicle; no serious or life-threatening injuries).

¹³⁰ See, e.g., John R. Hall et al., *The Mortality of Childhood Falls*, 29 *J. TRAUMA* 1273, 1273-1274 (1989) (in Cook County, falls were third leading cause of death in children 1-4 years old in 1983-1986; 41% of fatal falls occurred from falls of less than 3 feet, often while playing or from furniture, including 8 month old girl who fell off couch onto hard wood floor; two fatal falls occurred under hospital observation; 9 children were initially normal after falls from minor or medium heights and did not seek medical care until there was neurological deterioration, range 1 hour to 3 days; authors conclude that minor falls can be lethal and must be evaluated).

¹³¹ See, e.g., Barry Wilkins, *Head Injury—Abuse or Accident?*, 76 *ARCHIVES DISEASE CHILDHOOD* 393, 393 (1997) (determinants of injury severity may include fall height, nature of the surface, protective reflexes, whether the fall is broken, whether the child propelled himself, the mass of body and head, proportion of energy absorbed, whether some of the energy is dissipated in fractures, whether the contact is focal or diffuse, and whether there is secondary injury, including hypoxia/ischemia).

¹³² See, e.g., Plunkett, *supra* note 76; note 77 *supra* (describing two other videotaped falls); Patrick E. Lantz & Daniel E. Couture, *Fatal Acute Intracranial Injury, Subdural Hematoma, and Retinal Hemorrhages Caused by Stairway Fall*, 56 *J. FORENSIC SCI.* 1648, 1651-52 (2011) (case report of infant with a fatal head injury caused by a fall down stairs); Paul Steinbok et al., *Early Hypodensity on Computed Tomographic Scan of the Brain in an Accidental Pediatric Head Injury*, 60 *NEUROSURGERY* 689, 691 (2007) (reporting on radiology findings in five accidental fatalities, including a fall down stairs and a fall from a stool).

¹³³ See, e.g., Ommaya, *supra* note 96, at 226.

less than 3-4 feet) may occasionally cause death.¹³⁴

The issues are therefore: how rare are short fall deaths, and how should this affect the interpretation of individual cases? Proponents of the SBS/AHT diagnosis often contend that, while short falls can be fatal, the chances are so remote as to be inconsequential.¹³⁵ In making this argument, supporters generally cite a 2008 article by Dr. Chadwick and Gina Bertocci that estimates the annual fatality rate for short falls among young children at less than one in a million.¹³⁶ To create a “best estimate” of the mortality rate, the authors selected a single injury database compiled by the State of California.¹³⁷ Like other epidemiological research, its reliability depends upon the accurate categorization of cases as “accidental” or “abusive.” Since the time period of this database (1997-2003) encompasses the peak of shaken baby theory, this database may undercount short fall fatalities given the previously accepted belief that short falls could not kill.¹³⁸

¹³⁴ See John Plunkett, Forensic Pathologist, & Mark Dias, Professor of Neurosurgery, Keynote Presentation at the Penn State Hershey College of Medicine Second International Conference on Pediatric Abusive Head Trauma: Point/Counterpoint: Analysis of Outcomes from Short Falls (June 26, 2009), brochure available at <http://www.childdeathreview.org/Reports/2009PedAHTConference.pdf> (Dr. Dias replaced Dr. Jenny, who was unavailable). See also 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, 1214 (2008) (finding thirteen possible short-fall child fatalities listed in California database, six of which the authors believe may be valid).

¹³⁵ See David L. Chadwick, *Can a Short Fall Produce the Medical Findings of Shaken Baby Syndrome?*, NAT'L CTR. ON SHAKEN BABY SYNDROME, <http://www.dontshake.org/sbs.php?topNavID=3&subNavID=25&navID=278> (last visited Apr. 15, 2012).

¹³⁶ Chadwick, et al., *supra* note 135, at 1220. Chadwick identifies three classes of cases that can be attributed to trauma: accident (121 per million young children), homicide (22 per million young children) and short falls (0.48 per million young children). *Id.* Even if these rates are correct, this would mean that 0.48 out of every 143.48 cases of traumatic fatal injury, or about one in 300, is attributable to short falls. In the aggregate, nationwide, that would represent a significant number of incidents.

¹³⁷ *Id.* at 1214,1219. One study mentioned in Chadwick was discounted because the “fall histories [were] not validated” even though abuse had been ruled out by the police in all cases and two deaths had occurred under medical observation. *Id.* at 1218 (referring to Hall, et al., *supra* note 130).

¹³⁸ *Id.* at 1214. The authors noted that the injury coding in the database often did not match the more detailed information in the death certificates. *Id.* While the authors excluded cases incorrectly labeled as short fall deaths, they do not describe a corresponding effort to identify short fall deaths that may have been included in other categories, including

In short, the data may reflect nothing more than the biases of the old understanding.¹³⁹

Even if the Chadwick data is correct, however, it does not tell us whether any *particular* case is the result of accident or abuse. As Dr. Narang observes, “statistics embody averages, not individuals.”¹⁴⁰ In individual cases, the issue is whether an injured child who appears in the emergency room after a reported short fall is suffering the consequences of a fall or is the victim of abuse. In this context, the Chadwick article is often cited to suggest that the likelihood that the death was attributable to the fall is less than one in a million.¹⁴¹ In individual cases, however, it may be virtually certain that a short fall caused the injuries, *e.g.*, if the fall is confirmed by an independent witness or videotaped (as sometimes occurs with public surveillance equipment), even though the chances on average remain one in a million. More often, the medical evidence may confirm impact but cannot distinguish between a child who has fallen and hit his or her head and a child who has been hit on the head. The fact that fatal short falls are rare does not help us make this determination since child deaths are in and of themselves rare, and each cause (whether natural or accidental) is by definition even rarer.

In a large country such as the United States, moreover, small risks may translate into significant numbers. In 2010, there were approximately 12 million children under the age of 2 in the United States.¹⁴² Using Chadwick’s estimated mortality rate from short falls,

homicide.

¹³⁹ This is another example of the circularity that affects much of the research in this field. If deaths presenting with the triad following a reported short fall are typically diagnosed as SBS/AHT, the number of accidental short fall fatalities will appear to be vanishingly small. The rarity of short fall fatalities is then used to reject the caretaker’s history of a short fall and to support an SBS/AHT diagnosis. This circularity issue is addressed below.

¹⁴⁰ Narang, *supra* note 3, at 522 (quoting Jerome Groopman, HOW DOCTORS THINK 6 (2007)).

¹⁴¹ See, *e.g.*, Brief for Plaintiff-Appellant in Response to Non-Party Brief of Amici Curiae at 6, *State v. Louis*, 798 N.W.2d 319 (Wis. Ct. App. 2010) (Case No. 2009AP2502-CR) (“[Y]es, a short fall could conceivably cause an infant’s death, but it is exceedingly rare”).

¹⁴² The 2010 census recorded approximately 12 million children aged 0-2 in the U.S in 2010. Census Summary File 1, *Single Years of Age and Sex: 2010*, United States Census Bureau at http://factfinder2.census.gov/faces/tableservices/jsf/pages/productview.xhtml?pid=DEC_10_SF1_QTP2&prodType=table. Using Dr. Chadwick’s estimate of 0.48 deaths per

one would expect perhaps 6 short fall deaths in the 0-2 age group. If a substantial number of short fall deaths in this age group were misclassified as SBS/AHT deaths based on the assumption that short falls could not kill, and if babies and toddlers are more vulnerable to short falls than older children,¹⁴³ these figures could increase substantially. This would be consistent with the biomechanical studies and case reports, which confirm that the forces generated by the types of short falls described in SBS/AHT cases (fall from parent's arms, fall down stairs, etc.) typically exceed accepted head injury criteria and may be fatal.¹⁴⁴ Such deaths may be most likely to occur in children with pre-existing conditions, including chronic (old) subdural hemorrhages, coagulopathies (bleeding/clotting disorders) or pre-existing neurological impairment.

2. Timing ("lucid intervals").

Under the traditional SBS/AHT hypothesis, it was believed that the child would be immediately unconscious upon infliction of the injuries, which were assumed to consist of ruptured veins and axons.¹⁴⁵ The logical corollary was that whoever was with the child at the time of collapse must have inflicted the injuries.¹⁴⁶ This is, however, contrary to the well-known phenomenon of delayed deterioration from minor head injury, in which a prolonged period of normality or near normality may precede the collapse.¹⁴⁷ In 1998, Dr.

million children, the number of expected fatal short falls nationwide would be 5.76 (0.48 x 12) for children aged 0-2.

¹⁴³ Jenny, *supra* note 7, slide 56, at 19 (overwhelming evidence shows that the response to a given injury in an infant is much worse than that of an adult to a similar injury).

¹⁴⁴ See Jenny, *supra* note 173; Lantz, *supra* note 132.

¹⁴⁵ See *supra* notes 49-52 and accompanying text.

¹⁴⁶ See Imwinkelried, *supra* note 49, at 5 ("In effect, the testimony time stamps the injuries, powerfully incriminating the last adult in the child's presence before the onset of symptoms").

¹⁴⁷ See, e.g., J. W. Snoek et al., *Delayed Deterioration Following Mild Head Injury in Children*, 107 BRAIN 15 (1984) (reporting three delayed deaths in children apparently due to severe and uncontrollable unilateral or diffuse brain swelling). For this reason, hospitals typically urge parents and caretakers to monitor a child's symptoms after a head injury in order to detect delayed deterioration. See, e.g., Seattle Children's Hosp, *Head Injury Guidelines*, available at <http://www.seattlechildrens.org/medical-conditions/symptom-index/head-injury/> (guidelines directing parents to seek medical care immediately if child shows delayed

Gilliland concluded that there was an interval of more than 24 hours (and sometimes up to 72 hours or more) between the trauma and the collapse in approximately 25% of alleged shaking, shaking impact or impact cases.¹⁴⁸ Subsequent studies and case reports have confirmed that collapse may not be immediate, even in cases involving impact.¹⁴⁹

When the triad findings result from a natural disease process, the concept of a “lucid interval” may be meaningless because there may be no sudden precipitating event. Like any disease process, the natural mimics of abusive head trauma—ranging from stroke to metabolic or genetic disorders—may produce sudden and disastrous results, or may have a stuttering course, with a variety of warning signs and symptoms, followed by neurologic collapse. To determine the course of the disease, it is critical to obtain comprehensive and precise caretaker reports and to examine all records, including prenatal, birth, and pediatric records. This information must then be coordinated with the radiology images, neurosurgical reports and/or tissue slides, which can provide objective information on cause and timing. Often, as one explores the child’s history, it becomes apparent that multiple factors likely played a role in the collapse.

Today, there is no real dispute over whether lucid intervals can

neurological symptoms after head injury) (last visited Aug. 3, 2011).

¹⁴⁸ M.G.F. Gilliland, *Interval Duration Between Injury and Severe Symptoms in Nonaccidental Head Trauma in Infants and Young Children*, 43 J. FORENSIC SCI. 723, 723 (1998).

¹⁴⁹ See, e.g., Kristy B. Arbogast et al., *Initial Neurologic Presentation in Young Children Sustaining Inflicted and Unintentional Fatal Head Injuries*, 116 PEDIATRICS 180, 180 (2005) (on rare occasions, infants or toddlers may sustain a fatal head injury yet present to hospital clinicians as lucid before death); Scott Denton & Darinka Mileusnic, *Delayed Sudden Death in an Infant Following an Accidental Fall, A Case Report with Review of the Literature*, 24 AM. J. FORENSIC MED. PATHOLOGY 371 (2003) (9-month-old acted normally for 72 hours after fall before fatal collapse); Robert Huntington, Letter, *Symptoms Following Head Injury*, 23 AM. J. FORENSIC MED. PATHOLOGY 105 (2002) (reporting case of 13-month-old whose “severe intracranial injury symptoms...were delayed for several hours, during which time she was under our view and review in the hospital”). More recently, it has been noted that second impact syndrome—in which a minor impact occurring weeks to months after a more significant impact results in death—produces findings virtually identical to those in SBS/AHT cases. Robert C. Cantu & Alisa D. Gean, *Second-Impact Syndrome & a Small Subdural Hematoma: An Uncommon Catastrophic Result of Repetitive Head Injury with a Characteristic Imaging Appearance*, 27 J. NEUROTRAUMA 1557, 1557 (2010). This raises the possibility that the original trauma in some SBS/AHT cases may have occurred weeks to months before the collapse, possibly even at birth.

occur. Instead, the disputes about lucid intervals are more nuanced, usually arising over whether a lucid interval occurred in a particular case given the medical findings and symptoms. In a recent presentation, for example, Dr. Dias responded to the Gilliland research by noting that while children in the study experienced a period of lucidity following injury, all of the children who were seen by an independent observer “were described as *not normal*” during the interval.¹⁵⁰ However, the described symptoms, which included lethargy or fussiness, are signs of illness as well as head injury, and they provide little precision in timing.¹⁵¹ Such symptoms are not infrequently noted in children diagnosed with SBS, suggesting that some of these children may be ill rather than abused.¹⁵² Given these considerations, it has become increasingly difficult to time injuries or identify a perpetrator based on medical evidence alone.

3. Retinal hemorrhages.

In recent years, the focus in SBS/AHT cases has shifted from subdural hemorrhages and brain swelling, which are known to have many causes, to retinal hemorrhages. For many years, ophthalmologists and pediatricians testified that in the absence of severe trauma, retinal hemorrhages were highly suggestive or even diagnostic of shaking.¹⁵³ This position is puzzling since retinal hemorrhages are found in approximately one third of newborn babies¹⁵⁴ and in a wide range of conditions.¹⁵⁵ In adults, retinal

¹⁵⁰ Mark S. Dias, Presentation, *Concepts, Controversies & Conspiracy Theories in Abusive Head Trauma*, slide 34 at 12, New York City Abusive Head Trauma/Shaken Baby Syndrome Training Conference (Sept. 23, 2011) at http://www.queensda.org/SBS_Conference/SBC2011.html.

¹⁵¹ Gilliland, *supra* note 148, at 724. See also Huntington III, *supra* note 149, at 105.

¹⁵² See, e.g., *State v. Edmunds*, 746 N.W. 2d 590, 592 (2008) (during the hours before her death, the child did not feed normally and cried inconsolably).

¹⁵³ See, e.g., J.F. Geddes & John Plunkett, Letter, *The Evidence Base for Shaken Baby Syndrome*, 328 BRIT. MED. J. 719, 719 (2004) (“many doctors consider retinal hemorrhages with specific characteristics to be pathognomonic of shaking”; diagnosis is sometimes based on subdural or retinal hemorrhages alone”).

¹⁵⁴ M. Vaughn Emerson, et al., *Incidence & Rate of Disappearance of Retinal Hemorrhage in Newborns*, 108 OPHTHALMOLOGY 36, 37 (2001).

¹⁵⁵ See, e.g., Narang, *supra* note 3, Appendices B & C; Patrick E. Lantz & Constance A. Stanton, *Postmortem Detection & Evaluation of Retinal Hemorrhages*, 12 PROC. AM. ACAD. SCI. 271, 271

hemorrhages are closely linked to intracranial hemorrhages irrespective of cause, a phenomenon that is known as Terson syndrome.¹⁵⁶ To our knowledge, no explanation has ever been offered to explain why Terson syndrome would appear in adults but not in infants. Since infants are generally more vulnerable to illness or trauma¹⁵⁷ than adults, one might suspect that, if anything, children would be *more* susceptible to retinal hemorrhage than adults.

Since it was clear by 2006 that children also develop retinal hemorrhage in a wide range of conditions,¹⁵⁸ supporters of the SBS/AHT hypothesis modified their claim that retinal hemorrhages are highly suggestive of abuse. Instead, they argued that certain variants—specifically, retinoschisis (separation of the layers of the retina), retinal folds (lifting and folding of the retina) and/or extensive retinal hemorrhages (retinal hemorrhages that affect many retinal layers and extend to the ora serrata)—are highly suggestive or even diagnostic of abuse.¹⁵⁹ In recent years, however, this hypothesis has also begun to unravel. Today, it appears that the size and scope of retinal hemorrhages may be largely associated with edema and

(2006) (retinal hemorrhages present at autopsy in infants who died from meningitis, asphyxia/suffocation, prematurity/congenital conditions, heart disease, in utero hemorrhage, blunt force trauma, sudden infant death syndrome/resuscitation, apnea/gastroesophageal reflux, and birth-related causes); Henry E. Aryan et al., *Retinal Hemorrhage & Pediatric Brain Injury: Etiology & Review of the Literature*, 12 J. CLINICAL NEUROSCIENCE 624 (2005) (retinal hemorrhages associated with an ever-expanding list of conditions). It has also, of course, long been known that retinal hemorrhages and, less commonly, cerebral edema are linked to the lack of oxygen at high altitudes. See, e.g., Sankaranarayana P. Mahesh & Jeevan R. Mathura, Jr., *Retinal Hemorrhages Associated with High Altitude*, 362 N. ENGLAND J. MED. 1521, 1521 (2010); see also Fernando A. Moraga et al., *Acute Mountain Sickness in Children & Their Parents After Rapid Ascent to 3500 M (Putre, Chile)*; 19 WILDERNESS & ENVTL. MED. 287 (2008) (children more sensitive than adults to hypoxia from high altitudes).

¹⁵⁶ Albert Terson, *De l'hémorragie Dans le Corps Vitre au Cours de L'hémorragie Cerebrale*, 6 CLIN. OPHTHALMOL. 309 (1900).

¹⁵⁷ See, e.g., Jenny, *supra* note 7, slide 56, at 19 (infant response to injury is much worse than that of an adult); Centers for Disease Control and Prevention, *Protecting Against Influenza (Flu): Advice for Caregivers of Children Less than 6 Months Old* at <http://www.cdc.gov/flu/protect/infantcare.htm> (last visited 11/2/12) (infants younger than 6 months at higher risk of serious flu complications).

¹⁵⁸ See Lantz, *supra* note 135.

¹⁵⁹ See, e.g., Narang, *supra* note 3, at 548-553, 557.

time spent on life support rather than causation.¹⁶⁰ In addition, the severe, extensive retinal hemorrhages previously assumed to be diagnostic of SBS/AHT have also been identified in meningitis and an accidental short fall.¹⁶¹ The Atlas of Forensic Histopathology summarizes the current state of knowledge on retinal hemorrhages as follows:

The significance of retinal hemorrhage and optic nerve sheath hemorrhage is controversial. These hemorrhages are not, in and of themselves, sufficient to determine the presence of inflicted injury. Other circumstances under which retinal and optic nerve sheath hemorrhages may be found include resuscitation and cerebral edema. A recent retrospective study (Matshes, 2010) of 123 autopsies of children up to 3 years old showed retinal hemorrhage, optic nerve sheath hemorrhage, or both, in 18 cases. Of these, two were certified as natural deaths, eight as accidents, and eight as homicides. One finding of note was hemorrhage in six of seven cases without any head injury. There is a widespread belief among clinicians that skull fractures, subdural hematomas, and retinal hemorrhages do not occur in accidental short falls. In reality, all three have been found in cases of falls from short heights.¹⁶²

In short, it is becoming increasingly unlikely that the size, shape or location of retinal or optic nerve sheath hemorrhages will prove to be an accurate indicator of abuse.

Retinoschisis and retinal folds are similarly no longer deemed virtually diagnostic (pathognomonic) of shaking or abuse. The traditional theory was that absent an automobile accident or the like, retinoschisis or retinal folds could only be caused by the angular forces generated by the rapid acceleration and deceleration motion of

¹⁶⁰ Evan Matshes, *Retinal & Optic Nerve Sheath Hemorrhages Are Not Pathognomonic of Abusive Head Injury*, 16 PROC. OF THE AM. ACAD. FORENSIC SCI. 272, 272 (2010) (retinal hemorrhages and optic nerve sheath damage may be linked to cerebral edema and advanced cardiac life support and are not limited to children who die of inflicted head injuries).

¹⁶¹ Juan Pablo Lopez et al., *Severe Retinal Hemorrhages in Infants with Aggressive Fatal Streptococcus Pneumonia Meningitis*, 14 J. AM. ASS'N. PEDIATRIC OPHTHALMOLOGY STRABISMUS 97(2010); Lantz, *supra* note 135, at 1648, 1649.

¹⁶² PETER M. CUMMINGS ET AL., ATLAS OF FORENSIC HISTOPATHOLOGY 177 (2011); *see also* M. Vaughn Emerson et al., *Ocular Autopsy & Histopathologic Features of Child Abuse*, 114 OPHTHALMOLOGY 1384, 1384 (2007) (given our current lack of knowledge, "much of what we think we know about the ocular findings of child abuse will continue to be the result of speculation rather than based on sound evidence.").

shaking.¹⁶³ However, a series of case reports has now established that retinoschisis and retinal folds also occur in accidental injuries that do not involve rapid acceleration/deceleration forces but instead involve other types of forces, such as crush forces. In one case a fourteen-month-old child suffered a skull fracture, subdural hematoma, retinoschisis and retinal folds when a television fell on him.¹⁶⁴ In another, a four-month-old child suffered a fatal skull fracture with subdural hemorrhage and retinoschisis and retinal folds when a twelve-year-old child tripped and landed with her buttocks striking the infant's head.¹⁶⁵ In yet another case, a ten-week-old child suffered a skull fracture with subdural and subarachnoid hemorrhages, as well as retinal hemorrhages extending to the ora serata and retinal folds, when his mother, who was carrying him in a front-holding papoose, tripped and crushed his head between her chest and a wooden barrier.¹⁶⁶ Cases such as these have led researchers to conclude that, contrary to earlier beliefs, "there may be no retinal signs seen exclusively in non-accidental head injury."¹⁶⁷

4. Bruises, fractures and other findings.

In some cases, the triad is supplemented by bruises, fractures and other findings that can provide powerful confirmation of abuse. Ironically, however, such evidence may sometimes point in a

¹⁶³ See, e.g., Alex V. Levin, *Ocular Manifestations of Child Abuse* at 99-100, in Robert M. Reece and Stephen Ludwig, *Child Abuse, Medical Diagnosis and Management* (2nd Ed. Lippincott Williams & Wilkins 2001) (traumatic retinoschisis "highly specific for shaken baby syndrome and has never been described in any other condition of infants and young children in the shaken baby age range"; diagnosis aided by identification of paramacular folds).

¹⁶⁴ P. E. Lantz et al., *Perimacular Retinal Folds from Childhood Head Trauma*, 328 BRIT. MED. J. 754, 755-756 (2004) (statements in the medical literature that retinoschisis and perimacular retinal folds are diagnostic of shaken baby syndrome are not supported by objective scientific evidence).

¹⁶⁵ Gregg T. Lueder, et al., *Perimacular Retinal Folds Simulating Nonaccidental Injury in an Infant*, 124 ARCHIVES OPHTHALMOLOGY 1782, 1783 (2006).

¹⁶⁶ P. Watts & E. Obi, *Retinal Folds & Retinoschisis in Accidental & Non-Accidental Head Injury*, 22 NATURE 1514 (2008), available at <http://www.nature.com/eye/journal/v22/n12/full/eye2008224a.html>.

¹⁶⁷ *Id.* at 1514. As discussed below, the underlying problem is that the circularity and other confounding factors that affect the literature on subdural hemorrhages apply equally to the literature on retinal hemorrhages.

different direction. While bruises are often taken as confirmation of abuse, particularly in infants, in whom bruises are unexpected,¹⁶⁸ Dr. Michael Laposata, one of the nation's leading coagulation experts, has pointed out that it is rarely possible to differentiate on external examination between bruises caused by trauma and those caused by coagulopathies (bleeding disorders).¹⁶⁹ While a child who presents with bruises, subdural hemorrhage and retinal hemorrhage may indeed be the victim of abuse and should be evaluated accordingly, it is important to be aware that these features are also consistent with genetic or acquired coagulopathies, including disseminated intravascular coagulation.¹⁷⁰

Similar issues arise with skeletal findings. Contrary to popular belief, skull fractures may occur from birth trauma or household falls.¹⁷¹ Other fractures or bony abnormalities may result from accidental trauma, metabolic bone disease and/or nutritional deficiencies.¹⁷² In some cases, causation or vulnerability can be

¹⁶⁸ See, e.g., Naomi F. Sugar, et al., *Bruises in Infants & Toddlers: Those Who Don't Bruise Rarely Bruise*, 153 ARCHIVES OF PEDIATRICS & ADOLESCENT MED. 399 (1999) ("Bruises are rare in normal infants and precruisers and become common among cruisers and walkers. Bruises in infants younger than 9 months and who are not yet beginning to ambulate should lead to consideration of abuse or illness as causative").

¹⁶⁹ See generally Michael Laposata, *Overdiagnosis of Child Abuse Due to Undiagnosed Underlying Disease*, Am. Assoc. of Clinical Chemistry Annual Meeting (Dec. 2008) at http://www.aacc.org/resourcecenters/archivedprograms/expert_access/2008/december/Documents/1208EA.pdf; See also Martha E. Laposata & Michael Laposata, *Children with Signs of Abuse: When Is It Not Child Abuse?* 123 AM. J. CLIN. PATHOL., Supp. 1, S119, S120 (2005) (describing the "myriad of coagulopathies" that can mimic child abuse).

¹⁷⁰ See, e.g., *id.*; Marcel Levi & Hugo Ten Cate, *Disseminated Intravascular Coagulation*, 341 NEW ENGLAND J. OF MEDICINE 586, 586 (1999) (clinical conditions associated with disseminated intravascular association include sepsis, trauma, vascular disorders, reactions to toxins and immunological disorders).

¹⁷¹ See, e.g., Brian C. Patonay & William R. Oliver, *Can Birth Trauma Be Confused for Abuse?* 55 J. OF FORENSIC SCI. 1123 (2010); Ross Reichard, *Birth Injury of the Cranium & Central Nervous System* 18 BRAIN PATHOLOGY 565, 566 (2008) (incidence of skull fractures at birth is reported to be 2.9%); David S. Greenes & Sara A. Schutzman, *Occult Intracranial Injury in Infants*, 32 ANNALS EMERGENCY MED. 680, 684 (1998) (Duhaime reported that skull fractures were as likely to occur from falls of less than 4 feet as from falls of more than 4 feet; 18% of skull fractures in infants resulted from falls of less than 3 feet).

¹⁷² See Kathy A. Keller & Patrick D. Barnes, *Rickets vs. Abuse: a Nat'l and Internat'l Epidemic*, 38 PEDIATRIC RADIOLOGY 1210 (2008); Paul K. Kleinman, *Problems in the Diagnosis of Metaphyseal Fractures*, 38 PEDIATRIC RADIOLOGY S388, S390-S392 (2008); Andrew Hosken, *Call For Vitamin D Infant Death Probe*, BBC RADIO 4 TODAY (Jan. 26, 2012, at 3:06PM),

determined by testing and a careful medical history. In others, it may not be possible to differentiate between natural causes, accidental trauma and abuse on the basis of the medical findings alone.¹⁷³

5. Confessions.

As the differential diagnosis for the triad has expanded, the “case for shaking” as a mechanism of injury now rests largely on confessions.¹⁷⁴ SBS supporters argue that confessions prove that (a) some children with the triad were shaken; and (b) in the absence of a proven alternative, infants or children who present with the triad were almost certainly shaken.

The overriding problem is that confessions are not scientific evidence—and are rarely used as the basis for medical diagnoses—because the researcher cannot observe the underlying event. In the past decade, moreover, we have learned that confessions are not as reliable as once thought. Indeed, approximately 25% of the DNA exonerations in Innocence Network cases involved false confessions, guilty pleas or other incriminating statements to serious offenses

<http://www.bbc.co.uk/news/health-16726841> (parents acquitted of shaking child to death “after the jury learned that his fractures, supposedly telltale signs of abuse, could have been caused by his severe rickets. . . .Michael Turner QC, who defended Miss Al-Alas, told the BBC that he was shocked by the lack of knowledge about vitamin D deficiency of some of the expert witnesses at the trial, held at the Old Bailey”).

¹⁷³ See Alison M. Kemp et al., *Patterns of Skeletal Fractures in Child Abuse: Systematic Review*, 337 BRIT. MED. J. 1, 7 (2008) (stating that “no fracture on its own is diagnostic of child abuse”); Carole Jenny, *Clinical Report: Evaluating Infants & Young Children With Multiple Fractures*, 118 PEDIATRICS 1299 (2006) (citing Shea-Landry GL & Cole DE, *Psychosocial Aspects of Osteogenesis Imperfecta*, 135 CAN. MED. ASS’N J. 977-981 (1986) (“[B]one diseases associated with increased bone fragility can be subtle or difficult to diagnose. These children are usually preverbal and cannot give a cogent history of their experiences. If abuse has occurred, caregivers of young children may not be forthcoming with a truthful history. On the other hand, family members of a child having an undiagnosed bone disorder may not be able to explain any mechanism of injury and may be completely bewildered by the injuries. Many parents of children with genetic or metabolic bone disease report that they were initially accused of abusing their children”).

