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SUPERIOR COURT OF WASHINGTON FOR SNOHOMISH COUNTY

<p>STATE OF WASHINGTON, Plaintiff, vs. MICHAEL J. MORRIS, Defendant.</p>	<p>No. 09-1-01071-9 RESPONSE TO STATE'S MOTION TO TRANSFER DEFENDANT'S CrR 7.8 MOTION TO THE COURT OF APPEALS 73278-1</p>
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I. INTRODUCTION

In its Motion to Transfer, the State argues Mr. Morris had not made a substantial showing that his conviction was obtained in violation of the right to effective assistance of counsel and to a fair trial. Mr. Morris had made that showing.

"[A] claim of shaken baby syndrome is more an article of faith than a proposition of science." *Del Prete v. Thompson*, 10 F.Supp.2d 907, 957 fn. 10 (2014). That claim was made here, albeit by the name of abusive head trauma (AHT). As the record establishes, Mr. Morris' conviction was obtained through the use of unreliable and misleading expert opinion purporting to diagnose abuse as the cause of a child's injuries. Per the expert's diagnosis, Mr. Morris

RESPONSE TO STATE'S MOTION TO TRANSFER CrR 7.8
MOTION FOR RELIEF FROM JUDGEMENT - I

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229

abusively shook his daughter, hard enough to cause serious intracranial and retinal bleeding without leaving a mark on her body.

The causation opinion rested on the application of a hypothesis which has weak scientific support and which does not square with biomechanical principles, which rendered applying it here to “diagnose” causation unreliable. As such the opinion was inadmissible under Washington’s rules of evidence. Cloaked in the aura of a medical diagnosis and bolstered by misleading testimony on the supposedly firm reasons the diagnosis could confidently be made here, the unreliable expert opinion formed the entire basis of the State’s case against Mr. Morris. Counsel’s failure to keep out the inadmissible evidence resulted in a fundamentally unfair trial and confidence in the outcome is unwarranted. Additionally, the State’s introduction of unreliable evidence to convict where the limits of the diagnosis are known is equally problematic. Obtained with misleading and unreliable testimony the conviction violates due process.

II. ARGUMENT

A. Constitutional Errors Violating Mr. Morris’ Right to A Fair Trial Qualify for Relief Under CrR 7.8(b)(5).

Under CrR 7.8(b)(5), a court may grant relief from judgment for “any other reason justifying relief from the operation of the judgment.” Relief under this section is limited to extraordinary circumstances not covered by any other section of the rule. *State v. Brand*, 120 Wn.2d 365, 369, 842 P.2d 470 (1992). A defendant who is denied the constitutional right to effective assistance of counsel is entitled to relief under CrR 7.8(b)(5). *State v. Cervantes*, 100 Wn. App. 282 P.3d 98 (2012) (citing *State v. Martinez* 161 Wn. App. 436, 440-441, 253 P.3d 445 (2011)).

Mr. Morris does not need to show, as the State claims, a sudden, dramatic shift in the scientific community in order to be entitled to relief. Constitutionally ineffective trial counsel is a substantial irregularity in the proceedings, as are State violations of the defendant's due process right to a fair trial. The Court can grant relief based upon finding his constitutional rights to a fair trial and effective counsel were violated. Here, Mr. Morris has shown his constitutional rights were violated.

B. Mr. Morris Has Made a Substantial Showing that Trial Counsel was Ineffective in Failing to Challenge the Wholesale Admission of Misleading, Speculative and Unreliable Expert Testimony that Should Have Been Excluded or Limited

i. Dr. Feldman Testified A.M.'s Medical Findings Were Caused by Shaking

The State's argument that Dr. Feldman did not testify as to how A.M. was injured misrepresents the testimony. State Motion at 19. Dr. Feldman testified her injuries were the result of abusive head trauma (AHT). 6/3/11 RP 13. He said he did not know the exact mechanism since he was not there. 6/3/11 RP 16-17. However, Dr. Feldman explained it was abusive trauma by discussing, at length, how forceful shaking causes A.M.'s exact injuries—retinal hemorrhaging (RH) and subdural hemorrhage (SDH). Despite the caveat about not being there, the clear message was that Dr. Feldman determined, based on the clinical picture, that A.M. was abusively shaken by her last caregiver. This is exactly what the State argued in closing: "What [Dr. Feldman] said is violent shaking. Possibly joined with soft impact on a soft surface." 6/10/11 RP 772.

ii. The Reliability of Dr. Feldman's Causation Opinion Must be Assessed Under ER 702

Contrary to the State's argument, considering reliability when analyzing admissibility is not imposing a *Daubert* test. State Motion at 21. The Washington Supreme Court has explained that ER 702 requires "an assessment of admissibility of scientific evidence under the

helpfulness standard contained in the rule, thus providing in this jurisdiction the “best of both worlds.” *State v. Copeland*, 130 Wn.2d 244, 259-60, 922 P.2d 1304, 1314 (1996). *See also Reese v. Stroh*, 128 Wn.2d 300, 307-08, 907 P.2d 282, 286 (1995) (holding that where the objection the expert testimony is the application of an accepted theory or methodology to a particular medical condition, admissibility is weighed under the general reliability standards of ER 702 and ER 703).

ER 702 has a significant role in admissibility of scientific evidence aside from *Frye*.¹ *Copeland*, 130 Wn.2d at 260-261. Further, Washington’s evidence rules are fully capable of addressing the reliability of causation opinion. *Reese*, 128 Wn.2d at 308. When an expert’s errors in applying a methodology render the testimony unreliable, a trial court may use the rules of evidence, including ER 702, to exclude the testimony. *Lakey v. Puget Sound Energy, Inc.* 176 Wn.2d 909, 920, 296 P.3d 860 (2013).

For example, in *Lakey*, plaintiffs in a civil lawsuit proffered expert testimony that exposure to electromagnetic fields (EMFs) was a possible cause of various serious diseases. *See Id.* at 915. The expert reached his conclusions after doing a literature review but acknowledged he discounted studies and data that showed no EMF-disease link and did not consider toxicological studies. *Id.* at 916. The Court held the expert failed to follow proper methodology, rendering his conclusions unreliable and therefore inadmissible. *Id.* at 920. Additionally, where he also selectively sampled data within one of the studies, ignoring the larger pool of data within the study that showed no link, the expert’s “treatment of [the] data created an improper

¹ This is not to say that reliability cannot be assessed under *Frye* as well as ER 702 or that application of one excludes the other. They are related and both ultimately concerned with the reliability of the evidence. *See State v. Black*, 109 Wn.2d 336 (1987) (excluding under *Frye* evidence of rape trauma syndrome as means of proving rape because the syndrome was not a scientifically reliable means of proving a rape occurred).

false impression about what the study actually showed.” *Id.* at 921. His conclusions were unreliable and therefore not helpful to the trier of fact. *Id.*

iii. Dr. Feldman’s Causation Opinion is Unreliable and Inadmissible Under ER 702

Dr. Feldman was tasked with diagnosing what caused A.M’s injuries; he considered potential causes and ruled each one out until he landed at the cause, AHT. Logically, if the landed-upon cause is not actually capable of causing the injuries, the process is flawed from inception. The mere fact of rendering a “diagnosis” does not render the opinion helpful, as required under ER 702, if the diagnosis was made by applying unscientific data to the case-at-hand, while also ignoring data at odds with the diagnosis. As in *Lakey*, Dr. Feldman’s methods render his opinion unreliable and misleading. Unlike *Lakey*, Dr. Feldman did not just render an opinion as to a possible cause, but testified that abusive trauma was *the* cause.

The State asserts generally that AHT is a medically valid diagnosis. State Motion 16-17. For support, the State cites extensively to Dr. Sandeep Narang, an SBS/AHT advocate. According to the State, Dr. Feldman simply applied the recognized differential diagnosis method to arrive at the clinically valid diagnosis of AHT. *Id.* at 20-21; 27. The State also claims that the diagnosis was helpful because “an analysis of the medical findings was certainly beyond the understanding of the average layperson.” State Motion at 19.

Other than claiming AHT is generally accepted by others, the State’s analysis does not address whether the differential diagnosis method was reliably applied. The flaw in the State’s argument is that Dr. Feldman’s diagnosis was unreliably reached, for the reasons set forth here.

a. *Dr. Feldman relied on an unproved hypothesis*

Shaking as an explanation for SDH and RH began as a hypothesis, first proposed by Drs. Caffey and Guthkelch, who hypothesized, based on a 1968 study by Dr. Ommaya, that

infants might sustain whiplash-type injuries, including SDH and RH, from being violently shaken. *See* App. B (Guthkelch 2012); *See also* Ronald Uscinski, *Shaken Baby Syndrome: An Odyssey*, 46 *Neurol. Med. Chir.* 57 (2006) (providing a historical account of the hypothesis and research) (App. DD).² Notwithstanding that Dr. Guthkelch expressly was offering merely a hypothesis about *one* possible cause of subdural hematoma in infants, and that Dr. Caffey reached his conclusions based on evidence that even he acknowledged was “meager” and “manifestly incomplete,” the SBS hypothesis rapidly gained “acceptance and enormously widespread popularity,” with no real investigation or even question as to its scientific validity.” *Id.* (emphasis added).

Later, Dr. Ommaya himself noted the limits of his initial research, clarifying that it involved monkeys, not infants, and the monkeys had not been shaken, but instead been strapped in carts and impacted from the rear in an effort to gauge human thresholds to whiplash injury in car accidents. *See* App. D (Ommaya 2002 at 221-222). Dr. Ommaya further explained the study actually showed that SDH was *not* easily caused by whiplash and suggested it was misinterpreted by Drs. Guthkelch and Caffey in citing to it as scientific support for SBS. *Id.* (“[O]ur experimental results were referenced as providing the experimental basis of the ‘shaken baby syndrome’ (SBS) by Caffey, Guthkelch and others by analogy not realizing that the energy level of acceleration in our work related to speeds at motor vehicle crashes at 30 mph.”).

More than three decades later, the SBS/AHT hypothesis that shaking causes subdural bleeding and/or retinal hemorrhaging remains just that, a hypothesis. In 2012, Dr. Guthkelch had this to say about it:

² References to the appendices in Mr. Morris CrR7.8(b) brief follow the same format used in the brief. Appendices provided in this response are sequentially labeled starting with Appendix DD.

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."

App. B at 207.³ Dr. Feldman's opinion was far from an acknowledgement of not knowing. Instead, he presented this hypothesis as the basis of a firm medico-legal diagnosis, one sufficient to identify the perpetrator and his state of mind.

b. Dr. Feldman relied on a hypothesis at odds with biomechanics

The SBS/AHT hypothesis is grounded in biomechanics, both because it was rooted in the 1968 Ommaya study and because it describes a potential biomechanical phenomenon. *See* Uscinski, 46 *Neurol. Med. Chir.* at 58 (explaining the 1968 Ommaya biomechanics study provided the "sole source of experimental data from which the initial hypothetical shaking mechanism was drawn."). As such, the science of biomechanics is not only relevant, it is a critical part of the quest to evaluate whether shaking does in fact cause RH and SDH.

Yet, Dr. Feldman ignored the research, and specifically the research showing the level of force generated by shaking does not support shaking as a mechanism, even with impact on a soft surface. The State responds in a similar manner, arguing biomechanics does not undermine

³ Dr. Guthkelch further suggests that the primary findings be defined in terms of their medical features, which "would allow us to investigate causation without appearing to assume that we already know the answer." App. B at 202. He suggests that inferring abuse (and criminal intent) from the medical findings alone takes the hypothesis too far. *Id.* at 202-203. He suggests that given the complexity of the neuropathology of the infant brain, "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." *Id.* at 204. He suggests that the issue of what is supported by reliable scientific evidence "should be reviewed by individuals who . . . have a firm grounding in basic scientific principles, including the difference between hypotheses and evidence." *Id.* at 208.

the “wealth of literature and clinical experience that does accept shaking or shaking with impact as a mechanism for abusive head trauma.” State’s Motion at 24 (emphasis added).

The State does not dispute the research but wants to overlook it. Even if overlooking on-point research were acceptable, the literature that, contrary to biomechanical data, just “accepts” the opinion does not render the opinion more reliable. Additionally, the fact that “many researchers believe that shaking alone can cause SDH, retinal hemorrhage, and death” is unavailing. State Motion at 9 (quoting Appendix BB). “Science is not a democracy.”⁴ Evidence is evaluated on its merits, not on how many people believe in it.

Here, the biomechanical evidence in support of the shaking hypothesis is scant. The 1968 Ommaya study itself does not actually support shaking as a viable mechanism for the clinical findings. *See supra*, Section II(B)(iii)(a). Since then, many biomechanical studies have attempted and failed to validate the SBS hypothesis. *See App. F (Lloyd 2011)* (summarizing the research). In fact, while the findings of biomechanical studies “are consistent with the physical laws of injury biomechanics,” the results “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.” *App. G at 71 (Bandak 2005)*.

The State would have the Court overlook the research that so far disproves Dr. Feldman’s hypothesis. This includes the Prange 2003 study where, using a more biofidelic dummy, angular accelerations from shaking were well-below injury thresholds. *See App. H*. Additionally, measuring angular acceleration from drops of various heights and on various surfaces, impact on a soft surface did not reach levels high enough to cause subdural bleeding or axonal damage. Similarly, other researchers could not achieve injury-level accelerations by

⁴ Gregory A. Poland, M.D., and Robert M. Jacobson, M.D., *The Age-Old Struggle against the Antivaccinationists*, 364 N Engl J Med 97-99 (2011).

shaking without impact, even after modifying the neck in the dummy and using an exaggerated, gravity-aided shaking motion. *See* C. Z. Cory & B. M. Jones, *Can Shaking Alone Cause Fatal Brain Injury? A Biomechanical Assessment of the Duhaime Shaken Baby Syndrome Model*, 43 *Med., Sci. & Law* 317 (2003) (App. EE).

Even the research cited by the State does not support relying on the hypothesis in this case. *See* State Motion at 23-24. In the 2010 Finnie study, seven anesthetized baby lambs were shaken by holding “under the arms much like has been described for shaking,” *See* John W. Finnie et al., *Diffuse Neuronal Perikaryal Amyloid Precursor Protein Immunoreactivity in an Ovine Model of Non-Accidental Head Injury (the Shaken Baby Syndrome)*, 17 *J. Clinical Neuroscience* 237 (2010) (App. FF). The researchers shook the lambs as hard as they could for 30 seconds, waited for a period of time, then shook the lambs again for another 30 seconds, **until they had done it ten times over 30 minutes.** *Id.* at X (emphasis added). A small subdural hemorrhage was found in two shaken lambs, and minimal retinal hemorrhage was seen in only two lambs. *Id.* at 239. Thus, the only biomechanical study that could be said to provide support for the general shaking hypothesis does not support applying the hypothesis here, where Mr. Morris was alone with A.M. for 10 to 15 minutes.

The State claims Mr. Morris does not explain why an exact measure of force or injury threshold is necessary to conclude the injuries are the result of abuse. State Motion at 25-26. But the point is that Dr. Feldman’s vague testimony about forces, given when he explains the necessarily biomechanical phenomenon he says led to A.M.’s injuries, shows the explanation is not anchored to specific knowledge. Further, his own attempts to dismiss the lack of biomechanical support, if agreed with, provide yet another reason to doubt the shaking hypothesis. Dr. Feldman’s explanation for the failure of the hypothesis to be supported by

biomechanics is that “there are ... biomechanical studies that are gradually **better defining what the actual tissue thresholds are**, but as yet, there’s no study that defines **how infant brain tissue responds to repetitive shear forces back and forth.**” 6/03/11 RP 27-28 (emphasis added). In other words, the failure to validate shaking is a failure of biomechanics, not of the hypothesis.

For a hypothesis grounded in the results of a biomechanical study, the inability of biomechanics to define how infant tissue responds to shear forces back and forth is reason to doubt the hypothesis, not to place more trust in it. Additionally, the lack of knowledge flows both way. If the injury thresholds are not established and how infant tissue responds has not been defined, we do not know that shaking does not cause RH and SDH, but we also do not know that it does and how it does it.

Taking into account the biomechanical research, as one should, abusive shaking, possibly with soft impact, does not emerge as a valid, potential explanation for A.M.’s injuries, much less as *the* explanation.

c. Dr. Feldman relied on anecdotal and confessional data that does not validate the SBS/AHT hypothesis

At trial, when asked how it is that shaking is on the list of potential causes, Dr. Feldman explains he is relying on clinical experience that is published and on confessional studies. 6/3/11 RP 28. The State too claims that clinical observations and a “considerable body of literature” provide sufficient support. State’s Motion at 9-10. But reports about cases and studies using confessions do not validate the hypothesis for the reasons explored here.

As discussed in the 2003 Donohoe literature review, opinions based on clinical experience and descriptive reports rank the lowest in evidence-based medicine (EBM) standards. Donohoe 2003 at 239-241 (App. W). Relying on this data is unsound because case

reports and case studies are “universally regarded as an insufficient scientific basis for a conclusion regarding causation because case reports lack controls.” *Hall v. Baxter Healthcare Corp.*, 947 F. Supp. 1387, 1411 (D. Ore. 1996) (citing case law); *see also Siharath v. Sandoz Pharms. Corp.*, 131 F. Supp. 2d 1347, 1361 (N.D. Ga. 2001) (“Case reports are not reliable scientific evidence of causation, because they simply describe[] reported phenomena without comparison to the rate at which the phenomena occur in the general population or in a defined control group; do not isolate and exclude potentially alternative causes; and do not investigate or explain the mechanism of causation.”) (quoting *Casey v. Ohio Medical Prods.*, 877 F. Supp. 1380, 1385 (N.D. Cal. 1995)).

Additionally, the literature suffers from another problem identified by Donohoe 2003: Circularity. This refers to the problem of selecting cases by the presence of the medical features the study seeks to validate. “Not surprisingly, such studies tend to find their own case selection criteria pathognomonic [diagnostic] of SBS.” Donohoe 2003 at 239.

The State points to the supposedly supporting literature but does not address that circularity is an *acknowledged* problem and the reason the research turned to confessions as the proxy for abuse. *See App. X (Vinchon 2010 at 635-636) (acknowledging the circularity in most studies, as well as other biases rendering the value of the studies low).*⁵ Even Dr. Sandeep Narang, extensively cited by the State for the proposition that the diagnosis is well-supported by the literature, simply counters that circularity is inevitable. *See State Exhibit 6 at 562.* As if somehow this addresses the problem of circularity, he counters that other than SBS/ATH, no other explanation for the “associative findings” has been put forward.

⁵ As addressed in the Brief in Support of CrR 7.8(b) Motion, the Vinchon 2010 study is one Dr. Feldman used at trial to support the claim that A.M.’s exact combination of injuries is seen only in confirmed abuse cases, versus accidental cases. 6/3/11 RP 16. The study relies on judicial confessions without knowing the details of the confessions, including the mechanism of abuse. *See Vinchon 2010 at 642 (App. X).*

Dr. Feldman also relies on studies using confessional data. 6/3/11 RP 28. As Dr. Dias acknowledges, “the evidence base for shaking *is* confessions.” App. P at 330. But the merits of this data are likewise questionable. When Dr. Jan Leestma, a neuropathologist at Children’s Memorial Hospital at Northwestern University, closely examined the so-called SBS confession literature, he found that in the vast majority of the “confession” cases there was clear evidence of impact injury to the head—*i.e.*, the child’s injuries likely had not been caused by shaking at all or, at least, were likely partially attributable to an impact. He found that the confession literature only recorded 11 “pure” shaking cases and several of those were questionable because no details were given about the degree of shaking, for how long, or about the circumstances surrounding the confession. For example in some of the cases where the caretaker admitted shaking the infant, it turns out the “admission” was of bouncing the baby during play or attempts to revive the baby when it was found unconscious. *See* J.E. Leestma, *Case Analysis of Brain Injured, Admittedly Shaken Infants: 54 Cases*, 26 *Am. J. of Forensic Med. Path.* 199 (2005) (Appendix EE). Dr. Leestma concluded that “confessions” did not provide an adequate basis to establish the reliability of the SBS diagnosis. Yet, this is the data Dr. Feldman relies on to make his diagnosis.

Subsequent literature has expanded on the reasons why confessions do not scientifically validate SBS/AHT. *See, e.g.*, Keith A. Findley et al., *Shaken Baby Syndrome, Abusive Head Trauma, and Actual Innocence: Getting It Right*, 12 *Hous. J. Health L. & Pol’y* 209, 215 (2012) (App. GG) (explaining the several reasons why confessions do not validate SBS); Waney Squier, *The “Shaken Baby” Syndrome: Pathology and Mechanisms*, *Acta Neuropathol.* 1, 3 (2011) (reviewing so-called confession literature) (App. HH). *See also People v. Thomas*, 22 *N.Y.3d* 629, 646, 985 *N.Y.S.2d* 193, 202 (2014) (excluding proffered confession in SBS case

and noting the similarity between the medical findings and the confession “can be understood as a congruence forged by the interrogation.”); *Aleman v. Village of Hanover Park*, 662 F.3d 897, 907 (7th Cir. 2011) (describing a confession of slight shaking in an SBS case where the father was told the injury must have been caused by shaking as “worthless as evidence, and as a premise for an arrest.”)

Regarding the argument that confessions are unreliable as data, the State counters that the researchers’ decision to rely on judicial confessions means the confessions are reliable, and that it is unknown what percentage of the confessions are actually accurate. State Motion at 25. First, the researchers’ decision to use certain data tells us nothing useful about the data itself. It certainly does not mitigate potential problems with judicial confessions, including the known problem of false confessions. Second, not knowing to what degree the confessions are accurate or inaccurate is precisely the problem with relying on confessions. It is hard, if not impossible, to assess the quality of this research when the researchers themselves assume, but do not know, the quality of their data.⁶

In criminal cases, confessions in other, unrelated cases are worthless evidence. The State would never suggest when prosecuting a suspect that other alleged perpetrators’ confessions in similar cases is probative of guilt. Nonetheless, Dr. Feldman’s reliance on confessions to determine what happened here is not just accepted, but championed.

iv. The State’s Insistence That AHT is a Valid “Differential Diagnosis” Avoids the Question of Reliability

⁶ The State also points to Dr. Dias’ argument that to suggest shaking cannot cause the medical findings, one would have to illogically believe that all the confessed perpetrators lied. See State Motion at 25. The corollary is, of course, that to suggest shaking caused the injuries in every case where the caregiver denies shaking one would have to, also illogically, believe that every caregiver is lying.