¹⁷⁴ See, e.g., Dias, *supra* note 72, at 368 (“the consistent and repeated observation that confessed shaking results in stereotypical injuries that are so frequently encountered in AHT—and which are so extraordinarily rare following accidental/impact injuries—is the evidentiary basis for shaking”) (emphasis in original).

such as rape and murder.¹⁷⁵ False confessions are produced in part by the psychological techniques used in interrogation,¹⁷⁶ including, among other things, the presentation of real or fabricated proof of guilt sufficient to make a suspect feel that the situation is hopeless.¹⁷⁷ An accused who is convinced that he or she will be convicted and believes that confessing will minimize the consequences (or at least put an end to the questioning) may well make a rational choice to confess, even falsely¹⁷⁸—a type of confession recognized in the research literature as “coerced compliant false confessions.”¹⁷⁹

Confessions are particularly problematic in the child abuse area. First, there are remarkably few confessions—at least relatively few confessions that have been identified and examined in the research literature—relative to the large number of alleged shaking injuries (reportedly in the range of 1,200 to 1,500 per year in the United States).¹⁸⁰ One review of the child abuse literature from 1969 to 2001

¹⁷⁵ *False Confessions*, INNOCENCE PROJECT, <http://www.innocenceproject.org/understand/False-Confessions.php> (innocent defendants made incriminating statements, delivered outright confessions or pled guilty in about 25% of DNA exoneration cases). Indeed, in the Central Park jogger case, multiple defendants falsely confessed. See, e.g., Anton McCray, INNOCENCE PROJECT, http://www.innocenceproject.org/Content/Antron_McCray.php.

¹⁷⁶ See Mark Handler, Am. Assoc. of Police Polygraphists, PowerPoint Presentation, *Avoiding False Confessions & Defending Against Charges That You Obtained One* (2011) (on file with authors) (factors contributing to false confessions include investigator bias; pressure-filled interrogations; overconfidence on ability to tell truthful from deceptive subjects; certain coercive tactics; and context and subject characteristics that increase vulnerability).

¹⁷⁷ See, e.g., Richard J. Ofshe & Richard A. Leo, *The Decision to Confess Falsely: Rational Choice & Irrational Action*, 74 DENV. U. L. REV. 979, 986 (1996-1997) (“investigators elicit confessions from the innocent . . . by leading them to believe that their situation, though unjust, is hopeless and will only be improved by confessing”); Steven A. Drizin & Richard A. Leo, *The Problem of False Confessions in the Post-DNA World*, 82 N. C. L. REV. 891, 916 (2004) (“The most effective technique used to persuade a suspect that his situation is hopeless is to confront him with seemingly objective and incontrovertible evidence of his guilt, whether or not any actually exists”).

¹⁷⁸ Standard interrogation methods include cutting off denials of guilt and making the suspect believe that his situation is hopeless, followed by minimization strategies that present a confession as in his best interest. See, e.g., Ofshe, *supra* note 177, at 998-99.

¹⁷⁹ *Id.* at 998.

¹⁸⁰ See, e.g., NATIONAL CENTER ON SHAKEN BABY SYNDROME, <http://www.dontshake.org/sbs.php?topNavID=2&subNavID=10> (last visited Aug. 13, 2012) (stating that “[a]n estimated 1,200 to 1,400 children are injured or killed by shaking every year in the United States”); Tuerkheimer, *supra* note 51, at 10 (observing that an estimated 1,500 SBS diagnoses a year may provide “an outside parameter”).

found only 54 confessions to shaking, only 11 of which had no signs of impact.¹⁸¹ As the author concluded, 11 cases (in this study, approximately 1 every 3 years on average) does not permit valid statistical analysis or provide support for many of the commonly stated aspects of shaken baby syndrome.¹⁸² Three other articles—one in the U.S. and two in France—have addressed confessions to shaking but did not identify the confessions or the circumstances in which the confessions were obtained in sufficient detail to review their validity.¹⁸³ In two of these articles, moreover, the confessions did not reliably match the recorded medical findings, which included evidence of impact such as skull fractures, scalp swelling and bruising, underscoring the challenge with confessions.¹⁸⁴ In such cases, the confession may have understated the actions, or the shaking may have had nothing to do with the collapse.

Second, the definitions of “shaking” used in the literature and the courtroom are broad and ill-defined, and often include admissions to conduct that no one seriously argues could cause brain injury and death. As Professor Imwinkelried points out, Dr. Caffey’s seminal 1972 article includes “burpings,” a “confession” that a mother merely said “she and her husband ‘might have shaken [the infant] when he cried at night,’” and a case in which a mother said she “yanked a child to prevent him from falling off a bassinet onto the floor.”¹⁸⁵ As Professor Imwinkelried noted, “[i]t is debatable whether such conduct should be characterized as the kind of major, violent shaking events that supposedly cause shaken baby

¹⁸¹ Jan E. Leestma, *Case Analysis of Brain-Injured Admittedly Shaken Infants: 54 Cases, 1969-2001*, 26 AM. J. FORENSIC MED. PATHOLOGY 199, 199 (2005).

¹⁸² *Id.*

¹⁸³ Suzanne P. Starling et al., *Analysis of Perpetrator Admissions to Inflicted Traumatic Brain Injury in Children*, 158 ARCHIVES PEDIATRIC & ADOLESCENT MED. 454 (2004); Catherine Adamsbaum et al., *Abusive Head Trauma: Judicial Admissions Highlight Violent and Repetitive Shaking*, 126 PEDIATRICS 546 (2010); Matthieu Vinchon et al., *Confessed Abuse Versus Witnessed Accidents in Infants: Comparison of Clinical, Radiological, & Ophthalmological Data in Corroborated Cases*, 26 CHILDS NERVOUS SYS. 637 (2010).

¹⁸⁴ Starling, *supra* note 183, at 456; Adamsbaum, *supra* note 183, at 549.

¹⁸⁵ Imwinkelried, *supra* note 49, at 6 (quoting John Caffey, *On the Theory & Practice of Shaking Infants: Its Potential Residual Effects of Permanent Brain Damage & Mental Retardation*, 124 AMER. J. DISEASES CHILD 161, 163 (1972)).

syndrome.”¹⁸⁶ In other cases, the confessions are to mild shaking intended to revive a comatose infant.¹⁸⁷ As Judge Posner of the U.S. Court of Appeals for the Seventh Circuit pointed out recently in *Aleman v. Village of Hanover Park*, this type of shaking is the proper way to initiate infant CPR; hence, admitting to it hardly constitutes a confession to deadly criminal abuse.¹⁸⁸

Third, many of the confessions in child abuse cases involve interrogation techniques that are known to produce false confessions or plea bargains. Some interrogations include assertions that the medical evidence proves that a child was shaken and that only the accused could have done it. In *Aleman*, Judge Posner described such a scenario:

They told him [the suspect] the only possible cause of Joshua’s injuries was that he’d been shaken right before he collapsed; not being an expert in shaken-baby syndrome, Aleman could not deny the officers’ false representation of medical opinion. And since he was the only person to have shaken Joshua immediately before Joshua’s collapse, it was a logical necessity that he had been responsible for the child’s death. Q.E.D. *A confession so induced is worthless as evidence, and as a premise for an arrest.*¹⁸⁹

Sometimes these interrogation techniques may convince innocent parents or caretakers that they have committed a crime—a type of confession known in the research literature as “persuaded false confessions.”¹⁹⁰ When confronted with “proof” of shaking or impact,

¹⁸⁶ *Id.* at 6-7.

¹⁸⁷ *See, e.g., Aleman v. Village of Hanover Park*, 662 F.3d 897 (7th Cir. 2011) (Posner, J.) (description of gentle shaking to elicit response from collapsed infant was interpreted as confession to violent shaking).

¹⁸⁸ *Id.* at 902 (stating that “Aleman’s mild shaking of Joshua was the proper initiation of CPR.”) (citations omitted).

¹⁸⁹ *Id.* at 907 (emphasis added) (citing *Crowe v. County of San Diego*, 608 F.3d 406, 433 (9th Cir. 2010); *Wilkins v. DeReyes*, 528 F.3d 790, 800-02 (10th Cir. 2008); *see also* Emily Bazelon, *Shaken-Baby Syndrome Faces New Questions in Court*, N. Y. TIMES MAG. (Feb. 2, 2011), <http://www.nytimes.com/2011/02/06/magazine/06baby-t.html?pagewanted=all> (reporting the case of Dinesh Kumar, a Canadian father whose conviction was overturned after he had pled guilty to shaking his 5-week-old son to death; Kumar says that “at the time of his guilty plea, he believed he had no hope of prevailing against the damning testimony of the state’s pathologist, who has since been discredited for giving error-riddled testimony based on botched autopsies”).

¹⁹⁰ *Id.* at 999 (“persuaded” false confessions “are given after a person has become convinced

parents may search their memories for what they might have done, ultimately recalling minor incidents that are then viewed as confessions or changing histories.¹⁹¹ Some of these interrogations occur immediately after a child's death or serious injury, when distraught parents or caretakers may be particularly vulnerable to suggestion, manipulation or memory lapses.¹⁹²

Other "confessions" are provided as part of a plea bargain. As elegantly described by Professor Tuerkheimer, acknowledgements of guilt accompanying a plea bargain may simply represent a cost-benefit analysis, with a full and logical evaluation of the circumstances.¹⁹³ Since innocent defendants charged with killing or severely injuring a baby confront a high likelihood that a jury will return a guilty verdict, a rational defendant who is offered a "substantial discount" will accept the terms of the offer, notwithstanding factual innocence.¹⁹⁴

Finally, even if we assume that all shaking confessions are accurate and that shaking caused the collapse or death,¹⁹⁵ this still would not provide reliable evidence that the collapse or death in other cases was caused by shaking, any more than the confession of one bank robber to robbing a bank would provide reliable evidence that a defendant in another case was guilty of robbing a different bank. Today, we know that there are many alternative causes for

that it is more likely than not that he committed the crime, despite possessing no memory of having done so...[they] are elicited when an interrogator attacks and shatters a suspect's confidence in his memory"). These are known as internalized false confessions.

¹⁹¹ *Aleman*, 662 F.3d at 902.

¹⁹² Research confirms that emotionally challenged individuals are more susceptible to the pressures and suggestiveness of interrogations. *See, e.g.*, Richard A. Leo & Deborah Davis, *From False Confession to Wrongful Conviction: Seven Psychological Processes*, 38 J. PSYCHIATRY & L. 9, 38-40 (2010).

¹⁹³ Tuerkheimer, *supra* note 95, at 532-35.

¹⁹⁴ *Id.* at 534.

¹⁹⁵ This assumption is unlikely to be valid. For example, some shaking confessions occur in cases in which there is clear evidence of impact, including skull fractures and bruising. *See, e.g.*, Starling, *supra* note 183, at 456 (observing that 12% of "shaking only" confessions showed evidence of scalp or skull injuries). In other cases, the confession is to shaking around the time of the child's collapse, but the radiology and pathology establish that the injury was older. When the confessions do not match the injury, we do not know whether the confession was false or whether the shaking had nothing to do with the injuries, as in *Aleman*.

findings previously attributed to shaking and that very few medical findings are specific for inflicted trauma. An assumption that shaking caused the collapse or death in cases with confessions would not, therefore, suggest that shaking caused the findings in cases without confessions.¹⁹⁶ At most, this would simply place shaking on the lengthy and ever increasing list of potential causes.

6. *New hypotheses.*

In the past decade, researchers have struggled to differentiate between abuse, accidental trauma and natural causes. However, as Dr. Duhaime has pointed out, in this area, when you ask a question, you get an answer that more often than not leads to additional questions—a result that is very frustrating for those who want an answer and want it now.¹⁹⁷ Given the developments of the past decade, many more decades may pass—and many more hypotheses may be advanced and discarded—before we fully understand all of the causes of sudden infant death, with or without the triad. Today, we are still seeking answers to the questions that we have been asking for 40 years or longer—questions such as, why do some infants or toddlers suddenly collapse or die? Why do some of these children have subdural hemorrhages while others do not? What does the presence of the triad (or some elements of the triad) tell us about the cause of the collapse or death? And are there any findings that

¹⁹⁶ Dr. Dias suggests that the “common and consistent admission by the perpetrator to shaking the infant . . . overwhelmingly suggests that shaking is an important component of infant abusive TBI and is, in fact, sufficient to cause the intracranial injuries found in AHT. To suggest otherwise (as required by the biomechanical evidence) would require that every confessed perpetrator has to have been consistently and universally lying about the same phenomenon, something that defies logic and common sense.” Dias, *supra* note 72, at 369-370. However, the same analysis applies in the opposite direction: since most caretakers do not confess to shaking or any other form of abuse even when offered plea bargains but instead describe similar patterns, including short falls and/or sick or neurologically impaired babies, one would have to assume that these parents were consistently and universally lying about what they saw, a pattern that may indeed defy logic and common sense.

¹⁹⁷ Ann-Christine Duhaime, et al., *The Real Science: What Research is Telling Us about SBS/AHT: From Questions to Answers: Application of the Scientific Method to Abusive Head Trauma by Interdisciplinary Research Teams*, 11th International Conference on Shaken Baby Syndrome/Abusive Head Trauma Conference, National Center on Shaken Baby Syndrome (Sept. 12, 2010) (presentation notes on file with authors).

can accurately distinguish between accidents, abuse and natural causes? For decades, we thought we had answers to some of these questions: we thought that the presence of the triad, or some of its elements, proved that the child had been shaken. Today, the correct answer to these questions is, "we don't know." And, until we do know, we are, in Dr. Duhaime's words, simply "shooting in the dark."¹⁹⁸

As our knowledge has increased, and as we have learned that much of what we thought we knew was wrong, there has been increased recognition that, as currently described, SBS/AHT is a hypothesis, not a proven fact. As Dr. Peter Richards, a pediatric neurosurgeon at Oxford and strong supporter of the shaking hypothesis, testified recently:

We have enormous gaps in our knowledge. Anything anyone says is informed speculation, not scientifically proven fact, including what I say in the reports.¹⁹⁹

If accompanied by full disclosure, informed speculation may in some instances suffice for treatment. It is unclear, however, that it is sufficient to support legal findings of assault or murder.

¹⁹⁸ *Id.* at 14. In this remark, Dr. Duhaime was discussing the unilateral "big black brain," *i.e.*, the one-sided brain swelling found in approximately one-third of alleged SBS cases. Since shaking would be expected to damage both sides of the brain, the unilateral big black brain has always presented a pathophysiological conundrum. Ann-Christine Duhaime et al., *The Real Science: What Research is Telling Us about SBS/AHT, From Questions to Answers: Application of the Scientific Method to Abusive Head Trauma by Interdisciplinary Research Teams*, Eleventh International Conference on Shaken Baby Syndrome/Abusive Head Trauma (Sept. 12, 2010) (notes on files with authors).

¹⁹⁹ *Gloucestershire County Council and RH, KS and JS*, Case No. GF11C00125 (High Court of Justice, Family Division, Bristol District Registry, March 29, 2012) at ¶ 59 (addressing subdural hematoma in infants); *see also* Testimony of Dr. Richards, *Regina v. Freeston*, No. T20110348 (In the Crown Court at Portsmouth, May 2, 2012) at 42-43 (everything on this subject is informed opinion; my opinion is exactly the same, no better, no worse); 43 (Q: And you can't point to specific scientific findings that prove your opinion is right? A: That's correct.); 66 (acknowledging a change in the way people are approaching the whole question of the triad and non-accidental injury). The *Freeston* case was dismissed after Dr. Richards' testimony. (Transcripts on file with authors.)

III. THE MEDICAL EVIDENCE: OLD AND NEW

Despite many warning signals, Dr. Narang argues that the research associating the triad, or some elements of the triad, with SBS/AHT is sufficiently reliable to form the basis for medical diagnoses and criminal convictions.²⁰⁰ While acknowledging that some of this research is marred by circularity,²⁰¹ he identifies a number of articles that he believes are sufficiently reliable to meet the standards of evidence-based medicine and *Daubert*. Dr. Narang further asserts that the biomechanical, neuropathological and anatomical research that casts doubt on the SBS/AHT diagnosis is unreliable and that the SBS/AHT diagnosis should rest on the judgment of clinicians, particularly child abuse pediatricians.²⁰² In this section, we address each of these points.

A. Literature Supporting the AHT Diagnosis.

In the past decades, scores, if not hundreds, of medical articles have been published that examine the relationship between medical findings such as subdural and retinal hemorrhages and child abuse. Dr. Narang draws upon these studies to argue that highly significant statistical associations exist between subdural and retinal hemorrhages and child abuse, and that these associations are sufficient to support medical diagnoses of abuse and criminal convictions for assault or murder. While it is undeniable that a vast number of medical articles assert that their findings support the SBS/AHT hypothesis, this literature suffers from circularity and other methodological flaws. In this section, we describe the underlying methodology and its limitations, summarize the key studies, and identify some of the methodological and interpretive flaws that frequently appear in these studies.²⁰³

²⁰⁰ Narang, *supra* note 3, at 586-87.

²⁰¹ Narang, *supra* note 3, at 561.

²⁰² Narang, *supra* note 3, at 594-95.

²⁰³ These studies largely address AHT as broadly defined, rather than SBS. Thus, even

1. *The methodology.*

The studies cited by Dr. Narang follow the same basic methodology. In each study, the authors accept the basic premises of the SBS/AHT hypothesis and adopt criteria based on those premises to classify cases that present with subdural hemorrhage or other elements of the triad as accidental, abusive or natural. While the results of this classification vary depending on the precise criteria selected, the size of the sample and the sophistication of the analysis, each study found that if one adopts the SBS/AHT hypothesis, a relatively large percentage of cases resulted from abuse rather than accident. From these studies, Dr. Narang concludes that the presence of subdural and retinal hemorrhages is a statistically powerful indicator of abuse.²⁰⁴ This methodology does not, however, confirm the hypothesis or help us determine its validity. Nor does it tell us much about the diagnostic specificity of subdural and retinal hemorrhages. Instead, all that it tells us is what the resulting breakdowns would be if the hypothesis and the resulting classifications were correct.

This type of circular classification system can be used to “confirm” any hypothesis, irrespective of its validity. For example, one might hypothesize that dogs are by nature friendly and that they bite only if they have been abused or are in pain. The logical corollary is that dogs that bite must have been abused or are in pain. If one adopts these hypotheses, dogs that bite but show no signs of pain must have been abused. The given history of “no abuse” would therefore be deemed inconsistent with biting, the owners would be assumed to be lying, and the dogs would be classified as “abused.” If one further places into this category any dog that has ever bitten without evidence of pain, even as a puppy, the abuse rates for dogs might be extremely high, even approaching 100%. And the percentage of dogs for whom biting is a statistically reliable indicator of abuse would similarly be very high (theoretically 100%). This does

accepted at face value, they say nothing about the validity of shaking as the mechanism of injury and do not provide any support for the shaking hypothesis. As discussed below, because of methodological and interpretative problems, they also say relatively little about the causes and incidence of AHT.

²⁰⁴ Narang, *supra* note 3, at 541-48.

not, however, confirm the hypothesis that biting dogs have been abused or that biting is statistically diagnostic of abuse; instead, it simply confirms what the breakdown would be *if* the hypothesis were correct. The abuse rates and correlation of biting to abuse might drop rapidly if one accepted alternative explanations, such as breed predisposition; age (very young or very old); instinctive protection of territory; poor eyesight; and/or fear of strangers.

In the SBS/AHT studies cited by Dr. Narang, the authors implicitly or explicitly accept the SBS/AHT hypothesis that subdural and retinal hemorrhages are generally traumatic in origin and require considerable force. The studies then use classification systems derived from this hypothesis to classify the findings as accidental, abusive, or (in a few instances) natural. Thus, if the parent or caretaker describes a major accident, often characterized as equivalent to a motor vehicle accident or fall from a great height, the findings are classified as accidental. If the parent or caretaker cannot describe such an event, and particularly if the parent or caretaker describes a short fall or no trauma at all, the history is deemed to be inconsistent with the findings, and the case is classified as abusive. While some studies make an effort to eliminate natural causes, such as birth trauma, others do not. Overall, there is a general expectation that the parent or caretaker should be able to explain the medical findings—an expectation that is unrealistic in light of the broad range of causes.

2. The evidence.

In the studies cited by Dr. Narang, the researchers typically select a cohort of children who have been diagnosed with head injury based on the presence of intracranial findings. Some studies focus on a particular element of the triad, such as subdural or retinal hemorrhage; others include evidence of impact, such as skull fractures or bruises. Using various criteria, the researchers then categorize the findings as abusive, accidental, natural or undetermined, with most studies attributing the findings to abuse if no known medical cause is found and the history is considered inadequate to explain the findings. The criteria for inadequacy vary considerably. For example, some researchers accept three-foot falls

as a legitimate explanation for a subdural hemorrhage²⁰⁵ while others accept only major motor vehicle accidents or falls from great heights.²⁰⁶ Not surprisingly, the studies produce different breakdowns depending on the selection criteria, the sophistication of the analysis, and the inclusion of natural causes. The varying conclusions—producing abuse rates for subdural hemorrhages ranging from 28 percent²⁰⁷ to 81 percent²⁰⁸ in the studies discussed by Dr. Narang—are just one indication of the unreliability of “clinical judgment” across hospitals, countries and time spans—the precise problem that evidence-based medicine and *Daubert* seek to address.

There are, however, common themes. Essentially, if natural causes are excluded or ignored (as is often the case) and if the outliers are removed, most studies find that approximately half (35 percent²⁰⁹ to 60 percent²¹⁰) of the parents or caretakers can provide an “acceptable” traumatic explanation for a subdural hemorrhage while approximately half cannot. Since the researchers generally assume that subdural hemorrhages require more force than other head injuries (including skull fractures), the “abuse” rate for subdural hemorrhages is typically much higher than the “abuse” rate for skull fractures and other head injuries.²¹¹ This “abuse rate” is then used to

²⁰⁵ Duhaime, *supra* note 57, at 179, 180 (intradural or subdural hemorrhages classified as neither presumptive nor suspicious for inflicted injury if the history is of a fall greater than or equal to three feet).

²⁰⁶ Dimitra Tzioumi & R. Kim Oates, *Subdural Hematomas in Children Under 2 Years, Accidental or Inflicted? A 10-Year Experience*, 22 CHILD ABUSE & NEGLECT 1105, 1107 (1998) (motor vehicles accidents and falls from over eight feet considered sufficient to explain injuries).

²⁰⁷ Jakob Matschke et al., *Nonaccidental Head Injury is the Most Common Cause of Subdural Bleeding in Infants <1 Year of Age*, 124 PEDIATRICS 1587 (2009)

²⁰⁸ Duhaime, *supra* note 57, at 183. Cf. Alison M Kemp, *Abusive Head Trauma: Recognition and the Essential Investigation*, 96 ARCHIVES OF DISEASE IN CHILDHOOD EDUC. & PRAC. ED. 202, 205 (finding that “for a child under 3 years old with intracranial injury alone the probability of AHT was only 4%”).

²⁰⁹ Linda Ewing-Cobbs et al., *Neuroimaging, Physical, and Developmental Findings after Inflicted and Noninflicted Traumatic Brain Injury in Young Children*, 102 PEDIATRICS 300, 303 (1998).

²¹⁰ Kirsten Bechtel et al., *Characteristics that Distinguish Accidental from Abusive Head Trauma in Hospitalized Young Children with Head Trauma*, 114 PEDIATRICS 165, 176 (2004).

²¹¹ For example, in 1992, Duhaime categorized 24% of head injuries and 81% of subdural hemorrhages as abusive. Duhaime, *supra* note 57, at 181. This same pattern is found in more recent studies. In 2005, for example, Vinchon classified 38% of head injuries and 64% of subdural hemorrhages as abusive. M. Vinchon et al., *Accidental and Nonaccidental Head*

confirm the high correlation between subdural hemorrhages and SBS/AHT.

In this section, we briefly describe the key findings in a selection of studies cited by Dr. Narang on subdural hemorrhages.²¹² We then discuss some of the methodological problems with these studies.

a. Duhaime (1992).²¹³

This study examined 100 consecutively admitted children 24 months of age or younger with a primary diagnosis of head injury.²¹⁴ Subdural hemorrhages were classified as abusive if (i) they were accompanied by clinical or radiographic findings of focal impact with no history of trauma obtainable; (ii) the caregiver provided a history of a fall less than three feet when seen in association with a changing or developmentally incompatible history; *or* (iii) unexplained injuries such as healing long-bone fractures were present.²¹⁵ Under this classification system, all of the subdural hematomas deemed accidental resulted from motor vehicle accidents; falls under three feet were categorized as trivial and constituted one prong of the test to confirm abuse.²¹⁶ There appears to have been no consideration of natural causes, including birth injuries. This study classified 81% of the subdural hemorrhages in the study group as abusive and 19% as accidental.²¹⁷

Injuries in Infants: A Prospective Study, 102 J. NEUROSURGERY: PEDIATRICS 380, 381, 383 (2005). These and other studies are discussed below. See *infra* Part III.A.2.a-i.

²¹² While we focus on subdural hemorrhages in this section, the same methodological problems apply to the studies on retinal hemorrhages. See, e.g., *infra* note 271 and accompanying text.

²¹³ Duhaime, *supra* note 57.

²¹⁴ *Id.* at 179.

²¹⁵ *Id.* at 180.

²¹⁶ Consistent with Duhaime's earlier study (Duhaime, *supra* note 57), the authors concluded that shaking "does not generate sufficient deceleration forces" to cause subdural hemorrhages and brain injuries and that impact is required. Duhaime, *supra* note 57, at 183. They postulated that caretakers cause subdural hemorrhages by shaking, swinging or throwing the child, with the head stopping abruptly against a surface. *Id.* No biomechanical or empirical support is provided for this hypothesis. *Id.*

²¹⁷ Duhaime, *supra* note 57, at 184.

b. Ewing-Cobbs (1998).²¹⁸

This study examined 40 children ages one month to six years hospitalized for inflicted or noninflicted traumatic brain injury.²¹⁹ In determining abuse, the authors used a classification scheme similar to that of Duhaime (1992) to determine whether a caretaker's history was compatible or incompatible with the findings.²²⁰ Head injuries were classified as abusive if the caretakers described falls of under four feet or from arm height.²²¹ Children with documented prior histories of brain injury, metabolic/neurological disorders or prematurity (gestation of less than 32 weeks) were excluded from the study.²²² This study categorized 64% of the subdural hemorrhages in the study group as abusive and 36% as accidental (most commonly in motor vehicle accidents).²²³

c. Feldman (2001).²²⁴

This study examined 66 children less than three years of age with subdural hemorrhages or effusions.²²⁵ Histories that were considered to be incompatible with the findings included all cases with no history of trauma, all short falls, stairway falls, and an adult falling on a child.²²⁶ The acceptable histories included motor vehicle accidents, falls from 10 feet or more, and major accidents (kicked by horse, dresser fell on head, and hit on head by falling log).²²⁷ Children with previously known hemorrhagic disease, previous neurosurgical procedure, previously recognized perinatal brain injury, meningitis, brain atrophy, central nervous system infections, renal dialysis, or

²¹⁸ Ewing-Cobbs, *supra* note 209.

²¹⁹ *Id.* at 300.

²²⁰ *Id.* at 301.

²²¹ *Id.*

²²² *Id.*

²²³ *Id.* at 303.

²²⁴ Kenneth W. Feldman et al., *The Cause of Infant and Toddler Subdural Hemorrhage: A Prospective Study*, 108 PEDIATRICS 636 (2001).

²²⁵ *Id.* at 636.

²²⁶ *Id.* at 639.

²²⁷ *Id.*

severe dehydration/hyponatremia or cardiopulmonary bypass were excluded.²²⁸ This study categorized 59% of subdural hemorrhages in the study group as likely/highly likely/definite abuse; 23% as likely/highly likely/definite unintentional; and 18% as indeterminate.²²⁹

d. Wells (2002).²³⁰

This study included 293 children less than three years of age with intracranial hemorrhages that were evident on radiological examination. Intracranial hemorrhages were categorized as abusive if (i) the caretaker offered no explanation for the findings, (ii) the findings were in the authors' view incompatible with the stated mechanism; or (iii) there was a confession of abuse.²³¹ Children with a history of hemorrhage from prematurity, birth trauma, surgery or nontraumatic medical conditions were excluded.²³² This study categorized 50.5% of intracranial hemorrhages as abusive, 37.2% as accidental, and 12.3% as undetermined.²³³

e. Bechtel (2004).²³⁴

This study examined 87 children under 24 months admitted with a diagnosis of head injury and who had a CT scan.²³⁵ Head injuries

²²⁸ *Id.* at 637.

²²⁹ *Id.* at 638. Histories considered indeterminate included a 2-month-old who fell from a kitchen counter onto a hardwood floor while restrained in a bouncy seat (minor injuries consistent with the fall but no independent witness); a fall by a father onto a 7-month-old with the father's full weight landing on the child (indeterminate since the mother was momentarily out of sight); a 2-month-old who fell down 3 carpeted stairs with his father (witnessed by maternal grandmother; child also had chronic effusions and rib fractures that could have been perinatal); and a 4-month-old who was in a truck that was hit by a crane, throwing the infant to the floor with his mother landing on top of him (child also had chronic effusions from possible birth injury). *Id.* at 641-42.

²³⁰ Robert G. Wells et al., *Intracranial Hemorrhage in Children Younger Than 3 Years*, 156 ARCH. PEDIATR. ADOLESC. MED. 252 (2002).

²³¹ *Id.*

²³² *Id.* at 253.

²³³ *Id.* at 254.

²³⁴ Bechtel, *supra* note 210.