The State's response that Dr. Feldman reliably applied the "differential diagnosis" method to arrive at AHT, a medically valid diagnosis, is unavailing. State Motion at 20.

a. The State fails to address causation entirely

In response to the claim that Dr. Feldman's so-called differential diagnosis is really a differential etiology, the State simply asserts that SBS/AHT is a clinically valid diagnosis and that Dr. Feldman's diagnosis was meant to treat the patient.⁷ This ignores completely that Dr. Feldman did not just claim to diagnose the medical conditions affecting A.M., but he claimed to diagnose the cause. The term AHT clearly encompasses causation, and as such, the reliability of the diagnosis hinges on whether causation was reliably determined.

Dr. Sandeep Narang does not explain causation either but assumes it also. Citing the strong "association" of SDH and RH with trauma, Dr. Narang explains the "differential diagnosis" approach, and arrives at SBS/AHT as the default diagnosis when the known causes have been ruled out and the history given is inconsistent with the injuries. State Exhibit 6 at 570-572.⁸ This process assumes cause-in-fact. It also places the burden on the caregiver to explain the medical findings.

The methodology used by Dr. Feldman is clearly a differential etiology, *i.e.* the process of determining which of two or more causes is responsible for the patient's symptoms. *See Hendrix ex rel. G.P. v. Evenflo Co.*, 609 F.3d 1183, 1195, fn. 5 (11th Cir. 2010). The first step in a proper differential etiology is for the expert to compile a "comprehensive" list of causes that are each *capable* of explaining the clinical findings. *Id.* at 1195 (emphasis added); *Clausen v. M/V NEW CARISSA*, 339 F.3d 1049, 1057-58 (9th Cir. 2003). Importantly, for each such

⁷ The testimony makes clear Dr. Feldman was brought in to consult on the cause of the injuries. 6/2/11 RP 113; 117. He was not A.M.'s treating physician. *See* 6/2/11 RP 135.

⁸ For a complete discussion of the problems with Dr. Narang's article, see Findley (App. GG).

potential cause the expert “rules in” at this stage, that cause “must actually be capable of causing the injury.” *Hendrix*, 609 F.3d at 1195 (quoting *McClain v. Metabolife Int'l, Inc.*, 401 F.3d 1233, 1253 (11th Cir. 2005) (excluding potential cause “ruled in” by expert because it had not yet been established to be a potential cause of the injuries in question). “Expert testimony that rules in a potential cause that is *not* so capable is unreliable.” *Clausen*, 339 F.3d at 1058 (emphasis added).

As explained, AHT via shaking, “possibly” with impact on a soft surface—the proposed mechanism of abuse specifically advanced here—has not been established to be capable of causing A.M.’s medical findings. Attempts to confirm the biomechanics have shown the hypothesis to be “biomechanically improbable.” Appendix D (Ommaya 2002). By his own testimony, to rule it in, Dr. Feldman relied on anecdotal clinical data and on confessions. The researchers, in turn, do not know what the persons confessed to (is it even shaking?) and whether the confessions are accurate. Concluding from this data set that shaking, even with soft impact, actually caused the medical findings at issue here is a leap. As should be clear, Dr. Feldman’s determination is merely a hypothesis, one that he put on the table even though it is being hotly debated, has not been validated, and is at odds with biomechanics. Dr. Feldman’s differential diagnosis, ruling in as the cause-in-fact a hypothesis short in scientific support, is not reliable.

b. AHT is not a diagnosis just meant to treat the patient

Despite the State’s claim that SBS/AHT is a diagnosis for treating the patient, it is abundantly clear SBS/AHT has never been just a medical diagnosis. Instead, it is a diagnosis used primarily for prosecution, not treatment. Even the name signals its broader function. “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)." App. B at 202 (Guthkelch 2012). SBS, by contrast, is a name that focuses on the alleged cause of certain clinical findings. *Id.* Tightly tethering the concept of abuse to the medical findings has always been a focus of SBS/AHT advocates, even now that it is well-accepted that there are many other causes of the medical findings associated with SBS/AHT.

For example, in 2001, the Committee on Child Abuse and Neglect of the American Academy of Pediatrics (AAP) issued a policy statement that not only endorsed SBS, but said that a **presumption** of abuse should exist whenever a child younger than one year presented with intracranial injury and retinal hemorrhages. *Shaken Baby Syndrome: Rotational Cranial Injuries--Technical Report*, Pediatrics Vol. 108 No. 1 (July 2001) (emphasis added). By 2009, however, the shaking hypothesis had become controversial. Yet, instead of revisiting the SBS hypothesis in light of the controversy over the supposedly supporting science, the Committee issued another policy statement suggesting that physicians stop using the term Shaken Baby Syndrome and instead use the term Abusive Head Trauma. App. E (2009 AAP policy statement). This position paper, used by the State at trial, reveals that it made the name change not to more accurately reflect scientific discoveries, but rather to help criminal prosecutions despite mounting criticism of the scientific underpinnings of SBS: "Legal challenges to the term 'shaken baby syndrome' can distract from the *more important questions of accountability of the perpetrator* and/or the safety of the victim." *Id.* (emphasis added).⁹

The child abuse protection community has prosecuted SBS for over thirty years. So it is perhaps not surprising that, at this point, there are those who staunchly resist any challenge to the construct. In particular, there is a National Center on Shaking Baby Syndrome. The Center

⁹ In contrast, the National Association of Medical Examiners (NAME) did not renew its 2001 position paper. See App. GG at 232-233. The State relied on NAME's supposed support of the diagnosis even though by 2011 it was not accurate to say or imply that the 2001 position paper still endorsed the diagnosis.

advocates for SBS' reliability, trains law enforcement officers, and supports prosecutions. Every other year, the Center puts on international conferences for physicians, prosecutors and social workers to discuss new SBS developments that are dedicated to castigating each new batch of opposing literature as "biased," "misleading" and "unscientific."

For example, according to the Center's website, at the Twelfth International Conference one keynote address was titled: "While We Argue, Children Die: The Consequences of Misinformation."¹⁰ This address supposedly "set the tone for a meeting grounded in science." Other prominent presentations made were about how to respond to *Daubert* challenges and a panel that discussed the circumstances of perpetrator confessions gathered from around the world. The Fourteenth International Conference in 2014 had similarly focused presentations, with one keynote address titled: "Exonerating" the Guilty: Child Abuse and the Corruption of the False-Conviction Movement." Well-accepted medical diagnoses, of course, do not need international conferences to vouch for their existence.

The tethering of medicine and law is also apparent from the SBS literature. For example, there are manuals for prosecuting SBS cases, which are littered with pearls of junk science. *See, e.g.,* Brian Holmgren, *Prosecuting the Shaken Infant Case in The Shaken Baby Syndrome: A Multidisciplinary Approach*, 307 (2001) (providing prosecutors with ideas for physician testimony such as: the "expert can testify that the forces the child experiences [from shaking] are the equivalent of a 50-60 m.p.h. unrestrained motor vehicle accident, or a fall from 3-4 stories on a hard surface."). Similarly, pediatricians publish articles and book chapters dealing with legal issues, such as the *mens rea* of alleged shakers. *See, e.g.,* A. Levin, *Retinal Haemorrhages and Child Abuse*, in *18 Recent Advances in Pediatrics* 151 (2000) ("we know

¹⁰ Programs for the 12th and 14th International Conference on Shaken Baby Syndrome/Abusive Head Trauma, available at www.dontshake.org.

that the violence which results in SBS injuries is extreme . . . [and] beyond . . . that even the most distraught person would recognize as injurious.”).

In sum, SBS/AHT has always been a diagnosis that is not primarily medical or scientific, but instead one that intertwines medicine with law and child protection policy. That intertwining may be understandable, but the tendency for an unproven hypothesis to be shaped and perpetuated by forces other than objective science is undeniable and cannot be ignored in determining whether the diagnosis is sufficiently reliable to be admitted in a criminal case.

c. Dr. Feldman did not reliably rule out other causes

Dr. Feldman testified that part of a differential diagnosis is going through and eliminating other diagnoses that might cause the medical findings. 6/2/11 RP 122. As Dr. Feldman would have known, the list of diseases and conditions known to cause A.M.’s medical findings is long and growing. *See* State Exhibit 6 at Appendices B&C (Narang 2012) (listing the differential diagnosis of subdural and retinal hemorrhages respectively, including viral meningitis);¹¹ App. GG at 240 (explaining that by 2006 many differential diagnoses were widely recognized by supporters of the hypothesis, including accidental causes and a variety of illnesses and medical conditions).

In other words, it is known and recognized that non-abusive events can cause A.M.’s medical findings, and that the findings are not unique markers of abuse. After reviewing the imaging, radiologist Dr. Barnes identified several potential causes of A.M.’s medical findings. 6/7/11 RP 405. He could not rule out any of them based on the imaging alone. *Id.* at 401. Dr.

¹¹ Even though viral meningitis is a recognized differential diagnosis for RH and SDH, the prosecutor aggressively cross-examined Dr. Gabaeff on whether viral meningitis could lead to the medical findings, questioning at length Dr. Gabaeff’s explanation that this was accepted. *See* 6/8/11 602-608.

Gabaeff also identified potential causes including viral meningitis, which he believed was consistent with the whole clinical picture. 6/8/11 RP 503-505.

The State argues Dr. Feldman did a differential diagnosis but does not pretend, even for argument sake, that Dr. Feldman comprehensively identified and methodically ruled out other potential causes. Dr. Feldman's testimony makes clear he did not, since he focused on finding other injuries. He recommended lab tests that "primarily were focusing on whether there was any other injuries that didn't show up based on examination signs and symptoms." 6/2/11 RP 121. *See also* 6/3/11 RP 59 (explaining that A.M. had screening labs for bone integrity and for intra-abdominal trauma).

Relatedly, Dr. Feldman obtained a family history from the mother only (see 6/2/11 RP 134). He did not review A.M.'s pediatric records even though a child's history is important in making a diagnosis. 6/3/11 RP 52. A.M. had decreased appetite, a loose, foul-smelling diaper and congestion three days before, indicating a viral cold. *Id.* at 52;57. Notably, these and other symptoms A.M. had are consistent with viral meningitis. *Id.* at 55-57. Even though "a preexisting viral infection can subsequently settle in the area of the brain," and even though viral meningitis is on the accepted list of differential diagnoses for both SDH and RH, Dr. Feldman did not order the blood test for meningitis. *Id.* at 58-59. A.M.'s blood was drawn very often, but he considered the test "stupid." *Id.* at 59.

v. Dr. Freeman's Report Is Not Flawed and Undermines Dr. Feldman's Diagnosis

The State argues Dr. Freeman's report does not undermine Dr. Feldman's testimony. State Motion at 30. Specifically, the State claims the database does not take into account the difference between general and pediatric hospitals. *Id.* at 29. The State also claims Dr. Freeman failed to account for A.M.'s apnea and seizures, which other experts have found significant in

diagnosing abusive head trauma. *Id.* The State's analysis shows the purpose and point of the study was largely misunderstood.

Dr. Freeman's study measures whether Dr. Feldman's reliance on SDH and RH as indicators of abuse *when there are no other injuries indicating abuse*, as was the case here, is warranted. Indeed, Dr. Feldman did not rely on the medical findings as mere indicators, but as proof of abuse. To assess the validity of Dr. Feldman's assertions regarding the relationship between SDH, RH and abuse, one needs to look at a large and valid data set (that includes both abuse and non-abuse cases with RH and SDH) against which the accuracy of the determination can be tested. When Dr. Freeman did this, he found that RH and SDH, without more, are very poor proxies for abuse even when considered together. *See App. At 8-9.*

The Kids' Inpatient Database includes specialty hospitals. In fact, pediatricians themselves have relied on the Kids' Inpatient Database to study the occurrence of serious injuries due to physical abuse in hospitalized children. *See John M. Leventhal, MD et al, Using US Data to Estimate the Incidence of Serious Physical Abuse in Children, Pediatrics 2011-1277 (App. II).* Using this database is not flawed.

Regarding apnea and seizures, these were not considered by Dr. Freeman since the presence of these findings is not what Dr. Feldman relied on to suspect abuse. He thought the apnea was seizure-related actually. 6/2/11 RP 131. Whether or not the presence of these findings means his diagnosis is accurate depends on what other conditions, in addition to abusive head injury, seizures and apnea may indicate. For example, per Dr. Feldman, seizures can be a symptom of meningitis. 6/3/11 RP 57.

Dr. Feldman's claim at trial that RH and SDH are reliable indicators of abuse is wildly off the mark. *See, e.g., 6/3/11 RP 12-13; RP 16.* Relying on these medical findings, where there are no

other injuries and where Mr. Morris was alone with A.M. for 10-15 minutes, to reach his diagnosis of abuse was misguided. To suggest the medical findings reliably indicate abuse “is indeed a chasm too wide and deep to leap.” *State v. Black*, 109 Wn.2d 336, 348 (1987) (quoting *State v. Taylor*, 663 S.W.2d 235, 241 (Mo. 1984) (excluding rape trauma syndrome testimony because it does not reliably indicate rape).

vi. Counsel's Failure to Exclude the Testimony Was Not Strategic

Counsel knew Dr. Feldman’s testimony was critical. She asked for a hearing on the admissibility of Dr. Feldman’s testimony, which was settled with an agreement the State would not use the term “shaken baby syndrome” (SBS). The State did not agree to refrain from arguing A.M. was shaken, but simply not to use the term SBS.

The State argues counsel concluded she could not win such a motion. State’s Motion at X. However, “[c]ounsel can hardly be said to have made a strategic choice when s/he has not yet obtained the facts on which a decision could be made.” *Avila v. Galaza*, 297 F.3d 911, 920 (9th Cir. 2002) (quoting *Sanders v. Ratelle*, 21 F.3d 1446, 1457 (9th Cir. 1994). *See also Evans v. Lewis*, 855 F.2d 631 (9th Cir. 1988) (holding that the failure to investigate possible evidence cannot be deemed a trial tactic where counsel failed to view relevant, available documents).

Here, counsel’s decision to give up on the issue of admissibility altogether was premature. The date of the briefing establishes the issue of admissibility was settled before counsel interviewed Dr. Feldman and more than one year ahead of trial. App. J-L; 2/25/11 RP 8 (noting interview of Dr. Feldman is upcoming). Counsel decided not to challenge the admissibility of Dr. Feldman’s testimony before she had all the information on which to make a reasonable strategic decision. A decision considering admissibility under *Frye* only, more than

one year ahead of trial, before interviewing the witness and before trial preparation, is not a tactical decision where a maintainable challenge to the testimony remains wholly unassessed.

Further, there is no conceivable strategic decision that supports allowing the State to introduce unreliable causation testimony where causation is the critical issue. The cases cited by the State support the claim that counsel was deficient in this regard. In *State v. Nichols*, the Court addressed whether trial counsel was ineffective for failing to make a motion to suppress the evidence made during a pretextual stop. 161 Wn.2d 1, 162 P.3d 1122 (2007). The Court, after exploring at length whether under relevant caselaw the stop was pretextual, concluded that Nichols could not show it was. *Id.* at 8-14.

Thus, although counsel may legitimately decline to move for suppression on a particular ground if the motion is unfounded, that is not what happened here, where a legitimate question of admissibility existed. Counsel identified the State's controversial causation evidence needed to be vetted but retreated from the matter after a concession inconsequential to the issue. The State's argument that counsel successfully excluded potentially inflammatory evidence because Mr. Morris admitted he shook A.M. after she vomited and choked fails. Shaking is only "inflammatory" if one believes abusive shaking was entailed. Dr. Feldman clearly said it was even though he never used the term SBS.

In *State v. McNeal*, the court addressed trial counsel's failure to object to apparently inconsistent verdicts. The Court concluded the decision not to object was reasonable where the judge might well have ordered the jury to resume deliberations resulting in findings that would then allow the judge to impose a greater penalty. 145 Wn.2d 352, 363, 37 P.3d 280, 285 (2002). The Court, therefore, found a specific reason that could have supported counsel's decision not to object. Similarly, in *State v. Aho*, the Court considered trial counsel's failure to investigate

the effective dates of the relevant statutes in connection with the factual charging period. 137 Wn.2d 736, 745-46, 975 P.2d 512, 517 (1999). The Court found there was no conceivable legitimate tactic where the only possible effect of deficient performance was to allow the possibility of a conviction of a crime under a statute which did not exist and could not be applied during part of the charging period.

“The proper measure of attorney performance remains simply reasonableness under prevailing professional norms.” *Strickland v. Washington*, 466 U.S. 668, 688, 104 S. Ct. 2052 (1984). Counsel accepted damning testimony wholesale, without any limits and without ensuring the State, as the proponent of the evidence, could establish its admissibility. Constitutionally effective counsel would not have so readily ceded this terrain—whether expert testimony underpinning the entire case was even admissible—to the prosecution. As in *Aho*, the only possible effect of deficient performance was to allow the possibility of a conviction based on inadmissible testimony.

vi. Counsel's Failure to Challenge the Admissibility of Dr. Feldman's Opinion Testimony Prejudiced Mr. Morris

The State claims Mr. Morris was not prejudiced by counsel's failure to challenge admissibility because such a challenge would not have been successful. For the reasons set forth above, Dr. Feldman's opinion on causation was unreliable and therefore not helpful to the trier of fact. It is not admissible under ER 702 and applicable caselaw. Additionally, related considerations weigh in favor of excluding the testimony, disproving the State's claim that a motion would not have been successful.

a. *The burden of proof weighs in favor of excluding the opinion*

In a criminal case, “the Due Process Clause protects the accused against conviction *except upon proof beyond a reasonable doubt of every fact necessary* to constitute the crime

with which he is charged.” *In re Winship*, 397 U.S. 354, 364 (1970) (emphasis added). The State thus bore the burden of proving A.M.’s medical findings were the result of abusive shaking by Mr. Morris. Where, as here, the expert testimony constituted the proponent’s *only* evidence of causation, the court’s admissibility determination under ER 702 and other rules of evidence must consider the State’s burden of proof.

If conjecture is insufficient to help a jury determine proximate cause in a civil case, it is certainly insufficiently helpful in a criminal case requiring proof beyond a reasonable doubt. *See, e.g., Moore v. Hagge*, 158 Wn.App. 137, 148, 241 P.3d 787, 792 (2010) (expert’s summary judgment affidavit was unfounded; conjectural theories are insufficient to establish proximate cause); *Reese v. Stroh*, 128 Wn.2d at 309 (evidence establishing proximate cause in medical malpractice cases must rise above speculation, conjecture, or mere possibility); *McLaughlin v. Cooke*, 112 Wn.2d 829, 774 P.2d 1171 (1989) (evidence will be considered insufficient to support the trial verdict if it can be said that, considering all the medical testimony presented at trial, the jury must resort to speculation or conjecture in determining the causal relationship).

Proof beyond a reasonable doubt has “traditionally been regarded as the decisive difference between criminal culpability and civil liability.” *Jackson v. Virginia*, 443 U.S. 307, 315 (1979). Dr. Feldman’s opinion—a medico-legal hypothesis—is insufficient, since proof that leaves open the real possibility that Mr. Morris did not cause the medical findings could not satisfy the State’s burden of proof. Thus, Dr. Feldman’s expert opinion does not help the jury determine causation to the substantive standard.

b. *The danger of undue prejudice weighs in favor of excluding the opinion*

A trial court must evaluate both the relevance of the testimony and its prejudicial impact, excluding unnecessarily cumulative or unfairly prejudicial testimony. *See* ER 402, 403. *State v. Petrich*, 101 Wn. 2d 566, 575, 683 P.2d 173, 180 (1984). Here, the probative value of the shaking hypothesis must be weighed against the substantial prejudicial effect on a jury from testimony by an "expert witness" that unreliably determines causation. "Expert evidence can be both powerful and quite misleading because of the difficulty in evaluating it." *Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579, 113 S.Ct. 2786 (1993) (source omitted).

The admission of misleading expert testimony can be particularly prejudicial. This is because of the way juries perceive testimony given by an expert witness. *See Moore v. Hagge*, 158 Wn. App. at 155 (noting the danger that the jury may be overly impressed with a witness possessing the aura of an expert). Merely labeling a witness as an expert can make jurors more likely to believe the information the witness presents is legitimate. *See* N.J. Schweitzer and Michael J. Saks, *The Gatekeeper Effect: The Impact of Judges' Admissibility Decisions on the Persuasiveness of Expert Testimony*, 15 Psychol., Pub. Pol'y & L. 1 (2009).

For example, one study found that in jury simulations the decision to admit or exclude expert evidence "was the sole predictor of the perceived quality of the research," and admission of the evidence made jurors think evidence was of higher quality and more persuasive. *Id.* at 7-12. Further, studies have shown that traditional tools of the adversarial process like cross-examination and the use of opposing experts have a limited ability to counteract the effect expert testimony has on a jury, making it hard to limit the prejudice Rule 403 is designed to prevent. *See* Dawn McQuiston-Surrett and Michael J. Saks, *The Testimony of Forensic Identification Science: What Expert Witnesses Say and What Factfinders Hear*, 33

Law & Hum. Behav. 436 (2009). Thus, not only does the expert designation cause jurors to give the testimony an unearned air of legitimacy, but it also limits the ability of the defense to counter those flaws in the jury's mind.

The AHT diagnosis, purporting to diagnose causation, is unreliable. That this unreliable determination is presented as an expert medical "diagnosis" only serves to make it more prejudicial. Thus, balancing the probative value of the opinion against its prejudicial effect, the scales tip wholly towards excluding the evidence.

c. Due process concerns weigh in favor of excluding the opinion

For convictions resting on expert opinion, the unreliability of expert testimony clearly implicates due process. In *Han Tak Lee v. Glunt*, 667 F.3d 397 (3d Cir. 2012), petitioner raised a due process challenge to a murder conviction predicated on expert testimony regarding arson. The Court held that to merit relief, petitioner must show the expert testimony at trial undermined the fundamental fairness of the entire trial because the probative value of the evidence, though relevant, is greatly outweighed by the prejudice. *Id.* at 403. Finding on remand that the verdict rested almost entirely upon flawed and unreliable scientific evidence, the court found the petitioner made this showing and granted the writ. *Han Tak Lee v. Tennis*, 2014 WL 3894306 at *18-19 (M.D. Pa. June 13, 2014). Thus, where the proof of guilt rests on Dr. Feldman's causation opinion, and it is flawed and unreliable, preserving the fundamental fairness of the trial necessitates excluding it.

vii. Dr. Herlihy's Testimony is Not Enough to Sustain the Conviction

The State argues that even without Dr. Feldman's testimony there was sufficient evidence to conclude that A.M's injuries were caused by abuse. State's Motion at 30. This is incorrect. Dr. Herlihy did not time the retinal bleeding to the 10-15 minutes Mr. Morris was

alone with A.M. or even to the day. *See* 6/10/11 RP 334-335; 355 (explaining hemorrhages detected June 1, 2009 can be timed, reliably, to within one week).

There was evidence disputing Dr. Herlihy's conclusion that abuse caused the retinal bleeding. Even if it could be said Dr. Herlihy established this, her testimony alone could not establish Mr. Morris inflicted the abuse. The State clearly needed Dr. Feldman to tie the medical findings together and to opine they were caused by the same event happening during the very narrow window of time Mr. Morris was solely responsible for A.M.

C. Mr. Morris Made a Substantial Showing Trial Counsel was Ineffective in Failing to Correct Dr. Feldman's Misleading Testimony

Trial counsel's strategy was to question the soundness of Dr. Feldman's opinion. Trial counsel had a duty to carry out her chosen defense strategy competently. *See Alcala v. Woodford*, 334 F.3d at 870 (holding that counsel failed in his duty to present his chosen defense reasonably and competently). At trial, Dr. Feldman explained his reliance on the medical literature and pointed specifically toward certain studies to support his use of RH and SDH to diagnose abusive head trauma. The testimony, such as the claim that the Bhardwaj study showed RH is 95% specific for head injury, appeared very strong. 6/3/11 RP 12-13. Dr. Feldman used the Vinchon 2010 confessional study to similarly suggest that A.M.'s exact clinical picture, including no surface evidence of trauma, pointed strongly towards abusive shaking. 6/3/11 RP 16. Each of these specific claims bolstered Dr. Feldman's broad assertion that based on A.M.'s medical findings alone abuse could be confidently diagnosed.

Circular literature and confessional studies—which both these studies are—is not strongly supportive data. Trial counsel's failure to expose, or even explore, the flaws with the studies and with relying on them is deficient performance. The State concludes trial counsel's cross-examination omissions were strategic without articulating what conceivable strategy

would support letting this testimony go completely unchecked. There is not one. Further, in addition to the above deficiencies, the large majority of biomechanical studies went completely unexplored. As described above, Dr. Feldman's diagnosis in this case cannot be squared with biomechanics. Accordingly, this omission cannot be considered strategic when the defense strategy is to discredit the expert testimony.

For Dr. Feldman to conclude that A.M.'s injuries were the result of abuse inflicted by her last caregiver when she was alone with him requires the ability to reliably time the injuries to a 10-15 minute window. There is consensus, even amongst Dr. Feldman's child abuse colleagues, that symptoms may be delayed and that, although rare, even prolonged lucid intervals happen. *See* Brief in Support of CrR 7.8(b) Motion at 36-39. Given the very narrow window of time in this case, this was critical. Competent counsel would have used all the tools available on this topic to show the medical consensus that precisely timing injuries is not justified by what is known about the onset of symptoms. Trial counsel's failure to establish this allowed Dr. Feldman's narrow timing determination to be presented as sound, even though it is wildly outside what agreed-upon knowledge supports.

Prejudice is shown when "there is a reasonable probability that, but for counsel's unprofessional errors, the result of the proceeding would have been different." *Strickland v. Washington*, 466 U.S. 668, 694, 104 S. Ct. 2052 (1984). The Court must consider the prejudicial impact of each error cumulatively. *Id.* at 695. Here, viewed individually and cumulatively, given the significance of each issue to Dr. Feldman's opinion, there is a reasonable probability the result of the trial would have been different but for counsel's errors. Confidence in the outcome of the trial is unwarranted.

D. Mr. Morris Made a Substantial Showing that Dr. Feldman's Misleading and Unreliable Testimony Violated His Due Process Right to a Fair Trial

The State does not address the claim that Dr. Feldman's testimony was misleading, other than to say it was not. As described above and in the Brief in Support of CrR 7.8(b) Motion, Dr. Feldman's testimony did not accurately represent the strength of the evidence-base for shaking, especially as it applies in a case like this one lacking any evidence of impact or other signs of abuse. Without explaining or even addressing the limits of the stated support, Dr. Feldman's discussion of the medical literature created a false impression that the research strongly supported a definitive diagnosis of abuse. *See Lakey*, 176 Wn.2d at 921. Due process is violated when State introduces misleading testimony. *Hayes v. Brown*, 399 F.3d 972, 984 (9th Cir. 2005). Mr. Morris' conviction rests on Dr. Feldman's testimony, which certainly affected the judgment of the jury. It cannot be said that with Dr. Feldman's testimony misleadingly representing the support for his diagnosis, Mr. Morris received a fair trial. *See United States v. Agurs*, 427 U.S. 97, 103, 96 S.Ct. 2392 (1976)).

Further, the introduction of Dr. Feldman's unreliable testimony violated due process because the probative value of the evidence was greatly outweighed by the prejudice, undermining the fundamental fairness of the entire trial. *Han Tak Lee v. Glunt*, 667 F.3d 397, 403 (3d Cir. 2012).

III. CONCLUSION

For all the foregoing reasons, this Court should not transfer Mr. Morris' CrR 7.8(b) Motion to the Court of Appeals. The Court can address the merits of the motion and grant relief based upon the constitutional violations infecting Mr. Morris' trial. Mr. Morris has made a

sufficient showing warranting relief based on the record before the Court. However, if there are factual questions that need to be resolved to reach the merits, such as the extent to which Dr. Feldman's testimony should have been excluded or limited, this Court can hold a factual hearing and transfer is inappropriate.

Respectfully submitted this 18th day of November 2014

INNOCENCE PROJECT NW CLINIC



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APPENDIX INDEX

Appendix A	Mandate Letter
Appendix B	A.N. Guthkelch, <i>Problems of Infant Retino-Dural Hemorrhage With Minimal External Injury</i> , 2012
Appendix C	Duhaime 1987
Appendix D	Ommaya 2002
Appendix E	American Academy of Pediatrics policy statement re Abusive Head Trauma 2009
Appendix F	John Lloyd et al, <i>Biomechanical Evaluation of Head Kinematics During Infant Shaking Versus Pediatric Activities of Daily Living</i> , 2011
Appendix G	Faris A. Bandak, <i>Shaken Baby Syndrome: A Biomechanics Analysis of Injury Mechanisms</i> , 2005
Appendix H	Prange 2003
Appendix I	References on SBS/AHT
Appendix J	Motion for a Preliminary Determination of Whether or Not a Frye Hearing is Necessary
Appendix K	Defense Reply Brief
Appendix L	Response to Defense Reply Brief
Appendix M	State Exhibit 4, Written Statement
Appendix N	State Exhibit 2, Text Message
Appendix O	Susan Marguiles and Brittany Coates, <i>Biomechanics of Head Trauma in Infants and Young Children</i> , in <i>Child Abuse and Neglect, Diagnosis, Treatment and Evidence</i> , 2011
Appendix P	Mark S. Dias, <i>The Case for Shaking</i> , in <i>Child Abuse and Neglect, Diagnosis, Treatment and Evidence</i> , 2011
Appendix Q	Report of Dr. Michael Freeman
Appendix R	Michael D. Freeman et al., <i>Forensic Epidemiology: A systematic approach to probabilistic determinations in disputed matters</i> , 2008
Appendix S	Defense Memorandum in Support of a New Trial
Appendix T	8/2/11 E-mail Correspondence
Appendix U	Poplar Bluff Police Department Supplemental Narrative Report
Appendix V	Poplar Bluff Police Department Supplemental Narrative Report
Appendix W	Excerpt from redacted Missouri medical records
Appendix X	Vinchon 2010

Appendix Y	Gaurav Bhardwaj et al., <i>A Systematic Review of the Diagnostic Accuracy of Ocular Signs in Pediatric Abusive Head Trauma</i> , 2010
Appendix Z	M.G.F. Gilliland, <i>Interval Duration Between Injury and Severe Symptoms in Non-accidental Head Trauma in Infants and Young Children</i> , 1998
Appendix AA	Kristy B. Arbogast et al., <i>Initial Neurologic Presentation in Young Children Sustaining Inflicted and Unintentional Fatal Head Injuries</i> , 2005
Appendix BB	Kent Hymel et al., <i>Abusive Head Trauma</i> , in <i>Child Abuse and Neglect, Diagnosis, Treatment and Evidence</i> , 2011
Appendix CC	Trial Memorandum in Support of Defendant's Motion to Dismiss Pursuant to <i>State v. Green</i>
Appendix DD	Ronald Uscinski, <i>Shaken Baby Syndrome: An Odyssey</i> , 46 <i>Neurol. Med. Chir.</i> 57, 2006
Appendix EE	C. Z. Cory & B. M. Jones, <i>Can Shaking Alone Cause Fatal Brain Injury? A Biomechanical Assessment of the Duhaime Shaken Baby Syndrome Model</i> , 43 <i>Med., Sci. & Law</i> 317 (2003)
Appendix FF	John W. Finnie et al., <i>Diffuse Neuronal Perikaryal Amyloid Precursor Protein Immunoreactivity in an Ovine Model of Non-Accidental Head Injury (the Shaken Baby Syndrome)</i> , 17 <i>J. Clinical Neuroscience</i> 237 (2010)
Appendix GG	Keith A. Findley et al., <i>Shaken Baby Syndrome, Abusive Head Trauma, and Actual Innocence: Getting It Right</i> , 12 <i>Hous. J. Health L. & Pol'y</i> 209 (2012)
Appendix HH	Waney Squier, <i>The "Shaken Baby" Syndrome: Pathology and Mechanisms</i> , <i>Acta Neuropathol.</i> 1
Appendix II	John M. Leventhal, MD et al, <i>Using US Data to Estimate the Incidence of Serious Physical Abuse in Children</i> , <i>Pediatrics</i> 2011-1277

Exhibit DD

Shaken Baby Syndrome: An Odyssey

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Abstract

Shaken baby syndrome is evaluated in the context of its historical evolution and its veracity in referring to causal injury mechanisms. A rational assessment of the injury causation and consequent pathological states associated with the syndrome is presented. It is now evident that shaken baby syndrome evolved as a result of a faulty application of scientific reasoning and a lack of appreciation of mechanisms of injury. A brief explanation of the commonly understood usage and interface of scientific methodology and reasoning as applied to clinical medicine is given.

Key words: shaken baby syndrome, injury biomechanics, subdural hematoma

Introduction

Shaken baby syndrome is characterized as a constellation of clinical findings including subdural bleeding, retinal hemorrhages, and associated fractures of the extremities or ribs, with no external evidence of cranial trauma. This widely proclaimed yet still hypothetical supposition has become a virtually unquestioned assumption nowadays as a modality for causing inflicted intracranial injury in infants. In 1997 the author was asked to review the case of a child fatally injured supposedly by shaking, and in doing so researched the entire body of literature referencing the so-called "shaken baby syndrome." This article is a product of that effort, and in a sense represents an intellectual "odyssey."¹ The paper is divided into three parts. The first places the syndrome in the historical perspective of the original papers providing the initial description of shaken baby syndrome. The second part gives a brief discussion of the physical laws of motion governing injury

to relevant body structures, and encompasses a brief overview of the known pathophysiology of certain relevant forms of intracranial injury. The final section discusses the methodology of scientific reasoning and experimentation and its significance in the context of the immediate subject of the so-called inflicted shaking injury and the broader context of observing and understanding phenomena in our physical world.

Historical Perspective

The quantity of articles dealing with shaking as a putative mechanism for inflicting intracranial injury in infants has increased significantly since the 1970s when the original description of shaking first appeared in a paper published in *The British Medical Journal* in 1971 by Guthkelch,² Caffey,^{3,4} who is generally credited with identifying and characterizing injuries to infants by shaking, published extensively on the subject thereafter. However, it is in the

This excellent paper was presented on May 25, 2005 at the 33rd Annual Meeting of The Japanese Society for Pediatric Neurosurgery in Nara, chaired by Professor Toshiyuki Sakaki. All of the audience was greatly impressed by this new and unique concept of the so-called Shaken Baby Syndrome.

Shaken Baby Syndrome has now become a social issue in Japan and neurosurgeons are very much involved in its management. The topic was quite timely, so we thank Dr. Sakaki for selecting this paper as a special lecture for us.

Dr. Ronald H. Uscinski and I were residents together in neurosurgery at the Georgetown University Hospitals, Washington, D.C. in the early 1970s. I am very proud that such an excellent paper was produced in Washington, D.C. and presented in Nara.

Akira YAMAURA, M.D. (Advisory Board of *Neurologia medico-chirurgica*)
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Guthkelch article that shaking is invoked specifically as a mechanism for causing intracranial injury in infants with no external evidence of cranial trauma. Guthkelch hypothesized that "It seems clear that the relatively large head and puny neck muscles of the infant must render it particularly vulnerable to whiplash injury in this sort of situation," and moreover that "the rotation-acceleration strains on the brain would tend to occur fairly symmetrically also, in an anteroposterior direction. This may be the same reason why the infantile subdural haematoma is even more often bilateral..."

It is significant that as a mechanical justification for invoking shaking as a mechanism for causing intracranial injury in infants, the original and subsequent authors all reference a single paper by Ommaya et al., published in 1968.¹⁰ In this paper, Ommaya, a neurosurgeon studying head injury and building on earlier work by Holbourn,^{9,10} attempted to quantify experimentally the rotational acceleration necessary to cause intracranial injury via a whiplash in rhesus monkeys. The animals in Ommaya's experiment were placed in a contoured fiberglass chair with the head free to rotate; the chair was mounted on wheels and placed on tracks; and the apparatus was systematically impacted with a piston to simulate accelerations analogous to rear-end motor vehicle collisions, with the entire event photographed using a high-speed camera. Ommaya found intracranial injury in 18 of the animals, with concomitant neck injury in 11 of the 18. The purely isolated concept of rotational acceleration of sufficient magnitude to cause intracranial injury without impact and therefore without external evidence of injury was seized upon qualitatively by Guthkelch,⁹ Caffey,^{2,3} and others as the explanation for hitherto unexplained intracranial injury in infants. This concept was hypothesized and put forth by them as being the result of manual shaking.

Hence, the Ommaya paper emerges as the sole source of experimental data from which the initial hypothetical shaking mechanism was drawn. Significantly, Ommaya was actually attempting to quantify the requisite rotational acceleration necessary to cause head injury via whiplash movement of the head in humans during rear end motor vehicle collisions, with attendant vehicular impact. He never addressed the question as to whether human beings can shake infants with enough force to produce the acceleration necessary to cause intracranial injury. It is also significant that neither Guthkelch,⁹ Caffey,^{2,3} nor any subsequent investigators who have sought to identify and characterize ostensible shaking injuries to the infant head ever asked whether the infant torso and neck anatomy, quite

different physiologically from that of the rhesus monkey and of the "non-infant" human, could withstand such shaking. Nonetheless, the mechanism of shaking and the so named syndrome gained immediate acceptance and enormously widespread popularity, with no real investigation or even question as to its scientific validity.

The stage was set; the shaking hypothesis rapidly engendered numerous articles purporting to accept or validate the hypothesis. Ratification within the medical community was based principally on anecdotal reports and case studies. The nearly simultaneous establishment in the United States of the Mandated Reporting Laws¹¹ plus the emerging litigious atmosphere encompassing clinical medicine in America in effect rendered the medical reporting of all cases of even remotely suspected child abuse absolutely compulsory.

The combination of these factors, plus an unspoken, unproved, but increasingly pervasive assumption that all unexplained injuries in children were to be regarded as inflicted injuries, provided a new paradigm for a self-fulfilling prophecy. The hypothesis rapidly metamorphosed to a syndrome; its acceptance expanded exponentially, and "shaken baby" became a term synonymous and ultimately identical with intentional child abuse.

Injury Biomechanics

The causes of trauma obey the laws of injury biomechanics. These laws come from the generalized laws that govern motion, deformation, and forces existing in our universe. An example of one of these universal laws is Newton's second law of motion. Newton's second law governs the relationship between mass, acceleration, and force. In other words, given a mass such as a head, the acceleration of such a mass is governed by Newton's second law when there is a change in velocity divided by a change in time.

In 1943 the physicist Holbourn published a laboratory investigation of traumatic brain injury.⁹ Holbourn understood that the deformable brain was incompressible, hypothesized a rotational acceleration level beyond which injury would occur, and that a smaller mass of brain would require larger rotational acceleration. Ommaya himself alluded to this point in his paper,¹⁰ although this seems not to have been recognized by Guthkelch,⁹ Caffey,^{2,3} and others. In 1987 Duhaime et al.,⁷ using available data on scaled injury thresholds, demonstrated that shaking a mechanical model to cause intracranial injury in the form of concussion, subdural hematoma, and diffuse axonal injury, failed to reach such

thresholds. The model used three different examples of the infant neck in order to reproduce mobility. None of these examples addressed the potential for structural failure of the neck. Following the same line of thought from 1987, two of the same authors repeated the experiment,¹⁷ again using a model not addressing neck injury mechanisms, again focusing on rotational accelerations as the mechanism for causing intracranial injury as transmitted through the infant neck, and again demonstrating requisite accelerations to be not achievable by manual shaking.

While the above articles addressed experimentally the impossibility of causing intracranial injury in infants by manual shaking, no work addressed the potential consequence of such shaking activity on the infant neck, the critical link between the torso where the physical act of shaking is initiated, and the infant head where the injury is hypothesized to occur. Bandak precisely addressed this question in a quantitative manner in 2005.¹⁹ In focusing attention on the infant neck, and demonstrating thereby that any transmission of forces generated by shaking the infant torso must necessarily be transmitted through the underdeveloped infant neck to the disproportionately large head, Bandak showed clearly that cervical spinal cord or brainstem injury in the infant would occur at significantly lower levels of shaking accelerations than those purported in the shaken baby syndrome literature as a cause of subdural hematomas. It is now clear that if an infant is subjected to shaken baby syndrome accelerations one should expect to see injury in the infant neck before it is seen in the head. Moreover, such injury should include injury to the cervical spinal cord and brainstem, obviously with the expected accompanying clinical picture.

Based on the above cited material, it is clear that the hypothetical mechanism of manually shaking infants in such a way as to cause intracranial injury is based on a misinterpretation of an experiment done for a different purpose, and contrary to the laws of injury biomechanics as they apply specifically to the infant anatomy. Finally, manual shaking of an infant, if injurious, should result in an entirely different injury biomechanically, physiologically, and clinically, than hypothesized in 1971.

The "Unexplained Head Injury"

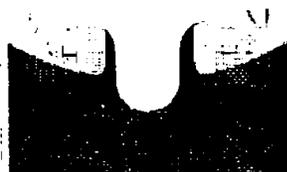
With regard to the cardinal aspect of inflicted injury by the hypothetical shaking mechanism, the "unexplained head injury," the following salient points are worthy of consideration. First, the so-called trivial head impact occurring after a fall of an

apparently short distance is believed in most instances to be an innocuous event. In fact this is not so. The free fall velocity from as little as 3 feet results in the equivalent of greater than 9 miles per hour against a hard surface, or more than twice the skull fracture energy for an infant, again as demonstrated by Bandak.¹⁹ Cadaver testing demonstrated skull fractures in infant specimens in every case when dropped from a height of 3 feet (84 cm), or the height of a changing table, onto a firm or hard surface. Fractures in the thin parietal bone were reliably produced when specimens were dropped even onto a softly cushioned surface.¹⁸⁻²⁰

Therefore, while the majority of such falls may be seen superficially as innocuous, there exists demonstrably proven potential for serious injury. This is compounded given the potential physiologic response to such injury including vomiting, aspiration, seizing, or other airway compromise with attendant potential for hypoxia, all further complicating the clinical picture. One concludes that rather than resorting to simple generalization, each case warrants careful and individual evaluation.

The second point is that subdural hematoma has hitherto been regarded as a cardinal sign in infants of inflicted injury. While acute subdural hematoma is typically seen after an obvious fall with impact, it must be differentiated from chronic subdural hematoma. Most neurosurgeons treating adult or pediatric patients are aware that chronic subdural hematoma certainly started acutely, but by definition its presence was either not recognized or its significance was not appreciated at the time of injury. This need not imply an intentional injury, and it is a matter worthy of some reflection that intentional injury is rarely diagnosed or even considered in an adult presenting with a chronic subdural hematoma. Yet the same injury with the same pathophysiology and the same pathologic anatomy is nowadays presumed to be intentional in the infant. The scientific grounding for this presumption remains unclear.

Some additional observations are noteworthy here. First, it has long been known among clinical neurosurgeons operating on patients with chronic subdural hematomas that at surgery fresh blood may be found in addition to the older blood comprising the hematoma. Second, it has also been demonstrated experimentally that chronic subdural hematomas enlarge by rebleeding from the neovascular membrane^{11-13,21} and that this bleeding has been shown to occur without accompanying trauma. Therefore, at clinical presentation a chronic subdural hematoma may exhibit fresh blood and this may be



mistakenly diagnosed as evidence of recent injury. Lastly, it is known that intracranial hemorrhage may occur even after an apparently uneventful vaginal birth,⁵¹ and it is also well known that a chronic subdural hematoma with well-developed outer and inner membranes is at least several weeks, or even months, old.

The above observations lead one to conclude that for an infant presenting with ostensibly unexplained intracranial bleeding with or without external evidence of injury under given circumstances, accidental injury from a seemingly innocuous fall, perhaps even a remote one, or even an occult birth injury, must be considered before assuming intentional injury.