²³⁵ *Id.* at 165.

were categorized as abusive if (i) there was no history of a traumatic event (fall, blow to head or motor vehicle crash); (ii) the history of a traumatic event was incompatible with developmental level; (iii) the inflicted injury was witnessed; (iv) there was a confession; or (v) there were other physical injuries consistent only with inflicted injuries (e.g., pattern bruises, occult rib or extremity fractures).²³⁶ In this study, virtually all of the cases classified as abuse had no history of significant trauma. Natural causes and birth injury were not addressed.²³⁷ This study categorized 40% of subdural hemorrhages in the study group as abusive and 60% as accidental.²³⁸

f. Hobbs (2005).²³⁹

This study included 186 children less than two years of age with subdural hemorrhages from the United Kingdom and the Republic of Ireland.²⁴⁰ Causation was determined by reporting clinicians and pathologists without predetermined criteria. This study classified 57% of subdural hemorrhages as abusive, 30% as natural (perinatal, meningitis and other medical conditions), 9% as undetermined and 4% as accidental.²⁴¹

g. Vinchon (2005).²⁴²

This study examined 150 children younger than 24 months of age hospitalized for craniocerebral traumatic lesions. The authors noted that the pathophysiology of subdural hemorrhages appeared to relate to the child's age rather than a specific cause of trauma. Twenty-one cases of birth trauma and five cases with natural causes (idiopathic macrocranium, hemophilia A) were identified. A disproportionate number of abuse cases had a history of perinatal illness (prematurity,

²³⁶ *Id.* at 166.

²³⁷ *Id.*

²³⁸ *Id.* at 168.

²³⁹ C J. Hobbs et al., *Subdural Haematoma and Effusion in Infancy: An Epidemiological Study*, 90 ARCHIVES DISEASE CHILDHOOD 952(2005).

²⁴⁰ *Id.*

²⁴¹ *Id.* at 954.

²⁴² Vinchon et al., *supra* note 211, at 380.

obstructed labor, hospitalization after birth), which the authors speculated might have led to poor parental bonding. The authors did not appear to consider that these children may have been suffering from birth injuries.²⁴³ This study classified 64.4% of subdural hemorrhages as abusive.

h. Matschke (2009).²⁴⁴

This study looked at subdural hemorrhages in fifty autopsies of infants under one year of age.²⁴⁵ Since this study addressed children who died, it would have encompassed the most severe head injuries. At autopsy, 62% of the subdural hemorrhages were attributed to natural causes, 30% to trauma, and 8% to undetermined causes.²⁴⁶ The natural causes consisted of coagulation disorders (28%), perinatal conditions (28%), infection (8%) and metabolic disorders (2%).²⁴⁷ In a retrospective review, the authors classified the trauma cases as abusive if they resulted in a confession, criminal conviction, or at least three of the following findings: (i) subdural hemorrhage; (ii) retinal hemorrhage; (iii) an inadequate history; (iv) serious external injury, *i.e.*, hematomas or lacerations; (v) unexplained fractures of the long bones, ribs or skull; or (vi) simple or gliding contusions.²⁴⁸ Histories viewed as inadequate included sudden collapse/found lifeless; falls from a baby buggy, couch or father's arms; accidental head bumps; and, in one case, a confession of beating and shaking to stop crying.²⁴⁹ Under these criteria, all but one of the trauma cases

²⁴³ Subdural hemorrhages, skull fractures, classical metaphyseal lesions (CMLs) and rib fractures may all be found at birth. *See, e.g.*, Rooks, *supra* note 109, (identifying subdural hemorrhages in nearly half of asymptomatic newborns); Rick R. van Rijn, *Birth-Related Mid Posterior Rib Fractures in Neonates: a Report of Three Cases (and a Possible Fourth Case) and a Review of the Literature*, 39 PEDIATRIC RADIOLOGY 30, 33 (2009) (fractures in full-term neonates are a well-known finding even after uneventful deliveries; CMLs and fractures of the clavicle, long bones, spine and skull have been reported from birth trauma); Reichard, *supra* note 171, at 566 (incidence of skull fractures at birth is reported to be 2.9%).

²⁴⁴ Matschke, *supra* note 207.

²⁴⁵ *Id.* at

²⁴⁶ *Id.*

²⁴⁷ *Id.*

²⁴⁸ *Id.* at 1588.

²⁴⁹ *Id.* at 1593, tbl. 1.

were considered to be abusive.²⁵⁰ Thus, overall, 28% of the subdural hemorrhages were classified as abusive and 2% as accidental.

i. Vinchon (2010).²⁵¹

This study collected 412 cases of traumatic head injury in children under 24 months of age, classifying 30% of head injury cases as abusive and 70% as accidental.²⁵² It did not separate subdural hemorrhage from other head injuries. Instead, it attempted to determine whether there were significant differences between confessed abuse cases and witnessed accidents.²⁵³ Forty-five cases of confessed inflicted head injury were compared with 39 cases of accidental trauma occurring in public places.²⁵⁴ The study found that 36.3% of the abuse cases (30 shaking, 15 beating) resulted in confessions obtained from judicial sources during or after the proceedings had been made public, as determined by a forensic pediatrician, while 13.5% of the accidents were corroborated by independent witnesses.²⁵⁵ In identifying SBS/AHT, the article endorsed the diagnostic value of what it called the "Ontario" triad, *i.e.*, subdural hemorrhage, retinal hemorrhage and no signs of impact,²⁵⁶ rather than the classic triad of subdural hemorrhage, retinal hemorrhage and encephalopathy. In this series, clinical signs of encephalopathy were often minimal and brain ischemia was detected by CT scan in only 27% of abuse cases.²⁵⁷ While the authors suggest

²⁵⁰ *Id.* at 1589.

²⁵¹ Vinchon, *supra* note 183.

²⁵² *Id.* at 639.

²⁵³ *Id.* at 638 (stating "The purposes of our study were to provide reliable elements for the differential diagnosis between [accidental trauma] and [inflicted head injury]...").

²⁵⁴ *Id.* at 639.

²⁵⁵ *Id.*

²⁵⁶ *Id.* at 643. The "Ontario" triad is based on an article by Michael Pollanen, Charles Smith and others. Charles Smith is the Ontario pathologist whose misdiagnosis of abuse in multiple cases in Ontario triggered the Goudge Inquiry. Michael S. Pollanen et al., *Fatal Child Abuse-Maltreatment Syndrome: A Retrospective Study in Ontario, Canada, 1990-1995*, 126 FORENSIC SCI. INT. 101 (2002).

²⁵⁷ This study did not control for confounding variables, such as the evolution of the intracranial pathology in the interval between the injury and clinical assessment or scan, which was significantly different in the two groups of patients. Vinchon, *supra* note 183, at

that the use of confessions avoids the problem of circularity, it is difficult to assess this claim since the confessions were not spontaneous and there is no information on their content or the conditions under which they were obtained.²⁵⁸ Based on confessions, the authors conclude that the presence of subdural hemorrhage, severe retinal hemorrhage and absence of impact provides “virtual certainty of abuse.”²⁵⁹

j. Other studies.

Other studies cited by Dr. Narang use similar procedures to categorize cases as abusive, accidental or natural, with some considering a broader range of causes than others.²⁶⁰ While fractures and bruises are often used to support findings of abuse, there is often relatively little effort to assess the age of these findings or to explore their relationship to nutritional deficiencies, coagulopathies or birth issues. Instead, most diagnoses of abuse continue to rest heavily on the inability of parents or caretakers to explain the medical findings—a process that is plagued with unknowns, even for medical professionals.

3. The flaws.

As even a brief review of the literature suggests, the numerous studies that have concluded that SBS/AHT is a frequent cause of the triad and that subdural hematomas and retinal hemorrhages are reliable indicators of abuse have methodological flaws that range from circularity to statistical mishaps.

641, tbl. 2.

²⁵⁸ The authors state that they had little data on the details, perpetrator, or mechanism of abuse. Under these conditions, it is impossible to verify causality or reliability. *Id.* (Vinchon, *supra* note 183, at 642).

²⁵⁹ *Id.* at 643.

²⁶⁰ For example, a small study from Spain excluded 15 babies with subdural hemorrhages from birth trauma, accidental trauma, or natural causes, including CNS infections and glutaric acidosis. In the 20 remaining cases, the study identified 3 cerebrovascular accidents (2 arteriovenous malformations and 1 sinus thrombosis) and 2 coagulation disorders. Victoria Trenchs et al., *Subdural Haematomas and Physical Abuse in the First Two Years of Life*, 43 PEDIATRIC NEUROSURGERY 352, 353-54, 354 (2007).

a. Circularity.

The primary defect is that virtually all of the SBS/AHT literature is circular. In study after study, doctors assume that, in the absence of a known medical explanation, subdural hemorrhages are caused by major trauma. Cases are then classified as abusive if the parents cannot describe a major trauma or substantiate a natural cause. As set forth in articles by leading child abuse pediatricians, these criteria were still being used in 2008. For example, Dr. Reece proposed that when the triad was present, the diagnosis of SBS was "highly probable" when *one* of the following is present: no history of trauma; a history inconsistent with the injuries; a history that changes over time; witnessed shaking and/or impact; confession to shaking and/or impact; or additional information supplied by a multidisciplinary child-protection team.²⁶¹ In a review, Dr. Hymel recommended omitting the second criterion (history inconsistent with the injuries) since that "presumes that we already know which histories are 'inconsistent' and which are 'consistent.'" Dr. Hymel suggested that additional research is needed to determine, with increasing precision, which histories are consistent and which are inconsistent.²⁶²

Under these standards, it is not surprising that some 50% of parents or caretakers cannot explain the findings to the satisfaction of the researchers. Contrary to Dr. Narang's suggestion, this does not prove that 50% of subdural hemorrhages are caused by abuse. All that it proves is that the researchers *believe* that this is so. One cannot validate a hypothesis based on a classification system that assumes the association that one wishes to prove. This is no different than deciding, *a priori*, that all male teenagers with long hair are drug users, assigning all male teenagers into "drug" and "drug-free" groups based on the length of their hair, and announcing that you have established a 100% correlation between long hair and drug use (and a corresponding 100% correlation between short hair and no drug use), with no effort to determine whether the correlation reflects

²⁶¹ Robert M. Reece, *What Are We Trying to Measure? The Problems of Case Ascertainment*, 34 AM. J. PREV. MED. S116, S118 (2008).

²⁶² Kent P. Hymel, *Sample Review, Epidemiology*, QUARTERLY UPDATE at <http://www.quarterlyupdate.org/epidemiology> (last visited July 24, 2012).

reality.

Since the circularity problem is well-recognized—Dr. Jenny pointed it out in 2002 and Dr. Narang agrees that “some circularity is inevitable” —Dr. Narang asserts that “numerous well-designed studies [have] set out to control circularity in their experimental design.”²⁶³ For instance, in 2004, Bechtel²⁶⁴ attempted to minimize circularity by using selection criteria based on “presenting history and physical examination findings.”²⁶⁵ As in other studies, however, “no history of traumatic event” was one of the criteria used to identify abuse,²⁶⁶ with 12 of the 15 reportedly abused children characterized as abused based on this criterion.²⁶⁷ Since there are many nontraumatic causes for subdural hemorrhages, this study almost certainly over-estimated the incidence of abuse.

Vinchon *et al.* later attempted to reduce circularity by examining cases of confessed abuse in France.²⁶⁸ While this might seem to be a logical improvement over earlier studies, the reliability of confessions is far from certain, as discussed above. Not surprisingly, the greatest incentive and pressure to confess may occur when the doctors, investigators and judiciary believe that the triad is strong evidence of abuse since, in these cases, the alleged abusers will likely be told—not just by the doctors, police and prosecutor but often by their own attorneys and even their own families—that the medical evidence is conclusive and the hope for acquittal is slim to nonexistent. In such cases, the attorney may advise—and a parent or caretaker may realistically conclude—that the best option is to accept fault irrespective of guilt. In this study, the high rate of confessions (36.3%) combined with a lack of information on the cases and the fact that all confessions appear to have been obtained during judicial proceedings raises concerns with the reliability of the data.²⁶⁹

²⁶³ Jenny, *supra* note 79, at 51-52; Narang, *supra* note 3, at 560-61.

²⁶⁴ Bechtel, *supra* note 210.

²⁶⁵ *Id.* at 166.

²⁶⁶ *Id.*

²⁶⁷ *Id.*

²⁶⁸ Vinchon, *supra* note 183.

²⁶⁹ *Id.* at 639.

Other researchers, such as Matschke, attempted to address circularity by using criminal conviction as one of the inclusion criteria.²⁷⁰ Since, however, such convictions are almost always based on the assumptions (and resulting medical opinions) that the research is designed to test, this criterion is entirely circular. This problem applies equally to the studies on retinal hemorrhages and other ocular findings since these studies use the same methodologies as the studies on subdural hemorrhages.²⁷¹

b. Rule-out diagnoses.

In 1996, SBS was a “rule in” diagnosis, *i.e.*, if the triad elements were found, SBS was automatically diagnosed, at least in the absence of a known alternative cause. Today, SBS/AHT is a “rule out”

²⁷⁰ Matschke, *supra* note 207, at 1588.

²⁷¹ In a recent review of the literature on retinal hemorrhages, the authors noted the potential for circular logic in all but 4 of the 20 studies reviewed. Gaurav Bhardwaj et al., *A Systematic Review of the Diagnostic Accuracy of Ocular Signs in Pediatric Abusive Head Trauma*, 117 *OPHTHALMOLOGY* 983, 985 (2010). However, these 4 studies used the same criteria as the studies on subdural hemorrhages and were also circular. Jane D. Kivlin et al., *Shaken baby syndrome*, 107 *OPHTHALMOLOGY* 1246 (2010) (SBS diagnosed by child advocacy physicians based on subdural hematomas and absence of history of major accidental trauma, accompanied in some cases by bone injuries); Kirsten Bechtel et al., *Characteristics that Distinguish Accidental from Abusive Head Trauma in Hospitalized Young Children with Head Trauma*, 114 *PEDIATRICS* 165 (2004) (criteria for abuse included clinical and radiological evidence of brain injury with no history of traumatic event or history of trauma incompatible with developmental level, witnessed inflicted head injury, confession, or evidence of other physical injuries); Elizabeth E. Gilles et al., *Retinal hemorrhage Asymmetry in Inflicted Head Injury: a Clue to Pathogenesis?*, 143 *J. PEDIATR.* 494 (2003) (injury characterized as inflicted if witnessed or accompanied by confession, felony conviction, or minimal or absent history of trauma); Vincent Pierre-Kahn et al., *Ophthalmologic Findings in Suspected Child Abuse Victims with Subdural Hematomas*, 110 *OPHTHALMOLOGY* 1718 (2003) (children with subdural hemorrhage who had no clinical or radiologic evidence of impact and no acceptable alternative explanation were presumed to have been shaken). A more recent review relied on some of the same studies and is also circular. SA Maguire et al., *Retinal haemorrhages and related findings in abusive and non-abusive head trauma: a systematic review*, *Eye* doi: 10.1038/eye.2012.213 (Oct. 19, 2012, epub ahead of print) (AHT determined by case conference, multidisciplinary assessment, admission or witnessed event; certain patterns of retinal hemorrhage far more common in AHT and extremely rare in accidental injury; however, no retinal sign is unique to abusive injury). While these studies conclude that there is an association between ocular findings and SBS/AHT, what they actually show is an association between eye findings and intracranial abnormalities, including subdural hemorrhage. Since the eye is an extension of the brain, this association is not surprising; however, it says nothing about causation.

diagnosis, *i.e.*, a diagnosis that can be made only if all other possible causes have been “ruled out” or excluded.²⁷² “Rule out” diagnoses are also known as diagnoses of exclusion or default diagnoses. By definition, these diagnoses occur when there is no laboratory test or direct evidence that would prove the diagnosis. If there were such a test or direct evidence, we would use them rather than going through the long, complex and ever-evolving list of “rule outs.”

Because “rule out” diagnoses cannot be confirmed, they run a significant risk of being wrong. For example, doctors believed for years that stomach (gastric) ulcers were caused by stress: when they could find no other cause, the default diagnosis was that it must be the patient’s fault.²⁷³ As it turned out, however, ulcers are predominantly caused by bacterial infections.²⁷⁴ Such misunderstandings of causation may do relatively little harm when there is no known treatment for the findings.²⁷⁵ In contrast, misdiagnoses of child abuse cause immediate and often irrevocable harm by removing children from their homes, imprisoning innocent parents and caretakers, and destroying families. Such misdiagnoses may also result in improper or inadequate treatment for conditions that, if properly diagnosed, may have been eminently treatable.

The potential error rate of rule-out diagnoses increases as the number of alternative diagnoses expands. In SBS/AHT, there are tens or hundreds of known “rule outs,” some of which can be identified only when the child is alive and others that can be

²⁷² See *e.g.*, Jenny, *supra* note 7; Narang, *supra* note 3, at 569.

²⁷³ See, *e.g.*, Press Release, The Nobel Prize in Physiology or Medicine (Oct. 3 2005) available 2005, Barry J. Marshall, J. Robin Warren, Nobel Prize website at http://www.nobelprize.org/nobel_prizes/medicine/laureates/2005/press.html (stress and lifestyle were considered the major causes of peptic ulcer disease before the discovery of *Helicobacter pylori* by Marshall and Warren, who received the Nobel Prize for their work).

²⁷⁴ *Id.*; see also MAYO CLINIC Staff, *Peptic Ulcer: Definition*, available at <http://www.mayoclinic.com/health/peptic-ulcer/DS00242> (doctors now understand that bacterial infection or some medications, not stress or diet, cause most peptic ulcers).

²⁷⁵ In the case of ulcers, one could argue that if an incorrect “rule out” diagnosis had not been propounded and widely accepted, the cause might have been discovered much more quickly. The failure to identify the true cause of ulcers also resulted in unnecessary surgery that may have increased morbidity and mortality. See, *e.g.*, J. R. Todd Jr., *Peptic Ulcer Disease, An 11 Year Study*, 63 J. NAT’L. MED. ASS’N. 40, 42 (1971) (discussing morbidity and mortality rates following Billroth II procedures).

identified only after death.²⁷⁶ As described by Dr. Narang, the “rule-out” procedure requires a detailed whole body physical examination and complete medical history, including a detailed history of the complaints surrounding the presenting symptoms; any history of trauma, infection and/or exposure to infection; a detailed history of prior illnesses, surgeries and hospitalizations; birth history; developmental history; a history of relevant family medical illnesses/disorders; and a comprehensive psychosocial history.²⁷⁷ In addition, the clinician must review the laboratory tests and radiology images and work with multiple agencies and medical specialties.²⁷⁸ These findings then form the basis for a differential diagnosis, or list of possible causes. Dr. Narang suggests that many “potential disorders can be eliminated through a detailed history, physical examination, and initial laboratory and radiologic” results.²⁷⁹ In so doing, the clinician must synthesize the information gathered 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.”²⁸⁰ This is a daunting task given the paucity of knowledge on the pathophysiology of the infant brain and the lack of evidence-based statistical information on causation. It is, moreover, unlikely that individual clinicians will have experience with the broad range of alternative causes, including

²⁷⁶ For example, seizure activity and some coagulation abnormalities can only be identified when the child is alive, while slides of the brain and meninges, which may reveal congenital abnormalities or pre-existing injury, can only be obtained after death.

²⁷⁷ Narang, *supra* note 3, at 569-571.

²⁷⁸ *Id.* at 573; see also Jenny, *supra* note 7, at 9 (recommending an even more detailed “rule out” procedure which includes a complete evaluation of past history, including prenatal history; a family history going back generations, including unexpected deaths, genetic or metabolic disease; a social history; a complete systems review, including medications, allergies, immunizations and feeding history; a review of exposures, including travel, pets and toxins; a minute-by-minute “incredibly detailed” history of recent events; a detailed head-to-toe physical exam; a review of old records, including birth records, growth charts, past imaging studies, lab results and hospitalizations; extensive laboratory testing and radiology imaging, including MRI, MRA and MRV; and consults with specialists in many fields, including hematology, metabolic, genetics and infectious disease, as needed. For children who survive, the clinician should follow the child’s long-term care; for those who do not, the clinician should attend the autopsy and consult with the medical examiner, as needed.)

²⁷⁹ Narang, *supra* note 3, at 573.

²⁸⁰ *Id.*

childhood stroke and rare genetic conditions.

Despite the wide range of alternatives, Dr. Narang suggests that at the end of this process “in the vast majority of cases, the common denominator for SDH’s and RH’s will be trauma,” in which case the clinician should distinguish between accidental and abusive head trauma by focusing on “inconsistencies.”²⁸¹ Dr. Narang defines inconsistency as (i) the absence of a history; (ii) a history that substantially changes or evolves; (iii) a history that is inconsistent with the child’s developmental capabilities; (iv) a history that is inconsistent with the pathophysiology of the injuries; or (v) a history that is inconsistent with the SBS/AHT literature.²⁸² Dr. Narang concludes that in the presence of such inconsistencies, “the clinician can diagnose ‘AHT/non-accidental trauma’ with a reasonable degree of medical certainty.”²⁸³

This process presents considerable challenges. For example, to determine if a particular injury is consistent with an accidental fall, the clinician must have a solid understanding of biomechanics and the unique characteristics of the fall; the unique characteristics and vulnerabilities of the child, including any genetic, nutritional or birth-related predisposing factors; the secondary metabolic response to injury; the anatomy of the developing brain; and the time course of the injury, including the impact of medical interventions.²⁸⁴ Since there is strong evidence that an infant’s response to a given injury is much worse than an adult’s response to a similar injury,²⁸⁵ what might appear to be minor or even trivial trauma in an adult may

²⁸¹ *Id.* at 573.

²⁸² *Id.* at 573-74

²⁸³ *Id.* at 574.

²⁸⁴ See, e.g., Wilkins, *supra* note 131, at 393 (determinants of injury severity for a fall may include the distance fallen, the nature of the surface on to which the child falls, forwards or sideways protective reflexes, whether a fall is in some way “broken,” whether the child propelled himself, the mass of the body and of the head, what proportion of the total kinetic energy is absorbed in compressing the ground and/or deforming the skull, brain or the rest of the body, whether the kinetic energy is dissipated in causing fractures, whether the contact with the ground is focal or diffuse, and the role of secondary brain injury such as hypoxic encephalopathy from an unprotected airway or ischemia from cerebral edema).

²⁸⁵ See Jenny, *supra* note 7, at 19 (there is overwhelming evidence that the response to a given injury in an infant is much worse than that of an adult to a similar injury).

produce serious consequences in an infant, particularly one with predisposing conditions.²⁸⁶ In looking at the absence of a history or a history that substantially changes or evolves, moreover, the clinician must assess the possibility that the parent or caretaker truly does not know what happened to the child and that “changes” in the story may reflect improper interviewing techniques or the efforts of parents and caretakers to search their memories to help the doctors and investigators determine what happened to the child. To examine these factors, clinicians must evaluate the conditions under which the information was obtained, as well as the psychological condition of the caretakers.

Given the consequences of an abuse diagnosis, doctors must be just as careful—and just as knowledgeable—in weighing these considerations as in ordering major surgery or terminating life support, for in each and every case, they hold the future of a family in their hands. If, at the end of the analysis, the answer to whether particular injuries are accidental, natural or abusive is “we don’t know,” that is what needs to be said, and no more.

c. Clinical judgment.

As Dr. Narang points out, it is not possible to conduct prospective randomized controlled studies in SBS/AHT research since it is not possible to violently shake babies for purposes of experimentation. Dr. Narang further points out that other medical diagnoses have not been validated by randomized controlled trials yet are widely accepted and uncontroversial.²⁸⁷ For example, a doctor may listen to a patient describe symptoms that have been

²⁸⁶ See, e.g., Joseph H. Piatt, *A Pitfall in the Diagnosis of Child Abuse: External Hydrocephalus,*

Subdural Hematoma, and Retinal Hemorrhages, 7 NEUROSURGERY FOCUS 4 (1999) (infants with external hydrocephalus may develop retinal and subdural hemorrhages spontaneously or from minor trauma); see also P.D. McNeely et al., *Subdural Hematomas in Infants with Benign Enlargement of the Subarachnoid Spaces Are Not Pathognomonic for Child Abuse*, 27 AM. J. NEURORADIOLOGY 1725 (2006) (subdural hematomas may occur either spontaneously or as result of minor or unrecognized trauma in infants with benign enlargement of the subarachnoid spaces); see also Sirotnak, *supra* note 10, at 203 (“spontaneous or trauma-induced intracranial hemorrhages can occur in various common inherited coagulation disorders and those induced by another disease process or medical therapy”).

²⁸⁷ Narang, *supra* note 3, at 531-32.

described as “migraine” and prescribe migraine treatment.²⁸⁸ If the description of the symptoms accords with that of other migraine patients and the treatment works, the doctor may reasonably diagnose migraine based on clinical experience.

Doctors do not, however, have this type of clinical experience with SBS/AHT. In exercising clinical judgment, doctors generally correlate the patient’s description of the symptoms and their onset (the patient history) with objective medical data (such as lab results) and response to treatment. Unlike a diagnosis of migraine, however, the SBS/AHT diagnosis is typically made in the context of patients who cannot talk, medical findings that lack definitive research, and a legal arena that demands near certainty (proof beyond a reasonable doubt). Since the parents or caretakers typically deny abuse, no one has seen it, and the infant obviously cannot verify it, there is no history to correlate with the findings.²⁸⁹ There is similarly no course of treatment that would confirm or disprove SBS or AHT. Unlike a diagnosis of migraine, a diagnosis of intentional injury cannot be verified by response to a specific treatment or medication. With no history to correlate with the findings and no treatment that would confirm the diagnosis, the SBS/AHT diagnosis lacks the safeguards that gird most clinical diagnoses, including migraine.²⁹⁰

d. Observer bias.

Observer bias refers to the innate cognitive biases that lead us to interpret data in ways that are consistent with what we expect to find.²⁹¹ Considerable research confirms that police investigators,²⁹²

²⁸⁸ *Id.*

²⁸⁹ One of the more unusual aspects of the SBS/AHT diagnosis is that clinicians typically reject the history provided by the caretakers and substitute their own description of the events preceding admission, in effect creating a new patient history that then becomes the lynchpin of the diagnosis.

²⁹⁰ As this suggests, SBS/AHT is not really a medical diagnosis but a legal conclusion. Doctors may reliably diagnose subdural hemorrhage, retinal hemorrhage and encephalopathy from radiology images and eye examinations. However, determining timing, causation and state of mind goes into areas that are more commonly reserved for pathologists, detectives, psychologists and juries.

²⁹¹ See, e.g., D. Michael Risinger et al., *The Daubert/Kumho Implications of Observer Effects in Forensic Science: Hidden Problems of Expectation and Suggestion*, 90 CAL. L. REV. 3 (2002).

scientists,²⁹³ and physicians²⁹⁴ are all subject to cognitive errors that lead us to seek, recall, and interpret data in ways that support our initial judgments or hypotheses, and to disregard or minimize information that is inconsistent.

As reflected in the studies cited by Dr. Narang, cognitive biases are unavoidable when physicians use “clinical judgment” to determine which cases are abuse and which are accidental or natural. In *Hobbs*, for example, the authors acknowledged that “there is no absolute or gold standard by which to define NAHI [nonaccidental head injury]”²⁹⁵ and declined to provide criteria for determining the causation of subdural bleeding.²⁹⁶ Instead, the authors deferred to the opinions of the treating physicians,²⁹⁷ who had been taught for decades that subdural hemorrhages in children were generally caused by abuse. Unsurprisingly, the treating physicians ascribed 57% of subdural hemorrhages and effusions to abuse.²⁹⁸ Even so, 57% is far from an overwhelming majority—far less than the 81% identified by Duhaime and far below the criminal standard for proof beyond a reasonable doubt—making it difficult to apply these “statistics” in any given case.²⁹⁹

Similar disparities arose in a study in which 570 doctors

²⁹² Karl Ask & Pär Anders Granhag, *Motivational Bias in Criminal Investigators’ Judgments of Witness Reliability*, 37 J. APPLIED SOC. PSYCHOLOGY 561 (2007); Karl Ask et al., *The “Elasticity” of Criminal Evidence: A Moderator of Investigator Bias*, 22 APPLIED COGNITIVE PSYCHOLOGY 1245 (2008); Keith A. Findley & Michael S. Scott, *The Multiple Dimensions of Tunnel Vision in Criminal Cases*, 2006 WIS. L. REV. 291 (2006).

²⁹³ Andrea Follmer Greenhoot et al., *Prior Beliefs and Methodological Concepts in Scientific Reasoning*, 18 APPLIED COGNITIVE PSYCHOLOGY 203 (2004); Itiel E. Dror & David Charlton, *Why Experts Make Errors*, 56 J. OF FORENSIC IDENTIFICATION 600 (2006).

²⁹⁴ Thomas S. Wallsten, *Physician and Medical Student Bias in Evaluating Diagnostic Information*, 1 MED. DECISION MAKING 145 (1981); Vicki R. LeBlanc et al., *Believing Is Seeing: The Influence of a Diagnostic Hypothesis on the Interpretation of Clinical Features*, 77 ACADEMIC MED. 567 (Oct. Supplement 2002); Jesse M. Pines, *Profiles in Patient Safety: Confirmation Bias in Emergency Medicine*, 13 ACADEMIC EMERGENCY MED. 90 (2006); Mark L. Graber et al., *Diagnostic Error in Internal Medicine*, 165 ARCHIVES OF INTERNAL MED. 1493 (2005).