A recent paper by Donohoe⁵² attempted to evaluate the available medical scientific evidence published from 1966 to 1998 wherein internationally accepted methods were used to determine the degree of confidence that accrues to claims regarding the condition termed "shaken baby syndrome." He concluded that some 32 years of cumulative material yielded inadequate scientific evidence to establish a firm conclusion on most aspects of causation, diagnosis, treatment, or any other matters pertaining to shaken baby syndrome. Donohoe's assessment focused on the methods and quality of the actual research. The scientific status of the syndrome itself was not addressed; rather it was the methodology supporting its validity that was found to be insufficient. Another paper by Leestma⁵³ searched all of the peer reviewed English language medical case literature and analyzed 324 cases that contained detailed individual case information. This search yielded 54 cases in which "some fashion of admission was noted that the injured baby had been shaken" (author's words). The details for all 54 cases were analyzed and of those, 11 cases were found wherein the reviewed material yielded no evidence of impact, and 12 cases had evidence of impact. The remaining cases provided either insufficient information or were excluded from the series for other reasons. After attempted statistical analysis of the material, no conclusions could be drawn by the author regarding which injuries occurred because of inflicted or accidental physical forces or by underlying or secondary disease processes, chiefly due to a paucity of data and inconsistent recording of relevant clinical information. That is, it was impossible to determine with scientific rigor what role shaking may have played in abusive head injury in these reported cases. Finally, it was not possible from the case analyses to infer that any particular form of intracranial or intraocular pathology was causally related to shaking, and that most of the

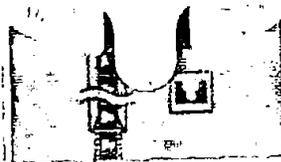
pathologies in allegedly shaken babies were due to impact injuries to the head and body.

Science and Shaking

The practice of clinical medicine is considered to be an artistic and a scientific endeavor. In its highest form this is accomplished through the elicitation of a careful and accurate history, and the performance of a thorough physical examination. It is in this manner that an appropriate diagnosis or differential diagnosis is made. In doing so, the physician must understand the principles underlying the normal and pathologic characteristics of the individual patient before him, and how to identify accurately, delineate, and ultimately integrate these characteristics in a way that elucidates the condition of his patient clearly and concisely. The understanding of such principles, however intricate and a priori compassionate, must be grounded in objectivity, logic, and rationality, and must be in conformity with known biologic and physical laws.

There is a balance between the qualitative aspect of caring for people who are sick, and the quantitative, ultimately cognitive understanding of science underlying the practice of clinical medicine. Although this latter understanding may be considered an applied science, it is grounded in principles of science nonetheless. Thus, within the framework of our approach to medicine, the same principles of scientific methodology and understanding are relevant as they are to understanding the nature of the world around us.

Advances in such scientific understanding may occur in two different ways. The first is by objective observation of phenomena occurring in nature, and correlation of this observation with what is already known of the physical universe to produce a more complete understanding and a higher order of comprehension. The second method is by experimentation under controlled conditions where investigators test hypotheses formulated in a methodical, logical, and rational way, in order to explain observed phenomena. This is how our understanding of the world advances, and this is also precisely how medicine advances. Verification of observation leads to verification by experimentation. This is sound scientific methodology. When this methodology produces descriptions and explanations that are in conformity, one has glimpsed a truth. When such descriptions and explanations are at variance, something is amiss, and truth is not identified.



References

- 1) Bandak FA: Shaken baby syndrome: a biomechanics analysis of injury mechanisms. *Forensic Sci Int* 151: 71-79, 2005
- 2) Caffey J: On the theory and practice of shaking infants. Its potential residual effects of permanent brain damage and mental retardation. *Am J Dis Child* 124: 181-169, 1972
- 3) Caffey J: The whiplash shaken infant syndrome: Manual shaking by the extremities with whiplash-induced intracranial and intraocular bleeding, linked with residual permanent brain damage and mental retardation. *Pediatrics* 54: 396-403, 1974
- 4) The Child Abuse Prevention and Treatment Act (CAPTA) of 1974, Public Law No 93-247 (January 31, 1974)
- 5) Cunningham F, Hauth J, Leveno K, Gilstrap L III, Bloom S, Wenstrom K: *Williams Obstetrics*, ed 22. New York, McGraw-Hill Medical Publishing Division, 2005, section V: p 682
- 6) Donohoe M: Evidence-based medicine and shaken baby syndrome: part I: literature review, 1966-1998. *Am J Forensic Med Pathol* 24(3): 239-242, 2003
- 7) Dubaime A, Gonnarelli T, Thibault L, Bruce D, Margulies S, Wisner R: The shaken baby syndrome. A clinical, pathological, and biomechanical study. *J Neurosurg* 68: 400-415, 1987
- 8) Guthkelch AN: Infantile subdural haematoma and its relationship to whiplash injuries. *Br Med J* 2(759): 430-431, 1971
- 9) Holbourn AH: Mechanics of head injuries. *Lancet* 9: 438-441, 1943
- 10) Holbourn AH: The mechanics of trauma with special reference to herniation of cerebral tissue. *J Neurosurg* 1: 191-200, 1944
- 11) Ito H, Komai T, Yamamoto S: Fibrinolytic enzyme in the lining walls of chronic subdural hematoma. *J Neurosurg* 48: 197-200, 1978
- 12) Ito H, Yamamoto S, Komai T, Mizukoshi H: Role of local hyperfibrinolysis in the etiology of chronic subdural hematoma. *J Neurosurg* 45: 26-31, 1976
- 13) Kawakami K, Chikama M, Tamiya T, Shimmamura Y: Coagulation and fibrinolysis in chronic subdural hematoma. *Neurosurgery* 25: 25-29, 1989
- 14) Leestma JE: Case analysis of brain-injured admittedly shaken infants: 54 cases, 1980-2001. *Am J Forensic Med Pathol* 26(3): 199-212, 2005
- 15) Merriam-Webster Online Dictionary, Springfield, Mass, Merriam-Webster, Inc. ©2005. Odyssey (noun). Available from: <http://www.m-w.com/dictionary.htm>
- 16) Ommaya AK, Faas F, Yarnall P: Whiplash injury and brain damage. *JAMA* 204: 75-79, 1968
- 17) Prange M, Coats B, Duhalme A, Margulies S: Anthropomorphic simulations of falls, shakes, and inflicted impacts in infants. *J Neurosurg* 99: 143-150, 2003
- 18) Weber W: [Experimental studies of skull fractures in infants]. *Z Rechtsmed* 92: 87-94, 1984 (Ger, with Eng abstract)
- 19) Weber W: [Biomechanical fragility of the infant skull]. *Z Rechtsmed* 94: 93-101, 1985 (Ger, with Eng abstract)
- 20) Weber W: [Predilection sites of infantile skull fractures following blunt force]. *Z Rechtsmed* 98: 81-93, 1987 (Ger, with Eng abstract)
- 21) Yamashima T, Yamamoto S, Friede R: The role of endothelial gap junctions in the enlargement of chronic subdural hematomas. *J Neurosurg* 59: 298-303, 1983

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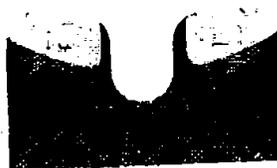


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Can Shaking Alone Cause Fatal Brain Injury? A biomechanical assessment of the Duhaime shaken baby syndrome model

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ABSTRACT

A biomechanical model of a one-month old baby was designed and tested by Duhaime and co-workers in 1987 in an attempt to assess the biomechanics of the shaken baby syndrome (SBS). The study implied that pure shaking alone cannot cause fatal head injuries, a factor which has been applied in criminal courts. In an attempt to test the validity of the model a preliminary study was undertaken in which a replica was constructed and tested. The broad description of the design and construction of the Duhaime model allowed for variations and therefore uncertainties in its reproduction. It was postulated therefore that differences in certain parameters may increase angular head accelerations. To further investigate this observation, an adjustable replica model was developed and tested.

The results indicated that certain parameter changes in the model did in fact lead to an increase in angular head acceleration. When these parameter changes were combined and an injurious shake pattern was employed, using maximum physical effort, the angular head acceleration results exceeded the original Duhaime et al. (1987) results and spanned two scaled tolerance limits for concussion. Additionally, literature suggests that the tolerance limits used to assess the shaking simulation results in the original study may not be reliable. Results from our study were closer to the internal head injury, subdural haematoma, tolerance limits. A series of end point impacts were identified in the shake cycles, therefore, an impact-based head injury measure (Head Injury Criterion – HIC) was utilized to assess their severity. Seven out of ten tests conducted resulted in HIC values exceeding the tolerance limits (critical load value, Stürztz, 1980) suggested for children.

At this present stage the authors conclude that it cannot be categorically stated, from a biomechanical perspective, that pure shaking cannot cause fatal head injuries in an infant. Parameters identified in

this study require further investigation to assess the accuracy of simulation and increase the biofidelity of the models before further conclusions can be drawn. There must now be sufficient doubt in the reliability of the Duhaime et al. (1987) biomechanical study to warrant the exclusion of such testimony in cases of suspected shaken baby syndrome.

INTRODUCTION

A clinical, pathological, and biomechanical study of the shaken baby syndrome (SBS) was conducted by Duhaime et al. in 1987. The biomechanical study required volunteers to 'violently' shake an infant model. The angular head acceleration output values did not exceed scaled tolerance limits for fatal infant brain injury, thus suggesting that pure shaking by a human cannot cause fatal brain injury in young infants. However, other clinical and pathological studies provide evidence to the contrary, suggesting that pure shaking (without an associated impact) can cause death in young infants (Hadley et al., 1989; Alexander et al., 1990; Gilliland and Folberg, 1996).

The biomechanical response characteristics of young infants were not investigated or modelled in the Duhaime et al. (1987) study. Although infant response data is sparse, there are some guidelines (scaled from adults) available from biomechanical studies within the automotive industry. Preliminary experiments conducted by the authors suggest that slight changes in the mechanical parameters of the Duhaime model might affect the angular head acceleration values obtained during shaking

experiments. This paper documents a re-investigation of the Duhaime et al. (1987) biomechanical model.

BACKGROUND

Guthkelch first described the mechanism of injury now known as the shaken baby syndrome in 1971. In 1972 Caffey described the shaking of infants and suggested as part of his conclusions that 'there are several features of infantile subdural haematomas which indicate that they are not usually caused by direct impact injuries to the head, but are caused by indirect acceleration-deceleration traction stresses such as whiplash-shaking of the head. These include bilaterality of subdural haematomas in 85% of infants (Ingraham and Matson, 1954) and frequent bilateral retinal haemorrhages. There is a striking lack of signs of impact injuries such as blows to the head. Usually there are no bruises to the face or scalp, no subperiosteal cephal-haematomas, and no fractures of the calvaria' (sic).

Duhaime et al. (1987) suggested that a history of shaking was usually lacking in so called 'SBS cases'. They reviewed all cases of SBS at the Children's Hospital of Philadelphia between January 1978, and March 1985. Thirteen of 48 suspected shake injuries resulted in fatalities, all had evidence of blunt head trauma at autopsy. The authors commented of their study that 'it is of interest that in more than half of our fatal cases, no evidence of external trauma was noted on the initial physical examination, which helped to contribute to the diagnosis of "shaken baby syndrome".'

The biomechanical section of the study consisted of a model of a one-month-old infant (with three different neck sections and two head types) instrumented with an accelerometer transducer to measure peak tangential head acceleration. Volunteers were asked to violently shake and subsequently to impact the rear of the head (occiput) of the model against a metal bar and a padded surface. The head acceleration output values were compared with an acceleration tolerance curve, scaled from animal experiments to the brain mass of an infant (500 gm). Duhaime et al. (1987)

concluded that 'the angular acceleration and velocity associated with shaking occurs well below the injury range, while the values for impacts span concussion, subdural [haematoma], and diffuse axonal injury ranges. This was true for all neck conditions with and without skulls'. 'It is our conclusion that the shaken baby syndrome, at least in its most severe acute form, is not usually caused by shaking alone.'

Duhaime et al. (1987) suggest that 'although shaking may, in fact, be part of the process, it is more likely that such infants suffer blunt impact' (that is, a shake and then an impact against a crib or other surface).

Literature on the shaken baby syndrome often fails to distinguish between 'pure shaking' and shaken impact baby syndrome. Therefore, for the purposes of this paper the SBS is defined as the manual shaking of an infant *with or without* associated head impact. Pure shaking describes the shaking of an infant *without* an impact of the head to an object, i.e. not excluding the impact of the chin-to-chest or occiput-to-back of the infant.

Bruce and Zimmerman (1989) summarised the biomechanical findings of Duhaime et al. (1987) and supported their conclusion, commenting that 'the physician should be suspicious of a report of pure shaking, as this is an unlikely scenario. If criminal prosecution is to occur, the charges should include impact injury as well as shaking'.

Hadley et al. (1989) commented on Duhaime et al's (1987) findings and conducted a study to investigate the problem further. All cases of non-accidental head injuries in infants were examined, with particular attention given to those with neurological symptoms, but no evidence of direct cranial trauma. All the patients defined as being in the 'isolated whiplash-shake injury subgroup' were very young infants (14 months or under). Hadley et al. (1989) pointed out that 'five of six patients on whom an autopsy was performed had no post-mortem evidence of direct cranial trauma'. The authors suggested that 'the discrepancies between this review and that... by Duhaime et al. (1987) may be explained in part by the younger age of the patients in our series

(median age, three months; range 1.5 to 14 months) versus theirs (median age, ten months; range three to 24 months). The high incidence of subdural haematoma in our 13 whiplash-shake injury patients and the high incidence of cervical cord injury in the six patients in whom autopsy was performed presumably also relates to the very young, immature patients in the population in our study'.

Hadley et al. (1989) suggest a note of caution in the argument for shaken-impact commenting that 'direct cranial trauma is not always required. We believe that a subgroup of patients, particularly the very young, may sustain severe neurological morbidity and mortality from rapid acceleration-deceleration injuries incurred from a whiplash-shake mechanism alone'.

Gilliland and Folberg (1996) reviewed a number of studies suggesting 'as more reports of systematic and ocular findings at death have been described it has become evident that many of the babies believed to have been shaken have suffered impact injuries (Duhaime et al., 1987; Rao et al., 1988; Hadley et al., 1989; Alexander et al., 1990; Elner et al., 1990; Massicotte et al., 1991; Munger et al., 1993; Bunde et al., 1994). To investigate whether shaking without direct head trauma is sufficient to inflict a lethal injury in infants Gilliland and Folberg (1996) reviewed findings from a large series of child deaths. The previously referenced studies indicated that 'many' and therefore not all 'of the babies believed to have been shaken have suffered impact injuries'. Their results (Gilliland and Folberg, 1996) showed that 'nine (11.3%) of the 80 head-injury deaths met the definition of death by the exclusive shaking mechanism of injury'. They suggested that their study 'confirmed earlier observations that some shaken babies do not have evidence of blunt head injuries (Rao et al., 1988; Hadley et al., 1989; Alexander et al., 1990; Massicotte et al., 1991; Munger et al., 1993; Bunde et al., 1994)'.

Another study by Jacobi (1986) was discussed where three infants (mean age six-months) were fatally injured from shaking alone (defined clinically). Gilliland and Fol-

berg (1996) suggested that 'shaking alone was not as often fatal as direct impacts but it was a lethal mechanism of injury in the child abuse deaths in children in Jacobi's study (1986)'.

Gilliland and Folberg (1996) criticise the biofidelity (i.e. human-like characteristics) of the Duhaime biomechanical model and suggest that 'the Duhaime et al. (1987) model is inadequate to explain death in children with no scalp and skull injuries after complete autopsy examination'.

Alexander et al. (1990) found that five out of nine of the fatally injured infants in their study had no evidence of impact at autopsy. The authors point out that 'the argument has been made that children with intracranial injuries but without detectable signs of external head trauma may have suffered an impact with a padded surface, such as a cushion or crib mattress, and this impact caused an intracranial injury. This cannot be completely refuted'... 'providing the burden of proof would seem at this point to fall to those who claim that impact must be present in all instances of serious intracranial injury'.

The results from the Duhaime biomechanical simulation are used to suggest that pure shaking has been shown to produce insufficient force to cause fatal head injury in infants. These results are sometimes used in alleged shaken baby syndrome cases to defend an assailant's actions, suggesting that even if shaking did occur, shaking alone cannot cause death in infants. However, Duhaime et al. (1987) did not categorically state this, they suggested that 'the shaken baby syndrome, at least in its most severe acute form, is *not usually* caused by shaking alone'. In 1996, Duhaime further attempted to clarify the conclusions drawn from the 1987 biomechanical simulation, stating that the researchers 'never said that beyond a shadow of a doubt shaking cannot cause injuries'. What they said was that 'shaking, at least with this model, produces angular decelerations which are too small to cause the target injuries for which there are established thresholds'. Again in 1998 Duhaime et al. suggested that 'although there is considerable controversy, the available evidence suggests that it is the sudden

deceleration associated with the forceful shaking of the head against a surface that is responsible for most, if not all, severe, inflicted brain injuries'. Again there is no categorical statement that pure shaking cannot ever produce fatal head injuries.

EXPERIMENTAL METHODOLOGY

1. Preliminary Investigations

A preliminary study was conducted which aimed to replicate the Duhaime model and allow direct observations of the biomechanical study, highlighting any problems associated with the construction and testing of the model. Analyses were also conducted on the reproducibility and repeatability of the experiments.

A second preliminary study involved a kinematic analysis of variation in the individual shaking styles/patterns. The study permitted an analysis of the effects of an increase in mass (corresponding to an increase in age of the infants) on volunteers' input accelerations to the model body. The results from both studies assisted in the formulation of a methodology for the main study.

During the construction, testing and analysis of the replica Duhaime model it was observed that volunteers demonstrated more varied shake patterns than just the standard anteroposterior (A-P) shake, suggested by Duhaime et al. (1987).

Head acceleration values derived from the replica study were higher than those recorded by Duhaime et al. (1987). In addition, some of the variant shake patterns demonstrated higher head accelerations than the A-P shake pattern.

To fully investigate the variation in volunteer shaking patterns, found during the preliminary study, a kinematic analysis was conducted using additional volunteers. Three models were constructed corresponding to the respective mean mass values of a one month-old, seven month-old and 18 month-old child. Eleven volunteers were requested to shake the model with maximum physical effort but no instruction was given as to the shake style or pattern to be adopted during testing. The study illustrated that individuals who receive no instruction as to how to shake the model

adopt very different patterns and in fact do not always adopt the anteroposterior shake pattern suggested by Duhaime et al. (1987). The accelerations achieved by all volunteers were reduced as the mass of the model (corresponding to a greater infant age) was increased. This suggested that the findings of Duhaime et al. (1987) should be interpreted only after the consideration of the mass of each individual child. For example, if an infant weighs 10 kg the findings of Duhaime et al. (1987) cannot be directly applied, since the level of input and body acceleration during shaking is likely to be reduced due to the greater mass of the infant. It is therefore imperative that research findings are applied appropriately in any given case.

It is noteworthy that during the shaking tests all seven female volunteers and some male volunteers showed signs of severe fatigue after ten seconds of violent shaking and reported that they would have found it difficult to continue for much longer. Therefore, further work may prove that the shake durations postulated to cause brain injury, sometimes suggested to be as long as one to two minutes, might be physically difficult to achieve.

In addition to the potential variability in acceleration values introduced by different shake patterns, it became apparent, during the production and testing of the Duhaime replica model, that there was considerable potential for variation in the way the model could be constructed from the vague description provided in the Duhaime et al. (1987) paper. It was suspected that the biomechanical characteristics of the neck, centre of gravity, chest and back padding and neck insertion point of the model might influence the level of peak angular head acceleration produced.

To assess the relative significance of each of the biomechanical parameters, an adjustable model was designed and constructed which allowed these pre-defined parameters to be changed from a standard model. Once the model was constructed a parametric study was conducted to ascertain the effect of changes to the model on the peak angular head acceleration, and therefore head injury level, during shaking tests.

2. Main study

The aim of the main study was to investigate:

'What factors significantly affect the angular head acceleration of a bio-mechanical model constructed to simulate the shaken baby syndrome?'

The adjustable model was designed to fulfil the following design specifications:

- The standard model must be a replica of the Duhaime et al. (1987) model.
- The model must be designed to allow replacement of the standard head with a modified head with a more realistic neck insertion point.
- The model must facilitate the changing of neck type from thin rubber neck to thick rubber neck and metal hinge neck.
- The model must allow for the adjustment of the mass attached to the thorax thus moving the centre of gravity of the body.
- The model must facilitate the changing of chest and back material.

Head

Standard head

The design and construction of a standard head followed the methods documented by Duhaime et al. (1987). In addition, it incorporated a metal collar to facilitate the attachment of the head to various neck configurations. The head-neck collar was placed in the desired position and resin (Fastcast, polyurethane UREOL 5202-1 A/B plus Filler DT 082, supplied by John Burn & Co. Ltd. (Birmingham, UK) was mixed and poured around the collar to secure it within the neck cavity of the model head. The final head mass (including head/neck collar) was 830 gm which fell within the range (770–870 gm) suggested by Duhaime et al. (1987). Head dimensions approximated to those of the Duhaime model.

Modified head

A modified head was constructed using the same methodology as the standard head. However, the head-neck collar was attached further inside the head than with the standard head at an accurate insertion point i.e. at the

OC junction (occipital condyles). The insertion position was estimated by scaling to the dimensions of a one-month-old infant's head using dimensions from anthropometric databases (Snyder et al., 1977; Norris and Wilson, 1995) and the actual dimensions of the model head using a method documented in Irwin and Mertz (1997) on the CRABI dummy (Child Restraint Air Bag Interaction). The head was cut to the required size (Cory, 2001) and shape to allow the head-neck collar to be placed in the required position. Resin was again used to hold the collar in place and to seal the remaining open parts of the head.

Necks

Three neck types were constructed to replicate those described by Duhaime et al. (1987), two rubber tube types and a metal hinge neck.

Rubber necks

The material used in the original study was 'commercially available hollow red rubber tubing' (Duhaime, 1998). The material used in the adjustable model was 'tubing-red rubber' supplied by Philip Harris-Scientific (Cardiff, U.K.). Neck dimensions approximated to those of the Duhaime model. As with the original model the thin rubber neck did not support the weight of the head in the upright position but did not kink when the head was allowed to fall unsupported. The thick rubber neck was able to support the weight of the head in the vertical position but allowed full passive movement of the head.

Both necks were cut to 4 cm (Duhaime et al., 1987) and mild steel tube plugs were bonded (using Araldite rapid adhesive) into ends of each neck to facilitate the attachment of the head to the neck and the neck to the thorax of the model. Once the adhesive had set, holes were drilled through each side of the neck and plug and screws were used to improve the strength of the join.

Metal hinge neck

The metal hinge neck was a replica of that used in the original study. It was a '360-degree steel hinge, 3.6 cm in width, placed in a horizontal plane to allow complete anteropos-

Table 1. Description of each model configuration for the shake tests.