²⁹⁵ *Hobbs*, *supra* note 239, at 954.

²⁹⁶ *Id.*

²⁹⁷ *Id.* at 952, 954.

²⁹⁸ *See id.* at 953 (noting findings of abuse in 106 out of 186 total cases examined).

²⁹⁹ *Id.* at 952.

(primarily pathologists and pediatricians) estimated the likelihood of abuse in 16 scenarios involving head injury.³⁰⁰ In this study, the doctors were asked to classify the head injuries as unintentional, inflicted or undetermined.³⁰¹ While no case produced complete agreement, a majority opinion was considered achieved if more than 50% of all survey respondents and more than 50% of experienced respondents³⁰² rated the injury as either unintentional or inflicted.³⁰³ Using these standards, a majority opinion was achieved in only eight of the sixteen scenarios, five of which were classified as inflicted and three of which were classified as unintentional.³⁰⁴ In general, pediatricians were more likely than pathologists to classify cases as inflicted.³⁰⁵ As the authors noted, the inability to achieve consensus in 50% of the cases may be an appropriate recognition of the uncertainties that persist in this challenging arena.³⁰⁶

Finally, observer bias influences the way in which we conduct

³⁰⁰ Antoinette L. Laskey, Michael J. Sheridan & Kent P. Hymel, *Physicians' Initial Forensic Impressions of Hypothetical Cases of Pediatric Traumatic Brain Injury*, 31 CHILD ABUSE & NEGLECT 329 (2007).

³⁰¹ *Id.* at 332. Respondents classified the hypothetical cases into seven categories ranging from definitive unintentional to definitive inflicted, which were then collapsed into the three broad categories of unintentional, inflicted or undetermined by the study authors. ("In an effort to identify case examples of widely acceptable criteria for research definitions of unintentional and inflicted pediatric TBI, the participants' responses were collapsed from seven forensic categories into three, according to the following conservative schema: definitive or probable unintentional TBI were labeled unintentional; possible unintentional, undetermined, or possible inflicted TBI were labeled undetermined; and probable or definitive inflicted TBI were labeled inflicted").

³⁰² The study classified as experienced those physicians who indicated they had devoted 50% or more of their professional time to activities directly related to child abuse for at least [fifteen] 15 years." *Id.* at 332.

³⁰³ *Id.*

³⁰⁴ *Id.* at 335.

³⁰⁵ See *id.* at 337 (noting that pathologists were consistently more likely than pediatricians to classify cases towards the unintentional end of the spectrum).

³⁰⁶ See *id.* at 338. Dr. Karen Kafadar, Chair of the Department of Statistics at Indiana University, has further observed that 16 scenarios is not a large set of scenarios, so the actual agreement rate could be even lower. She notes: "Success' (i.e., at least 50% agreement among the raters) in 8 of the[cases] leads to an estimated success rate of $8/16 = 50\%$, with a 95% confidence interval ranging from $(4/16 - 0.25)$ to $(12/16 - 0.75)$. So, if $8/16 = 50\%$ sounds less than ideal, in fact the 'true' 'success rate' could be as low as 25%, and is rather unlikely to exceed 75%." Email from Dr. Karen Kafadar to Keith Findley, July 20, 2012.

research. To determine whether subdural or retinal hemorrhages are correlated with abuse, it is critical to determine whether and under what conditions these findings occur in children (or adults) who are not abused. Not surprisingly, the major scientific breakthroughs in SBS/AHT research have come through the examination of groups in which abuse is impossible or unlikely. Thus, from Geddes we learned that the swollen brains and thin subdural hemorrhages previously believed to be diagnostic of abuse are also found in infants who died from respiratory tract infection, perinatal asphyxia, gastroenteritis or sudden infant death syndrome (SIDS),³⁰⁷ from Rooks we learned that thin subdural hemorrhages are present in 46% of asymptomatic newborns,³⁰⁸ from Lantz, Matshes and Lopez we learned that retinal hemorrhages are found in many types of deaths,³⁰⁹ and from Holmes-Morton we learned that these findings may be associated with genetic abnormalities.³¹⁰ As this suggests, if we want to determine the full range of causes associated with the triad, we must go outside the child abuse arena and conduct studies that are free from observer bias and that look for the findings associated with abuse in children who collapse or die from natural causes.³¹¹

³⁰⁷ Geddes, *supra* note 70, at 1300.

³⁰⁸ Rooks, *supra* note 109, at 1083.

³⁰⁹ Lantz, *supra* note 135, at 271; Lopez, *supra* note 161, at 98.

³¹⁰ See, e.g., D. Morton Holmes et al., *Glutaric Aciduria Type I: A Common Cause of Episodic Encephalopathy and Spastic Paralysis in the Amish of Lancaster County, Pennsylvania*, 41 AM. J. MED. GENETICS 89 (1991); D. Holmes Morton, *Through My Window—Remarks at the 125th Year Celebration of Children's Hospital of Boston*, 94 PEDIATRICS 785 (1994); D. Holmes Morton et al., *Pediatric Medicine and the Genetic Disorders of the Amish and Mennonite People of Pennsylvania*, 121 AM. J. MED GENETICS Part C 5 (2003).

³¹¹ Since children who are asymptomatic or who are diagnosed with medical conditions do not routinely receive CT scans or eye examinations, we do not know the prevalence or characteristics of retinal and subdural hemorrhages in the general population or in specific medical conditions. We do know, however, that the more we look, the more we find. See, e.g., Lantz, *supra* note 135, at 271; Matshes, *supra* note 207 (finding retinal hemorrhages in natural, accidental and abusive deaths); Lopez, *supra* note 161 (finding severe retinal hemorrhages in *Streptococcus pneumoniae* meningitis); Rooks, *supra* note 109, at 1083 (finding subdural hemorrhages in 46% of asymptomatic newborns); Laura Rooms et al., *Hemophagocytic Lymphohistiocytosis Masquerading as Child Abuse: Presentation of Three Cases and Review of Central Nervous System Findings in Hemophagocytic Lymphohistiocytosis*, 111 PEDIATRICS e636 (2003) (reporting three cases of hemophagocytic lymphohistiocytosis).

e. Reversing the burden of proof.

Through a strange alchemy of legitimate confusion and flawed methodology, the burden of proof is reversed in SBS/AHT cases. The 2001 AAP Technical Report made the burden-shifting presumption explicit, stating that “data regarding the nature and frequency of head trauma consistently support the need for a presumption of child abuse when a child younger than [one] year has suffered an intracranial injury.”³¹² Once this presumption is in place, the burden is on the parents to “prove” an alternative explanation.

In so doing, Dr. Narang states that “[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.”³¹³ This standard raises two concerns. First, it assumes that the medical findings are traumatic and that doctors are able to accurately assess the biomechanical plausibility of the event. Second, in explaining the findings, parents are at a considerable disadvantage since they typically lack medical expertise and do not know what elements of the history might be important. Unlike doctors, moreover, who are encouraged to change their diagnoses as they acquire new information, parents are not permitted to add to the history as they learn more about the findings since this is viewed as a “changing story” and confirmation of abuse. This is especially problematic since the medical personnel and police often insist that the initial history cannot account for the injuries and pressure the caretaker to search his or her memories for additional details or other possible explanations. When the caretaker attempts to comply, however, any new details or possible explanations are viewed as a “changing story” and confirmation of abuse. Often, this is a circle from which there is no escape.

initially misdiagnosed as suspected child abuse).

³¹² Comm. on Child Abuse and Neglect, *supra* note 82, at 206.

³¹³ Narang, *supra* note 3, at 560.

f. Interpretive error: statistical misunderstandings.

Even if the studies cited by Dr. Narang and others did not suffer from circularity and other methodological flaws, they still would not provide a reliable statistical basis for diagnosing SBS/AHT. The statistical errors fall into two categories: misperceiving the significance of the *P-value*, and failing to avoid what is known as the Prosecutor's Fallacy.

(i) *P-value*.

Dr. Narang claims that the studies he cites have tremendous statistical power because they achieve *P-values* of .05 or better.³¹⁴ While that does indeed sound overwhelming, reliance on the *P-value* can be misleading. The *P-value* means that a finding is statistically significant based on the improbability that the conclusion attributed to a specific variable was caused by chance, using the standard threshold criterion of .05 (*i.e.*, the chance of a random rather than significant correlation is only 5%).³¹⁵ The articles cited by Narang conclude that there is only a very small chance that the higher rates of subdural and retinal hemorrhage seen in cases involving abuse (as opposed to accidents or natural causes) are due to chance, indicating that the correlation is real rather than artificial (*i.e.*, produced by chance).³¹⁶ Even if the causes were accurately classified, however, this measure provides no indication of the *strength* of the correlation for it does not distinguish between weak correlations (*e.g.*, subdural and/or retinal hemorrhages are 3% more likely in abuse cases than non-abuse) and strong ones (*e.g.*, such findings are 80% more likely in abuse cases).³¹⁷ Yet the strength of the correlation is precisely what

³¹⁴ *Id.* at 536-37, 544-47.

³¹⁵ *Id.*

³¹⁶ *Id.*

³¹⁷ Dr. Karen Kafadar, Chair of the Department of Statistics at Indiana University, notes, for example, that, given enough data, remarkably small correlations—largely meaningless for any practical purposes—might nonetheless be deemed statistically significant based on their *p-value*. She explains: "An estimate of correlation of 0.07 could be "statistically significantly different from zero" at significance level 0.05 if the estimate of 0.07 were based on 1000 data

is needed to satisfy fact finding requirements in criminal cases, which requires proof beyond a reasonable doubt. Statistical significance is necessary but not sufficient to support this evidentiary standard.

(ii) The Prosecutor's Fallacy.

Dr. Narang's article makes a fundamental logical error that is so common that it has its own name: the Prosecutor's Fallacy.³¹⁸ It is the same mistake as saying: "Because lawyers tend to be literate people, literate people tend to be lawyers."³¹⁹ For example, Dr. Narang cites several studies for the proposition that AHT is more likely to cause subdural hematomas in infants than accidental trauma.³²⁰ Even if these studies accurately assess causation, it would be an improper application of statistics to conclude that an infant who presents with a subdural hematoma is likely to have been abused.

Bayesian statistics teach that to determine the predictive value of an association—in this case, the likelihood that the presence of subdural or retinal hematomas indicates abuse—one must know not only the correlation between subdural hematoma and abuse but also the prior probability, or base rate, of abuse.³²¹ If the base rate of abuse is much smaller than the base rate of non-abuse, even an extraordinarily high correlation between subdural hematomas and abuse would not make abuse more likely than non-abuse when a child presents with a subdural hematoma.³²² Professor James Wood

points. But most people would not get terribly excited about a correlation coefficient of 0.07." Email from Dr. Karen Kafadar to Keith Findley, July 20, 2012.

³¹⁸ See *McDaniel v. Brown*, 130 S. Ct. 665, 670 (2010); William C. Thompson & Edward L. Schumann, *Interpretation of Statistical Evidence in Criminal Trials: The Prosecutor's Fallacy and the Defense Attorney's Fallacy*, 11 LAW & HUM. BEHAV. 167, 170-71, 181-82 (1987); Michael I. Meyerson & William Meyerson, *Significant Statistics: The Unwitting Policy Making of Mathematically Ignorant Judges*, 37 PEPP. L. REV. 771, 778 (2010) (the "'prosecutor's fallacy' ... incorrectly reverses events in a conditional probability to create a direct statement about the defendant's probability of guilt that is not implied by the evidence. In logical reasoning, such an error is called 'transposing the conditional'" (footnotes omitted).

³¹⁹ Meyerson, *supra* note 318, at 778.

³²⁰ See *supra* pages 177-87.

³²¹ For a general overview of Bayesian statistics, see J. ARTHUR WOODWARD ET AL., *INTRODUCTION TO LINEAR MODELS AND EXPERIMENTAL DESIGN* 13-15 (1990).

³²² For a discussion of base rates, see James M. Wood, *Weighing Evidence in Sexual Abuse Evaluations: An Introduction to Bayes's Theorem*, 1 CHILD MALTREATMENT 25 (1996); Michael J.

puts it this way: "Exactly the same evidence may lead to quite different conclusions, depending on the rate of abuse in the group being evaluated."³²³

A simple illustration makes this point. Suppose that an airport machine that checks for explosives hidden in checked bags is 99% accurate in detecting explosives; that is, it has a one percent false positive and a one percent false negative rate. This means that the machine will sound an alarm 99 times if 100 bags with explosives are fed through the machine, and will sound an alarm only once if 100 bags without explosives are fed through the machine. In other words, bags containing explosives are 99 times as likely to make the alarm sound as bags not containing explosives. If the alarm sounds, how likely is it that the bag contains explosives? Probably not very likely at all. If one million bags are checked by machine, one of which contains explosives (a number that is almost certainly too high), there would be approximately 10,000 *false* alarms for every *true* alarm. By the same token, if the number of children with subdural hematomas from accidental or natural causes is significantly greater than the number with subdural hematomas from abuse, then Dr. Narang is wrong to assume from the studies he cites that subdural hematomas most likely indicate abuse.

The studies in Dr. Narang's article illustrate this point. In these studies, the correlation of subdural hematoma to abuse is very high but the base rate of abuse compared to non-abuse—to the extent it is revealed in the studies—is sometimes relatively modest, suggesting that subdural hematomas are at best only weakly diagnostic of abuse. Bechtel et al., for example, studied 82 children admitted for head trauma and concluded that 15 (18%) of the injuries were inflicted and 67 (82%) were "accidental."³²⁴ Bechtel then reported that 80% (12/15) of the "inflicted" group had subdural hematomas while only 27% (18/67) in the "accidental" group had subdural hematomas.³²⁵ From this, Dr. Narang concludes that, with a *P-value* of .001, "the

Saks & D. Michael Risinger, *Base rates, the Presumption of Guilt, Admissibility Rulings, and Erroneous Convictions*, 2003 MICH. ST. L. REV. 1051 (2003).

³²³ Wood, *supra* note 322, at 26.

³²⁴ Bechtel, *supra* note 210, at 165.

³²⁵ *Id.* at 167.

association of SDH's with inflicted injury was highly statistically significant."³²⁶ But that is only part of the story. When one factors in the low base rate of abuse, the conclusion is quite different. To compute the posterior probability of abuse, which more accurately reflects the diagnostic significance of subdural hematoma, one has to multiply the base rate by the likelihood ratio, which represents "the relative probability of coming across a particular piece of evidence in one group rather than in another."³²⁷ Here, since 80% of purported inflicted cases have subdural hematomas and 27% of accidental cases have subdural hematomas, the likelihood ratio is 80:27, or 2.96:1. But because the base rate of abuse is only 18%, the true likelihood of abuse given subdural hematoma is only 35%.³²⁸ One can make the same calculation in a different manner: since 18 of the subdural hematomas identified by Bechtel were accidental and 12 were inflicted, subdural hematomas were 50% more common in accident cases than in abuse cases. Either way, subdural hematoma is not diagnostic of abuse since most cases with this finding are non-abusive.³²⁹

A similar analysis applies to other studies. In the Matschke study, for example, the authors looked at 715 infant deaths, finding subdural hematomas in 50 of them.³³⁰ Unlike the Bechtel study, the Matschke study attempted to identify all causes of the subdural hematomas, not just those attributed to trauma. Of the 50 cases with subdural hemorrhage, 15 (30%) were identified as traumatic and 35

³²⁶ Narang, *supra* note 3, at 545.

³²⁷ Wood, *supra* note 322, at 26.

³²⁸ The formula for computing the probability of abuse, also known as the posterior odds, using Bayes's theorem, is: Prior Odds (here, the base rate) x the Likelihood Ratio = Posterior Odds. See Wood, *supra* note 322, at 29. With prior odds (the base rate) of abuse of 1:5.56 (18%), and a likelihood ratio of 2.96:1, the posterior odds are: $1/1.56 \times 2.96/1 = 2.96/1.56$. That computes to a probability of abuse of about 35%, because converting odds into probability is accomplished by adding the numerator and the denominator of the odds together (2.96 plus 1.56 = 8.52) and dividing the numerator (2.96) by that total: $2.96/8.52 = .35$ (35%). See Wood, *supra* note 322, at 28-29.

³²⁹ The Bechtel study had only two classifications: inflicted or accidental. If some of the abuse cases were natural in origin, the base rate of inflicted abuse would have been even smaller.

³³⁰ Matschke, *supra* note 207, at 1587.

(70%) were attributed to other causes.³³¹ Of the 35 cases that were not identified as traumatic, the subdural hemorrhages were attributed to bleeding/clotting disorders, perinatal events, infections, metabolic diseases, or (in 8% of the cases) undetermined causes.³³² A simple counting reveals that the study does not support the conclusion of its authors, which Dr. Narang quotes for the proposition that “most SDH’s are attributable to trauma.”³³³ To the contrary, the data show that most SDH’s are attributable to non-traumatic events, by a ratio of 70% to 30%.³³⁴ As this suggests, while Dr. Narang is undoubtedly correct that some children who have been abused will have subdural hemorrhages, he commits the Prosecutor’s Fallacy when he claims that children who have subdural hemorrhages are likely to have been abused. Instead, this is just one of many possible causes.

(iii) Improper classifications.

These statistical misunderstandings assume even greater importance when superimposed on statistics that likely misclassify a significant number of medical findings as abusive. At present, we have no reliable statistics on the incidence of abusive head injuries. Instead, what we have are estimates of what the incidence would be if various hypotheses prove to be correct. Without some method of properly and accurately classifying the medical findings previously associated with shaking, there is no valid statistical basis for estimating the incidence of abusive head trauma in general, let alone the likelihood that abusive head trauma has occurred in specific cases.

³³¹ *Id.* at 1587.

³³² *Id.* at 1589.

³³³ Narang, *supra* note 3, at 542 (citing Matschke, *supra* note 207, at 1594).

³³⁴ The Matschke study goes on claim that over 90% of the trauma cases were attributable to abuse. Matschke, *supra* note 207, at 1593. However, the study uses criteria that likely lead to an overestimation of the rate of abuse. *See* note 161, Matschke *supra* 207, at 1588, and related text. In any event, the study’s conclusion that abuse is the most common cause of subdural bleeding in infants depends on dividing the natural causes into separate categories. If combined, they constitute 36% of cases, a greater proportion than that of alleged abuse.

B. The Skeptics: New Research, Old Anatomy

Two types of study cast doubt on the old SBS hypothesis: (1) studies that point out the lack of support for the traditional hypothesis, and (2) studies that identify specific problems with the hypothesis and/or suggest alternative causes. Dr. Narang dismisses both types of studies, suggesting that they were improperly conducted or are unsupported by the evidence.

1. *Studies that identify the lack of support for the traditional SBS hypothesis.*

Dr. Narang focuses on Dr. Donohoe's 2003 study, "Evidence-Based Medicine and Shaken Baby Syndrome Part 1: Literature Review, 1966-1998,"³³⁵ which he dismisses as poor scholarship.³³⁶ Specifically, he claims that Dr. Donohoe failed to capture the breadth of SBS/AHT medical research by using only the search term "shaken baby syndrome" in the Medline database and internet search.³³⁷ Since, however, Dr. Donohoe was examining the evidence base for SBS, not for all types of traumatic brain injury, it was appropriate to search for articles using the phrase "shaken baby syndrome."³³⁸ It was not until after Dr. Donohoe's analysis—and may have been partly as a result of his analysis—that the medical community began moving away from shaking as a mechanism and adopting more expansive terminology. Dr. Narang does not identify any research on shaking that Dr. Donohoe (or for that matter the participants in the 2002 NIH conference) missed. Without identifying the missing

³³⁵ Donohoe, *supra* note 100.

³³⁶ Narang, *supra* note 3, at 534.

³³⁷ *Id.* Dr. Narang contends that Dr. Donohoe should have searched for 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." *Id.* at 533-534. Such expanded searches would have dramatically altered Dr. Donohoe's inquiry, broadening its scope far beyond his objective of identifying the research basis for shaken baby syndrome.

³³⁸ Dr. Donohoe examined SBS research through 1998, a period in which SBS was an increasingly popular foundation for criminal convictions. As Dr. Donohoe observed, 1998/1999 is also regarded as "the turning point in acceptance of the tenets and practice of EBM [evidence based medicine]." Donohoe, *supra* note 100, at 239.

literature, Dr. Narang's criticism appears to be semantic rather than substantive.

Dr. Narang further criticizes Dr. Donohoe's observation that none of the SBS research achieved the "best evidence" standards of "Level 1," which includes randomized controlled trials.³³⁹ We all agree that such studies are not possible since one cannot violently shake a child—let alone a large sample of children—to see what happens. Dr. Narang thus notes that "even the most ardent [evidence based medicine] advocate would admit that the best quality of evidence that can be expected in diagnostic studies is 'Level 2.'"³⁴⁰ While Dr. Narang is correct that Level 1 evidence cannot be achieved in SBS research, this does not mean that Dr. Donohoe was incorrect to note that none of the SBS literature achieved Level 1 status and that none exceeded Level 3.³⁴¹ Instead, the lack of high quality evidence requires that clinicians and researchers exercise considerable caution in endorsing particular diagnoses or hypotheses, particularly when the adverse consequences are high. Rather than urging greater caution, however, Dr. Narang urges the courts to substitute the clinical judgment of pediatricians and others, which is by nature subjective, for the objective medical evidence envisioned by evidence-based medicine and *Daubert*. This suggestion would lower the level of proof in child abuse cases and almost certainly result in mistaken diagnoses and false convictions—the very problems that evidence-based medicine and *Daubert* were attempting to address.

2. Studies that identify problems with the SBS/AHT hypothesis.

Dr. Narang also criticizes studies that identify errors in the SBS literature, including the neuropathological studies conducted by Dr. Geddes and the more recent work on infant anatomy by Dr. Squier (a pediatric neuropathologist and a co-author), Dr. Mack (a pediatric radiologist) and Dr. Eastman (a clinical pathologist), claiming that this work is unsupported by the evidence. However, this research is

³³⁹ Narang, *supra* note 3, at 535.

³⁴⁰ *Id.*

³⁴¹ Donohoe, *supra* note 100, at 241 (by the end of 1998, no evidence on the subject of SBS exceeded QER III-2).

extensively referenced to the medical literature. Once again, Dr. Narang does not identify any errors in the articles or the supporting literature.

In criticizing the work of Dr. Geddes, Dr. Narang selects his targets curiously. Dr. Narang does not discuss, or even mention, the groundbreaking research of Dr. Geddes and her colleagues in which they found that the brain swelling in alleged SBS/AHT cases was in most cases hypoxic-ischemic rather than traumatic, and that the subdural hemorrhages were typically thin, bilateral, and quite different in appearance from the traumatic hemorrhages found in older children and adults.³⁴² These observations, which are now generally accepted, called into question the traumatic origins of two of the three components of the SBS triad. Instead, Dr. Narang attacks Geddes III,³⁴³ in which Dr. Geddes and her co-authors suggested a “Unified Hypothesis” to explain the mechanism of subdural hemorrhage and brain damage in allegedly abused infants. In Geddes III, the authors examined fifty non-traumatic infant deaths from infection, hypoxia and sudden infant death syndrome as well as three “shaken baby” deaths. Since all of the SBS deaths and most of the natural deaths showed intradural rather than subdural bleeding, the paper suggested the mechanism might be vascular leakage from veins within the dura rather than the traumatic rupture of bridging veins. The paper further suggested that the intradural bleeding might result from a cascade of events combined with immaturity and hypoxia-induced vascular fragility.³⁴⁴ Contrary to Dr. Narang’s

³⁴² Geddes, *supra* note 70, at 1304 (observing that “axonal damage occurs in the brains of both head-injured subjects and in controls in much the same distribution...this is not ‘DAI’ [diffuse axonal injury]; but diffuse vascular or hypoxic-ischaemic injury, attributable to brain swelling and raised intracranial pressure”); Geddes, *supra* note 52, at 1297 (subdural hemorrhages found in cases of alleged non-accidental trauma are “materially different from those seen in adults, and are rarely ‘massive’...They are almost invariably bilateral thin films of blood over the cerebral hemispheres, which do not require neurosurgical intervention”).

³⁴³ Geddes, *supra* note 70.

³⁴⁴ *Id.* at 19 (“our observations in the present series indicate that, in the immature brain, hypoxia both alone and in combination with infection is sufficient to activate the pathophysiological cascade which culminates in altered vascular permeability and extravasation of blood within and under the dura. In the presence of brain swelling and raised intracranial pressure, vascular fragility and bleeding would be exacerbated by additional haemodynamic forces, such as venous hypertension, and the effects of both

assertion, Dr. Geddes did not recant this suggestion in her courtroom testimony but simply made clear that it was a hypothesis, akin to the SBS hypothesis, albeit more closely aligned with the anatomy of the infant brain.³⁴⁵

Like the Geddes studies, Squier and Mack's description of the "immature vascular plexus" is firmly rooted in anatomical research.³⁴⁶ Indeed, this is an observational study of the kind described by Dr. Narang as "not just the norm but the cornerstone of medical diagnoses."³⁴⁷ As Professor Goldsmith pointed out in 2001 and Dr. Reece pointed out in 2002, research on the physiology and pathophysiology of the central nervous system is essential to understanding the issues associated with SBS/AHT.³⁴⁸ While Dr. Narang suggests that the existence of a highly vascularized immature dural plexus is simply a hypothesis, this description of the anatomy is based on microscopic examinations and resin casts, which are illustrated in the Squier and Mack articles.³⁴⁹ Their descriptions are further confirmed by decades of anatomical research on the dura.³⁵⁰

sustained systemic arterial hypertension and episodic surges in blood pressure").

³⁴⁵ In her testimony, Dr. Geddes stated that "[the 'unified hypothesis'] is not fact; it is hypothesis but, as I have already said, so is the traditional explanation.... [W]e do use the word "hypothesis" throughout [the paper]." *R v Lorraine Harris, Raymond Charles Rock, Alan Barry Joseph Cherry, Michael Ian Faulder*, 1 Cr App R 5, [2005] EWCA Crim 1980, Case Nos: 200403277, 200406902, 200405573, 200302848, at <http://www.bailii.org/ew/cases/EWCA/Crim/2005/1980.html>.

³⁴⁶ Waney Squier & Julie Mack, *The Neuropathology of Infant Subdural Haemorrhage*, 187 FORENSIC SCI. INT. 6 (2009); Julie Mack, Waney Squier & James T. Eastman, *Anatomy and Development of the Meninges: Implications for Subdural Collections and CSF Circulation*, 39 PEDIATR RADIOL. 200 (2009).

³⁴⁷ Narang, *supra* note 3, at 531-532.

³⁴⁸ Goldsmith, *supra* note 73 ("Intimate collaboration is urged between biological specialists, medical professionals and biomechanicians to investigate crucial unsolved problems related to head injury, such as the rate of blood absorption from broken vessels by the body as a function of age, and the rate of effusion from ruptured vessels"); *Inflicted Childhood Neurotrauma*, *supra* note 84, at VIII ("[T]he contributions of basic scientists doing bench research related to the physiology and pathophysiology of the central nervous system are welcome and essential to the generation of understanding about these phenomena").

³⁴⁹ Squier, *supra* note 346, at 8; Mack, *supra* note 346, at 203-205.

³⁵⁰ See, e.g., Erna Christensen, *Studies on Chronic Subdural Hematoma*, 19 ACTA PSYCHIATRICA ET NEUROLOGICA 69, 74 (1944) ("[t]he outermost fibrillary layer of the dura contains arteries as well as veins; the arteries are running in looping streaks, accompanied by two veins which open into the superior sagittal sinus. The arteries as well as the veins form anastomoses, the

Squier and Mack further pointed out the thin “subdural” bleeds traditionally associated with SBS/AHT in infants are unlikely to be caused by bridging vein rupture since the quantity of blood is too small given the volume of blood carried within these veins.³⁵¹ They also noted that there is no “subdural space”, as hypothesized in traditional SBS theory; instead, the arachnoid and the dura are contiguous. Based on the anatomy, Squier and Mack observed that the blood-rich network of vessels in the inner layer of the immature dura may be the source of thin film bleeds found in infants, which are quite distinct from the thick, space-occupying subdural hemorrhages found in older children and adults. Dr. Narang does not identify any errors in these descriptions of the anatomy, which have been presented without objection at conferences on both sides of the debate.³⁵² These observations have, moreover, been widely accepted

vessels branching dichotomically. Fine capillaries and arteries run obliquely through the dural tissue to the inner side where a nicely arranged, long-meshed capillary net is found, the junctions of which form ampullary blood-filled dilatations; and these ampullary dilatations constitute the connecting link between the capillary and venous systems. On the outer aspect a more wide-meshed capillary network is seen; and at the transition between the two capillary layers a few tiny vessels are seen”); J.A. Hannah, *The Aetiology of Subdural Hematoma: An Anatomical and Pathological Study*, 84 J. NERV. MENT. DIS. 169, 171 (1936) (“[c]ontrary to the usual conception, that the dura is a comparatively avascular structure, its blood supply is richer and much more complicated than would appear necessary to supply a structure, the functions of which are merely to support the brain and to act as an endosteum to the skull bones); C. W. Kerber & T.H. Newton, *The Macro and Microvasculature of the Dura Mater*, 6 NEURORADIO. 175, 179 (1973) (the dura contains “a vascular network which is complex and far in excess of the expected metabolic needs of a membrane furnishing only mechanical support); Hui Han et al., *The Dural Entrance of Cerebral Bridging Veins into the Superior Sagittal Sinus: an Anatomical Comparison between Cadavers and Digital Subtraction Angiography*, 49 NEURORADIO. 169, 169 (2007).

³⁵¹ Squier, *supra* note 346, at 7-8 (rupture of the large caliber veins carrying large volumes of blood from the brain to the dural sinuses would be unlikely to produce the thin film haemorrhages characteristic of the young infant). The infant brain receives a large proportion of the cardiac output, creating substantial regional blood flow (averaging 40 ml/100 g per minute in a 6-month-old). The parasagittal bridging veins, which are strong and few in number, are responsible for draining a large proportion of the blood that flows through the supratentorial cortex. *Bilateral* subdural hemorrhages would require the rupture of multiple bridging veins, all of which would bleed at a relatively rapid rate. Since the bilateral thin film subdural hemorrhages in infants are typically small, sometimes no more than 5 cc, bridging vein rupture is an implausible explanation for these hemorrhages. See also Max Wintermark et al., *Brain Perfusion in Children: Evolution with Age Assessed by Quantitative Perfusion Computed Tomography*, 113 PEDIATRICS 1624 (2004).

³⁵² See Julie Mack, *Alternatives to Bridging Vein Rupture: Embryology and Function of the Infant*

even by the strongest supporters of the SBS/AHT hypothesis.³⁵³

3. *A shifting paradigm.*

Broadly speaking, the research dynamic between supporters and skeptics of the SBS/AHT hypothesis can be characterized as follows—supporters publish great quantities of research, in which selection criteria and clinical judgment based on the SBS/AHT hypothesis are used to differentiate abuse from accidents and natural causes. By failing to consider the wide range of known alternative causes or the unique pathophysiology of the infant brain, the studies almost certainly overestimate the incidence of abuse. Dr. Narang aggregates this data and presents it as persuasive statistical evidence that subdural and retinal hemorrhages are reliable indicators of abuse. In making these claims, Dr. Narang also fails to consider the base rates of abuse and non-abuse when making statistical claims about the diagnostic power of subdural and retinal hemorrhages. Nonetheless, irrespective of its evidentiary basis and statistical validity, the sheer volume of this research serves to intimidate those who are not familiar with its methodological shortcomings.

At the same time, researchers and clinicians who question the SBS/AHT hypothesis or suggest alternatives based on biomechanical studies or the anatomy of the infant brain routinely confront personal and professional attacks on their motivation, competence and integrity.³⁵⁴ These attacks have slowed the research and deterred

Dura, Presentation, EBMS Symposium (February 21, 2009) (brochure on file with authors); Waney Squier, Presentation, *The Pathology of Infant Subdural Hemorrhage and Brain Swelling*, EBMS Symposium (February 22, 2009) (brochure on file with authors); Julie Mack, Keynote Presentation, *The Dural Venous Plexus: Implications of Subdural Collections*, Second International Conference on Pediatric Abusive Head Trauma (June 26, 2009) brochure at <http://www.childdeathreview.org/Reports/2009PedAHTConference.pdf>.

³⁵³ See, e.g., Thomas L. Slovis and Stephen Chapman, *The pathophysiology does not denote the mechanism*, *Editorial*, 39 PEDIATR RADIOL. 197-198 (2009) (“At the end of the day, the article of Mack et al. makes us revisit the pathophysiology of subdural collections and subdural hematomas based on anatomy”); Thomas L. Slovis et al., *The creation of non-disease: an assault on the diagnosis of child abuse*, 42 PEDIATR RADIOL. 903-905 (2012) (referencing workshop on areas in which new data has changed our understanding, e.g., subdural hematoma can occur from bleeding dural veins and not only bridging veins, citing Mack et al *supra* note 346).

³⁵⁴ These attacks appear to be largely coordinated by the NCSBS. See, e.g., Holmgren, *supra*

others from addressing these important issues.³⁵⁵ What Dr. Narang and other supporters of the SBS/AHT hypothesis fail to mention, however, is that despite these vociferous attacks, most of the work they have attacked in the past has been absorbed into the mainstream, slowly but certainly shifting the paradigm. As this suggests, the recent changes in terminology are not semantic but instead reflect the slow process of discarding previous “truths” about SBS.

At present, the new paradigm includes general agreement on the following points:

- Subdural hemorrhages in infants are not caused exclusively or almost exclusively by shaking or inflicted trauma.
- The dura is far more complex than previously understood, with some hemorrhages previously identified as subdural arising within the dura.
- Thin subdural hemorrhages are found in nearly half of asymptomatic newborns, confirming that they are not always symptomatic and can occur without brain damage.
- Rebleeds of chronic subdural hematomas can and do occur.
- Retinal hemorrhages are not caused exclusively or almost exclusively by shaking or other forms of trauma.
- Retinal folds and retinoschisis are not diagnostic of abuse.

note 41 (Pinocchio slides and sing-along); Colin Welsh, Presentation, *A National Co-ordinated Approach to Cases of Non-Accidental Head Injury in the UK*, 11th International Conference on Shaken Baby Syndrome, sponsored by the National Center on Shaken Baby Syndrome (Sept. 2010) (describing efforts of New Scotland Yard and child abuse prosecutors to silence experts who question the diagnosis) (notes on file with authors); Brian K. Holmgren, *Irresponsible Expert Testimony*, NCSBS website at http://dontshake.org/sbs.php?topNavID=3&subNavID=28&subnav_1=96&navID=115.

³⁵⁵ In a recent discussion of an SBS case on the Fifth Estate, a Canadian investigative program, a defense attorney said that he had talked to 50-60 experts who questioned SBS theory, but that only two were willing to testify for fear of being blackballed. Television Program, *Diagnosis Murder, THE FIFTH ESTATE* (January 13, 2012) available at <http://www.cbc.ca/fifth/2011-2012/diagnosismurder/>.

- The brain swelling in alleged SBS/AHT cases is hypoxic-ischemic rather than traumatic.
- Impact, even on a padded surface, generates more force than shaking.
- Short falls can present with the triad and result in death.
- Lucid intervals can occur in trauma cases.
- The concept of a lucid interval does not apply when the triad arises from natural causes.
- There is a long list of alternative causes for the triad, ranging from birth trauma to genetic abnormalities, infection and childhood stroke.

As the new paradigm emerges, new cases must be evaluated—and old cases re-evaluated—with the same commitment to meticulous diagnosis found in any other complex area of medicine. Our understanding of the medicine and the biomechanics of injury must be combined with a recognition that many fundamental questions remain unanswered. In the meantime, we must strive to make the best possible decisions under conditions of uncertainty—conditions that require us to balance the unthinkable harm of child abuse against the equally unthinkable harm of destroying families and imprisoning innocent parents and caretakers based on a flawed hypothesis.

To this end, in 2011 two of our co-authors—Dr. Barnes and Dr. Squier—published invited reviews of the literature in their own areas of expertise, pediatric neuroradiology and pediatric neuropathology. These reviews describe our current state of knowledge on the medical findings previously attributed to shaking as well as the ever-expanding list of alternative diagnoses.³⁵⁶

IV. MEDICAL AND LEGAL STANDARDS OF RELIABILITY

While we now have a better understanding of potential causes for subdural hemorrhage, retinal hemorrhage and encephalopathy, the issue has become: how much of this evidence is sufficiently

³⁵⁶ Barnes, *supra* note 12; Squier, *supra* note 12.

reliable for medical diagnosis and courtroom testimony?

A. Medical Diagnosis: Art or Science?

As Dr. Narang recognizes, there has been a shift in medicine towards the objective examination of the quality of the evidence supporting established theories. The movement known as evidence based medicine represents an effort to examine the reliability of the evidence on which doctors make diagnoses and order treatment.³⁵⁷

Under the standards of evidence-based medicine, clinicians formulate questions, conduct literature searches to identify the best available evidence, and critically assess the reliability of that evidence.³⁵⁸ In so doing, clinicians need to distinguish high from low quality primary studies, identify knowledge gaps and frame questions to fill those gaps, and apply the research evidence to the particular patient.³⁵⁹ Evidence-based medicine guidelines assist in this process by providing a hierarchy of evidence, ranging from randomized controlled trials to unsystematic clinical observations.³⁶⁰

While randomized controlled trials of child abuse are not possible, a review of the literature indicates that the problem goes much deeper: the real problem is that the literature cited in support of the SBS/AHT hypothesis falls at the bottom of the hierarchy of evidence and rests almost entirely on assumptions and hypotheses, combined with emotionally compelling demonstrations and

³⁵⁷ See, e.g., David L. Sackett et al., *Evidence Based Medicine: What It Is and What It Isn't*, 312 BRIT. MED. J. 71, 71 (1996) (“[e]vidence based medicine is the conscientious, explicit, and judicious use of current best evidence in making decisions about the care of individual patients”); Frank Davidoff et al., *Evidence Based Medicine*, 310 BRIT. MED. J. 1085, 1085 (1995) (“clinical decisions [in evidence based medicine] should be based on the best available scientific evidence...and the clinical problem—rather than habits or protocols—should determine the type of evidence to be sought”).

³⁵⁸ *Id.*; see also Robert C. Hawkins, *The Evidence Based Medicine Approach to Diagnostic Testing: Practicalities and Limitations*, 26 CLIN. BIOCHEM. REV. 7 (2005); Guyatt, *supra* note 1, at 1290-1296.

³⁵⁹ See Guyatt, *supra* note 1, at 1290, 1293 (clinicians should seek evidence from as high in the appropriate hierarchy of evidence as possible and apply it to the particular circumstances of the patient); Hawkins, *supra* note 358, at 8 (clinicians must determine whether the research used independent reference standards and was applied to a population of patients comparable to the patient in question).

³⁶⁰ Guyatt, *supra* note 1, at 1292; see also Phillips, *supra* note 92.

anecdotal evidence, largely in the form of confessions. Recent research has made clear that many of the underlying assumptions are inconsistent with the anatomy and physiology of the infant brain.

To address the lack of an objective evidence base for the SBS/AHT hypothesis, Dr. Narang recommends that the clinical judgment of child abuse pediatricians be substituted for evidence-based medicine. This proposal circles back, however, to the original problem: even the most popular clinical judgments can be wrong, as evidenced by a long list of misguided clinical judgments, ranging from lobotomies to ulcers to hormone replacement therapy.³⁶¹ Organizational acceptance of clinical judgments is not, moreover, persuasive. As Daniel Kahneman, the Nobel Prize winning Professor of Psychology and Public Affairs at Princeton University, points out, this problem is not unique to medicine: history has shown that “people can maintain an unshakeable faith in any proposition, however absurd, when they are sustained by a community of like-minded individuals.”³⁶² In this case, the reluctance to apply the standards of evidence-based medicine to SBS/AHT has been exacerbated by the efforts of advocacy groups dedicated to the promulgation of the SBS/AHT hypothesis and the criminal prosecution of SBS/AHT cases.³⁶³ While we support their commitment to the prevention of child abuse, this commitment should not substitute subjective beliefs for objective scientific evidence. Instead, the commitment must be to getting it right.

Given the current state of knowledge, what is it reasonable for medical personnel to suggest? Is this simply one of the areas in which “the evidence is so sparse, that EBM simply cannot be instructive either for Medicine or Law”?³⁶⁴ The answer to this question depends on the facts of the case and the proposed solutions.

³⁶¹ See, e.g., Guyatt, *supra* note 1, at 1293 (hormone replacement therapy does not help prevent coronary artery disease despite several observational studies that had shown “dramatically positive results”).

³⁶² DANIEL KAHNEMAN, THINKING, FAST AND SLOW 217 (2011).

³⁶³ Of these, the most prominent is the NCSBS, which since the 1990s has taken a lead role in training prosecutors, doctors and social workers. Active participants in the NCSBS have been involved in the NAME and AAP policy statements and the more recent certification of child abuse pediatricians.

³⁶⁴ Narang, *supra* note 3, at 521-522.

SBS/AHT cases range from cases with obvious head trauma (facial bruising, skull fracture and/or soft tissue swelling) to cases in which seemingly healthy children have suddenly and inexplicably collapsed. Sometimes the history and a meticulous review of the medical records provide a likely answer; other times, it is not possible to determine causation based solely on the medical evidence.

In the face of such uncertainty, we must look closely at the costs and benefits of the proposed solutions. The answers are simplest when we are dealing with prevention. Because violent shaking is dangerous and has no known benefits, there are few costs and many potential benefits associated with educating parents that they should never shake a child. Because short falls can be fatal, parents should also be warned that children should not be placed on counters or couches, or in other places from which they might fall or where other children or adults might fall on them.

Similar principles apply to treatment. Because the body cannot always distinguish between trauma and illness, we need to constantly examine and re-examine our treatment protocols to ensure that we are providing the best possible care to children who present with the triad or one of its components. If the head findings are primary, we need to be able to quickly and accurately distinguish between the various possibilities (e.g., injury, infection or stroke) so that we can provide appropriate treatment. If the head findings are secondary, we need to promptly identify and treat the underlying illness or condition if the child is to survive.

The burden shifts when the solution is to destroy families and imprison parents. Based on what we now know, it is inappropriate for medical professionals to diagnose shaking or abusive head trauma based solely or primarily on the presence of subdural hemorrhage, retinal hemorrhage and/or encephalopathy. When a child abuse referral or diagnosis is made based on these findings, it should be clearly disclosed that there are many possible causes for these findings; that the issues are complex and poorly understood; and that an SBS/AHT diagnosis based exclusively or primarily on these findings rests on good-faith beliefs and hypotheses, rather than science.

B. Daubert: Is SBS/AHT Ready for the Courtroom?

As Dr. Narang states, in determining reliability for admissibility purposes under *Daubert*, courts may consider: (1) whether a theory or technique can be (and has been) tested (also known as falsifiability or testability); (2) whether the theory or technique has been subject to peer review and publication; (3) whether there is a known or potential error rate; and (4) whether there is general acceptance in the relevant scientific community.³⁶⁵ In addition, the courts must consider whether the theory is “sufficiently tied to the facts of the case.”³⁶⁶

Dr. Narang does not argue that the medical literature on SBS/AHT meets the technical standards of *Daubert* (particularly factors 1 and 3) but argues that the courts should instead accept the “clinical judgment” of doctors, particularly child abuse pediatricians, that abuse has occurred. According to Dr. Narang, this interpretation is supported by *Kumho Tire v. Carmichael*,³⁶⁷ which according to Dr. Narang “tethered” the admissibility standard of expert testimony to the standards of medical practice, including the SBS/AHT studies on which he relies. This analysis is, however, incomplete.

To begin, *Daubert* governs only the general *admissibility* of scientific or expert testimony about the causes of injury or death in SBS/AHT cases. Increasingly, the legal issues do not focus on admissibility but focus instead on the case-specific significance of the evidence once it is admitted. These issues include whether medical opinions based on disputed medical issues are legally or factually sufficient to support convictions under the “beyond a reasonable doubt” standard and whether previously obtained convictions should be re-examined given the new scientific understanding of the limitations of the triad as a diagnostic tool and the very real possibility of alternative explanations for a child’s injuries or death.³⁶⁸ As a legal matter, in *Cavazos v. Smith*, six of the nine Supreme Court justices acknowledged flaws in the evidence but held that the

³⁶⁵ *Daubert*, *supra* note 2.

³⁶⁶ *Id.*

³⁶⁷ *Kumho Tire Co. v. Carmichael*, 526 U.S. 137 (1999).

³⁶⁸ *See Tuerkheimer*, *supra* note 51.

disputed SBS science presented at trial met the minimal due process standards for sufficiency of the evidence, at least as of the trial date.³⁶⁹ Today, given the many challenges to the old SBS theory, the factual sufficiency of the evidence has become an increasingly significant question, as has the question of how to handle old convictions—a question not addressed by the majority in *Smith* beyond the narrow holding that the old expert opinions constituted sufficient evidence to convict as of the trial date and the suggestion that Ms. Smith seek clemency, which has since been granted. Given the changes in the science, old SBS/AHT convictions are now being challenged based on newly discovered evidence, actual innocence, ineffective assistance of counsel and other similar claims.³⁷⁰

In arguing admissibility under *Daubert*, moreover, it is unclear what Dr. Narang believes should be admitted. Evidence that some brain injuries in children are of traumatic origin, sometimes even intentionally inflicted? Evidence that subdural hematomas and retinal hemorrhages are seen in cases of inflicted abuse? Evidence that shaking can cause the triad and can lead to injury or death? Evidence that subdural hematomas and retinal hemorrhages are diagnostic of shaking or abuse in the absence of a major motor vehicle accident, fall from a multistory building or other proven alternative? Some of these questions are not controversial, and the evidence clearly satisfies the *Daubert* standard. Others are

³⁶⁹ *Smith* did not address the quality of the science, and admissibility was not an issue. Instead, the Court merely purported to apply, in a very straightforward manner, the deferential and forgiving constitutional standard for assessing sufficiency of the evidence under *Jackson v. Virginia*. *Cavazos v. Smith*, *supra* note 119, at 6. Under that standard, evidence will be deemed sufficient if, taking the evidence in the light most favorable to the prosecution, a reasonable jury could have found guilt beyond a reasonable doubt. Because the State offered experts who opined that the child died of SBS, the Court held that the jury could have found guilt if it credited those expert opinions, which the jury was free to do. The three dissenters—Justices Ginsburg, Sotomayor and Breyer—disagreed, suggesting that the changes in the literature and the fact-intensive character of the case called for a full briefing and consideration of the issues. *Cavazos v. Smith*, dissent, *supra* note 119, at 8, 9.

³⁷⁰ *State v. Edmunds*, 746 N.W. 2d 590, 596 ¶ 15 (2008) (granting a new trial based on newly discovered evidence because “a significant and legitimate debate in the medical community has developed in the past ten years over whether infants can be fatally injured through shaking alone, whether an infant may suffer head trauma and yet experience a significant lucid interval prior to death, and whether other causes may mimic the symptoms traditionally viewed as indicating shaken baby or shaken impact syndrome”); *State v. Louis*, 332 Wis.2d 803 (Wis. Ct. App. 2011) (unpublished disposition).

undermined by the research.

Dr. Narang's analysis of admissibility under *Daubert* further attempts to assess admissibility without limiting the evidence to be introduced or the purpose for which it is proffered. Under *Daubert*, however, any determination of admissibility must include an assessment of the significance of the evidence as it applies "to the task at hand."³⁷¹ As Professor Michael Risinger explains, under *Daubert* and *Kumho*, "reliability cannot be judged globally, 'as drafted,' but only specifically, 'as applied.' *The emphasis on the judgment of reliability as it applies to the individual case, to the 'task at hand,' runs through the opinion like a river.*"³⁷² Because Dr. Narang's global analysis does not identify the specific propositions he wishes introduced or their application to the "task at hand," it tells us little about the admissibility of particular evidence in particular cases.

In determining these issues, clinical judgment cannot trump scientific research. To the contrary, under *Daubert*, the role of judgment or experience is limited:

When a witness is called to . . . make conclusions or inferences about adjudicative facts in the case at hand, the testimony is based in part on experience, but in part on some translation scheme to mediate between previous experiences and a particular conclusion in this case. In those circumstances, reliability is dependent on both sufficient experience and a reliable translation system. Perhaps where there are real-world, practice-based, empirically unambiguous indices of success or failure in coming to one's conclusions, we might rationally rely upon experience not only to provide the expert's data base, but also to authenticate the reliability of the conclusory skills involved. . . .

*[But], in circumstances when experience alone does not resolve the main doubts about reliability, it would be irrational, and therefore an abuse of discretion to rely upon it.*³⁷³

It is also insufficient to rely on the fact that some professional groups accept or endorse the diagnosis of SBS/AHT. As Professor Risinger points out:

³⁷¹ See *Kumho*, *supra* note 367 (quoting *Daubert*, *supra* note 2).

³⁷² D. Michael Risinger, *Defining the "Task at Hand": Non-Science Forensic Science after Kumho Tire Co. v. Carmichael*, 57 WASH. & LEE L. REV. 767, 773 (2000) (footnote omitted; emphasis added).

³⁷³ *Id.* at 775-76 (emphasis added).

[A]dherence to such standards cannot establish reliability [for admissibility purposes] when, as is often the case, it is the very reliability of the standard practice that is in issue. The guild test does at least claim to deal with reliability of the process beyond individual experience, but the reliability judgment is delegated to a group that, by definition, already believes in the process. The guild test trades the *ipse dixit* of the individual for the *ipse dixit* of the group.³⁷⁴

For this reason, *Kumho Tire* recognizes the inadequacy of general acceptance by a community when the issue is the reliability of the discipline and/or its application to the case at hand.³⁷⁵

In this response we do not take a position on the appropriate application of *Daubert* or other legal standards to particular hypotheses. We note, however, that there are essentially two possibilities. One could exclude both sides of the debate from the courtroom because there is inadequate information to make a conclusive diagnosis. Or, as is presently the case, experts with differing perspectives can argue it out in the courtroom, leaving it to judges and juries to sort out the intricacies of the infant brain and the complexities of biomechanics, as advocated by some prominent legal scholars, including Professor Edward Imwinkelried.³⁷⁶ This approach presents two problems. First, trying and retrying undecided scientific issues on a weekly basis is extraordinarily expensive and inevitably results in inconsistent and “fluky” justice.³⁷⁷ Second, and perhaps more important, if doctors cannot agree on these complex and unresolved issues, it is unlikely that jurors or judges can do any better.

What cannot be allowed is for supporters of the SBS/AHT hypothesis to present their hypotheses in the courtroom without making clear the limits of their knowledge and without the provision of competing presentations that are equally well-grounded and are often more consistent with the anatomy and physiology of the infant brain. Given the deference that judges and juries often give to expert opinion—a topic that is well-covered by Dr. Narang—the failure to present evidence from critics of the SBS/AHT hypothesis would

³⁷⁴ *Id.* at 777.

³⁷⁵ *Id.* at 778.

³⁷⁶ See Imwinkelried, *supra* note 49.

³⁷⁷ Tuerkheimer, *supra* note 51, at 523.

almost certainly increase the number of false convictions in an area that is likely already riddled with false convictions.³⁷⁸

C. The Costs of Misdiagnosis.

The costs of misdiagnosing child abuse are obvious. If we under-diagnose child abuse, abusive parents will go unpunished and children will be left in unsafe homes. If we over-diagnose abuse, we destroy families and imprison innocent parents and caretakers. But there is a third often under-recognized cost of misdiagnosis: if we identify the wrong problem, we will inevitably apply the wrong solution. For example, when infection or stroke is misdiagnosed as abuse, the focus almost inevitably shifts from appropriate treatment to interrogations and arrests. If the misdiagnosis becomes systemic, this may be accompanied by a broader failure to identify medical problems that may ultimately prove to be preventable or treatable.

V. THE PATH FORWARD

As we work towards a new paradigm, we must bear in mind that the misdiagnosis of SBS/AHT is extraordinarily harmful, and that there is no self-corrective mechanism. Typically, any suggestion of SBS/AHT results in the automatic removal of the child and/or the child's siblings from the home. In addition to the emotional anguish, families often lose their savings and homes in frantic attempts to reclaim their children while facing prison sentences up to and including the death penalty. While these costs may be justified if a child has been abused or murdered, one should be quite certain that the abuse did indeed occur before imposing these costs, particularly

³⁷⁸ While Dr. Narang dismisses the Goudge Inquiry in Ontario, Canada as consisting of "a few recent case reports of wrongful convictions" (Narang, *supra* note 3, at 515), the inquiry identified significant shortcomings in the field of pediatric forensic pathology and the diagnosis of shaken baby syndrome in particular. See Inquiry into Pediatric Forensic Pathology in Ontario (Sept. 2008) at <http://www.attorneygeneral.jus.gov.on.ca/inquiries/goudge/index.html>. The final report recommended a review of shaken baby and pediatric head injury convictions given the changes in SBS knowledge over the past two decades. See Consolidated Recommendations, Inquiry into Pediatric Forensic Pathology in Ontario 86 at http://www.attorneygeneral.jus.gov.on.ca/inquiries/goudge/report/v1_en_pdf/Vol_1_Eng_CR.pdf. Given the composition of the reviewing panel, it is unclear whether this review will lead to meaningful reform.

given a legal system that is ill-equipped to correct past mistakes.³⁷⁹

In this case, the suggestion that shaking may harm vulnerable infants—a suggestion originally made by Dr. Guthkelch—was eminently sensible and holds true today. The SBS corollary—that shaking can be presumed from specific medical findings, including subdural hemorrhage—was plausible and widely accepted, including by Dr. Barnes and Dr. Squier, two of the co-authors of this article. Research conducted over the past decades has, however, established that the SBS hypothesis was based on a misunderstanding of biomechanics and the infant brain, and that there are many alternative causes. The shift in terminology from SBS to AHT has not solved this problem since it is *harder*—not easier—to defend against mechanisms that are not specified and that therefore cannot be tested or even debated.

We suggest four paths forward: research, collaboration, acknowledgment of the complexities, and learning to work under conditions of uncertainty.

A. Research

While we may never reach the levels of certainty demanded by evidence-based medicine or *Daubert*, we can certainly do better than we have done in the past. The research that Professor Goldsmith suggested in his NIH presentation in 2001 is as applicable today as it was then, and many of his suggestions align with those of Dr. Narang. Promising avenues include:

1. Studies on the anatomy and physiology of the infant brain, including the tolerance and failure limits of bridging veins, the role of cerebral spinal fluid, the mechanisms of retinal hemorrhage, and the role of biochemical cascades.
2. Analysis of other diseases and medical conditions that

³⁷⁹ See, e.g., Tuerkheimer, *supra* note 51, at 544 (“While not always expressly articulated, commitment to the finality of criminal convictions is deeply embedded in our criminal law structures and jurisprudence”); Cavazos v. Smith, *supra* note 119, at 7 (upholding conviction in Shirley Smith case despite acknowledging that “[d]oubts about whether Smith is in fact guilty are understandable”).

“mimic” SBS/AHT. While children are not little adults, they are subject to many of the same illnesses and medical conditions, including stroke, infection and nutritional deficiencies. We need to prevent, diagnose and treat these conditions rather than automatically ascribing them to abuse.

3. Careful, complete and nonjudgmental interviews of parents and caretakers, who often hold the clues to the correct diagnosis.
4. The development of protocols for investigating known alternative causes and identifying new causes.
5. Maintenance of a national registry on SBS/AHT cases, with retention of medical records, radiology images, blood samples and tissue samples. Videotaped autopsies would also be helpful. This would allow us to obtain accurate numbers and would provide a basis for ongoing evidence-based medical scrutiny and judicial review.

B. Working Together

To date, the child abuse community has been divided into hostile camps. If the medical issues are to be addressed, however, we need to work together. To do this, we endorse Dr. Guthkelch's recommendation that we adopt descriptive medical terminology that does not attempt to answer the question that is being asked. It is very difficult to have professional discussions on the cause of medical findings that are named “shaken baby syndrome” or “abusive head trauma” since these terms assume the causation.

Second, we need to continue to have less antagonistic professional discussions. The biannual conferences conducted by Penn State Hershey are a good start. At these conferences, the organizers invite one or more presenters with diametrically opposed viewpoints to debate important issues. Often, the opposing camps are not as far apart as one might think. At the joint conference in Jackson Hole in 2009, for example, Dr. Plunkett and Dr. Dias quickly reached agreement that short falls can indeed be fatal, albeit rarely.³⁸⁰

³⁸⁰ Plunkett, *supra* note 267.

Another constructive conversation occurred at a conference sponsored by the Queens District Attorney's Office in New York in September 2011. While the presenters and audience consisted largely of supporters of the SBS/AHT hypothesis, a panel composed of representatives from both sides of the debate discussed the key issues in a professional manner, sometimes reaching the same conclusions. For example, all of the panelists agreed that violent shaking may cause serious injury or death; that the triad is not diagnostic of abuse; and that each case requires an extended inquiry into the child's medical history and findings.

Third, personal and professional attacks on those with opposing views must stop. New ideas and a willingness to question traditional understandings are a precondition to scientific progress. If we are to ensure the wellbeing of children and families, our commitment to "getting it right" requires that we put aside our preconceptions and consider new ideas, including those contrary to our most cherished beliefs. While there is always resistance to new ideas, every mistake—and every delay in correcting our mistakes—imposes heavy costs on children and families. Debate and disagreement are essential, but there is no room for *ad hominem* attacks or efforts to prevent the dissemination of new research.

Finally, this debate needs to be taken to the broader legal, medical and scientific communities. Since we now know that our initial understanding of SBS/AHT was flawed, we need the advice and support of other specialties, including scientists and doctors who are not so closely involved in the debate. An independent review of the validity and basis for the SBS/AHT diagnosis by the National Academy of Sciences would be a good start. Discussions at major Children's Hospitals and other teaching hospitals would also be useful. In the legal arena, it is important to keep lawyers and the judiciary abreast of the advancing medical science and for prosecutors, judges and child protection agencies to consider the facts of each case rather than relying exclusively on medical hypotheses.