Model name	Neck type	Position of centre of gravity from the head	Chest and back padding	Head type	Shake type
Standard model	Thin rubber neck (medium resistance)	24 cm	Silicone	Standard head	Standard anteroposterior – low physical effort
Thick rubber neck model	Thick rubber neck (high resistance)	24 cm	Silicone	Standard head	Standard anteroposterior – low physical effort
Hinge neck model	Hinge neck (low resistance)	24 cm	Silicone	Standard head	Standard anteroposterior – low physical effort
High centre of gravity model	Thin rubber neck (medium resistance)	22 cm	Silicone	Standard head	Standard anteroposterior – low physical effort
Cotton wool chest and back model	Thin rubber neck (medium resistance)	24 cm	Cotton wool	Standard head	Standard anteroposterior – low physical effort
Modified head model	Thin rubber neck (medium resistance)	24 cm	Silicone	Modified head	Standard anteroposterior – low physical effort
Parameter combination model	Hinge neck (low resistance)	22 cm	Cotton wool	Standard head	Gravity assisted – maximum physical effort

terior angulation of the head. The centre of rotation was 3.3 cm below the estimated level of the skull base (approximating the C-6 vertebral level) (Duhaime et al., 1987). The total hinge neck length was 4 cm. A nylon sleeve was added to the inside of the hinge to reduce the effects of friction.

Body

Frame

The design of the frame facilitated the attachment of body masses, different neck types, shoulder and chest and back pouches. Anthropometric studies were consulted for approximate dimensions for shoulder width and shoulder to buttock height for the one-month-old age group (Snyder et al., 1977; Steenbekkers, 1993; Pheasant, 1988; Beusenberget al., 1993).

A T-bar design was utilised with an upper horizontal (shoulder) section and a midline vertical (vertebral) section. Body masses were constructed to add to the T-bar to ensure total body mass of 3-4 kg (Duhaime et al., 1987) and to enable the centre of gravity of the model to be adjusted from a more central position (standard model mass configuration) to a higher centre of gravity.

Shoulder, chest and back pouches

A similar shape and material (cotton) to the Duhaime model was used to design and construct the body section for the adjustable model.

The two types of chest and back padding were cotton wool and silicone. The cotton wool was 50% cotton and 50% viscose and the silicone was a two-part system (Burnco Silicone rubber with catalyst type F (fast), supplied by John Burn & Co. Ltd. Birmingham, U.K.).

Details of the model configurations can be found in Table 1. More detailed information on the design and construction of the model can be found in Cory (2001).

Instrumentation and data capture

An accelerometer (Bruel & Kjaer type 4369) was attached to the thorax of the adjustable model (sampling at 5000 Hz) to record acceleration and thus allow the subsequent calculation of velocity. In accordance with the Duhaime model, tangential head acceleration was recorded utilising a piezoelectric accelerometer (PCB Piezotronics Model no. 339B10) attached to the vertex in a coronal plane through the centre of the neck. *Acknowledge*

(BIOPAC Systems, Inc., CA 93117, USA) software was utilised for analysing the accelerometer data.

Test Procedures

Shake type and holding pattern

In the first series of tests (parametric tests) the volunteer was asked to hold the model at a position equivalent to under the arms of an infant and shake it in an anteroposterior direction keeping the shake pattern the same throughout all tests. The volunteer adopted a low acceleration shake type to allow multiple consecutive controlled tests and to maintain the shake characteristics as uniform and constant as possible. All test procedures were the same for each parameter change on the adjustable model.

In a second series of tests the model was configured such that the parameters which had produced the greatest accelerations individually were combined and a model constructed which would produce the greatest acceleration as a whole (the worst case scenario). In addition, the volunteer was asked to use a shake pattern, which had been shown to produce the greatest level of acceleration, the 'gravity assisted' shake pattern. In the 'gravity assisted' shake pattern the arms are extended such that the model is elevated above one shoulder and accelerated downwards to below waist level (using gravity to assist). This results in the back of the head (occiput) impacting with the back of the model. The volunteer's arms are then pulled upwards, returning to the original position above the shoulders (with the volunteer's head tilted to avoid collision with the model) and inwards to induce chin-chest impact at the opposite endpoint of the shake cycle.

RESULTS

A series of shaking tests were conducted to determine significant biomechanical parameters on an adjustable model of an infant. Although the volunteer was instructed to shake the model in the same manner for each test, some variation from test to test was expected. Therefore, the variation between

tests in peak body acceleration (i.e. the input acceleration imparted to the body of the model by the volunteer) may affect the peak angular head (output) acceleration (i.e. acceleration of the head of the infant model). A rationalising method was employed to account for the slight increase/decrease in body acceleration. The ratio of the two values was calculated [peak head acceleration (rad/s^2)/ peak body acceleration (m/s^2)]. This 'ratio' value resolves the differences being attributed to a change in input acceleration and not parameter change. Therefore, the 'ratio' value was utilised to assess the statistical significance of parameter changes to the model.

Ratio

The Levene's test was used to test for differences in variance between the standard model ratio results and ratio values for models with each subsequent parameter change. The appropriate Student's t-Test (that is, for equal or unequal variance) was then used to test for significant differences between the standard model ratios and each set of ratio results for every parameter change. Table II shows the ratio value and the t-Test results for each parameter change.

Figure 1 shows results for the parametric study plotted in the ratio format, that is, angular head acceleration (rad/s^2) against linear body acceleration (m/s^2).

Adjusting all significant parameter changes-parameter combination model

It can be seen from the results presented in Table II that the hinge neck, high centre of gravity and the cotton wool chest and back models showed significant differences to the 'ratio' values, from the standard model configuration. Therefore, as documented in Table I the parameter combination model included the hinge neck, high centre of gravity and cotton wool chest and back. The shaking type was changed to 'gravity assisted' with maximum physical effort from the shaker.

Figure 2 shows results for the parameter combination model plotted in the format used by Duhaime et al. (1987), that is, angular head acceleration (rad/s^2) against angular head

Table II. Statistical results for ratio values of standard model tests compared to ratio values for each adjusted parameter test.

Model type	Mean (ratio)	Standard deviation	Variance	T-test used	T-test result (P value) when compared to standard model	Significant difference to standard model (P<0.05)
Standard model	31.01	4.30	18.51	-	-	-
Thick rubber neck model	32.50	3.02	9.13	Assuming unequal variances	0.128	No
Hinge neck model	72.09	13.44	180.55	Assuming unequal variances	0.000	Yes
Higher centre of gravity neck model	34.18	3.44	11.80	Assuming unequal variances	0.003	Yes
Cotton wool chest and back model	42.54	4.27	18.23	Assuming equal variances	0.000	Yes
Modified head model	32.72	4.12	16.97	Assuming equal variances	0.121	No

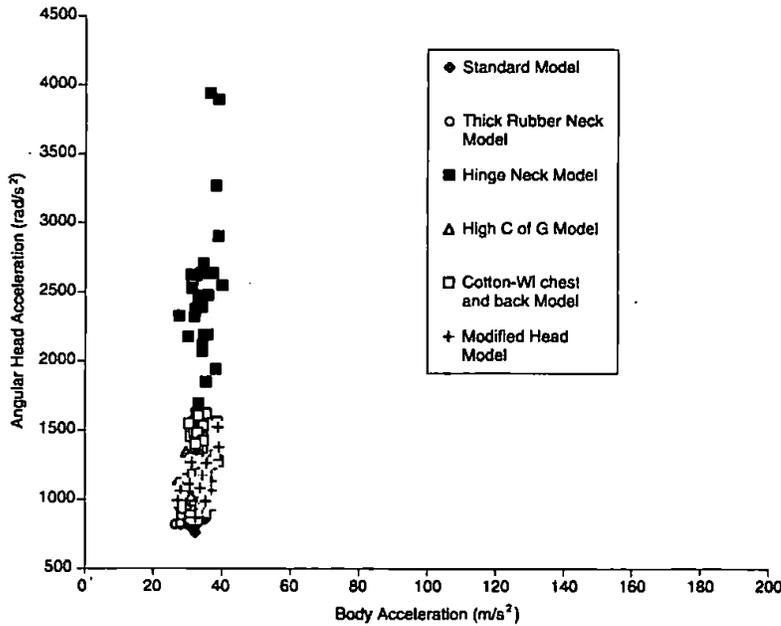


Figure 1. Adjustable model results showing effect of each parameter change on angular head acceleration (rad/s²).

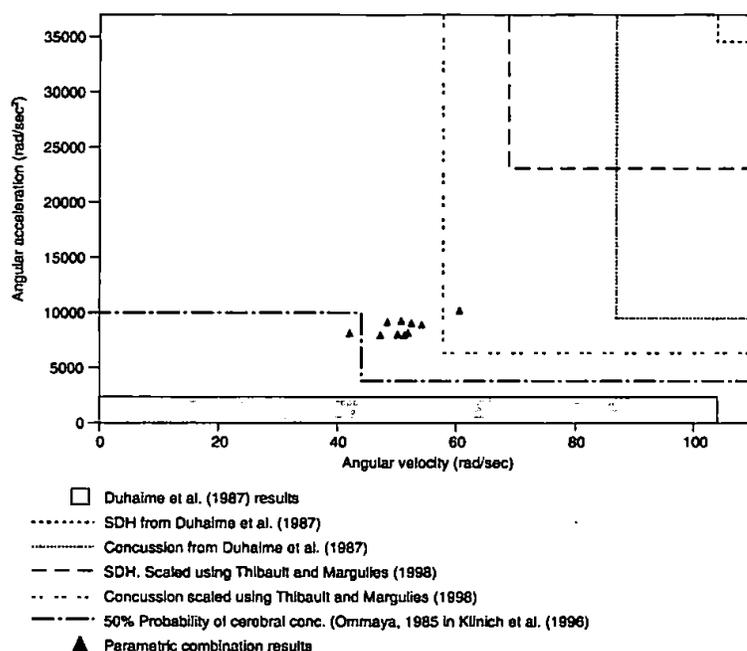


Figure 2. Shaking test results for parameter combination model showing comparison with Duhaime et al. (1987) results and tolerance limits for concussion and Subdural Haematoma (SDH) scaled for a one-month-old infant.

velocity (rad/s). The shaded region on the graph indicates the range of results from the original 1987 Duhaime et al. study. The internal head injury tolerance limits used by Duhaime for concussion and subdural haematoma are shown along with more recent tolerance limits suggested by Thibault and Margulies (1998). Also, another tolerance limit for 50% probability of onset of cerebral concussion (Ommaya, 1985) scaled using the method documented by Klinich et al. (1996), for a one-month-old's head dimensions is shown. All tolerance values were derived from animal studies.

Table III shows the results recorded from the body and head accelerometer data and the calculated Head Injury Criterion (HIC) values for the parameter combination model shake tests.

RELEVANT BIOMECHANICAL RESEARCH FOR ASSESSING SHAKEN BABY SYNDROME SIMULATION RESULTS

Our preliminary studies show that shaking the model produced both chin-to-chest and back of head (occiput)-to-back impacts. If this were to occur in infants during shaking, that is, if it is anatomically possible, it will have profound implications to the argument that currently ensues as to whether pure shaking alone can produce fatal head injury, since it introduces a series of end-point impacts.

Other evidence of end-point impacts

Other studies have reported end-point impacts. Janssen et al. (1991) conducted physical crash tests using a TNO (the Netherlands Organisation for Applied Scientific Research)

Table III. Results recorded from the body and head accelerometer data and the calculated Head Injury Criterion (HIC) values for the parameter combination model shake tests.

Test-Name	Results for model head					Results for model body			
	Peak tangential head AccN (m/s ²)	Peak angular head AccN (rad/s ²)	Peak tangential head velocity (m/s)	Peak angular head velocity (rad/s)	Time duration of peak head curve (ms)	Head Injury Criterion (HIC)	Peak body AccN (m/s ²)	Peak body velocity (m/s)	Time duration of peak body curve (ms)
Para-1	1385.47	8149.80	7.16	42.13	13	684	71.51	3.53	103
Para-2	1577.35	9278.53	8.63	50.78	11	1001	91.33	6.18	129
Para-3	1558.02	9164.85	8.24	48.47	11	952	96.14	5.89	121
Para-4	1738.86	10216.83	10.30	60.59	15	1421	73.97	4.02	144
Para-5	1515.65	8915.66	9.22	54.24	17	1039	67.98	4.02	120
Para-6	1366.93	8040.74	8.53	50.20	17	823	54.74	4.61	161
Para-7	1389.10	8171.15	8.83	51.94	18	866	79.66	5.69	174
Para-8	1353.00	7958.80	8.04	47.32	18	735	66.81	5.49	136
Para-9	1542.92	9075.98	8.93	52.51	17	1008	71.22	5.98	143
Para-10	1353.58	7962.26	8.73	51.36	18	846	64.06	5.79	172
Mean	1488.16	8693.45	8.66	50.95	16	938	73.74	5.12	140

P 3/4 (nine-month-old) child dummy. High-speed film analysis of the tests showed that the dummy's chin impacted the upper torso. These impacts not only affected the accelerations of the dummy's head, but also influenced the induced neck forces'. In parallel with the experimental work, a 2D Mathematical Dynamics Model (MADYMO) of the P 3/4 (nine-month-old) dummy was used to obtain a better understanding of the response of the child physical dummy. 'Particular emphasis was placed on analysing the effect of chin-to-chest contacts...'

It is noteworthy that end point impacts, chin-to-chest and occiput-to-back, will produce increased tensile neck forces. Recently published work by Geddes et al. (2001a) documents the disruption of the craniocervical junction from both pure shaking and shaken-impact scenarios. 'Our study shows that infants of two to three months typically present with a history of apnoea or other breathing abnormalities, show axonal damage at the craniocervical junction, and tend also to have a skull fracture [not in the 'shaken-only' cases], a thin film subdural haemorrhage, but lack extracranial injury'. All reported cases

were fatal as the study was conducted on post-mortem subjects. Geddes et al. (2001b) suggest that 'it may not be necessary to shake an infant very violently to produce stretch injury to the neuraxis. It is true that the more vigorous the shaking, the greater the stretch that would take place at the extremities of movement, and the worse the damage produced'.

Duhaime (1996) suggested the 'impact' of the back of the head (occiput)-to-back as a possible injury mechanism in the shaken baby syndrome. Commenting 'that much of the injury, subdural bleeding, occurs because, when the back of head strikes, the open lambdoid suture in infants indents into the brain causing tissue strains at the posterior deep bridging veins (the vein of Gallen, internal cerebral veins and straight sinus). Much of the bleeding seen, especially in the posterior interhemispheric fissure, has nothing to do with angular acceleration/deceleration, but is due to failure of the deep draining veins in the centre of the brain caused by an occipital impact. If that is the case, everything that was done with respect to injury thresholds may be wrong. But it's still impact'.

Problems associated with scaling tolerance limits

It is not clear from Duhaime et al. (1987) exactly which studies were utilised to scale from primates to infants to obtain tolerance values for pure angular head acceleration. However, Gennarelli et al. (1982) and Thibault and Gennarelli (1985) were referenced in the study and both studies used apparatus that ensured a purely angular acceleration was imparted to the subject. Additionally, in a personal correspondence with S.S. Margulies (1999) it was suggested that Gennarelli and Thibault (1982) was used as the source data for the subdural haematoma tolerance limits in the Duhaime et al. (1987) study. Gennarelli and Thibault (1982) commented of the apparatus used in the experimental set-up that 'the system provides nonimpact-distributed inertial loading conditions so that the effects of acceleration are studied in isolation'. Gennarelli et al. (1982) utilised apparatus that 'provided a nonimpact, distributed acceleration load to move the head in a controlled pathway'.

In a series of primate experiments they utilised a controlled mechanism (head in helmet linkage system) for inducing rotational (angular) acceleration of the head. The tests purposefully prevented the possibility of contact between the chin-to-chest or occiput-to-back. Even if the head were allowed free motion (as in the sled tests, Ommaya et al., 1967; Ommaya and Hirsch, 1971), structural and anatomical differences between adult primates and human infants may significantly reduce confidence in a direct comparison; for example, the relatively large neck muscles of adult primates may passively reduce head rotation and thus may prevent or greatly reduce any contact of the chin or back of the head (occiput) to the body. Therefore, an impact of the chin-to-chest or occiput-to-back type mechanism may not be intrinsic in animal tests.

If an impact or series of impacts of this type were to occur during pure shaking, impact tolerance limits should be used, based on impact tests, rather than the currently applied angular acceleration (shaking) data. This point can be illustrated by comparing thresh-

old values for these two mechanisms. For a pure 'impulsive/indirect' angular acceleration, scaled for a three-year-old's brain mass, values of 8140 rad/s² for 10 ms are applied as an injury threshold (scaled from Ommaya et al., 1967; Stürtz, 1980). However, for an impact-induced angular acceleration, values of 2008 rad/s² for 10 ms are applied (scaled from Ommaya and Hirsch, 1971; Stürtz, 1980). The different tolerance values reflect the fact that the brain is less tolerant to impact induced angular acceleration than it is to pure indirect angular acceleration.

Stürtz (1980) commented, of the direct values (induced by impact), scaled for three and six-year-olds, that 'because a child's skull is less rigid, the direct application of the force means a generally higher endangering of the child. Therefore, the derived values for children could still be considerably reduced'. With a one-month-old child this comment is even more relevant.

Duhaime et al. (1987) assessed their shaking and shaken impact results using a scale with tolerance limits for cerebral concussion, subdural haematoma, and diffuse axonal injury scaled from subhuman primates, for the brain mass of a one-month old infant. Duhaime et al. (1987) suggested 'a tolerance scale... has been developed for the subhuman primate by Thibault and Gennarelli (1985)'. The scaling relationship shown in Thibault and Gennarelli (1985) is referenced as 'the one proposed by Holbourn (1943)'.

Holbourn's (1943) method was also documented and discussed by Ommaya et al. in 1967 and in many further publications by Ommaya and other authors. Ommaya et al. (1967) commented of the method that it could be used for 'extending the results of experiments on concussion-producing head rotations on lower primate subjects to predict the rotations required to produce concussion in man'.

The scaling data was subsequently applied to the paediatric population by Stürtz (1980).

Recent research has raised questions about the applicability of the scaling laws when scaling from human adults to infants. Prange et al. (1999) investigated a number of assump-

tions made during Holbourn's original scaling (1943, 1956), documented by Ommaya et al. (1967). Discussing both their findings and those of Thibault and Margulies (1998), Prange et al. (1999) suggested that these 'data demonstrate that the assumption of identical material properties in the model and prototype used by Ommaya et al. (1967) does not hold true for scaling between paediatric and adult inertial head injury'. Additionally, they concluded that 'the geometry of the adult brain was also found to be significantly different from the infant brain. This shows that the assumption of similar geometry between infants and adults also fails'. The authors suggested that their research 'provides a foundation for the study of the unique etiology and pathophysiology of paediatric brain injury'.

Ommaya made a further assumption, that 'the skull is very stiff, such that deformations of the skull do not contribute heavily to the strains of the enclosed brain' (Ommaya et al., 1967). Problems may arise however, if this assumption is applied to the paediatric population since a more compliant paediatric skull may produce strains in the enclosed brain (Thibault and Margulies, 1998; Margulies and Thibault, 2000).

Another concern is that the tolerance limits used to assess engineering models of shaking are scaled from data collected during biomechanical research within the automotive industry, which are based on a single whiplash event or a single impact event. This may be quite unlike a shaking episode during child abuse, where there may be a greater number of shakes and impacts, the cumulative effects of which are not assessed within the tolerance limits scaled from animal surrogates and adult cadavers. The authors suggest that the possible breakdown in material properties of cranial bridging veins and nerve axons with repeated tensile and/or shear and/or compressive strains may increase the potential for injury, compared to that of a single insult.

It is also imperative to acknowledge that current biomechanical analyses are viewed only from a mechanical perspective and fail

to consider any subsequent pathophysiological consequence which may result from a primary injury.

Therefore, when tolerance limits are scaled to assess simulations involving infant models, they do not assess the risk of fatality considering all possible mechanisms of injury for the paediatric population, only the effects a single insult of pure angular head acceleration, that is, one possible mode of injury.

Also, if experimentation were to show a significant impact at the chin-to-chest and occiput-to-back, the authors suggest that future tests should consider both the effects of pure angular acceleration and impact (linear) acceleration using a head impact model, for example the Head Injury Criterion (HIC). If the impact tolerance limit were surpassed, even though the pure angular tolerance limits were not, the implication may be that fatal head injury may occur in pure shaking from injury mechanisms (chin-to-chest, occiput-to-back impacts) not identified by Duhaime's study in 1987. The effects of multiple impacts/shakes cannot be properly assessed until further research is conducted in this area. However, the authors suggest the potential damage of multiple impacts/shakes of the same acceleration magnitude would at least be equal to, and possibly even greater than, a single insult.

Head Injury Criterion (HIC)

Prasad and Mertz (1985), compared a collection of skull fracture and brain injury data with their corresponding HIC values and suggested that at an HIC value of 1000 there is a 16% risk of life-threatening brain injury in an adult population.

Stürtz (1980) simulated ten pedestrian accidents involving children (age not specified) using an anthropometric dummy. The simulations resulted in an HIC value of 840 being suggested as the critical load value of a child's head, rather than the 1000 value applied to adults. A 'critical load value' was defined as 'the load on the body under which an initial

considerable damage of the organism takes place – destruction of a cell; irreversible injury – for instance when bone fractures occur or primary organs rupture’.

Mode of shaking

In the Duhaime et al. (1987) study, models were ‘held by the thorax facing the volunteer and were shaken in the anteroposterior (A-P) direction, since this is the motion most commonly described in the shaken baby syndrome’. It must be noted that any minor deviation in this shaking pattern would result in the head moving in directions other than in the A-P plane. Since the measurement apparatus used in the original study was capable of measuring only in the A-P plane, measurement and assessment of even minor perturbations would require measurement apparatus capable of measuring head acceleration in those other directions. For example, if evidence were provided which suggested that a child was held so that its side was facing the assailant and the head accelerated laterally, this alternative mechanism would have to be considered.

Gennarelli et al. (1982) found that the direction of head motion (with acceleration remaining constant within narrow limits) was an important factor in producing the brain injury, Diffuse Axonal Injury (DAI), in primates, commenting, ‘of the sagittal [A-P] group, 85% had a good recovery, while of the lateral group 84% had persisting coma or severe disability’. Margulies and Thibault (1992) scaled DAI thresholds to lateral rotation for a 500 gm brain mass (i.e. a one-month-old) which indicate a lower tolerance to lateral rotational acceleration than the angular rotational acceleration also scaled to a 500 gm brain mass in the Duhaime et al. (1987) paper. Therefore, any deviation from the standard anteroposterior shake pattern could predispose an infant to a greater injury and, therefore, should be noted and included in any subsequent simulation and/or opinion.

For a thorough review and explanation of head impact injury models and tolerance values see Cory et al. (2001).

DISCUSSION

Effect of model parameter changes on ratio-results

- The ratio-results from the adjustable model tests showed that three factors, the metal hinge-neck, high centre of gravity and cotton wool chest and back padding parameters, significantly increase the ratio-results, compared to the standard model. These results emphasise a requirement that models for the investigation of the SBS simulate an infant as accurately as possible in terms of mass distribution (centre of gravity) and response (biofidelity of neck, chest and back).
- The three parameter changes, centre of gravity, neck type and chest and back padding, were adjusted on the same model and subjected to an increased shake effort and altered shake pattern. The shake tests produced results higher than those produced in the original Duhaime et al. (1987) study, also surpassing two scaled tolerance limits for concussion. On a cautionary note, evidence has arisen from literature that raises doubts about the validity of scaling tolerance limits from primates and adults to infants. It is possible that these limits are inaccurate to some unknown degree. As the results from the parameter combination model are closer to the limits (for subdural haematoma) than the Duhaime model results, this factor becomes more significant in the issue of whether pure shaking alone is capable of causing fatal head injuries to infants.

In the Discussion and Conclusion the ‘ratio-results’ are defined as dimensionless values, indicating a comparable level of angular head acceleration for all parameter changes.

Impact of chin-to-chest and back of head (occiput)-to-back

- The contact between the chin-to-chest and occiput-to-back of the model during shaking tests indicates that there may be another injury mechanism in ‘pure shaking’, not previously investigated. Evidence (Ommaya and Hirsh, 1971; Stürz, 1980) suggests that

if an impact occurs, before rotational (angular) acceleration, the head injury tolerance may be reduced, i.e. less additional rotational acceleration may be required, in addition to the impact, to cause a fatal brain injury.

- In light of the fact that there is an impact between the chin-to-chest and occiput-to-back during the shake tests conducted in this study, the angular acceleration tolerance limits may not be applicable in all shaking scenarios. The controlled mechanism of inducing the rotational (angular) acceleration of the head, in the previously mentioned primate tests, was designed to purposefully prevent the possibility of contact between the chin-to-chest or occiput-to-back. Since impact was clearly identified in the experimental series, for the parameter combination model, the results were assessed using an impact based tolerance limit (Head Injury Criterion (HIC)). Forty per cent of the results were over the HIC threshold of 1000 and 80% were over the HIC critical loading value of 840 suggested by Stürztz (1980) for children.

The term shaken baby syndrome (SBS) defines the assailant's actions on the model/infant, but it does not adequately describe all the possible mechanisms of injury. The term SBS has evolved to incorporate shaking and head impact(s) with an external object or surface. This study has suggested a series of impacts occur between the head and body during pure shaking, a back of head (occiput)-to-back impact, then a whiplash of the head, followed by a chin-to-chest impact. It may be possible to induce a purely angular acceleration of the head using different shaking mechanisms. However, it is suggested that in a frenzied attack (as simulated in the parameter combination tests), with many shake cycles, it is highly likely that endpoint impacts would occur. We are primarily concerned, in this study, with the question of causing *fatal head injury* during 'pure shaking' and therefore the main emphasis is on the worst case scenario.

It is suggested that when the 'pure shaking' of an infant is described in a biomechanical

context a better term, which adequately describes the most likely mechanism, would be impact-whiplash-impact (IWI). The authors would like to note that no suggestion is being made that this be added to the already abundant descriptions of the SBS entrenched in the medical literature. It is suggested that the term described above is more useful in the description of the biomechanical mechanisms present in violent 'pure shaking' scenarios.

Problems with scaling

- There is some evidence from literature that the assumptions made originally to scale from animals to adult humans do not necessarily apply to scaling from adults to infants. It is suggested that if these assumptions are not valid, when the scaling law is applied between human adults and human infants (Prange et al., 1999), they must also be invalid when scaling directly from primates to human infants.
- Thibault and Margulies (1998) have put forward a new method for scaling, based on the results of recent research on paediatric (surrogate) brains. These changes lower the tolerance limits for infants.
- The results were compared with tolerance limits from the automotive industry where the effects of a single event (whiplash and/or impact) were measured, rather than the multiple insults associated with the SBS. The cumulative effect of many shakes and/or impacts is unknown.

CONCLUSION

The 1987 Duhaime et al. study documented a biomechanical simulation of the shaken baby syndrome. During the study angular head accelerations surpassing fatal head injury tolerance limits could not be produced in infant models from shaking alone. The simulation was conducted over 16 years ago and although it was a valuable first step to a better understanding of the biomechanics of the shaken baby syndrome, because it has been widely applied in the courts and literature, the model was in need of re-assessment.

To test the validity of the model a preliminary study was undertaken in which a replica

was constructed and tested. The vague description of the design and construction of the Duhaime model in the original paper allowed for the possibility that variations might be introduced during its reproduction. It was postulated therefore, that differences in certain parameters might affect angular head accelerations.

The authors conducted a parametric study with an adjustable replica of the Duhaime model to answer the following question:

'What factors significantly affect the angular head acceleration of a bio-mechanical model constructed to simulate the shaken baby syndrome?'

Experimental results suggested that three parameters significantly increased the level of angular head acceleration: metal hinge-neck, high centre of gravity and cotton wool chest and back material.

These findings emphasise the requirement that future models of the shaken baby syndrome accurately simulate an infant, in terms of the biomechanics of mass distribution (centre of gravity) and response (biofidelity of neck, chin-to-chest and occiput-to-back contact points).

The angular head acceleration and velocity results from the parameter combination model, with increased shake effort and altered shake pattern, surpassed the Duhaime et al. (1987) results and spanned two scaled tolerance limits for concussion. As this adjustable replica 'Duhaime' model produces different acceleration values from the original study, it is evident that changing certain parameters affects angular head acceleration. However, it cannot be claimed that either model (i.e. the original Duhaime et al. (1987) model or the parameter combination replica Duhaime model) is biofidelic. It is currently unknown whether an improved level of biofidelity in some parameters would increase or decrease the angular head accelerations produced during pure shaking. However, it can be suggested that if these parameters do affect the results they must be designed to be as biofidelic as possible for reliable conclusions to be drawn.

The model produced in 1987 was very

simplicistic and was not designed to resemble a human infant in terms of mechanical response (biofidelity). In the last 16 years much research has been conducted in the area of modelling children using mechanical crash test dummies and computer models. It is now known that to run meaningful simulations, models must be based on appropriate data, to design and assess (calibrate) the head, neck, and thorax for biofidelity. The Duhaime et al. (1987) study has been widely quoted (197 times, Science Citation Index 2003) in other papers on the subject and is often quoted in SBS litigation. However, although it has been criticised in the literature it has not previously been properly reproduced and systematically assessed. The authors suggest that the current study has provided evidence to suggest that changes in the biomechanical properties of the model influence the results for angular head accelerations. Neglecting these factors produces a model with an unknown resemblance to an infant, therefore, any simulation results obtained with such a model will be meaningless and conclusions drawn unreliable.

The conclusions drawn from the current study emphasise the need for the design and construction of a biofidelic infant model to simulate shaking before results can be reliably quoted in the literature and/or applied in a court of law. However, there are many other problems associated with the Duhaime et al. (1987) biomechanical study and future studies on the biomechanics of the shaken baby syndrome:

- Evidence suggests that the tolerance limits used to assess the shaking simulation results in Duhaime et al. (1987) may not be reliable. The degree to which they are inaccurate is unknown.
- The results of all shake tests conducted during this study identified clear impacts at the chin-to-chest and occiput-to-back sections of the shake cycle. Therefore, the (possibly inaccurate) tolerance limits utilised by Duhaime et al. (1987) may not be applicable in the assessment of shaking simulations due to the impacts identified in this study.

- Although the current model was not bio-fidelic, the impact tolerance limits suggested for children were surpassed in 80% of the parameter combination model shake test results which indicates that endpoint impacts, if identified in future tests, should be assessed with impact-based tolerance limits.
- Even though 80% of the results surpass the impact tolerance limit the cumulative effect of multiple impacts (from many shake cycles) cannot be assessed as the tolerance limits are based on single impacts (as in car crash scenarios). Therefore, although some shaking/impact results might be below fatal head injury tolerance limits, the effect of repeated consecutive sub-lethal loading is unknown.

It is suggested that further research into the design, construction and assessment of a model for SBS research is required to develop a biofidelic infant model, in light of the research conducted and child data published since the Duhaime et al. (1987) study.

The application of data from animal surrogate experiments, adult cadaver experiments and scaling calculations has greatly assisted in overcoming the problems associated with the paucity of child data, when developing child-safe environments. However, extreme caution should be exercised when applying the data in a medico-legal context. In addition, since the Duhaime model has an unknown level of biofidelity, presentation of the study in evidence in any criminal prosecution runs the risk of its prejudicial effect outweighing its probative value and may result in any arbiter of fact wrongly interpreting the evidence.

Therefore, at this present stage, the authors conclude that it cannot be categorically stated, from the Duhaime et al. (1987) study, that 'pure shaking' cannot cause fatal head injuries in an infant. There must, therefore, be sufficient doubt in the reliability of the Duhaime et al. (1987) biomechanical study to warrant the exclusion of such testimony in cases of suspected shaken baby syndrome.

REFERENCES

- Alexander R., Sato Y., Smith W. and Bennett T. (1990) Incidence of impact trauma with cranial injuries ascribed to shaking. *AJDC* 144, 724-6.
- Beusenberg M.C., Happee R., Twisk D. and Janssen E.G. (1993) Status of injury biomechanics for the development of child dummies. *SAE Paper No* 933104.
- Bruce D.A. and Zimmerman R.A. (1989) Shaken impact syndrome. *Pediatr. Ann.* 18, 482-94.
- Bundez D.L., Farber M.G., Mirchandani H.G., Park H. and Rorke L.B. (1994). Ocular and optic nerve hemorrhages in abused infants with intracranial injuries. In: Gilliland M.G. and Folberg R. (1996). Shaken babies - some have no impact injuries. *J. For. Science* 41, 114-6.
- Caffey J. (1972) On the theory and practice of shaking infants: Its potential residual effects of permanent brain damage and mental retardation. *AJDC* 124, 161-9.
- Cory C.Z. (2001) A biomechanical assessment of the Duhaime shaken baby syndrome model. Doctor of Philosophy Thesis, University of Wales, Cardiff, U.K.
- Cory C.Z., Jones M.D., James D.S., Leadbeatter S. and Nokes L.D.M. (2001) The potential and limitations of utilising head impact injury models to assess the likelihood of significant head injury in infants after a fall. *Forensic Sci. Int.* 123, 89-106.
- Duhaime A.C., Gennarelli T.A., Thibault L.E., Bruce D.A., Margulies S.S. and Wisner R. (1987) The shaken baby syndrome: A clinical, pathological, and biomechanical study. *J. Neurosurg.* 66, 409-15.
- Duhaime A.C. (1996) Research on the pathophysiology of the shaking-impact syndrome. *The Pediatric Trauma and Forensic Newsletter* 4, 73-7.
- Duhaime A.C. (1998). Personal Correspondence.
- Duhaime A.C., Christian C.W., Rorke L.B. and Zimmerman R.A. (1998) Current Concepts: Non-accidental head injury in infants - The 'shaken-baby syndrome'. *N. Engl. J. Med.* 338, 1822-9.
- Elnor S.G., Elnor V.M., Arnall M. and Albert D.M. (1990). Ocular and associated systematic findings in suspected child abuse. In: Gilliland M.G. and Folberg R. (1996) Shaken babies - some have no impact injuries. *J. For. Science* 41, 114-6.
- Geddes J.F., Hackshaw A.K., Vowles G.H., Nickols C.D. and Whitwell H.L. (2001a) Neuropathology of inflicted head injury in children: I. Patterns of brain damage. *Brain* 124, 1290-8.
- Geddes J.F., Vowles G.H., Hackshaw A.K., Nickols C.D., Scott I.S. and Whitwell H.L. (2001b) Neuropathology of inflicted head injury in children: II. Microscopic brain injury in infants. *Brain* 124, 1299-306.
- Gennarelli T.A. and Thibault L.E. (1982) Biomechanics of acute subdural hematoma. *J. Trauma* 22, 680-6.
- Gennarelli T.A., Thibault L.E., Adams J.H., Graham D.L., Thompson C.J. and Marcincin R.P. (1982) Diffuse axonal injury and traumatic coma in the primate. *Ann. Neurol.* 12, 564-74.

- Gilliland M.G. and Folberg R. (1996) Shaken babies – some have no impact injuries. *J. For. Science* 41, 114–6.
- Guthkelch A.N. (1971) Infantile subdural haematoma and its relationship to whiplash injuries. *BMJ* 2, 430–1.
- Hadley M.N., Sonntag V.K.H., Rekate H.L. and Murphy A. (1989) The infant whiplash-shake injury syndrome: a clinical and pathological study. *Neurosurgery* 24, 536–40.
- Holbourn A.H.S. (1943) Mechanics of head injuries. *Lancet* 438–41.
- Holbourn A.H.S. (1956) Private Communication, 13th Oct. to Dr. Sabina Strich. In: Ommaya A.K., Yarnell P., Hirsch A. and Harris E. (1967). Scaling experimental data on cerebral concussion in subhuman primates to concussive thresholds for man. *Proc. 11th Stapp Car Crash Conf. Anaheim, CA*.
- Ingraham F.D. and Matson D.D. (1954). Neurosurgery of Infancy and Childhood. In: Caffey J. (1972). On the theory and practice of shaking infants: Its potential residual effects of permanent brain damage and mental retardation. *AJDC* 124, 161–9.
- Irwin A.L. and Mertz H.J. (1972) Biomechanical bases for the CRABI and Hybrid III Child Dummies. *Child Occupant Protection, 2nd Symposium Proceedings P-316, Orlando, Florida, SAE, November 12, Paper No. 973317*.
- Jacobi G. (1986). Damage patterns in severe child abuse with and without fatal sequelae. In: Gilliland M.G. and Folberg R. (1996). Shaken babies – some have no impact injuries. *J. For. Science* 41, 114–6.
- Janssen E.G., Nieboer R., Verschut R. and Huijskens C.G. (1991) Cervical spine loads induced in restrained child dummies. *35th Stapp Car Crash Conf., P-251, SAE, Paper No, 912919*.
- Klinich K.D., Saul R., Auguste G., Backaitis S. and Kleinberger M. (1996) Techniques for developing child dummy protection reference values. *Report by the child injury protection team, NHTSA, October 1996*.
- Margulies S.S. (1999) Personal Correspondence.
- Margulies S.S. and Thibault L.E. (1992) A proposed tolerance criterion for diffuse axonal injury in man. *J. Biomechanics* 25, 917–23.
- Margulies S.S. and Thibault K.L. (2000) Infant skull and suture properties: Measurements and implications for mechanisms of pediatric brain injury. *J. Biomechanical Engineering* 122, 364–71.
- Massicotte S.J., Folberg R., Torczynski E., Gilliland M.G.F. and Luckenback M.W. (1991) Vitreoretinal traction and perimacular retinal folds in the eyes of deliberately traumatized children. In: Gilliland M.G. and Folberg R. (1996). Shaken babies – some have no impact injuries. *J. For. Science* 41, 114–6.
- Munger C.E., Peiffer R.L., Bouldin T.W., Kylstra J.A. and Thompson R.L. (1993) Ocular and associated neuropathologic observations in suspected whiplash shaken infant syndrome. In: Gilliland M.G. and Folberg R. (1996). Shaken babies – some have no impact injuries. *J. For. Science* 41, 114–6.
- Norris B. and Wilson R. (1995) CHILDATA, The handbook of child measurements and capabilities – data for design safety, *DTI Consumer Safety Unit, June 1995, ISBN 0-9522 571-1-4*.
- Ommaya A.K., Yarnell P., Hirsch A. and Harris E. (1967) Scaling experimental data on cerebral concussion in subhuman primates to concussive thresholds for man. *Proc. 11th Stapp Car Crash Conf. Anaheim, CA*.
- Ommaya A.K. and Hirsch A.E. (1971) Tolerance of cerebral concussion from head impact and whiplash in primates. *J. Biomechanics* 4, 13–21.
- Ommaya A.K. (1985) Biomechanics of head injury: experimental aspects. In: Nahum A.M. and Melvin J. (eds.) *The Biomechanics of Trauma, N.J. Prentice-Hall*. pp.245–79.
- Pheasant S.T. (1986) Bodyspace: anthropometric ergonomics and design. In: Norris B. and Wilson R. (1995). CHILDATA, The Handbook of Child Measurements and Capabilities – Data for Design Safety, *DTI Consumer Safety Unit, June 1995, ISBN 0-9522 571-1-4*.
- Prange M.T. and Margulies S.S. (1999) Pediatric rotational brain injury: The relative influence of brain size and material properties. *Proc. of 43rd Stapp. Car Crash Conf. SAE, pp 333–41*.
- Prasad P. and Mertz H.J. (1985) The position of the United States delegation to the ISO Working Group 6 on the Use of HIC in the Automotive Environment. *SAE, Paper No. 851246*.
- Rao N., Smith R.E., Chou J.H., Xu X.H. and Kornblum, R.N. (1988) Autopsy findings in the eyes of fourteen fatally abused children. In: Gilliland M.G. and Folberg R. (1996). Shaken babies – some have no impact injuries. *J. For. Science* 41, 114–6.
- Snyder R.G., Schneider L.W., Owings C.L., Reynolds H.M., Golomb D.H. and Schork M.A. (1977) Anthropometry of infants, children, and youths to age 18 for product safety design, *UM-HSRI-77-17 Final Report Contract CPSC-C-75-0068 to Consumer Product Safety Commission, May 31. <http://www.itl.nist.gov/div894/ovrt/projects/anthrokids/anthrokids.html>*
- Steenbekkers L.P.A. (1993) Child development, design implications and accident prevention, No.1 in *Physical Ergonomics Series*. In: Norris B. and Wilson R. (1995). CHILDATA, The Handbook of Child Measurements and Capabilities - Data for Design Safety, *DTI Consumer Safety Unit, June 1995, ISBN 0-9522 571-1-4*.
- Stürtz G. (1980) Biomechanical data of children. *24th Stapp Car Crash Conf. Proc., SAE, Paper No. 801313*.
- Thibault K.L. and Margulies S.S. (1998) Age-dependent material properties of the porcine cerebrum: effect on pediatric inertial head injury criteria. *J. Biomechanics* 31, 1119–26.
- Thibault L.E. and Gennarelli T.A. (1985) Biomechanics of diffuse brain injuries. In: *Proc. of the Fourth Experimental Safety Vehicle Conference*. New York, American Association of Automotive Engineers.

Exhibit FF



Laboratory Study

Diffuse neuronal perikaryal amyloid precursor protein immunoreactivity in an ovine model of non-accidental head injury (the shaken baby syndrome)

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ABSTRACT

Non-accidental head injury ("shaken baby syndrome") is a major cause of death and disability in infants and young children, but it is uncertain whether shaking alone is sufficient to cause brain damage or an additional head impact is required. Accordingly, we used manual shaking in an ovine model in an attempt to answer this question since lambs have a relatively large gyrencephalic brain and weak neck muscles resembling a human infant. Neuronal perikaryal and axonal reactions were quantified 6 hours after shaking using amyloid precursor protein (APP) immunohistochemistry. Neuronal perikaryal APP was widely distributed in the brain and spinal cord, the first time such a diffuse neuronal stress response after shaking has been demonstrated, but axonal immunoreactivity was minimal and largely confined to the rostral cervical spinal cord at the site of maximal loading. No ischaemic-hypoxic damage was found in haematoxylin and eosin-stained sections.

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1. Introduction

In Western industrialised countries, traumatic head injury is the leading cause of death and disability in infancy and childhood¹ and inflicted head injury comprises almost 25% of all head injuries in children less than 2 years of age admitted to hospital.²

Caffey first identified a causal link between shaking and infant subdural and retinal haemorrhages, and long-bone fractures.^{3,4} Subsequently, the neuropathological triad of subdural and retinal haemorrhages and acute encephalopathy was termed the "shaken baby syndrome" (SBS).⁵ It has also frequently been designated "non-accidental head injury" (NAHI)⁶ or, by those who contend that a head impact is a precondition for the development of this lesion complex, the "shaken-impact syndrome".⁷ However, none of these lesions is pathognomonic of inflicted head trauma.⁸

Death occurs in 10% to 40% of patients with NAHI.⁹ The most severe presentation is that of the collapsed, apnoeic baby or one showing severe respiratory distress, but there may be more subtle and non-specific signs, including lethargy, irritability, seizures, vomiting and inappetence. Survivors frequently experience chronic neurological problems such as cognitive and behavioural disturbances, cerebral palsy, blindness and epilepsy.^{10,11}

One of the dominant controversies in the SBS is whether a head impact is necessary to produce pathology or whether shaking alone is sufficient to injure the brain. Its resolution is frequently impeded by difficulty eliciting the circumstances surrounding

these injuries due to denial or obfuscation by the perpetrators.¹² A recent survey of the literature spanning three decades¹³ revealed that abuse was admitted in <20% of cases and that there was evidence of cranial impact in 80%. Other studies^{14–16} have concluded that head impact is not essential. Furthermore, impact of the head with a soft surface may not produce contact injuries, but sufficient angular deceleration may be generated to damage neural tissue due to the sudden deceleration of the head.¹⁷ Biomechanical studies have shown that head impact generates a very much higher loading than shaking.¹⁸ However, while some⁷ argue that shaking is insufficient to injure the brain, others¹⁹ contend that shaking alone cannot be excluded as sufficient cause.

There is currently no satisfactory biomechanical model in which to investigate the pathogenesis of SBS or potential therapeutic intervention strategies.²⁰ Accordingly, since sheep have a relatively large gyrencephalic brain resembling that of humans, we used neonatal lambs to examine neuronal perikaryal and axonal changes in the brain resulting from shaking alone. Neuronal perikaryal reactions and axonal injury in these brains were detected using amyloid precursor protein (APP) immunohistochemistry.