C. Acknowledging the Complexities

For decades, the SBS hypothesis provided a clear and simple explanation for the collapse or death of children who presented with subdural hemorrhage, retinal hemorrhage and brain swelling. We

now know, however, that its premises were wrong. The SBS hypothesis was based on a three-component model that did not reflect or recognize the complexities of the infant brain. In its original form, SBS taught that subdural hemorrhages were caused by the traumatic rupture of bridging veins in the "subdural space." However, the small thin subdurals typically found in infants are too small to represent the rupture of bridging veins, there is no subdural space between the dural and arachnoid membranes, and the "sub"dural hemorrhages in infants more likely originate in the venous dural plexus. The SBS hypothesis also taught that retinal hemorrhages in children were caused by the traumatic rupture of retinal veins. However, retinal hemorrhages in children are also seen in natural diseases and appear to reflect the same causes as retinal hemorrhages in adults, including lack of oxygen, thrombosis, increased intracranial pressure and time spent on life support. Finally, the SBS hypothesis taught that brain swelling was caused by the traumatic rupture of axons (nerve fibers) throughout the brain. However, we have known for more than a decade that the brain swelling is due to lack of oxygenated blood from any cause. All of this knowledge was neglected because it did not fit the model.

As our analyses become more anatomically correct, we are finding that there is no single model. Instead, the cases vary widely. A few cases present with large space-occupying subdural hemorrhages, as one would expect from ruptured bridging veins, but most present with thin intradural/subdural hemorrhages or thrombosed (clotted) veins with surrounding leakage. The ocular findings range from small unilateral retinal hemorrhages to bilateral multilayered retinal hemorrhages with retinoblastoma. The brain findings range from no brain damage at all to swollen hypoxic-ischemic brains with no hope of recovery. In some cases, all of the findings are acute (new), while in others some or most of the findings are weeks to months old, or even older. The clinical histories are equally diverse: some children were healthy until their collapse; others had seizures, feeding difficulties or neurological impairments from birth; and yet others were symptomatic for days or weeks before collapse. In some cases, the collapse occurred when the child and a caretaker were alone; in others, the child and the caretaker were alone for minutes, if at all.

Given the heterogeneity of the medical findings and factual settings, one should be skeptical of a “one size fits all” diagnosis. One should also be skeptical of diagnoses that rest on three isolated findings without considering the characteristics of the developing brain and the relationship between the brain and the rest of the body. In so doing, one should remember that:

If one were to name the universal factor in all death, whether cellular or planetary, it would certainly be loss of oxygen. Dr. Milton Helpern, who was for twenty years the Chief Medical Examiner of New York City, is said to have stated it quite clearly in a single sentence: “Death may be due to a wide variety of diseases and disorders, but in every case the underlying physiological cause is a breakdown in the body’s oxygen cycle.” Simplistic though it may sound to a sophisticated biochemist, this pronouncement is all-encompassing.³⁸¹

In infant deaths, like all other deaths, the medical question is “what caused the lack of oxygen?” —not “who did it?” In our effort to determine why the child lacked oxygen—a question that has hundreds of possible answers and may sometimes prove unanswerable—we must treat each case the same way as we treat any other complex diagnosis: we must consider the lab results, the history, and all of the medical findings, bearing in mind the complexities of the human body and the physiological cascades that occur when this tightly regulated system goes awry. We must also carefully sort out, to the best of our ability, which findings help determine the cause of injury or death and which are secondary to an ongoing process and/or medical intervention. To do anything less is a disservice to children, families and our system of justice.

Today, everyone agrees that the “triad” of findings previously attributed to shaking may reflect abuse, accident or natural causes. What we don’t know is how many cases—or sometimes which cases—fall into each of these categories. More than a decade ago, the Five Percenters suggested that 5% of SBS cases were misdiagnosed as child abuse³⁸²—a figure that many thought was high. Based on the

³⁸¹ SHERWIN B. NULAND, *HOW WE DIE: REFLECTIONS ON LIFE’S FINAL CHAPTER* 67 (1994). Professor Nuland teaches surgery and the history of medicine at Yale University.

³⁸² Beth Hale, *Falsely Branded a Baby Batterer—Now Rioch Edwards-Brown’s a Fighter for Justice*, *DAILY MAIL*, Nov. 24, 2011, at <http://www.dailymail.co.uk/femail/article-2065430/INSPIRATIONAL-WOMEN-OF-THE-YEAR-Falsely-branded-baby-batterer-->

changes in the literature over the past decade, however, this figure may be even higher. But is it 10%, 25%, 50% or even 95%? The answer to this question is: we don't know. And until we do know, we cannot use statistics to address the issues, let alone to diagnose individual cases.

D. Working Under Conditions of Uncertainty.

While we would all like a "gold standard" that distinguishes quickly and accurately between abuse, accident and natural causes, the medicine is uncertain and evolving, and the cases are complex. As we continue to search for answers, we need to make the best possible decisions under conditions of uncertainty. Dr. Narang suggests that we do this by emphasizing clinical judgment, leaving the resolution of the disputed medical issues to judges and juries. We suggest that the costs of this approach are too high and that we instead need to make clear the limits of our knowledge while expanding our knowledge base. In essence, this is what doctors and lawyers do when we treat patients or advise clients. It should be no different in the courtroom, where the safety of children and the future of entire families hangs in the balance.

Appendix E



Shaken baby syndrome: A biomechanics analysis of injury mechanisms

Faris A. Bandak*

Department of Neurology, A1036 F. Edward Hébert School of Medicine, Uniformed Services, University of the Health Sciences, 4301 Jones Bridge Road, Bethesda, MD 20814, USA

Received 2 November 2004; received in revised form 2 February 2005; accepted 8 February 2005

Abstract

Traumatic infant shaking has been associated with the shaken baby syndrome (SBS) diagnosis without verification of the operative mechanisms of injury. Intensities for SBS have been expressed only in qualitative, unsubstantiated terms usually referring to acceleration/deceleration rotational injury and relating to falls from great heights onto hard surfaces or from severe motor vehicle crashes. We conducted an injury biomechanics analysis of the reported SBS levels of rotational velocity and acceleration of the head for their injury effects on the infant head-neck. Resulting forces were compared with experimental data on the structural failure limits of the cervical spine in several animal models as well as human neonate cadaver models. We have determined that an infant head subjected to the levels of rotational velocity and acceleration called for in the SBS literature, would experience forces on the infant neck far exceeding the limits for structural failure of the cervical spine. Furthermore, shaking cervical spine injury can occur at much lower levels of head velocity and acceleration than those reported for the SBS. These findings are consistent with the physical laws of injury biomechanics as well as our collective understanding of the fragile infant cervical spine from (1) clinical obstetric experience, (2) automotive medicine and crash safety experience, and (3) common parental experience. The findings are not, however, consistent with the current clinical SBS experience and are in stark contradiction with the reported rarity of cervical spine injury in children diagnosed with SBS. In light of the implications of these findings on child protection and their social and medico-legal significance, a re-evaluation of the current diagnostic criteria for the SBS and its application is suggested.

© 2005 Elsevier Ireland Ltd. All rights reserved.

Keywords: Injury; Infant; Shaken; Baby; Rotational; Acceleration/Deceleration; Syndrome; Neck

1. Introduction

Shaking an infant to the point of severe brain injury is usually associated in the literature with the diagnosis referred to as the shaken baby syndrome (SBS). Infant shaking is in fact a potentially very injurious mechanical event. Consequently, its analysis and assessment requires knowledge and training in *Injury Biomechanics*. This scien-

tific discipline deals with the mechanical damage processes and causations of injury. Therefore, Injury Biomechanics is central to the study of the mechanisms of injury in the SBS.

The current description of the SBS in the literature evolved over a period of nearly a half a century with some reports attributing its genesis to Caffey [1–3] a pediatric radiologist, who had the notion that an association between chronic subdural hematoma and long bone fracture in children should be a red flag for child abuse. Caffey's notion remained less known for about 10 years until he encountered the case of Virginia Jaspers, a nurse caretaker who confessed

* Tel.: +1 301 299 7357.

E-mail address: fbandak@usuhs.mil.

Table 1
Head injuries in the infant vs. the adult

Infant	Adult	Both
Deformation based		
Tears in subcortical white matter	Epidural hematoma	Lesions in the corpus callosum and brain stem
Separation of a suture	Basilar skull fractures	Traumatic axonal injury
Suture-to-suture linear fractures	Diffuse axonal injury	Linear skull fractures
Ping pong fractures		
Bilateral skull fractures		
Pressure based		
Coup hemorrhages	Coup and contre-coup hemorrhages	
Coup contusions	Coup and contre-coup contusions	
Coup acute subdural hematoma		
Relative motion based		
		Acute subdural hematoma ^a
		Localized intracranial hemorrhages ^a

^a Represents different biomechanisms for the infant and adult.

to shaking a 2-week-old infant who died. Jasper's confession is a legalistic characterization and thus did not provide scientific support for Caffey's notion but did help start the use of the SBS label in the literature. It is unclear from the literature that Caffey envisioned this label to evolve into the SBS diagnosis as seen and applied today.

Kempe [4] contributed to the current description of SBS by introducing the "Battered Child Syndrome" and the concept that inconsistency between clinical observations and reported event history should signal abuse. However, a fundamental element of the meaning behind accurate "history" has to do with the biomechanical causes of injury. Clearly, the assessment of the mechanical causation of injury requires training and experience in Injury Biomechanics, a distinct discipline not taught in medical school. Lack of education and experience in Injury Biomechanics, amongst other factors, has led in practice to the proliferation and propagation of inaccurate and sometimes erroneous information on SBS injury mechanisms in the literature.

Another factor was added by Guthkelch [5] who synthesized the accumulating SBS medical literature to conclude that it is possible to infer shaking without impact as a cause of injury when an infant presents with subdural hematoma and retinal hemorrhages. He did not conclude that only shaking could cause such injuries. At this point in its evolution, the SBS began to develop in the literature into the *injury causation signature* that is widely described and used today. More specifically, an infant presenting with, at a minimum, acute subdural hematoma (ASDH) and retinal hemorrhages with "inconsistent" or "un-explained" biomechanical history is commonly diagnosed with SBS. Such diagnosis puts the physician in the difficult position of evaluating injury causation to determine if the reported history is biomechanically "consistent" or "explainable" without the benefit or even availability of an Injury Biomechanics assessment.

In this study, we will present a biomechanics analysis of infant shaking and its consequences on the head-neck to

determine if it is possible for the fragile infant neck to withstand SBS-defined levels of head accelerations without injury.

2. Biomechanical classification of head injury

The SBS diagnosis has been primarily linked to injuries of the head. Table 1 shows the types of head injuries occurring in infants and adults. Generally, head injuries can be classified in groups with similar biomechanical genesis (Fig. 1). Biomechanical forces acting on the head can be dynamic or static (Fig. 1) and since shaking is a dynamic event, static forces (Fig. 1) will not be discussed here. Dynamic head loadings are categorized as either contact or non-contact meaning direct loading to the head and head loading through the neck respectively. The mechanical features leading to a particular head injury or set of injuries distinguishing primary and secondary are shown in Fig. 1. Primary injuries are those caused directly by the mechanical insult and secondary injuries result as that part of the pathophysiological progression following primary injury. *The boundary between primary and secondary injury in SBS has not been clearly defined.*

Fig. 1 shows injuries that have been related in the literature to local intracranial brain motion, gross intracranial brain motion, or both. For instance, a direct impact to the head resulting from a fall on a flat, hard surface produces a local indentation of the skull impinging on the brain and producing local brain deformations and pressures. Another consequence of the contact is the stopping of the moving head. The stopping force is communicated to the brain through a local path starting first with the area of contact on the scalp over the skull through the subarachnoidal cerebro-spinal fluid (CSF) layer surrounding the brain and eventually reaching the brain. The higher the fall, the faster, to a practical limit, the head impact velocity, and so the

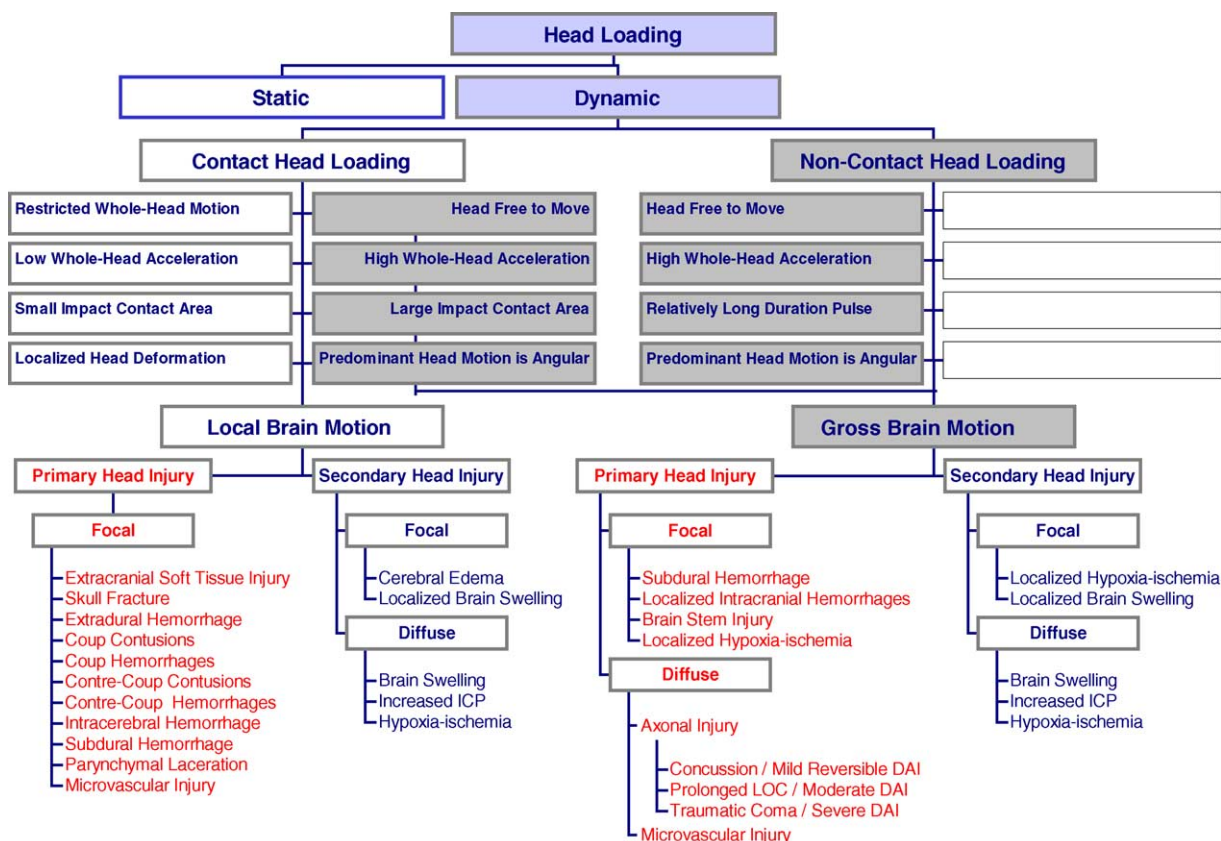


Fig. 1. Biomechanical classification of head injuries.

greater and faster the stopping force transmission to the brain. Whole-brain movement is eventually stopped against higher intracranial coup pressure and lower contre-coup pressure. The extent of gross intracranial brain movement also depends on how much rotational energy is involved in the impact [6]. Forces on the head through the non-contact mechanism pass through the neck and therefore inherently have a rotational component though, practically, much lower peak accelerations and much longer durations are achievable through this type of loading. Theoretically, injuries associated with rotational head accelerations are common to both the contact and the non-contact categories (Fig. 1) if sufficient magnitudes and rates are reached. Head injuries from non-contact loading are producible through contact loading but the converse is generally not true.

The non-contact form of head loading has been the cornerstone of the Shaking Baby Syndrome definition. In its purest form the SBS, as described in the medical literature [7], represents rotational head accelerations from a sequence of mechanical events paraphrased as follows:

An infant is gripped by the chest or shoulders and forcefully shaken back and forth whipping the head in the antero-posterior direction. The nearly non-existent muscle strength

of the infant neck makes the head highly susceptible to high head-whipping rotational acceleration so severe that the brain moves relative to the interior surface of the skull resulting in torn bridging veins and so acute subdural hematoma.

While SBS has taken on other labels in the literature adding or substituting terms like “whiplash” and “impact,” it still maintains the shaking component as the central causation substratum of this diagnosis.

3. Biomechanical aspects of the infant anatomy

Discussion of infant shaking calls for a brief overview of the infant anatomy deemed relevant to the biomechanics of shaking. The head of the infant, weighing up to one third of the total body weight, is effectively a nearly unsupported mass. As a practical matter of common safety, caution is required of a caretaker when picking up or generally moving an infant. Caretakers must provide head support to compensate for the fragility, laxity, and the lack of infant neck muscle strength since it provides minimal resistance to any externally induced relative motion between the head and the thorax.

The massive-head and weak-neck attributes of the infant create the potential for severe neck injury under certain dynamic conditions. The flat, shallow, articular surfaces, cartilaginous nature, and incomplete ossification of the cervical vertebrae increases the potential for relative displacement between vertebrae and puts more burden on the weak infant cervical spine ligaments and on the infantile spinal cord. The neck as a whole, with the characteristics described above can stretch significantly beyond subluxation limits without ligamentous rupture. In addition, it can be stretched more than the spinal cord is able to accommodate [8] thus potentially injuring the spinal cord under distraction forces. Atlanto-axial and atlanto-occipital dislocation, dens fractures, and cord transections can occur from excessive stretch of the neck as has been reported for circumstances of breech extraction deliveries [9]. The infantile ligaments control the articulation and govern the mobility and range of motion of the cervical spine. These ligaments are vital to the stability of the atlanto-occipital joints governing the articulation at the cranio-vertebral junction. The cranio-vertebral junction houses the cervico-medullary portion of the spinal cord and thus presents a vulnerability to commonly fatal craniospinal injuries under serious traumatic excursions into the spinal canal. This canal is large in C1 compared to the segments below. It is occupied equally by the dens and the cord both taking up two thirds of the space with the remaining one third free space. Consequently, there is some room for the cord to move under atlanto-occipital displacement. However, displacements exceeding the available free space can injure the cord.

The chest of the infant is defined in the SBS literature as a possible grip area for the shaker. The infant's thin thoracic wall and the cartilaginous elastic nature of the ribs make the

chest more vulnerable to large deformations and chest indentations which can cause injury and affect internal organs. The chest circumference is generally smaller (0.5 in. or so) than the head circumference at birth with its value reaching that of the head circumference at about 1 year of age. The neonatal chest is nearly circular and slowly becomes elliptical with age making it wider in the coronal plane at about 6 months of age. The location of the heart in an infant is of course different from that of the older child. It is located at the midpoint between the head and the buttocks and after the fourth year, the heart descends downward as the thorax grows. The chest prepares biomechanically for this descent by becoming very bony with ossified ribs forming the protective rib cage, which also descends downward providing similar protection for the kidneys, the spleen, and other organs.

Integrated into the chest structure is the thoracic part of the vertebral column. The infant spinal column has a single curve composed of the thoracic, the cervical, and the lumbar portions of the column [10]. This important feature is relevant to the infant's biomechanical response to shaking versus that of the older child. The lower portion of the spinal column, referred to as the sacral region, forms another but smaller curve. The vertebral column eventually assumes the familiar S-shape as the skeletal structure develops and the pelvis tilts forward creating the thoraco-lumbar inflection point in the familiar S-shape.

4. Injury biomechanics analysis of infantile SBS

In order to evaluate the operative injury mechanisms in the brain of an infant under SBS-defined head acceleration,

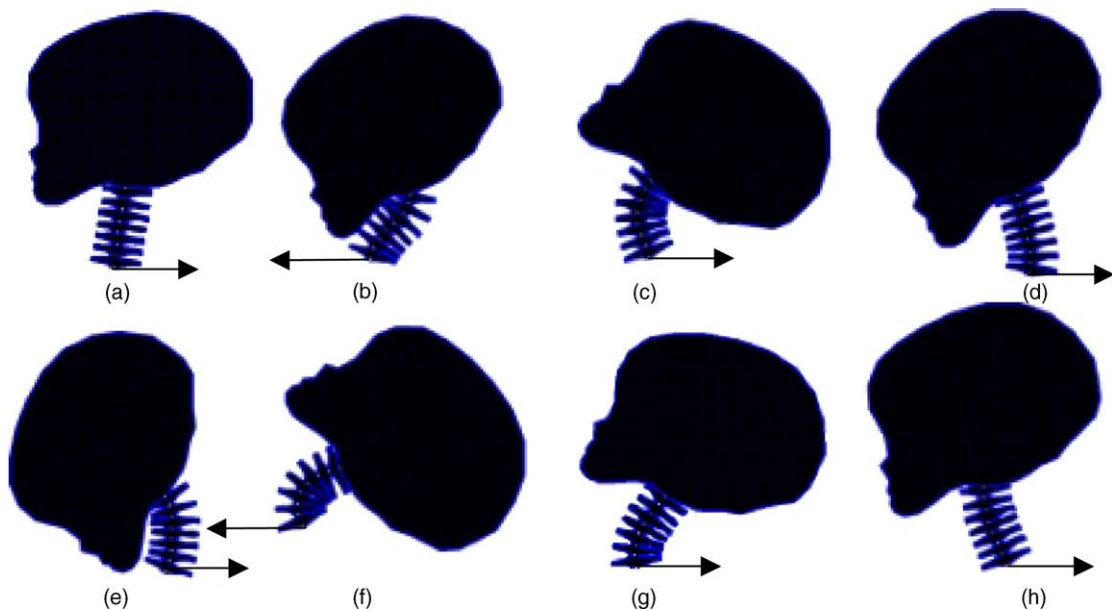


Fig. 2. Several time-history snapshots of the head-neck during chest shaking.

we must obtain the loadings that actually reach the brain along the path starting at the grip points of the shaker’s hands, through the thorax, spinal column, neck, and eventually the head. Fig. 2 shows, schematically, the back and forth head motion resulting from shaking. The grip forces act on the thorax and load the ribs. They are transmitted through the posterior joints connecting the ribs to the thoracic spinal column and so shake the column imposing that back-and-forth on the base of the cervical spine portion of the spinal column. The resulting forces enter the neck at C7. We did not address possible injury to any part of the body below C7 in this study. This conservative position was taken even though the required chest-shaking intensity of SBS can cause injuries in the infant below C7.

5. SBS head loading

We have necessarily described SBS in terms of head loading because the head has been central to the definition of the SBS injuries. Chest shaking forces reach the head through the neck starting at C7 and moving back and forth in wave fashion (Fig. 2) eventually transmitting forces to the cranio-vertebral junction. Along this path, the force acts on soft, flat, incompletely ossified vertebrae, and soft ligaments. The forces reach the cranio-vertebral junction and induce the infant skull to move back and forth with little resistance to pivot about the atlanto-occipital joint. The overall head motion is governed by the neck which significantly influences

the head trajectory and the center of rotation. In fact, the neck must completely bear the load that keeps the head and thorax from separating. For the infant, this type of chest-shaking head motion can be thought of as a kind of “dragging” motion where the neck, acting as a tether, pulls the unsuspecting head in response to the induced motion of the chest. This has not been described as the mechanism for SBS.

To understand what has been described in the literature for SBS accelerations, consider an illustrated example of a sphere with mass m connected to a rod with length r as shown in Fig. 3. If point B starts to move in a straight line in the horizontal direction, a force from the rod will act on it to cause it to also move inwards in the radial direction resulting in the composite, familiar motion along a circular arc ultimately setting mass m in rotation about point A. At any point along the arc, one can evaluate the acceleration of mass m . This acceleration has two main components, a tangential component (along the arc), a_t , and a normal (in the radial direction) component, a_n , as shown in Fig. 3. The figure shows that the mass is tending to escape the circular arc and go free but is constrained by the rod to move along the arc. Naturally then, the mass m is exerting a force with components $F_n = ma_n$ and $F_t = ma_t$ on the rod. If for instance, the outward acceleration a_n has a magnitude of 50 G and the value for mass is 1.36 kg, then the force acting on the rod is equal to 665 N (150 lbf). Clearly, the rod will give way if it is not strong enough to bear such a force. Under similar circumstances the neck experiences forces as the massive head is accelerated.

$a_n = v^2/r$	Normal acceleration (velocity squared over the radius)
$a_t = r (d^2\theta /d^2t)$	Tangential acceleration (radius times the angular acceleration)
$F = ma$	Newton’s Second Law (mass times acceleration)
$F_n = ma_n$	Normal Force (distraction force)
$F_t = ma_t$	Tangential Force (shear force)

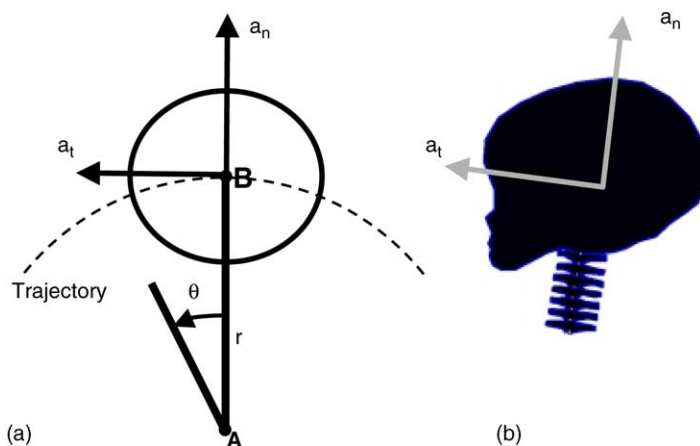


Fig. 3. Force diagram on (a) sphere and (b) head-neck schematically indicating rotational head motion about point A.

The reported SBS back-and-forth head motion of this type along an arc produces an exchange between a_n and a_t in a periodic fashion reaching peaks at alternating times along the arc. Relating these to forces acting on the rod, we see that the rod has to resist forces tending to both stretch and bend it. While more complicated biomechanically, the actual shaking response of the infant head also imposes shear and tensile forces as well as compressive forces on the infant neck.

To evaluate the neck forces produced by SBS-type head accelerations, we considered the range of rotational head acceleration and velocity levels cited in the literature. Jenny and co-authors [11] attempting to show that higher accelerations can be achieved by human manual shaking reported that peak rotational accelerations of 6000–13,252 rad/s^2 and rotational velocities of 120–153 rad/s are possible through the manual human shaking of a dummy surrogate representing a Japanese infant. Spivack used the work of Duhaime et al. [12] relating rotational velocities of 50–120 rad/s and approximately 30,000- rad/s^2 rotational acceleration to SBS. SBS is cited in the literature as having forces that are as great as those of falls from great heights, by some accounts more than 9 m (30 ft), onto hard surfaces or from high-speed motor vehicle crashes [13]. These contentions of SBS load levels have not been substantiated biomechanically with some reports refuting their validity at all [12,14].

To illuminate and compare simplified head motion energies of familiar events such as car crashes, biking, walking, etc., with shaking, we calculated the free head velocity associated with each event as shown in Table 2. The table reflects conservative but realistic estimates of maximum free head velocity for each of the events. Ironically, the values show that shaking head velocity is less than the maximum free-fall velocity from height of 1 m (3(1/4) ft). This comparability of energies raises further questions concerning the forces reported for SBS and might account in part for the misconceptions and controversial views on the head injury severity of “short falls.” The energy from velocity is called kinetic energy and we refer to it here as *unrealized* damage energy meaning it can be converted to potentially damaging energy through a rapid change in magnitude and/or direction. For the case of the infant head, this velocity change can come from the action of the neck on the head during chest

shaking as we explained earlier or it can come from an impact action applying force directly on the head such as that from a fall. Table 2 shows the energies as multiples of units of failure energy where the failure energy was taken to be equal to the skull fracture energy in the infant. Skull fracture energy was chosen since skull fracture has been used as a conservative indicator of impact intracranial injury in infants [15,16].

A range of angular accelerations and velocities was taken to be between 5000 and 15,000 rad/s^2 for rotational acceleration and between 50 and 150 rad/s for rotational velocity. This range was chosen because it is conservatively representative of the *lower* values cited in the literature for SBS and reported to be below thresholds postulated in the SBS literature for subdural hemorrhages. The SBS forces were obtained and compared with the current biomechanical data related to infant injury thresholds.

6. Results and discussion

The shaken baby syndrome, as a diagnosis, has become virtually synonymous with inflicted cerebral trauma. The injury mechanisms of the SBS have historically not been linked to cervical spine injury even though its early evolution was bolstered by Caffey’s [3] interpretation of Ommaya’s whiplash work [17,18]. Caffey interpreted this work as a demonstration that brain injury could occur from head whipping by chest shaking without contact head impact. Caffey translated Ommaya’s results without considering Injury Biomechanics, into an explanation for a confession of shaking.