2. Materials and methods

2.1. Experimental protocol

Seven anaesthetised and ventilated, 7–10-day-old lambs were manually grasped under the axilla and vigorously shaken with enough force to snap the head back and forth onto the chest, similar to the actions believed to occur in the SBS.^{4,17} This shaking also re-

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sulted in considerable lateral and rotational head movement. Each lamb was shaken in this manner regularly (10 times of 30 seconds duration) over a 30-minute period, then placed quietly in the sphinx position for 6 hours under anaesthesia. There was no head impact. Three, age-matched control lambs were not shaken, but otherwise subjected to the same experimental protocol.

Lambs were maintained under anaesthesia until killed by perfusion fixation of the brain with 4% paraformaldehyde containing 0.02% heparin. Brains remained *in situ* for 2 hours and were then immersed in 10% buffered formalin for 7 days. Rostral cervical spinal cord and both eyes (including optic nerves) were also collected. Brains and cords were sectioned into 5 mm whole coronal slices and paraffin-embedded. Eyes were routinely processed for light microscopy.

2.2. Immunohistochemistry

Axonal and neuronal perikaryal reaction in these brains was evaluated using amyloid precursor protein (APP) immunohistochemistry. Brain sections were incubated overnight with a monoclonal antibody to APP at a dilution of 1:3000, stained with 3,3'-diaminobenzidine tetrahydrochloride (DAB, Sigma-Aldrich, St Louis, MO, USA) using avidin-biotin peroxidase (Vector ABC kit; Vector Laboratories, Burlingame, CA, USA) and counterstained with haematoxylin. An APP-positive control brain, and a negative control with the primary antibody excluded, accompanied each staining procedure. Duplicate sections were stained with haematoxylin and eosin (H&E).

2.3. Morphometry

Neuronal perikaryal and axonal reaction was assessed using a semi-quantitative grid system. This method produces a detailed topographical overview of the neuronal cell body and axonal response. A transparent graticule comprised of 4-mm grid squares, each with a unique reference number, was placed over each sec-

tion. On average, there were 10 coronal slices of the double hemispheres and seven of the cerebellum and brainstem producing, in total, approximately 1100 grid squares representing the entire surface area of the brain sections. The cervical spinal cord was analysed in a similar manner. The graticule had reference marks so correct alignment could be made with the underlying slide and independent evaluation of brain sections conducted. A central and peripheral reference point was made on each glass slide and these were then matched up with corresponding reference points on the transparent graticule. The detection of any APP immunostaining of axons in a grid square or APP reactive granules occupying at least 50% of the neuronal perikaryon resulted in a positive score. Axonal injury was only assessed in white matter as axons were sometimes difficult to distinguish from APP-positive dendrites in grey matter. The number of positive grids was then summed and the percentage of APP positive grids for neuronal cell bodies and axons calculated, producing an APP score ranging from 0 to 100. The APP reaction was independently assessed by two pathologists, blind to whether the lambs had been shaken or were controls. These data were statistically analysed by an analysis of variance (ANOVA) and Student's *t*-test.

This project was approved by the Animal Ethics Committee of the Institute of Medical and Veterinary Science, Adelaide.

3. Results

At necropsy, the only significant macroscopic finding was focal subdural haemorrhage, confined to a 1 × 1 cm area, in two shaken lambs (lambs 1 and 5). Microscopically, neuronal perikaryon APP immunoreactivity (Fig. 1) was widely distributed (Table 1), including cerebral cortical neurons, cerebellar Purkinje cells and brainstem neurons. The neuronal perikaryal APP reaction in the treated group (mean ± standard deviation, 43.3 ± 15.8) was higher than that of the control group (2.3 ± 0.6) ($p < 0.01$, ANOVA and Student's *t*-test). There was no evidence of neuronal loss in areas showing increased APP expression. By contrast, there were very

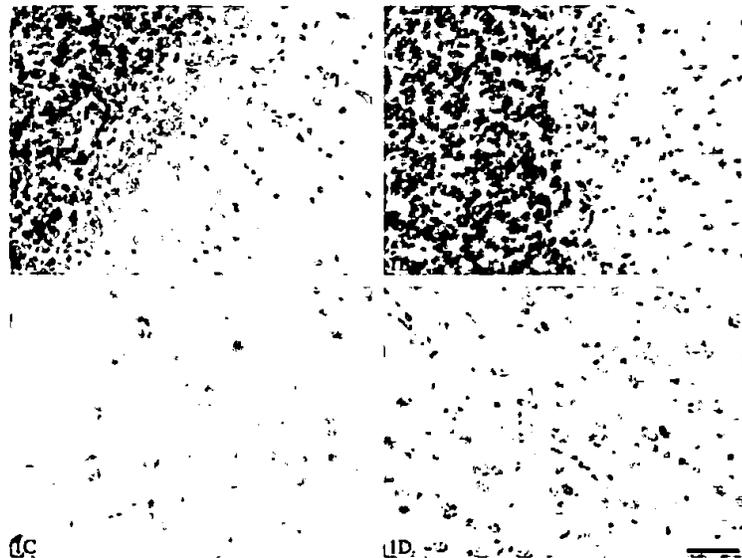


Fig. 1. Amyloid precursor protein (APP) immunostaining of (A, B) the cerebellar and (C, D) cerebral cortices showing (A) numerous immunopositive (brown) Purkinje cells and (C) cortical neurons in shaken lambs, while these neurons are immunonegative in (B, D) control lambs. Bar = 50 μ m.

Table 1
Percentage of grid squares containing amyloid precursor protein-positive axons and neurons

Lamb No.	Axonal reaction	Neuronal reaction
Control		
1	0	2
2	0	2
3	0	3
Shaken		
1	6	76
2	6	38
3	8	45
4	3	41
5	3	44
6	2	28
7	3	31



Fig. 2. Amyloid precursor protein (APP) immunostaining showing only a few APP-positive (brown) axons in the brainstem of a shaken lamb. Bar = 50 μ m.

few immunopositive axons (often <1% positive grids and never >2%) and they were usually single (Fig. 2) and randomly distributed in hemispheric and cerebellar white matter, midbrain, and brainstem (Table 1). However, APP-positive axons were more common, albeit still very limited in number, in the rostral cervical spinal cord at the site of maximal stress. No parenchymal haemorrhage or recognizable ischaemic-hypoxic damage was found in H&E-stained brain and spinal cords. Minor retinal haemorrhage was observed in two lambs (lambs 1 and 5) and no APP-positive axons were detected in optic nerves. In control lambs, no APP-positive axons were observed (and thus statistical analysis could not be performed) and neuronal APP expression was minimal (2–3% positive grid squares).

4. Discussion

The results of this study showed that vigorous whiplash shaking of lambs produces widespread neuronal perikaryal APP expression. This generalized upregulation of neuronal APP probably represents a non-specific, acute stress response to trauma^{21,22} as it is unlikely that all these APP-immunoreactive neurons were irreversibly damaged. Rapidly developing and widely distributed neuronal perikaryal APP expression was also found in an ovine head impact model²³ due to upregulation of APP messenger RNA.²⁴ We believe this to be the first report of such a diffuse, acute phase neuronal reaction in the brain to shaking.

By contrast, axonal injury (AI) was minimal in these lambs, occurring as individual APP-positive fibres randomly distributed in the white matter, although it was minimally more common in

the rostral cervical spinal cord at the site of maximal loading during shaking. The craniocervical joint may be particularly vulnerable in infants <3 months of age and damage at this site may provide a substrate for the common presenting signs of apnoea and respiratory distress.^{25–27} Diffuse (multifocal) traumatic AI (dTAI) was not found in shaken lambs and, although a few studies^{15,28,29} have reported diffuse axonal injury (DAI) in patients with SBS, it is generally accepted that dTAI occurs rarely in infants <1 year of age.^{27,30}

Death in most patients with NAHI is now attributed to diffuse cerebral swelling resulting from global hypoxic brain damage.^{16,26,30} In one large study, 75% of abused babies presented with severe apnoea and most died from raised intracranial pressure (ICP) secondary to brain swelling.²⁵ Global hypoxic brain damage was commonly found upon microscopic examination and, although one-third had vascular AI related to increased ICP, dTAI was rarely observed. No identifiable ischaemic-hypoxic injury was found in shaken lambs.

Neuronal perikaryal APP expression in these shaken lambs was similar to that found in lambs, albeit somewhat older (4–5 weeks), impacted to different regions of the head with a humane stunner.³¹ In the latter, neuronal APP upregulation was widely distributed and APP scores after temporal, frontal and occipital impacts were 40, 78 and 74, respectively. However, APP-positive axons in this ovine impact-acceleration model were also widespread, with APP scores of 12, 35 and 22, respectively, much higher than in the shaken lambs. Most impacted lambs also had contusions, unlike shaken lambs in the present study.

The response of a child's brain to an insult often differs from that of an adult.¹ A disproportionately large infant head, weak cervical muscles and a relatively large subarachnoid space permit significant differential movement of the immature brain within the skull during shaking¹⁷ and the incompletely myelinated white matter and higher brain water content predispose the infant brain to shear injury.³² The thin, pliable infant skull also transmits impact forces more readily to deeper brain structures.³³ Shaking is an acceleration-deceleration type of injury which is similar to whiplash, but of longer duration. It is the sudden deceleration of the brain that is believed to cause intracranial injury¹⁷ and brain damage is accentuated if additional contact forces are applied to the head.

A small subdural haemorrhage was found in two shaken lambs, due to tearing of fragile bridging veins between the cortical surface and dural sinuses during shaking,¹⁷ but retinal haemorrhages were minimal and only seen in two animals. However, subdural and ocular haemorrhage, although common in patients with NAHI, is not specific for trauma.¹¹

There have been very few animal models attempting to replicate brain injury due to head shaking. Raghupathy and Margulies³⁴ and Raghupathy et al.³⁵ subjected neonatal pigs to head rotational acceleration in the axial plane. A single, mild rotational acceleration of the head produced AI in frontal lobes only, while consecutive rotations produced additional AI in parietal and temporal lobes, the corpus callosum, hippocampus and basal ganglia. Neuronal perikaryal changes were not described. Smith et al.³⁶ and Bonnier et al.³⁷ used a rotating shaker to produce more severe brain injury in neonatal rats and mice, respectively, but these are lissencephalic species and the high mortality rate (27%) in the Bonnier et al.³⁷ study suggested that rotational loading may have been much greater than occurred in lambs or piglets. The length of time from shaking to necropsy was also much greater in these rodent studies. Moreover, while the mechanical devices used in these studies facilitated reproducibility of shaking, they do not produce the type of head shaking believed to occur in real-world instances of human infant abuse.

In conclusion, this lamb model of the SBS showed widespread neuronal perikaryal APP immunoreactivity, consistent with an acute stress response, but minimal APP immunopositive AI.

References

1. Ward JD. Paediatric head injury. In: Narayan RK, Wilberger JE, Povlishock JT, editors. *Neurotrauma*. New York: McGraw-Hill; 1996. p. 859–87.
2. Duhaime AC, Alario AJ, Lewander WJ, et al. Head injury in very young children: mechanisms, injury types, and ophthalmologic findings in 100 hospitalised patients younger than 2 years of age. *Paediatrics* 1992;90:179–85.
3. Caffey J. On the theory and practice of shaking infants. Its potential residual effects of permanent brain damage and mental retardation. *Am J Dis Child* 1972;124:161–9.
4. Caffey J. The whiplash shaken infant syndrome: manual shaking by the extremities with whiplash-induced intracranial and intraocular bleeding, linked with residual permanent brain damage and mental retardation. *Paediatrics* 1974;54:396–403.
5. American Academy of Paediatrics Committee on Child Abuse and Neglect. Shaken baby syndrome: inflicted cerebral trauma. *Paediatrics* 1983;92:872–5.
6. Blumberg PC, Reilly PL, Vink R. Trauma. In: Louis DN, Love S, Ellison DW, editors. *Greenfield's neuropathology*. London: Edward Arnold; 2008. p. 792–6.
7. Duhaime AC, Gennarelli TA, Thibault LE, et al. The shaken baby syndrome. A clinical, pathological and biomechanical study. *J Neurosurg* 1987;66:409–15.
8. Richards PG, Betocchi GE, Bonshok RE, et al. Shaken baby syndrome. *Arch Dis Child* 2006;91:205–6.
9. Jayawant S, Rawlinson A, Gibbon F, et al. Subdural haemorrhages in infants: population based study. *BMJ* 1998;317:1558–62.
10. Ghahreman A, Bhasin V, Chaseling R, et al. Nonaccidental head injuries in children: a Sydney experience. *J Neurosurg* 2005;103:213–8.
11. American Academy of Paediatrics, Hymel KP; Committee on Child Abuse and Neglect; National Association of Medical Examiners. Distinguishing sudden infant death syndrome from child abuse fatalities. *Paediatrics* 2006;118:421–7.
12. Krous HF, Byard RW. Shaken infant syndrome: selected controversies. *Paediatr Dev Pathol* 1999;2:497–8.
13. Leestma JE. Case analysis of brain-injured admittedly shaken infants: 54 cases, 1969–2001. *Am J Forensic Med Pathol* 2005;26:199–212.
14. Gilliland MGF, Folberg R. Shaken babies – some have no impact injuries. *J Forensic Sci* 1996;41:114–6.
15. Shannon P, Smith CR, Deck J, et al. Axonal injury and the neuropathology of the shaken baby syndrome. *Acta Neuropathol* 1998;95:625–31.
16. Gehrmichen M, Schleiss D, Pedal I, et al. Shaken baby syndrome: re-examination of diffuse axonal injury as a cause of death. *Acta Neuropathol* 2008;116:317–29.
17. Duhaime AC, Christian CW, Rorke LB, et al. Nonaccidental head injury in infants – the “shaken baby syndrome”. *N Eng J Med* 1998;338:1822–9.
18. Ommaya AK, Goldsmith W, Thibault L. Biomechanics and neuropathology of adult and paediatric head injury. *Br J Neurosurg* 2002;16:220–42.
19. Cory CZ, Jones BM. Can shaking alone cause fatal brain injury? A biomechanical assessment of the Duhaime shaken baby syndrome model. *Med Sci Law* 2003;43:317–33.
20. Gerber P, Coffman K. Nonaccidental head trauma in infants. *Childs Nerv Syst* 2007;23:499–507.
21. Gentleman SM, Nash MJ, Sweeting CJ, et al. Beta-amyloid precursor protein (beta APP) as a marker of axonal injury after head injury. *Neurosci Lett* 1993;160:139–44.
22. Mattson MP. Cellular actions of beta-amyloid precursor protein and its soluble and fibrillogenic derivatives. *Physiol Rev* 1997;77:1081–132.
23. Van den Heuvel C, Lewis S, Wong M, et al. Diffuse neuronal perikaryon amyloid precursor protein immunoreactivity in a focal head impact model. *Acta Neurochir Suppl* 1998;71:209–11.
24. Van den Heuvel C, Blumberg PC, Finnie JW, et al. Upregulation of amyloid precursor protein messenger RNA in response to traumatic brain injury: an ovine head impact model. *Exp Neurol* 1999;159:441–50.
25. Geddes JF, Hackshaw AK, Vowles GH, et al. Neuropathology of inflicted head injury in children. I. Patterns of brain damage. *Brain* 2001;124:1290–8.
26. Geddes JF, Whitwell HL. Inflicted head injury in infants. *Forensic Sci Int* 2004;146:83–8.
27. Geddes JF, Vowles GH, Hackshaw AK, et al. Neuropathology of inflicted head injury in children. II. Microscopic brain injury in infants. *Brain* 2001;124:1299–306.
28. Vowles GH, Scholtz CL, Cameron JM. Diffuse axonal injury in early infancy. *J Clin Pathol* 1987;40:185–9.
29. Gleckman AM, Bell MD, Evans RJ, et al. Diffuse axonal injury in infants with nonaccidental craniocerebral trauma. Enhanced detection by beta-amyloid precursor protein immunohistochemical staining. *Arch Pathol Lab Med* 1999;123:146–51.
30. Reichard RR, White CL, Hladik CL, et al. Beta-amyloid precursor protein staining nonaccidental central nervous system injury in paediatric autopsies. *J Neurotrauma* 2003;20:347–55.
31. Finnie JW, Van den Heuvel C, Gebski V, et al. Effect of impact on different regions of the head of lambs. *J Comp Pathol* 2001;124:159–64.
32. Dobbing J. The late development of the brain and its vulnerability. In: Davis JA, Dobbing J, editors. *Scientific foundations of paediatrics*. London: Heinemann; 1981. p. 744–59.
33. Case ME, Graham MA, Handy TC, et al. Position paper on fatal abuse head injuries in infants and young children. *Am J Forensic Med Pathol* 2001;22:112–22.
34. Raghupathi R, Margulies SS. Traumatic axonal injury after closed head injury in the neonatal pig. *J Neurotrauma* 2002;19:843–53.
35. Raghupathi R, Mehr MF, Helfaer MA, et al. Traumatic axonal injury is exacerbated following repetitive closed head injury in the neonatal pig. *J Neurotrauma* 2004;21:307–16.
36. Smith SL, Andrus PK, Gleason DD, et al. Infant rat model of the shaken baby syndrome: preliminary characterization and evidence of the role of free radicals in cortical haemorrhage and progressive neuronal degeneration. *J Neurotrauma* 1998;15:693–705.
37. Bonnier C, Mesples B, Carpentier S, et al. Delayed white matter injury in a murine model of the shaken baby syndrome. *Brain Pathol* 2002;12:320–8.

Exhibit GG

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

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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.

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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&sc=1&sq=maggie+jones+who+was+abused&st=c&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 273 (Stephen Lazortiz & 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 Lazaritz & 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 OF OPHTHALMOLOGY, (MAY 2010), available at http://one.aao.org/ce/practiceguidelines/clinicalstatements_content.aspx?cid=914163d5-5313-4c23-80f1-07167ec62579 (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 CRM. 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/AJFT 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-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. Flunkett (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-L0>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.c. the NAME paper was withdrawn in 2006; the AAP paper was modified in 2009.

⁸² See AM. ACAD. OF PEDIATRICS, INFLICTED CHILDHOOD NEUROTRAUMA: PROCEEDINGS OF A CONFERENCE SPONSORED BY DEPT 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. RELIAB. 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 Duhalme (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); Ominaya, *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 C. 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., Sirotiak, *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 & C. 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*, 736 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., Sankaranarayanan 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 36, 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 Cruise 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 Nail and Internal 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-Abas, 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 Shua-Landry CL & 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 *Crove 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 file 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. Klm 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/hypernatremia 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 13 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. S67 (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 Unmitting 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: $1/5.56 \times 2.96/1 = 2.96/5.56$. 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 5.56 = 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"); *Infllicted 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 RADIOLOGY 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 RADIOLOGY 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 513), 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/vol1_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 Riach 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.

Exhibit HH

The “Shaken Baby” syndrome: pathology and mechanisms

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Abstract The “Shaken Baby” syndrome (SBS) is the subject of intense controversy; the diagnosis has in the past depended on the triad of subdural haemorrhage (SDH), retinal haemorrhage and encephalopathy. While there is no doubt that infants do suffer abusive injury at the hands of their carers and that impact can cause catastrophic intracranial damage, research has repeatedly undermined the hypothesis that shaking per se can cause this triad. The term non-accidental head injury has therefore been widely adopted. This review will focus on the pathology and mechanisms of the three physiologically associated findings which constitute the “triad” and are seen in infants suffering from a wide range of non-traumatic as well as traumatic conditions. “Sub” dural bleeding in fact originates within the deep layers of the dura. The potential sources of SDH include: the bridging veins, small vessels within the dura itself, a granulating haemorrhagic membrane and ruptured intracranial aneurysm. Most neuropathologists do not routinely examine eyes, but the significance of this second arm of the triad in the diagnosis of Shaken Baby syndrome is such that it merits consideration in the context of this review. While retinal haemorrhage can be seen clinically, dural and subarachnoid optic nerve sheath haemorrhage is usually seen exclusively by the pathologist and only rarely described by the neuroradiologist. The term encephalopathy is used loosely in the context of SBS. It may encompass anything from vomiting, irritability, feeding difficulties or floppiness to seizures, apnoea and fulminant brain swelling. The spectrum of brain pathology associated with retinal and

subdural bleeding from a variety of causes is described. The most important cerebral pathology is swelling and hypoxic–ischaemic injury. Mechanical shearing injury is rare and contusions, the hallmark of adult traumatic brain damage, are vanishingly rare in infants under 1 year of age. Clefts and haemorrhages in the immediate subcortical white matter have been assumed to be due to trauma but factors specific to this age group offer other explanations. Finally, examples of the most common causes of the triad encountered in clinical diagnostic and forensic practice are briefly annotated.

Keywords Shaken baby syndrome · Subdural haemorrhage · Retinal haemorrhage · Infant encephalopathy · Axonal injury · Subcortical haemorrhage · Cerebral venous sinus thrombosis · Subpial haemorrhage

The triad

Shaken Baby syndrome is generally, but not exclusively, diagnosed in infants under 1 year of age, the peak age being 10–16 weeks. Boys represent 65% of cases and are younger at presentation [153, 158]. The diagnosis is characterised by the triad of retinal haemorrhage (RH), thin-film bilateral or multifocal subdural haemorrhage (SDH) and encephalopathy. A mechanistic explanation and pathological description of the three components of the triad will be discussed in the context of our current understanding of the anatomy and physiology of the brain and its coverings in the first year of life. Not all babies presenting with the triad will die, and neuroradiology rather than neuropathology is the cornerstone of diagnosis in babies who survive. Interpretation of imaging depends on

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understanding the neuropathology and wherever possible reference will be made to the correlation of pathological with radiological appearances.

The origins and history of the “Shaken Baby” hypothesis

In the early 70s Guthkelch [63] and later Caffey [18] suggested that this triad could result from whiplash or shaking injury. Guthkelch, noting that not all babies with SDH had external marks of injury to the head, suggested that shaking rather than striking the infant might be the cause. Extrapolating from the results of contemporaneous biomechanical studies on adult primates, he suggested that the whiplash of shaking may shear bridging veins leading to bilateral thin-film subdural haemorrhage, which he noted was quite unlike the unilateral subdural bleeding typically described in adults. He wrote “Moreover, since one would expect that the child is often grasped more or less symmetrically by chest or limbs the rotation-acceleration strains on the brain would tend to occur fairly symmetrically also, in an anteroposterior direction. This may be the reason why infantile subdural haematoma is even more often bilateral—for example in 14 of 18 cases (78%) of my earlier series—than subdural haematoma in adults for which the proportion of bilateral cases does not exceed 50%”.