The Ommaya whiplash animal model primarily addressed the primate head as an adequate surrogate for the adult human brain under the types of loading tested. While an argument can be made for this model substitution for the case of adult human head, the neck is another matter. Astonishingly, Caffey did not give consideration to the head-neck features of the primate model used to study whiplash. In fact, the animal model used had quite opposite whiplash-related features compared to the infant when the head-neck is concerned. Specifically, the rhesus monkey has a small,

Table 2
Head velocities and normalized failure energy associated with familiar dynamic events compared with shaking and falls

	Height (m)	Velocity (m/s)	Normalized impact energy (Nm/N)	1.3 N head weight (E_{SF}) ^a	2.3 N head weight (E_{SF})
Free fall (30 ft)	9.10	13.36	9.10	13.00	23.00
Powered vehicles 48 km/h (30 mph)		13.36	9.10	13.00	22.99
Adult biking 32 km/h (20 mph)		8.90	4.04	5.77	10.20
Adult running 24 km/h (15 mph)		6.70	2.29	3.27	5.78
Toddler biking 16 km/h (10 mph)		4.40	0.99	1.41	2.49
Toddler running 9.7 km/h (6 mph)		2.70	0.37	0.53	0.94
Free fall (3 ft)	0.91	4.09	0.85	1.22	2.15
Shaking 11–15.5 km/h (6.8–9.5 mph; 10–14 ft/s)		4.31	0.95	1.35	2.39

^a E_{SF} is the energy associated with skull fracture and so the column is in units of E_{SF} .

Table 3
Neck distraction forces vs. head weight and center of rotation

Neck distraction force (N)						
Head weight (kg)	0.68		1.34		1.59	
Neck length (cm)	3.81	6.35	3.81	6.35	3.81	6.35
Low end of reported SBS rotational acceleration range	1027	1711	1711	2852	2395	3992
High end of reported SBS rotational acceleration range	9240	15399	15399	25665	21559	35931

relatively less massive, head on quite a strong neck while the infant on the other hand has a relatively more massive head on a floppy, weak, neck. Consequently, the infant neck is far more susceptible to whiplash injury than that of the rhesus monkey under the similar chest-shaking head accelerations. Nearly half of the concussed animals in Ommaya’s [17]

whiplash experiments experienced cervical spine or brain stem injuries even with the disproportionately strong neck relative to the human infant.

Ommaya’s experiments [17] as well as follow-up primate experiments by Ommaya’s previous co-workers, Gennarelli and Thibault [19] supported a rotational acceleration

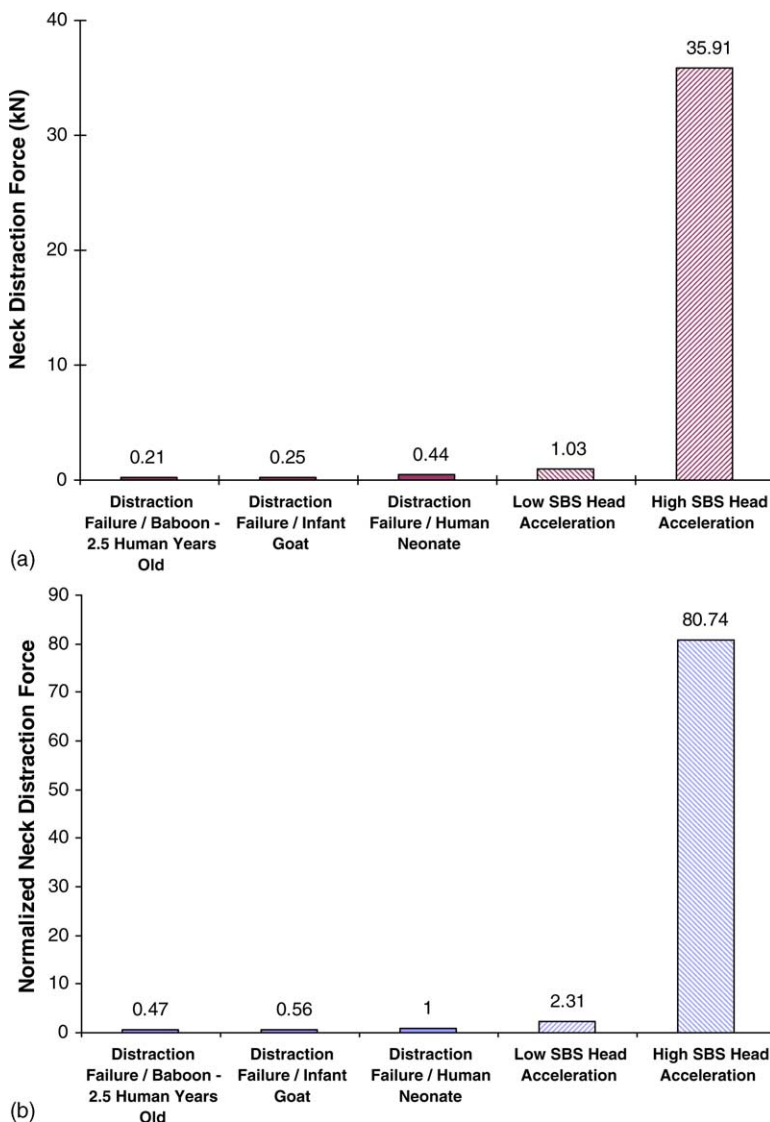


Fig. 4. SBS neck distraction force vs. (a) Structural failure of the cervical spine and (b) normalized to human neonate distraction failure data.

mechanism for the generation of cranial subdural hematomas. This is the much-heralded differential rotational skull-brain motion mechanism that causes parasagittal bridging veins to rupture and thus hemorrhage below the dura. This mechanism was postulated for the adult head with a fully ossified, stiff, skull which plays a major role in its activation. It is important to note that the Gennarelli-Thibault experiments were conducted in a way that protected the neck from head whipping forces. In those experiments, the head of the primate was potted in a metal cylinder which was constrained to accelerate and then decelerate along a prescribed arc in a prescribed time frame. In this way, the neck was not subjected to the forces of the accelerated free head as it would be if the loadings were applied at the chest. This is precisely and erroneously the presumed SBS head motion where equivalent rotational accelerations of the infant human head were calculated by scaling this type of data. The important question when using the results of these experiments to interpret infant shaking injury is whether it is naturally possible for a free human head to reach such head accelerations through chest shaking without neck injury. This question was not addressed by those primate experiments nor has it been addressed quantitatively in the literature. It was addressed in this study for the case of infant shaking.

Our results for SBS head accelerations imposed for different head masses and centers of rotation (Table 3) representative of infants show that the resulting forces produce both distraction and localized hyper-flexion and hyper-extension forces on the infant neck and that these forces increase with increasing head mass. Therefore, it can be concluded that for the same acceleration and same neck size, a heavier head is more likely to produce a shaking neck injury.

Using a range of values for head mass and center of rotation for infants, and peak acceleration and peak velocity for SBS, we obtained neck distraction forces ranging from a lower bound of 1027 N to an upper bound of 35,910 N (Table 3). These values were compared with experiments on several animal species and on neonate cadavers as shown in Fig. 4. The experimental results show that the cervical spine becomes susceptible to severe injury at values as low as 209 N for the baboon at 3 years equivalent human age [20,21]. Fig. 4 shows failure values of 249 N for the infant goat neck [22] and 445 N for the human neonate neck [9]. It is important to note that these experimental values approximate force levels that would cause major structural failure of the cervical spine for each species. Serious cord injury or even transection can be expected to occur at even lower force levels [8]. The important observation here is the order of magnitude differences between what is reported in the SBS literature as necessary levels to cause injury and the actual magnitudes of the operative forces.

These force levels conservatively indicate that severe cervical spinal cord or brain stem injury in the infant can occur at significantly lower levels than invoked by the

current SBS literature as a cause of subdural hematomas. These results are quite consistent with the laxity and fragility of the infantile cervical spine and the lack of muscle strength of the neck. They are also consistent with the established experience that neck injury does not usually occur as a result of direct loading to the neck. The more common causes of neck injury are by action that involves the head. Whether by direct head impact induced accelerations, or head accelerations from indirect chest shaking, it is generally the forces on the head that transmit to neck injury forces. This is the essence of the term “whiplash” that is quite familiar to neck injury victims of rear-end motor vehicle crashes. Ironically, whiplash has been intrinsically embraced in the definition of the Shaken Baby Syndrome without reference at all to cervical spine injury.

7. Conclusions

This study resulted in the following findings:

1. Head acceleration and velocity levels commonly reported for SBS generate forces that are far too great for the infant neck to withstand without injury.
2. Head velocity from human manual shaking is of the same order as free fall head velocity from a height of about 1 m (approximately 3-ft).
3. Shaking head accelerations can potentially cause severe, if not lethal, cervical spinal cord or brain stem injury in the infant at levels *well below* those reported for the Shaking Baby Syndrome.
4. Given that cervical spine injury is reported to be a rare clinical finding in SBS cases, the results of this study indicate an SBS diagnosis in an infant with intracerebral but without cervical spine or brain stem injury is questionable and other causes of the intracerebral injury must be considered.
5. Re-consideration and clarification of the relative significance of each of the terms “shaking” and “impact” as used in the SBS diagnosis of cerebral trauma in an infant is warranted.
6. Cervical spine and/or brain stem injury should be included amongst the factors considered in the determination of consistency of reported history in cases where infant shaking is suspected. It should be kept in mind that such injury is not exclusive to shaking as the sole mechanical cause. Traumatic shaking is just one of the causes.
7. The rotational head acceleration mechanism for the intracerebral injuries of the SBS is inconsistent with the findings of this study. A non-impact, shaking-only, mechanism of primary intracerebral injury in an infant has not yet been described and requires further research. We have shown that infant shaking can cause primary brain stem or cervical spinal cord disruption which is known to lead to secondary intracerebral injury manifest-

ing as the familiar apneic presentation which is followed by hypoxic-ischemic response and cerebral edema.

In light of the findings of this study, re-evaluation of the present diagnostic criteria for the SBS merits serious attention for its implications on child protection and for the social and medicolegal significance of its application.

References

- [1] J. Caffey, Multiple fractures in the long bones of infants suffering from subdural hematoma, *Am. J. Roentgen.* 56 (1946) 163.
- [2] J. Caffey, On the theory and practice of shaking infants: its potential residual effects of permanent brain damage and mental retardation, *Am. J. Dis. Child.* 124 (1972) 161.
- [3] 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, *Pediatrics* 54 (1974) 396–403.
- [4] C.H. Kempe, F.N. Silverman, R.F. Steele, W. Droegemueller, H.K. Silver, The battered child syndrome, *JAMA* 181 (1962) 17–24.
- [5] A.N. Guthkelch, Infantile subdural hematoma and its relationship to whiplash injuries, *Br. Med. J.* 2 (1971) 430–431.
- [6] F.A. Bandak, On the mechanics of impact neurotrauma: critical review and synthesis, In: Bandak et al., (Ed.), *Traumatic Brain Injury: Bioscience and Mechanics*, Mary Ann Liebert, 1996, pp. 139–153.
- [7] American Academy of Pediatrics, Committee on child abuse and neglect, Shaken baby syndrome: Rotational cranial injuries – technical report, *Pediatrics*, 2001 108, 1, 206–210.
- [8] W.E. Stern, R.W. Rand, Birth injuries to the spinal cord: report of two cases and review of the literature, *Am. J. Obstet. Gynecol.* 78 (1959) 498.
- [9] M. Duncan, Laboratory note: on the tensile strength: a critical of the fresh adult fetus, *Br. Med. J.* 2 (1874) 763–764.
- [10] W.H. Johnson, J.A. Kennedy, *Radiographic Anatomy of the Human Skeleton*, Williams and Wilkins, Baltimore, 1961.
- [11] C. Jenny, T. Shams, N. Rangarajan, T. Fukuda, *Injury Biomechanics Research, 30th International Workshop*, Ponte Vedra Beach, Florida, 2002, pp. 129–143.
- [12] A.-C. Duhaime, T.A. Gennarelli, L.E. Thibault, D.A. Bruce, S.S. Margulies, R. Wiser, The shaken baby syndrome: a clinical, pathological, and biomechanical study, *J. Neurosurg.* 66 (1987) 409–415.
- [13] A.C. Duhaime, J. Alario, et al. *Pediatrics* 90 (1992) 179–185.
- [14] M.T. Prange, B. Coats, A.C. Duhaime, S.S. Margulies, Anthropomorphic simulations of falls, shakes, and inflicted impacts in infants, *J. Neurosurg.* 99 (2003) 143–150.
- [15] S.A. Schutzman, P. Barnes, A.-C. Duhaime, D. Greenes, C. Homer, D. Jaffe, R. Lewis, Evaluation and management of children younger than 2-years-old with apparently minor head trauma: proposed guidelines, *Pediatrics* 107 (5) (2001) 983–993.
- [16] K. Quayle, D. Jaffe, N. Kupperman, B. Kaufman, B. Lee, T. Park, W. McAlister, Diagnostic testing of acute head injury in children: When are head computed tomography and skull radiographs indicated? *Pediatrics* 99 (5) (1997).
- [17] A.K. Ommaya, F. Faas, P. Yarnell, Whiplash injury brain damage, *JAMA* 204 (1968) 285–289.
- [18] A.K. Ommaya, P. Yarnell, Subdural hematoma after whiplash injury, *Lancet* (1969) 237.
- [19] T.A. Gennarelli, L.E. Thibault, *Biomechanics of acute subdural hematoma*, *J. Traum.* 22 (1982) 680–686.
- [20] D. Nuckley, M. Eck, S. Hersted, R. Mizra, R. Ching, Tensile mechanics of the developing baboon cervical spine, in: F.A. Bandak (Ed.), *Injury Science Research Proceedings of the 28th International Workshop on Human Subject Biomechanics*, Atlanta, Georgia, 2000, pp. 85–89.
- [21] R. Ching, J. Nuckley, S. Hersted, M. Eck, F. Mann, Tensile mechanics of the developing cervical spine, *Stapp. Car Crash. J.* 45 (2001) 329–336.
- [22] R. Mayer, F. Pintar, N. Yoganadan, Pediatric neck tensile strength characteristics using a caprine model, in: F.A. Bandak (Ed.), *Injury Biomechanics Research, Proceedings of the 27th International Workshop on Human Subject Biomechanics*, San Diego, California, 1999, pp. 87–92.

Glossary

Injury Biomechanics: Biomechanics is the subset of the scientific discipline of Mechanics that deals with the forces, motions, deformations, ruptures, fractures, breaks, etc. of living tissue. The science of Biomechanics applies at the microscopic (cellular, sub-cellular, etc.) and the macroscopic (tissue, organ, full body, etc.) scales. Injury Biomechanics is the application of Biomechanics to the understanding of the causation and mechanism of injury.

Normal: in this case “normal” means in a direction perpendicular to the arc.

G: is the designated symbol for one unit of gravitational acceleration. On earth, the value of *G* is 32.2 ft/s²

N: refers to a Newton, the unit of force or weight.

Rad: is the symbol for radian, which, like degrees, is a measure of angle. Approximately 6 radians represents one revolution or 360 degrees. The actual value for one revolution is 2π radians where π has an approximate value of 3.14.

E_{SP}: infant skull fracture energy.

Appendix F

BRIEF COMMUNICATION

M.G.F. Gilliland,¹ M.D.

Interval Duration Between Injury and Severe Symptoms in Nonaccidental Head Trauma in Infants and Young Children*

REFERENCE: Gilliland MGF. Interval duration between injury and severe symptoms in nonaccidental head trauma in infants and young children. *J Forensic Sci* 1998;43(3):723–725.

ABSTRACT: Forensic pathologists are frequently asked to describe the interval between injury and the onset of symptoms in child abuse head injury deaths. A prospective, postmortem study examined the interval between injury and onset of symptoms in 76 head injury deaths in which this information was available. The head injury deaths were divided by mechanism of injury. The mechanisms were shake (no impact), combined shake and blunt impact, and blunt impact (no history of shaking). The interval was less than 24 hours in 80% of shakes, 71.9% of combined, and 69.2% of blunt injuries. The interval was greater than 24 hours in more than 25% of each of these latter groups and was more than 72 hours in four children. The variable intervals between injury and severe symptoms warrant circumspection in describing the interval for investigators or triers of fact. It should be noted that in all of the cases where information was supplied by someone other than the perpetrator, the child was not normal during the interval.

KEYWORDS: forensic science, child abuse, head injury, interval to symptoms

As more head injury child deaths are recognized as abusive and therefore investigated, forensic pathologists are more frequently asked to determine the time of injury. This information is used to identify or exclude possible perpetrators. Many forensic pathologists have had the experience of investigating several such deaths and finding that the interval between injury and presentation is brief. In 1995 Nashelsky and Dix found minimal data to substantiate or contradict the concept that the interval is very short (1). Howard, Bell and Uttley reported the intervals from injury to neurosurgical evaluation for 28 children with subdural hemorrhage in 1993 (2). They found two of the three children with documented shaking injury had intervals within 24 hours but the third was 72 hours. For the other 25 infants with subdural hemorrhage 13 presented in 24 hours, three in 24–72 hours, and nine after more than 72 hours. The present study was undertaken to examine the interval from injury to symptom onset.

¹East Carolina University School of Medicine, Department of Pathology and Laboratory Medicine, Brody 7 S 10, Greenville, NC.

*Presented at the National Association of Medical Examiner's Meeting, September 14, 1996, Traverse City, MI.

Received 2 May 1997; and in revised form 16 July, 15 Sept. 1997; accepted 15 Sept 1997.

Methods

A prospective, postmortem study investigated 169 child deaths and examined this interval in the 76 head injury deaths with such information available. These children with head injuries are a subset of a larger group of children reported previously (3).

Sample Selection

One hundred seventy-five of nearly 400 deaths of young children investigated at the Dallas County Medical Examiner's Office from 1982 to 1989 were studied prospectively. Case selection depended on random assignment of cases and on the prosecutor's willingness to participate in the study. Nineteen pathologists contributed one or more cases each by the end of case collection. All child deaths were equally likely to be included in the study. The deaths included diagnoses of child abuse, suspected child abuse, apparent accidental trauma, and apparent natural death. History, autopsy findings, and ocular findings were gathered and reviewed for the more general study. Children whose immediate cause of death was head injury were selected to examine the interval from injury to severe symptoms.

Symptom Onset Definition

The onset of severe symptoms was identified as the time when an external event occurred or the caretaker called for medical assistance. In these young children the symptoms were extreme: unresponsiveness, difficulty breathing, cardiorespiratory collapse. The persons identifying the symptoms were usually the caregiver calling or presenting for emergent medical attention. In some cases the identifiers were persons witnessing an external event such as a motor vehicle collision. The need for emergent medical attention was confirmed by the health care workers who evaluated the children and found them unresponsive, commonly without vital signs or with failing vital signs.

Mechanism of Injury

The deaths caused by head trauma were divided by mechanism of injury as described previously (4). The factors used in the definition included: finger marks or rib fractures; history of shaking; subdural and/or subarachnoid hemorrhage; and evidence of impact (contusions, subscalpular hemorrhage, skull fractures). The mechanisms so defined were shake (no impact with two of the following—finger marks or rib fractures, subdural or subarachnoid hemorrhage, history of shaking), combined shake and blunt impact

(impact with finger marks or rib fractures, history of shaking), and blunt impact (no finger marks or rib fractures, no history of shaking).

Results

Forty-six percent were less than one year old, 22% were between one and two years of age, and 32% were over two years of age. Forty-two were white; 24 were black; 7 were of Hispanic origin; and 3 were of other ancestry. Forty-one of the 76 children were male.

Five of the infants had exclusive shaking mechanism of injury. Both shaking and blunt mechanisms were identified in 32 infants and children. Exclusively blunt mechanisms of injury were identified in 39 of the infants and children.

The interval was less than 24 hours in all but one of the five shaken infants. It was less than 24 hours in 71.9% of 32 infants with combined, and 69.2% of 39 with blunt injuries (Table 1). The interval was greater than 24 hours in more than 25% of the groups with a blunt force component and extended more than 72 hours in four children with blunt trauma as a part of the mechanism—one with combined shake and blunt mechanisms, and three with exclusively blunt mechanism.

The 22 cases with intervals longer than 24 hours were reviewed to determine if any symptoms had been described prior to the catastrophic collapse leading to death or brief hospitalization prior to death. Ten of these children were described as lethargic or otherwise abnormal during the interval. The other twelve were in the care of the presumed perpetrator and had no credible description of their condition.

These findings are depicted graphically in Fig. 1. The columns with no volume are the graphical representation of zero.

Discussion

The interval from injury to catastrophic or near-catastrophic collapse requiring medical attention, or death is observed to be short, less than 24 hours, in almost all the babies with shaking as the exclusive mechanism of injury. This correlates with our understanding of the effect of violent shaking causing global disruption of the nervous system. Diffuse axonal injury can be demonstrated if life support is maintained. The expression "violent" is appropriate, although some find it objectionable (5,6).

In this study some of the infants with blunt force as part or the exclusive mechanism of injury presented more than 24 hours after injury. Blunt injuries are not necessarily as immediately disruptive of the nervous system and brain functioning as violent shaking. Secondary phenomena including brain swelling and edema produce symptoms. Although brain swelling and edema can develop

Interval: Injury to Presentation

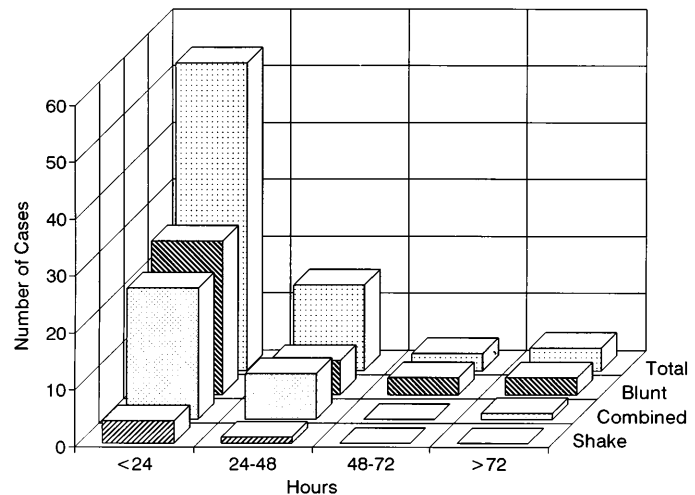


FIG. 1—The graphic display confirms the impression that most of the children will present with severe symptoms in an interval of less than 24 hours after injury.

very rapidly, in less than 24 hours, delayed onset of symptoms is not uncommon.

The proportion of children presenting beyond 24 hours was not as great as found by Howard et al. (2) in their retrospective clinical study of 28 infants and young children identified as having subdural hematoma after presenting for neurosurgical evaluation. Six of the children in their study died within a week of hospitalization, and two others 8 and 9 years later. No autopsy information was provided. Nine of the children survived neurologically intact (2). Thus, the cases of Howard et al. were not as severely injured and do not serve as a comparable group for fatally injured children.

Conclusion

Enough variability in the interval between injury and the time of severe symptoms or presentation for medical care in fatally injured children exists to warrant circumspection in describing such an interval for investigators or triers of fact. Our data indicate that the interval is brief (less than 24 hours), in almost $\frac{3}{4}$ of cases of head injury death, especially in shaking injuries. However, in more than $\frac{1}{4}$ of the cases, the interval from injury to the onset of severe symptoms is longer. In all cases in which the children were seen by an independent observer after injury, they were described as not normal.

Acknowledgments

Critical review is provided by Irma Fiordalisi MD Associate Professor of Pediatrics at East Carolina University School of Medicine and Pediatric Intensivist at Children's Hospital of Eastern North Carolina.

References

1. Nashelsky MB, Dix JD. The time interval between lethal infant shaking and onset of symptoms. *Am J Forensic Med Pathol* 1995; 16(2):154-7.

TABLE 1—Interval from injury to severe symptoms.

Interval in Hours	Mechanism of Injury			Total
	Shake	Combined	Blunt	
Less than 24	4	23	27	54
24 to 48	1	8	6	15
48 to 72	0	0	3	3
More than 72	0	1	3	4
Total	5	32	39	76

2. Howard MA, Bell MA, Uttley D. The pathophysiology of infant subdural haematomas. *Br J Neurosurg* 1993;14:201–7.
3. Gilliland MGF, Luckenbach MW, Chenier TC. Systemic and ocular findings in 169 prospectively studied child deaths: retinal hemorrhages usually mean child abuse. *Forensic Sci Int* 1994;68:117–32.
4. Gilliland MGF, Folberg R. Shaken babies—some have no impact injuries. *J Forensic Sci* 1996;41(1):114–6.
5. Taff ML, Boglioli LR, DeFelice JF. Letter: Commentary on “Controversies in shaken baby syndrome and on Gilliland MGF and Folberg R, shaken babies—some have no impact injuries.” *J Forensic Sci* 1996;41(5):729–30.
6. Gilliland MGF, Folberg R. Author’s response. *J Forensic Sci* 1996;41(5):730.

Additional information and reprint requests:
M.G.F. Gilliland M.D., Professor
East Carolina University School of Medicine
Department of Pathology and Laboratory Medicine
Brody 7 S 10
Greenville, NC 27858-4354
e-mail: mgilliland@brody.med.ecu.edu

Appendix G



James R. Gill MD is the Chief Medical Examiner for the State of Connecticut.

Author Affiliations: New Hampshire Office of Chief Medical Examiner, Concord, NH (TA), Brody School of Medicine at East Carolina University, Department of Pathology and Laboratory Medicine, Greenville, NC (MG), Harris County Institute of Forensic Sciences, Houston, TX (JL), New Mexico Office of the Medical Investigator, Albuquerque, NM (EM), Mayo Clinic - Anatomical Pathology, Rochester, MN (RR).

Contact Dr. Gill at: jgill@ocme.org

Acad Forensic Pathol
2013 4 (2): 206-213

© 2014
National Association of Medical Examiners

National Association of Medical Examiners Position Paper: Recommendations for the Postmortem Assessment of Suspected Head Trauma in Infant and Young Children

James R. Gill MD, Thomas Andrew MD, M.G.F. Gilliland MD, Jennifer Love PhD, Evan Matshes MD, R. Ross Reichard MD

ABSTRACT: The National Association of Medical Examiners convened a panel to create a position paper for recommendations for the investigation of infant deaths due to inflicted head trauma. The correct certification of both the cause and manner of death is dependent upon an evaluation of all available data including information derived from the investigation, scene, postmortem examination, and ancillary studies. This paper provides recommendations for the forensic pathologist on what constitutes the dataset to be produced during the postmortem examination of infants who have died of, or have apparently died of, inflicted head trauma. Specifically, this paper describes 1) procedures, 2) ancillary laboratory tests, and 3) forms of documentation that are important in the investigation of these deaths. The evaluation and documentation of such infant deaths involves the production of a detail oriented and thoroughly documented examination that is independently reviewable to support the multitude of inquiries that may follow from the public and the criminal justice system.

KEYWORDS: Forensic pathology, Head trauma, infant, Homicide

INTRODUCTION

The National Association of Medical Examiners (NAME) was founded with "the dual purposes of fostering the professional growth of physician death investigators and disseminating the professional and technical information vital to the continuing improvement of the medical investigation of violent, suspicious and unusual deaths" (1). One method of fulfilling this mission is the publication of autopsy standards and position papers (2-5). NAME has previously published position papers on recommendations for the investigation and certification of cocaine, heat-related, and opioid deaths (3-5).

Fatalities due to traumatic brain injury (TBI) among infants/young children, particularly homicides, are among the most challenging investigations for a forensic pathologist for many reasons: any death in this age group may elicit passionate responses even by trained professionals, the decedent may have been seen recently by a physician, a caregiver may be the perpetrator, and the wrong diagnosis may result in a wrongful

prosecution, dissolution of families, and putting other children at risk. As such, NAME convened a panel to generate recommendations for the investigation of these deaths. Complementary to a thorough death investigation, the goal of the autopsy is not only to aid in the determination of the cause and manner of death, but also to produce an independently reviewable dataset that allows the original forensic pathologist (and others who follow) the best opportunity to review primary data and potentially answer unanticipated future questions. As Moritz stated, "If a negative or positive postmortem finding is so important that it may make the difference between the freedom or imprisonment, or the life and death of someone, every attempt should be made to protect, preserve, and record it for others to see and evaluate" (6).

Autopsy protocols have been developed for cardiac, metabolic, and sudden infant deaths (7-13) to ensure consistency and comprehensive examinations that look for a broad range of diseases. Similarly, this paper's aim is to describe an autopsy protocol to facilitate a thorough, independently reviewable investigation of pediatric



1.	Photography including overall full-body external color images with close-ups of specific findings and pertinent negative findings.
2.	Anatomical (skeletal) survey with, as indicated, an internal examination of the musculoskeletal system to document and/or exclude soft tissue or bone injury.
3.	Description (color, size, location) and photographic documentation of intracranial hemorrhage (epidural [EDH], subdural [SDH], or subarachnoid [SAH]).
4.	Examination of the formalin-fixed brain, cranial dura, and spinal cord and the ability to consult with a board-certified neuropathologist.
5.	Description of the eyes and optic nerves both grossly (with photographic documentation) and microscopically. <ul style="list-style-type: none"> A. Optic nerve sheath: describe the extent and location (subdural, subarachnoid, intradural, extraocular, orbital fat) of any hemorrhage. B. Retina: describe any hemorrhage/injury including the layers involved (preretinal, retinal, subretinal), extent (few, numerous, extensive), and distribution (posterior pole, equatorial, and peripherally, including whether they abut the ora serrata). C. A neuropathologist or ophthalmic pathologist consultation may be useful.
6.	Describe injury and hemorrhage of the anterior and posterior neck.
7.	Medical record review as available for correlation with the history and autopsy findings.

head injury deaths. NAME has already published general autopsy standards that are applicable to infants/children with apparent head trauma. This traumatic brain injury protocol is not meant to replace those preexisting standards but to provide detailed techniques, procedures, and other recommendations for these investigations. Each case is unique, and this protocol is not meant as a substitute for professional judgment. The following protocol, however, may be considered practice recommendations for these investigations endorsed by the NAME Board of Directors.

Infants die suddenly and unexpectedly for a myriad of reasons that span the spectrum from natural disease to inflicted injury. Infant/childhood deaths due to TBI may have no history or external evidence of trauma; therefore, unexpected and unexplained infant/childhood deaths are thoroughly evaluated at the outset as they may be homicides or due to rare natural disease. As such, these deaths undergo a uniformly thorough investigation and documentation through the initial stages of the evaluation, documentation, and evisceration. As pathologic findings emerge during the course of the examination and prosection, the necessity and/or utility of ancillary dissections and studies will become apparent, and their selection can be tailored by the forensic pathologist.

The subsequent protocol describes a progression of relevant examinations and processes for the thorough description and documentation of pertinent findings (Table 1). Although the focus of the paper is on the central nervous system, the

importance of the detection and documentation of other disease processes and injuries involving the torso and extremities is vital.

DISCUSSION

Initial Autopsy Investigation

The initial autopsy investigation includes digital photography and full body radiologic imaging performed prior to the internal examination.

Digital Photography

Integral to the development of a dataset that facilitates independent case review is high quality, color photography. Unlike film photography, digital imaging allows the prosecutor to know immediately if the image is properly captured (14). Therefore, digital photography is the preferred method for photographic documentation for these investigations. A core list of recommended photographs is included in Table 2. In general, all external surfaces of the body are photographed with close-up photographs of specific findings. Important internal images of injury and pertinent negative findings include subscalpular views, subdural/epidural/subarachnoid hemorrhage, skull, brain (external and representative cross sections), and eyes, including the optic nerves, and subcutaneous and skeletal injury.

Plain Film Radiography

As skeletal injury may not be detected during a standard autopsy, healed and healing fractures



1	Identification photo (face)
2	Both sides of the face
3	Extended anterior neck
4	Chest, abdomen, and back
5	Genitals and perianal region
6	Arms and legs including wrists, ankles, and hands
7	Layered soft-tissue plane dissections (chest and abdomen, scalp)
8	Exposed pericranial surfaces following usual reflection of the scalp
9	Exposed cerebrocranial surfaces following pericranial membrane removal
10	Epidural, subdural, and subarachnoid hemorrhages
11	Exterior surfaces of the brain and spinal cord
12	Representative cross sections of the brain and spinal cord
13	The cervical spine and/or cervical nerve roots, as indicated
14	External and internal surfaces of the eyes

*A case number should be visible in each photograph and a reference scale included in at least one photograph of major wounds and injury to allow for 1:1 reproduction (15).

may be missed (16). Therefore, full body radiographs, which may aid in the detection of these lesions, are indicated when investigating a death with potential inflicted trauma. A radiologic skeletal survey is necessary for the evaluation of unexplained infant/childhood deaths that come to the attention of the medical examiner/coroner (ME/C) (16, 17). A pediatric skeletal survey protocol is provided in Table 3 (if board-certified radiologists are not available to define the pediatric skeletal survey). Forensic pathologists are trained to examine plain film radiographs. As some pediatric fractures may be subtle and disease may simulate fractures, consultation with a pediatric radiologist may be useful in some instances.

Advanced Radiologic Techniques

Postmortem computed tomography (CT) and magnetic resonance imaging (MRI) offer the ability to provide unobstructed and three-dimensional visualization of body lesions. Some offices have in-house scanners or have arranged access with local hospitals. While these procedures likely provide superior results for certain injuries over plain films, at the current time, the strengths and weaknesses of each modality have yet to be fully defined in the forensic autopsy setting. In addition, plain film radiographs continue to be the choice for the clinical evaluation for most fractures in children. The use of CT/

1	Three views of the skull: a. Anterior-Posterior (AP) b. Towne's (30° angle view of the mandibular condyles and the midfacial skeleton) c. Lateral
2	Two views of the cervical spine: a. AP b. Lateral
3	Two views of the trunk / torso: a. AP b. Lateral
4	Two views of the ribs: a. LPO (Left posterior oblique) b. RPO (Right posterior oblique)
5	Four views of the upper extremities: a. Left upper extremity b. Right upper extremity c. Left hand d. Right hand
6	Four views of the lower extremities: a. Left lower extremity b. Right lower extremity c. Left foot d. Right foot

MRI remains at the discretion of the forensic pathologist and subject to the availability of facilities and funding for such modalities (18). While postmortem CT/MRI currently may be a useful adjunct, the external and internal autopsy exami-



nation remains the best method for the postmortem diagnosis of injury (19). Since some infants or children who ultimately die from injuries may have a survival period in the hospital, antemortem advanced imaging often has been obtained. Admission imaging and other hospital CT and MR scans may provide valuable information and should be sought and reviewed.

External and Internal Examination

Depending on the age of the infant/young child, certain body measurements (e.g., head circumference) in addition to length and weight may be useful. A search for trace evidence and sexual assault evidence collection, if indicated, is done prior to washing the body to better visualize injuries. An external and internal examination is performed per the NAME Forensic Autopsy Performance Standards which also are followed when describing an injury (e.g., type, location size, shape, pattern) (15).

Specialized Autopsy Investigations When Suspicious Findings are Encountered

As the autopsy progresses, findings suspicious for inflicted traumatic brain injury (ITBI) may be detected. If so, the following specific examinations and documentation of particular findings are recommended. These are described by body region.

Head

Scalp/subscalpular: the number, location, and size of scalpular and subscalpular hemorrhages are best documented through a combination of the photograph(s) and narrative description(s). Correlation should be made between sites of hemorrhage and medical intervention (i.e., prior surgery). Other discrete areas of hemorrhage may occur, such as along cranial sutures from diastasis due to marked brain swelling. The documentation should convey the information necessary for users of the autopsy report to determine when hemorrhage is either due to a secondary process or a direct result of trauma. If there is concern for facial injury not apparent externally, a formal face dissection is possible (20). Detection of otherwise unknown or underappreciated impact sites of the face may change the context of the infant death from one of non-impact to one with demonstrable impact. Intraoral examinations may reveal frenulum, inner cheek, and tongue injuries. Shaving of hair allows for better evaluation and documentation of scalp injuries. Careful examination of the ears includes looking inside and behind the ears.

Skull: documentation of the location, dimension, type (e.g., linear, depressed, comminuted, diastatic) of fracture(s) is achieved through the narrative report, digital photography, and radiologic images; some forensic pathologists may choose to supplement their reports with diagrams. Skull fractures may be subtle in the young pediatric population and therefore removal of the pericranial membranous soft tissues along with the cranial dura allow them to be better identified and examined in detail. Microscopic sections help confirm and document gross findings and may be useful for assessment of the stage of healing.

Hemorrhage: the type [epidural (EDH), subdural (SDH), or subarachnoid (SAH)], location, size, color, and adherence are documented through appropriate use of narrative description and digital photography. The size of SDH and EDH, for example, may be documented by volume, weight, or three-dimensional measurements. The effects of the hemorrhage on the brain (e.g., compression, herniation, shift) are noted in the autopsy report. Microscopic sections help confirm and document gross findings and may be useful for assessment of the stage of healing (21, 22).

Brain: for optimal results, the brain and cranial dura should be fixed in formalin (usually for a minimum of 10-14 days). Forensic pathologists are trained to diagnose and describe traumatic brain injuries. As some neuropathologic diseases may be subtle or mimic trauma, consultation with a board-certified neuropathologist may be warranted. Express mail delivery services, gross photography, tissue retention, and microscopic slides allow for consultation even after the initial brain dissection. Pertinent positive and negative gross and microscopic findings include: hypoxia-ischemia, contusion, contusion hematomas, diffuse axonal injury, presence/absence of brain swelling and herniation (type and extent), intracerebral hemorrhage (location and extent), vascular malformations, congenital anomalies, and other focal lesions. Examination of the cerebral dura includes description of any pathology (e.g., subdural blood, surgical defects) and evaluation (opening) of the sinuses (e.g., thrombosis). Microscopic examination of the dural sinus may help distinguish thrombus from postmortem coagulated blood.

Microscopic sections for suspected inflicted traumatic brain injury include evaluations of pathology identified grossly and sampling that allows assessment of possible natural disease processes, hypoxic-ischemic brain injury, and traumatic axonal injury. Hypoxic-ischemic brain injury in infants/children has a characteristic distribution.



of injury, and those regions should be included in the evaluation in addition to regions typically involved in adults. A list of locations for histologic sampling is included in Table 4. Diagnosis of diffuse traumatic axonal injury (dTAI) requires traumatic axonal injury in multiple locations including the corpus callosum, cerebral hemispheric white matter, and brainstem. Infants have been reported to have traumatic axonal injury isolated within the brainstem (23). Histologic sampling for dTAI in infants and children includes routine hematoxylin and eosin (H&E) sections. If available, examination with amyloid precursor protein (APP) immunostain may be useful in select instances. A list of locations for histologic sampling for dTAI is included in Table 5. Limited sampling has been demonstrated to preclude the diagnosis of dTAI; therefore, if traumatic axonal injury is present but insufficient to diagnose dTAI, additional (bilateral) histologic sections should be evaluated. Assessment of APP immunostaining patterns due to trauma and a variety of other processes has previously been reported (23-25). Microscopic evaluation for natural disease processes includes examination of neocortex, deep gray structures, brainstem, and cerebellum. Histologic sampling and use of special stains, however, may vary depending on the clinical history and gross pathology identified at autopsy. Marked non-perfused brain changes (so-called "respirator brains"), for example, may dramatically affect the gross evaluation and hinder histologic sampling and diagnosis.

Histologic evaluation of the cerebral dura is useful to assess gross pathology and evaluate for more subtle processes. Evaluation for intracellular iron deposition may facilitate assessment of the stage of healing in SDH and EDH. The interface between sub/epidural blood and ongoing tissue healing is the best site for histologic evaluation. Although there are criteria for the stages of healing of a SDH, many variables affect this process (e.g., size of hematoma, source of blood, age of the individual), resulting in a somewhat predictable but potentially variable course (22). Retention of representative cortical regions, deep gray structures, brainstem, and cerebellum may prove useful. In addition, samples from contralateral white matter regions (e.g., parasagittal white, posterior limb of internal capsule) may be necessary to diagnose or exclude dTAI.

Spinal cord: the spinal cord is examined at all three levels (cervical, thoracic, and lumbar). Some have recommended removal of the brain with the spinal cord still attached (26), but it is unclear if this technically difficult dissection yields any diagnostic value.

1	Border zone (ie, frontoparietal region)
2	Deep gray structures (basal ganglia/thalamus)
3	Hippocampus (including subiculum)
4	Midbrain (inferior colliculus)
5	Pons
6	Cerebellum

1	Posterior corpus callosum (near splenium)
2	Parasagittal white matter
3	Posterior limb of the internal capsule
4	Midbrain, pons, and medulla

Ocular examination: monocular indirect ophthalmoscopy is a noninvasive and nondestructive technique to view the retina prior to autopsy and the equipment is less expensive than that needed for ophthalmic endoscopy (27), however, it does require additional equipment and training. A gross dissection and microscopic examination of the eyes also is possible at autopsy. The interpretation of retinal hemorrhages is beyond the scope of this paper. Forensic pathologists, however, have a duty not only to diagnose and interpret, but also to document and preserve findings. As an autopsy is often the best and sometimes only chance to diagnose, confirm, or exclude certain findings, pathologists, as the independent documenters of facts, are best to err on the side of documentation. A recent prospective clinical study indicated that the assessment of retinal hemorrhages may be useful as a predictor of iTBI (28). Pending the publication of additional research, at a minimum, the ocular examination serves as a quality assurance measure that may confirm or refute clinically diagnosed retinal hemorrhages. Therefore, the removal and examination of the eyes in instances of inflicted or suspected-inflicted traumatic brain injury is recommended.

A technique for removal and examination of the eyes has previously been published (29). After removal, the eyes and periocular soft tissues are fixed in formalin prior to microscopic examination. For optic nerve sheath hemorrhage, the location (subdural, subarachnoid, intradural, extraocular, orbital fat) is documented in the autopsy report. For retinal hemorrhages, the involved lay-



ers (preretinal, retinal, subretinal), extent (few, numerous, extensive), and distribution (posterior pole, equatorial, and peripherally, including whether they about the ora serrata) are described. Special stains, such as Prussian blue to identify hemosiderin, may be useful in some instances. Collection of vitreous will disturb the retina and should be postponed until adequate examination of the retina, the optic nerve, and brain is done and the value of the vitreous analysis for the specific case is considered. Photography with appropriate lighting can aid in the documentation of ocular findings.

Neck

Infants and children who die of inflicted injuries may have injuries of the anterior and posterior neck. In addition to the standard anterior neck dissection, a posterior neck dissection also may reveal internal injury. Injuries of the neck have been proposed to explain potential mechanisms of death and techniques have been described for the examination of the anterior and posterior neck intrinsic spine structures/nerve roots (30-37). If the prosecutor is concerned for intrinsic structural spinal/nerve root injury, then these structures may be evaluated by various methods. One method is an *en bloc* dissection of the cervical osseous and neural structures with formalin fixation (37). Another is an *in situ* method that removes the spinal cord and attached ganglia without the surrounding bone and soft tissues (38). In this technique, the laminae are cut and the spinous processes removed. The lateral aspects of the neural arches are then removed by cutting the articulating facets and pedicles of the vertebrae. The freed sections of bone are removed and the spinal cord is removed with the ganglia attached.

Trunk and Upper and Lower Extremities

The NAME autopsy performance standards include procedures and descriptions for trunk injuries. A subcutaneous examination of the arms, legs, and back and buttocks may improve detection of occult subcutaneous and deep hemorrhage (39).

Of particular importance in pediatric ITBI are the identification and description of rib and other skeletal fractures. In addition to radiographs, an *in situ* skeletal examination may be useful to further document or exclude injury. An *in situ* examination may include exposure of the shaft and epiphyseal cartilages of the ribs, clavicles, long bones, and scapulae (40). Traumatized or abnormal structures may be removed for additional analysis including gross (i.e., dry bone) or histologic examination (41-43). When possible, a

description of the stage of healing (i.e., bone callus formation) is included in the autopsy report. Forensic pathologists are trained to examine plain film radiographs and bones. As some osseous findings may be unusual normal anatomic variants, consulting with a board-certified forensic anthropologist may be useful. Other useful techniques and examinations include: stripping the parietal pleural lining to better visualize rib fractures, resecting the spinal column with the medial ribs for further evaluation for posterior rib fractures, and histologic sections of cutaneous/subcutaneous injuries (Prussian blue can identify hemosiderin).

Ancillary Studies

Depending upon the circumstances and autopsy findings, ancillary studies for infectious (e.g., viral, bacterial cultures), hereditary, metabolic, or thrombophilic diseases may be indicated.

Records

Review of the medical records including antemortem CT/MRI reports should be done as they may provide relevant clinical information. Review of reports generated by other agencies such as child protective services and law enforcement investigation findings also may guide the forensic pathologist's investigation and therefore these should be requested.

CONCLUSION

The investigative value of each of the described studies is often unknown at the onset of the autopsy. Many procedures performed during an autopsy create irreversible changes to tissues and thus proactive documentation is critical. Ultimately, the forensic pathologist must use medical judgment on how to conduct each autopsy examination but would be wise to heed the admonition that "I would rather explain why I did an examination than why I did not." In these instances, it may be stated that the examination was done in accordance with recommendations endorsed by the Board of Directors of the National Association of Medical Examiners.

DISCLOSURES

The opinions and conclusions of this paper have been reviewed and approved by the National Association of Medical Examiners Board of Directors and as such are endorsed by NAME. These opinions and positions are based on a consensus of the current literature, knowledge, and prevailing theories on this topic. As Scientific knowledge and experience grow, NAME reserves the



right to revise or update these opinions. The process by which NAME position papers are initiated, written, reviewed, and approved is publicly available at <https://netforum.avectra.com/temp/ClientImages/NAME/2c26a527-f992-4f70-9d03-7941bff5319d.pdf>. All scientific position papers endorsed by the National Association of Medical Examiners automatically expire five years after publication unless reaffirmed, revised, or retired at or before that time. This work is a product of NAME and as such, was not subjected to *Academic Forensic Pathology Journal* editorial review.

The editors and publication staff do not report any relevant conflicts of interest.

REFERENCES

- National Association of Medical Examiners [Internet]. Marceline (MO): The Association; c2005-2014 [cited 2014 Jan 9]. Available from: <https://netforum.avectra.com/web/DynamicPage.aspx?WebCode=LoginRequired&Site=NAME>
- Peterson GF, Clark SC. Forensic autopsy performance standards. *Am J Forensic Med Pathol*. 2006 Sep; 27(3):200-25.
- Stephens BG, Jentzen JM, Karch S, et al. National Association of Medical Examiners position paper on the certification of cocaine-related deaths. *Am J Forensic Med Pathol*. 2004 Mar; 25(1):11-3.
- Donoghue ER, Graham MA, Jentzen JM, et al. Criteria for the diagnosis of heat-related deaths: National Association of Medical Examiners. Position paper. National Association of Medical Examiners Ad Hoc Committee on the Definition of Heat-Related Fatalities. *Am J Forensic Med Pathol*. 1997 Mar; 18(1):11-4.
- Davis GG, National Association of Medical Examiners and American College of Medical Toxicology Expert Panel on Evaluating and Reporting Opioid Deaths. National Association of Medical Examiners position paper: recommendations for the investigation, diagnosis, and certification of deaths related to opioid drugs. *Acad Forensic Pathol*. 2013 Mar; 3(1):77-83.
- Moritz AR. Classical mistakes in forensic pathology. *Am J Clin Pathol*. 1956 Dec; 26(12):1383-97.
- Basso C, Burke M, Fornes P, et al. Guidelines for autopsy investigation of sudden cardiac death. *Virchows Arch*. 2008 Jan; 452(1):11-8.
- Lee AH, Gallagher PJ. Post-mortem examination after cardiac surgery. *Histopathology*. 1998 Nov; 33(5):399-405.
- Rinaldo P, Yoon HR, Yu C, et al. Sudden and unexpected neonatal death: a protocol for the postmortem diagnosis of fatty acid oxidation disorders. *Semin Perinatol*. 1999 Apr; 23(2):204-10.
- Byard RW, Krous H. Sudden infant death syndrome problems, progress, and possibilities. London: Arnold; c2001. Appendix I, International standardized autopsy protocol for sudden unexpected infant death; p. 319-33.
- Sadler DW. The value of a thorough protocol in the investigation of sudden infant deaths. *J Clin Pathol*. 1998 Sep; 51(9):689-94.
- Bove KE. Practice guidelines for autopsy pathology: the perinatal and pediatric autopsy. Autopsy Committee of the College of American Pathologists. *Arch Pathol Lab Med*. 1997 Apr; 121(4):368-76.
- Ackerman MJ, Tester DJ, Driscoll DJ. Molecular autopsy of sudden unexplained death in the young. *Am J Forensic Med Pathol*. 2001 Jun; 22(2):105-11.
- Oliver WR. Considerations for gross autopsy photography. *Acad Forensic Pathol*. 2011 Jul; 1(1):52-81.
- Peterson GF, Clark SC. Forensic autopsy performance standards. Marceline (MO): National Association of Medical Examiners; 2006. 27 p.
- McGraw EP, Pless JE, Pennington DJ, White SJ. Post-mortem radiography after unexpected death in neonates, infants, and children: should imaging be routine? *AJR Am J Roentgenol*. 2002 Jun; 178(6):1517-21.
- Adamsbaum C, Mejean N, Merzoug V, Roy-Salmon C. How to explore and report children with suspected non-accidental trauma. *Pediatr Radiol*. 2010 Jun; 40(6):932-8.
- Noite K, Mfady G, Zimwala R, et al. Postmortem X-ray computed tomography (CT) and forensic autopsy: a review of the utility, the challenges, and the future implications. *Acad Forensic Pathol*. 2011 Jul; 1(1):4-50.
- Molina DK, Nichols JJ, Dimairo VI. The sensitivity of computed tomography (CT) scans in detecting trauma: are CT scans reliable enough for courtroom testimony? *J Trauma*. 2007 Sep; 63(3):625-9.
- Collins K. Special autopsy dissections. Northfield (IL): College of American Pathologists; 2010. 50 p.
- Munro D, Merritt H. Surgical pathology of subdural hematoma. *Arch Neurol Psych*. 1934; 35:65-78.
- Hirsch CS, Ambrustmayer V, Spitz and Fisher's medicolegal investigation of death. 4th ed. Springfield (IL): Charles C Thomas; c2006. Chapter 19. Part 1, Trauma of the nervous system; p. 994-1077.
- Reichard RR, White CL 3rd, Hladik CL, Dolinak D. Beta-amyloid precursor protein staining of nonaccidental central nervous system injury in pediatric autopsies. *J Neurotrauma*. 2003 Apr; 20(4):347-55.
- Geddes JF, Vowles GH, Beer TW, Ellison DW. The diagnosis of diffuse axonal injury: implications for forensic practice. *Neuropathol Appl Neurobiol*. 1997 Aug; 23(4):339-47.
- Reichard RR, White CL 3rd, Hladik CL, Dolinak D. Beta-amyloid precursor protein staining in nonaccidental pediatric medicolegal autopsies. *J Neuropathol Exp Neurol*. 2003 Mar; 62(3):237-47.
- Judkins AR, Hood IG, Mirchandani HG, Rorke LB. Technical communication: rationale and technique for examination of nervous system in suspected infant victims of abuse. *Am J Forensic Med Pathol*. 2004 Mar; 25(1):29-32.
- Lantz PE, Adams GG. Postmortem monocular indirect ophthalmoscopy. *J Forensic Sci*. 2005 Nov; 50(6):1450-2.
- Minns RA, Jones PA, Tandon A, et al. Prediction of inflicted brain injury in infants and children using retinal imaging. *Pediatrics*. 2012 Nov; 130(5):e1227-34.
- Gilliland MG, Levin AV, Enzenauer RW, et al. Guidelines for postmortem protocol for ocular investigation of sudden unexplained infant death and suspected physical child abuse. *Am J Forensic Med Pathol*. 2007 Dec; 28(4):323-9.
- Matshes EW, Evans RM, Finckard JK, et al. Shaken infants die of neck trauma, not brain trauma. *Acad Forensic Pathol*. 2011 Jul; 1(1):82-91.

- 31) Geddes JF, Vowles GH, Hackshaw AK, et al. Neuropathology of inflicted head injury in children. II. Microscopic brain injury in infants. *Brain*. 2001 Jul; 124(Pt 7):1299-306.
- 32) Hadley MN, Sonntag VK, Reigate HL, Murphy A. The infant whiplash-shake injury syndrome: a clinical and pathological study. *Neurosurgery*. 1989 Apr; 24(4): 536-40.
- 33) Shannon P, Smith CR, Deck J, et al. Axonal injury and the neuropathology of shaken baby syndrome. *Acta Neuropathol*. 1998 Jun; 95(6):625-31.
- 34) Geddes JF, Hackshaw AK, Vowles GH, et al. Neuropathology of inflicted head injury in children. I. Patterns of brain damage. *Brain*. 2001 Jul; 124(Pt 7):1290-8.
- 35) Adams VI. Autopsy technique for neck examination. I. Anterior and lateral compartments and tongue. *Pathol Annu*. 1990; 25 Pt 2:331-49.
- 36) Adams VI. Autopsy technique for neck examination. II. Vertebral column and posterior compartment. *Pathol Annu*. 1991; 26 Pt 1:211-26.
- 37) Matshes EW, Joseph J. Pathologic evaluation of the cervical spine following surgical and chiropractic interventions. *J Forensic Sci*. 2012 Jan; 57(1):113-9.
- 38) Peterson JEG, Love JC, Wolf DA, et al. Proceedings of the American Academy of Forensic Sciences 66th annual scientific meeting. Colorado Springs: American Academy of Forensic Sciences; c2014. G51, Ganglia and nerve root hemorrhage in cases of pediatric blunt head injury; p. 346.
- 39) Spitz W, Spitz D. Investigation of deaths in childhood. In: Spitz W, Spitz D, editors. *Spitz and Fisher's medicolegal investigation of death*. 4th ed. Springfield (IL): Charles C Thomas; 2006. p. 1325.
- 40) Love JC, Sanchez LA. Recognition of skeletal fractures in infants: an autopsy technique. *J Forensic Sci*. 2009 Nov; 54(6):1443-6.
- 41) Love JC, Derrick SM, Wiersma JW. *Skeletal atlas of child abuse*. New York: Humana Press; 2011. 136 p.
- 42) Kleinman FK, Marks SC, Blackburne B. The metaphyseal lesion in abused infants: a radiologic-histopathologic study. *AJR Am J Roentgenol*. 1986 May; 146(5):895-905.
- 43) Lonergan GI, Baker AM, Morey MK, Boos SC. From the archives of the AFIP. Child abuse: radiologic-pathologic correlation. *Radiographics*. 2003 Jul-Aug; 23(4):811-45.



Appendix H

Potential Experts and Areas of Expertise

Dr. Ronald H Uscinski, neurosurgeon
(301) 656-8590
(703) 207-8512

For more information: <http://health.usnews.com/doctors/ronald-uscinski-11295>

Dr. Patrick Barnes, pediatric neuroradiologist
Stanford University School of Medicine
pbarnes@stanford.edu

(650) 723-8087

(650) 497-8601

(650) 497-8376

For more information: <https://med.stanford.edu/profiles/patrick-barnes>

Dr. Julie Mack, radiologist
Penn State Medical Center
(717) 531-8991

juliemack@comcast.net

For more information: <http://health.usnews.com/doctors/julie-mack-126045>

Dr. Michael Krasnokutsky, radiologist
(360) 918-2667

mkrasn@gmail.com

For more information: <http://health.usnews.com/doctors/michael-krasnokutsky-787167>

Dr. Shaku Teas, forensic pathologist
(708) 366-4389

shakuteas@gmail.com

Dr. Douglas Smith, retired medical doctor
(734) 369-3386

Douglas2k@comcast.net

Dr. Steve Rundell, biomechanical engineer
Explico Engineering
(248) 563-7151

steve@explico.com

For more information: <https://www.explico.com/experts/steve-rundell>

Dr. Chris Van Ee, biomechanical engineer
Design Research Engineering
(248) 668-5533

chrisv@dreng.com

For more information:

<https://static1.squarespace.com/static/57a909ef20099e1140916521/t/5af38eac03ce64b99ddd8b04/1525911213048/Van+Ee+CV+2017+03.pdf>

Other Potential Resources

University of Michigan Innocence Clinic

Director and staff attorneys very familiar with these issues (734) 763-9353, or contact staff attorney Imran Syed directly at (734) 763-0577, or by e-mail at galad@umich.edu

Attorney Katherine H. Judson

Former SBS/AHT Litigation Coordinator for the Wisconsin Innocence Project and current Director of Center for Integrity in Forensic Sciences

(608) 736-2437

Attorney Heather Kirkwood

Background in civil litigation, but now consults especially regarding appropriate experts for these cases

Tel. (206) 286-9138

Cell (206) 372-8230

hkirkwood2@comcast.net

Further Reading/Watching

Washington Post Piece:

<http://www.washingtonpost.com/graphics/investigations/shaken-baby-syndrome/>

New York Times Piece:

<https://www.nytimes.com/2011/02/06/magazine/06baby-t.html>

Presentations from a symposium on child abuse evidence hosted at University of Michigan:

<http://sites.lsa.umich.edu/npcae/presentations/>

Scenes of a Crime, documentary film about an abusive head trauma murder case involving a false confession

More information at: <http://scenesofacrime.com/>

Trailer at: <https://vimeo.com/37160802>

Appendix I

Identifying, Investigating, and Litigating Cases Involving Abusive Head Trauma

January 28, 2021

CHECKLIST

- Identify whether your case involves allegations of shaking, a diagnosis of Shaken Baby Syndrome or Abusive Head Trauma, or a conclusion that head injury was necessarily non-accidental and intentionally inflicted
 - May require discovery
- Discovery
 - You will need to request all of the regular discovery materials, but you might also need:
 - Medical records from the instant case
 - Child's medical records from birth
 - Mother's medical records from pregnancy
 - Autopsy report
 - Including all photos taken during all parts of the autopsy and all autopsy slides
 - CPS/DHS records
 - Summaries and/or reports from any expert the other party plans to call
 - You may need to file motions or more specific requests than in a typical case
- Investigation
 - Familiarize yourself with the medical controversy involving Shaken Baby Syndrome and Abusive Head Trauma
 - Review journal articles
 - Review all discovery materials, interview client, witnesses, etc.
 - Consult with experts or attorneys who specialize in these cases
 - Meet with potential experts in the relevant fields, which may include:
 - Forensic pathology
 - Neuropathology
 - Radiology
 - Pediatrics
 - Biomechanics
 - Childhood development
 - Retain necessary experts and, if appropriate, file a request for funds to pay your expert(s)
- Prepare your defense
 - Consider *Daubert* challenges
 - Consider filing pretrial motions related to experts qualifications, limiting their opinions to their fields of expertise
 - Prepare to cross-examine experts, possibly using journal articles
 - Present expert testimony to support your defense
- **Make sure to create a record every step of the way**