Duhaime studied both biomechanical and clinical aspects of Shaken Baby syndrome and wrote perhaps the most exhaustive studies and reviews of the condition in the late 80s. In a review of 57 patients with suspected shaking injury, all 13 who died had evidence of impact head trauma. Eight had skull fractures and bruises, five had externally visible scalp bruises and six had “contusions and lacerations” of the brain. Her biomechanical studies led her to conclude that the acceleration force generated by impact exceeded that caused by shaking by a factor of 50. The following year, Duhaime wrote “it is our opinion, based on the clinical data and the studies outlined, that the “Shaken Baby syndrome” is a misnomer, implying a mechanism of injury which does not account mechanically for the radiographic or pathological findings” [38].

Others have repeated these biomechanical studies and shown that an adult shaking a dummy cannot generate the forces considered necessary to produce subdural bleeding [31, 125]. In contrast, Roth, using finite element modelling of the material properties of bridging veins and the angular velocities measured by Prange [125], calculated that shaking could generate sufficient force to cause BV rupture. The peak force considered necessary to do so in this model was equivalent to that generated by a 1.25 feet fall [133].

Duhaime devised an algorithm for the diagnosis of non-accidental injury on which many subsequent studies have been based. It assumes that a short fall cannot explain the triad: “falls clearly described as less than 3 feet in height were designated as “trivial” trauma and when given as an explanation of a high-force injury, along with variability in the history or a developmentally incompatible scenario, non-accidental injury was presumed” [39]. Clinical and biomechanical studies have demonstrated the error in this assumption; there are many reports of babies suffering intracranial bleeding, sometimes fatal, after low falls [3, 55, 66, 168] and laboratory studies have shown that the forces generated by even a 25-cm fall are twice those generated by maximal shaking and impact onto a soft surface [147].

An exhaustive study of the published literature over a period of 32 years found only 54 cases of confessed shaking, of which only 11 had no evidence of impact and could be considered pure shaking. There are only three published reports of witnessed shaking; all three infants were already collapsed before the shaking event [91, 140].

Since the initial work of Guthkelch, the importance of “rotational forces” as the mechanism of intracranial injury has been emphasised. Many have mistakenly assumed that rotational forces require shaking. There is no doubt that rotation is a potent cause of intracranial injury, but virtually any impact to the head will also cause rotation because the head is hinged on the neck. Holbourn wrote in 1943 “rotations are of paramount importance” and “If the head is so well fixed that it cannot rotate at all when it receives a blow there will be no rotational injury” [70]. It is reasonable to assume that the infant with a weak neck would be even more vulnerable to hinging of the head on the neck than the adult with developed musculature and full head control. While rotational acceleration/deceleration is important in causing brain damage, there is absolutely no evidence that it requires shaking or swinging. While shaking does cause rotational forces, their magnitude is insufficient to cause intracranial injury; biomechanical studies have shown that impact and falls cause far greater rotational forces [31, 37, 125].

Neuropathological studies have had enormous implications for the shaking hypothesis. Geddes showed that brain swelling and HII were virtually universal in babies thought to have suffered non-accidental injury, but very few had traumatic axonal injuries. Where, the present axonal injury was at the craniocervical junction [57, 58]. The clinical implication is that the signs of encephalopathy are due to hypoxia and brain swelling. As the pace at which swelling occurs is variable, there is an opportunity for a “lucid interval”, in contrast to immediate concussion as expected from diffuse axonal injury. In the majority of infants with the triad, the brain damage is non-specific and unless there

is injury at the craniocervical junction, the diagnosis of shaking can be no more than speculation.

In 2009 the American Academy of Paediatricians, followed in early 2011 by the UK Crown Prosecution Service, accepted that the term Shaken Baby syndrome should be dropped because it did not exclusively explain the triad of findings, although confessions supported the role of shaking. The term non-accidental head injury (NAHI) has since been widely adopted [27, 32].

While shaking is no longer a credible mechanism for NAHI, there remains no doubt that inflicted head injury does occur, but its clinical recognition remains problematic. “There is no diagnostic test for inflicted brain injury, the diagnosis is made on a balance of probability and after careful exclusion of other possible causes of the clinical presentation” [99] and “there is no absolute or gold standard by which to define NAHI” [69].

In arguing “The Case for Shaking” Dias [35] began with the statement “Unfortunately, nobody has yet marshalled a coherent and comprehensive argument in support of shaking as a causal mechanism for abusive head injury” and concluded “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 base for shaking”.

This very definite statement indicates that, 40 years after it was first proposed, the shaking hypothesis now rests upon confession evidence.

How reliable are confessions? Clinical evidence of impact is found in up to 63% of confessions of shaking only [35] and when imaging evidence is considered “No correlation was found between repetitive shaking and SDH densities.” [2]. This review will take a pragmatic approach, addressing the evidence provided by detailed examination of the tissue in babies who manifest the triad.

Brain examination in the triad

The foregoing discussion illustrates the considerable responsibility for the pathologist who may be presented with a clinical diagnosis based on dubious criteria, the reliability of which will depend on the extent to which other possible causes have been excluded.

The differential diagnosis of a baby with the triad is wide and includes birth difficulties, coagulopathy, arterial occlusive disease and venous thrombosis, metabolic and nutritional disorders, infections, hypoxia–ischaemia (e.g. airway, respiratory, cardiac, or circulatory compromise) and seizures. Multifactorial and secondary cascades are common, for example “trivial trauma” in the context of predisposing or complicating medical conditions such as

prematurity, pre-existing subdural haemorrhage, coagulopathy and infectious or post-infectious condition (e.g. recent vaccination). Death occurring within the context of a recent vaccination should be reported to the appropriate agencies.

A complete and thorough review of current and past medical history involving scrutiny of perinatal and neonatal records, laboratory tests and the clinical management of the child is required. If the baby dies the gold standard is a thorough and complete autopsy where neuropathology has a key role.

Many alternative diagnoses may not have been considered or test results may not be available before pathological opinion is required. Once pathological conclusions have been reached, they can be assessed in the context of all the available information.

Neuropathologists may not have the advantage of attending the autopsy to see for themselves the evidence from other sites and particularly to be present when the skull is opened to identify bleeding and its potential sources. The brain may be received whole and fixed; if giving a second opinion, only blocks and slides may be submitted. Residual fixed brain slices should be requested; they frequently yield evidence that may not have been appreciated on the first examination. In my experience, cortical veins and focal congestion or thrombosis are often overlooked. The dura and spinal cord are essential parts of the examination.

Sampling

Standard representative blocks should be taken from all brain areas and all levels of the brainstem and spinal cord, with at least three blocks from each level of the brainstem and the cervical cord, to include nerve roots and dorsal root ganglia.

The dura must be carefully examined by naked eye as well as microscopically. Old, healing subdural membranes can be difficult to see with the naked eye as they form a thin, light brown and often uniform layer (Fig. 1b). The dural sinuses must be carefully examined and sampled. Intradural bleeding is the most common posteriorly, in the spongy tissue around the torcula, in the posterior falx and in the tentorium and these areas, as well as convexity dura, should be sampled.

Dura from the spinal cord is informative for two main reasons. First, unlike the cranial dura, it is not routinely stripped from the underlying arachnoid barrier membrane during autopsy. Foci of the normal *in vivo* apposition of these membranes, as well as minor bleeding into the subdural compartment may be seen in spinal dura (Fig. 2). Second, it is common for intracranial subdural blood to track into the spinal subdural compartment, and sometimes this blood is the only evidence of old subdural haemorrhage.

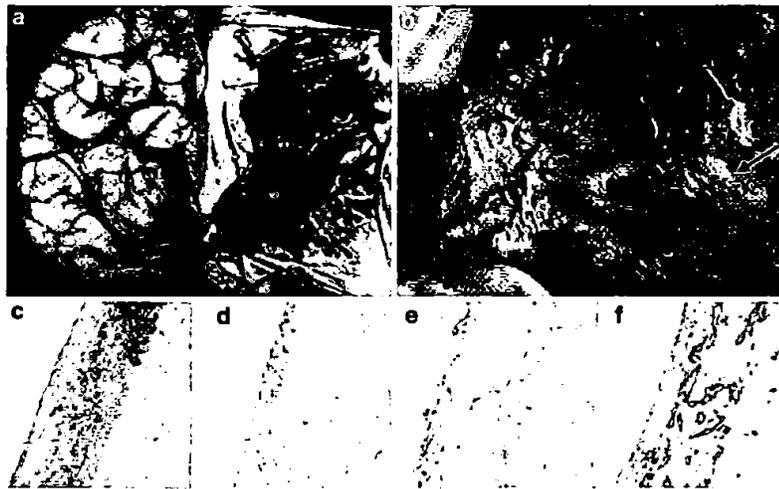


Fig. 1 Healing subdural haemorrhage. **a** Fresh bleeding into a thin, healing subdural membrane. The dura has been lifted off the brain at autopsy. There is a large area of fresh bleeding on the arachnoid surface of the dura. A faint brown/yellow tinge at the edges indicates older bleeding. **b** Female 10 months old with head trauma 3 weeks before death. **b** A thin, light brown membrane covers the deep dural surface. The membrane could easily be overlooked but where a small area has lifted its delicate nature can be appreciated as well as the contrast with the normal light grey dura beneath (*arrow*). **c** Fifteen months baby with head trauma 4 weeks before death from inflicted

abdominal injury. **c** Male 3 months: H&E stained section showing fresh bleeding into a membrane on the subdural surface. The uniform pink dura is on the right side of the panel. **d** Perl's positive material is most abundant on the free edge of the membrane with fresh bleeding beneath. **e** CD 34 demonstrates vessels in the membrane crossing the underlying fresh haemorrhage and, at its junction with the dura. The dura shows finely speckled fibroblast staining. **f** CD31 highlights the wide, sinusoidal vessels within the membrane and straddling the fresh bleed. The dura itself is unstained. (**c-f** 4×)

Staining methods

Brain and spinal cord

As well as standard H&E stain, the most helpful stains for identifying subtle areas of early tissue damage are CD68 and β APP, which draw the eye to even the smallest collections of macrophages and damaged axons. Reticulin and CD34 stains demonstrate proliferating capillaries in damaged tissue, and reticulin is invaluable for demonstrating subpial bleeding. Endothelial markers CD31 and CD34 demonstrate reactive blood vessels; smooth muscle actin (SMA) is a useful adjunct in the detection of very early organisation of intravascular clot.

Dura

A somewhat different panel of stains is required if subtle healing subdural membranes are to be identified. Both thin, early membranes and old-fibrosed membranes are hard to differentiate from normal dura with H&E alone. A recommended panel includes Perl's stain for iron, CD 68 for macrophages and an endothelial marker to show newly formed and reactive vessels. CD31 is preferred to CD34 as an endothelial marker, since the latter also labels

dural fibroblasts, making interpretation more difficult (Fig. 1c, d). Perl's stain indicates altered blood and previous haemorrhage, and is generally identifiable from 48 h after the bleed. Sometimes this stain is negative even when there appears to be a well-developed healing membrane. There are two explanations for this paradoxical finding: Perl's stain may be less reliable in certain fixation protocols and, rarely, CSF leaks promote development of a non-haemorrhagic but vascular subdural membrane [110]. Neurofilament stain demonstrates the luxuriant innervation of the infant dura; evidence of its involvement in the regulation of venous outflow from the brain and of functions as yet unknown, but clearly far more than just providing physical protection for the brain.

Subdural haemorrhage

At the outset, it is important to appreciate the anatomical location of subdural bleeding, which, in fact, originates within the deep layers of the dura. The skull bones, periosteum and meninges develop by condensation from the same mesenchymal layer and the dura forms a single functional unit with the arachnoid barrier layer [98]. In life there is no subdural space; "the traditional concept of a



Fig. 2 The dura-arachnoid interface. **a** Fetal spinal cord. The arachnoid (A) is being lifted off the dura (D) by fresh bleeding (arrows). There is a little fresh subarachnoid bleeding and the nerve root is congested (4 \times). **b** Fetal cranial arachnoid. The barrier layer is an avascular membrane (A). Above it loosely adherent flaky cells of the dural border layer are seen (arrow). The subarachnoid space is relatively cellular (10 \times). **c** Spinal cord. Several contact sites between

dura and arachnoid are indicated by arrows. Note fresh bleeding between the cells of the dural border layer at the right of the picture (10 \times). **d** Spinal dura showing adherent arachnoid cells (arrow). The dura is less vascular than the cranial dura. Note fresh bleeding into the epidural fat (4 \times) (c and d are from the spinal cord of a male baby of 4 months who died after prolonged seizures. There was no evidence of proximate trauma)

virtual, slit-like subdural space is in error" [53, 65]. When the dura is stripped from the brain surgically, at autopsy and by bleeding, the loosely adherent cells of the dural border layer are torn apart and an artificial space is created. A careful examination of the outer surface of the arachnoid membrane will reveal cellular remnants of the dural border cell layer. Similarly, adherent arachnoid cells can sometimes be identified on the deep dural layers. (Fig. 2)

Distribution and patterns of dural and subdural haemorrhage

When a baby presents to hospital, it is often the radiological diagnosis of SDH that raises the question of NAHI and significantly influences subsequent management. The importance of this element of the triad places an onus on the pathologist to establish and describe the sources and nature of infant SDH.

The typical pattern of subdural haemorrhage in babies with the triad is of a bilateral thin film over the cerebral convexities and in the posterior interhemispheric fissure [22, 40, 189].

The distribution of subdural blood is not a reliable indication of its cause. Rather, radiological studies have

demonstrated that the distribution is a function of age and that redistribution occurs by gravity and sedimentation [33, 47, 167]. MRI studies show spinal subdural haemorrhage in almost half of babies with intracranial subdural haemorrhage, sometimes in direct continuity with posterior fossa blood. The location in the most dependent spinal areas, dorsally at the thoracolumbar level, indicates gravitational redistribution [86]. The spinal dura extends beyond the dorsal root ganglia where it blends with the sheath of the nerve roots, allowing subdural blood to track out into them (see Fig. 10).

The bilateral widespread thin film distribution of infant SDH differs from the adult form, where subdural haemorrhage generally forms a unilateral localised mass within the convexity dura [63]. There are several potential explanations for this difference. First, the mechanism for SDH in an infant may be fundamentally different from that in an older child or an adult with a mature skull. Second, the infant dura is far less collagenised than the adult dura, and its fibroblasts are widely separated by a loose matrix (Fig. 3) allowing ready dispersion of blood. Finally, infant subdural haemorrhage is frequently not solid but "thin and easily tapped" [155, 167] allowing easy dispersion.

SDH does not need to be large or space occupying to cause clinical symptoms. Although the blood is physically

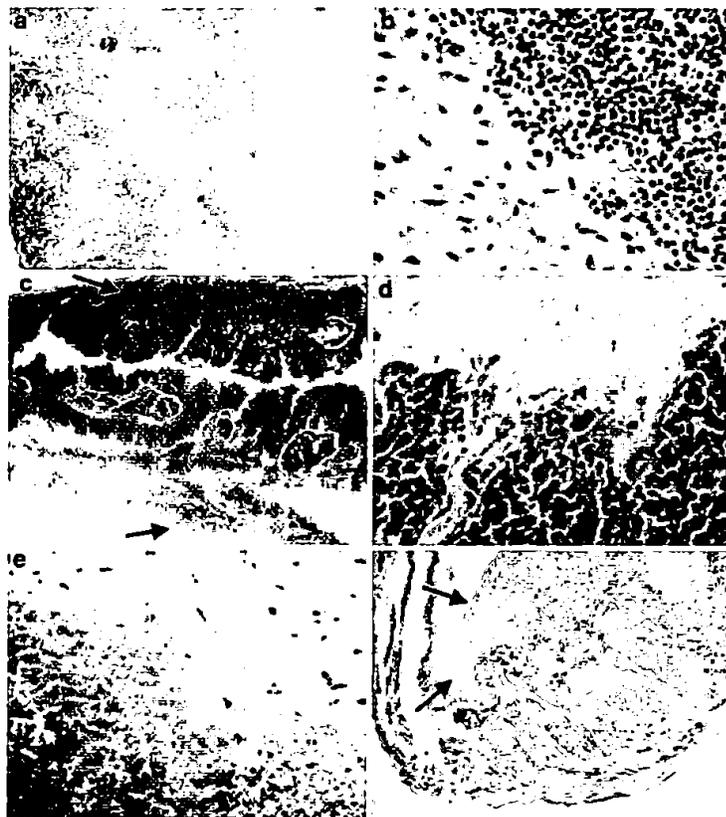


Fig. 3 Intradural bleeding. **a–d** Dural bleeding at 20 weeks gestation and 20 months of postnatal life. **a, b** The foetal dura is cellular, with delicate, loosely interwoven fibroblast-like cells and little collagen. This allows ready dispersion of blood (foetus 20 weeks) (**a** 4 \times , **b** 10 \times). **c, d** 20 month baby with acute demyelinating encephalomyelopathy with severe brain swelling but no trauma. There is extensive bleeding into the dura, most of it originating in the plexuses between the dural leaflets. In several areas, blood extends to the free edges of the dura (*arrows*). **d** The cells of the dura are less numerous than in the foetus, the dura consists largely of dense bands of collagen; in

d red cells are seen between these dense fibrous bands (**c** 2 \times , **d** 10 \times). **e** There is fresh bleeding in the lower left of the panel but also many flecks of Perl's positive material between the fibres of the dura indicating much earlier bleeding, probably from birth. 5 month infant with sinus thrombosis. (Perl's stain 10 \times). **f** Infant of 3 months. Elastic van Gieson stain demonstrates intradural blood as yellow, standing out against the bright pink collagen of the dura. The fibres of the dura are split apart by fresh blood which is also spilling on to the dural surface (*arrows*) (4 \times)

separated from the surface of the brain by the arachnoid barrier layer, it causes cerebral irritation, and clinical manifestations may occur without obviously raised intracranial pressure.

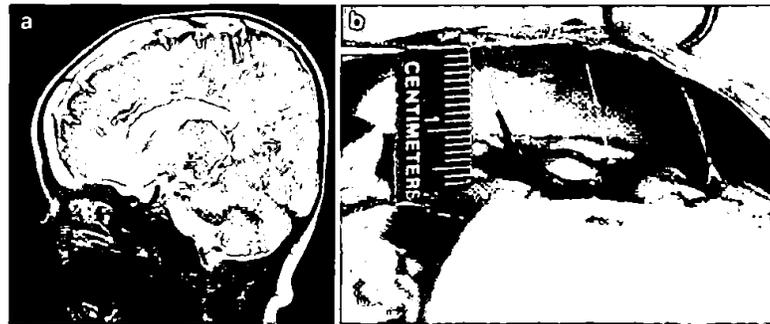
Autopsy identification of subdural haemorrhage

The volume of blood seen on scans may be very small, 2–3 ml of blood are sufficient for its radiological identification; between 1 and 80 ml (median 10 ml) was found in babies with blunt force injuries to the head [111]. This poses

a problem for the pathologist as this small volume of blood could readily be overlooked as the skull is opened and large vessels are cut, as they inevitably will be if special autopsy techniques are not employed. "At autopsy, the subdural hemorrhage may consist of only 2 to 3 ml of blood and may not be observed if the prosector does not personally inspect the subdural space as the calvarium is being removed. Extreme caution should be taken to not misinterpret as premortem subdural hemorrhage the blood draining from the dural sinuses when these are incised at autopsy" [22].

Needling the cisterna magna before opening the skull may identify extracerebral blood or fluid collections. In

Fig. 4 Radiological and autopsy demonstration of bridging veins. **a** MRI: sagittal view demonstrating five bridging veins (arrows) crossing large extracerebral fluid collections in both the subarachnoid and subdural compartments. **b** By carefully opening the skull at autopsy with parasagittal cut it is possible to demonstrate the integrity of bridging veins



order to examine the bridging veins, the skull is opened by parasagittal cuts lateral to the superior sagittal sinus [82]. By carefully lifting the midline bony strip, the bridging veins can be visualised and any extracerebral blood and fluid identified before veins or sinuses are cut (Fig. 4). Some claimed to be able to establish the integrity of bridging veins by retrograde dye injection via the superior sagittal sinus [104, 154].

Origin of subdural haemorrhage

The four most important potential sources of subdural bleeding are the bridging veins (BV), the dura itself, the vascular membrane of a healing subdural haemorrhage, and a ruptured intracranial aneurysm.

Bridging veins

It is widely believed that subdural bleeding results from mechanical tearing of bridging veins [23]. However, it is extremely hard to find any pathological description of ruptured BV in infants with SDH. Cushing, describing his surgical and pathological observations in the newborn wrote “In two of the cases that I have examined I have satisfied myself that such ruptures were present. “A positive statement, however, cannot be given even for these cases, since the dissection and exposure, difficult enough under any circumstances, owing to the delicacy of the vessels, is the more so when they are obscured by extravasated blood”. We have not moved a long way on this issue in the last century. Voigt [171] described disruption of bridging veins in adults, but they were characterised by local subarachnoid bleeding, and he wrote “Most striking in these cases is the absence of a noteworthy subdural hematoma”. Duhaime [38] did not demonstrate BV rupture in her autopsied cases, but hypothesised that the point of bridging vein rupture is in the subarachnoid space, giving rise to both subarachnoid and subdural bleeding. Bell [9]

illustrates a thrombosed bridging vein which she described as the site of traumatic rupture, but venous thrombosis is common in hypoxic and ventilated infants and does not provide robust evidence of BV rupture. Maxeiner [105] claimed to demonstrate ruptured BV using autopsy dye injection studies. His images indicate that the dye is in the subarachnoid rather than the subdural compartment and his autopsy description of less than 5 ml of blood in the subdural space where “nearly all the parasagittal bridging veins were completely torn” suggests that his methods are unreliable. Unless the BV are visualised before brain removal, artefactual rupture at autopsy cannot be excluded.

Not only are there no convincing pathological examples of BV rupture associated with thin film subdural bleeding but also there are physiological and anatomical objections to this hypothesis. Bridging veins are few in number—about 8–11 each side—and carry high blood flow (Fig. 4). In a 6-month baby, nearly 260 ml of blood flows into the dural sinuses per minute and the majority of cortical blood flows via the parasagittal BV into the superior sagittal sinus, where flow rate is 9.2 cm per second in the infant [90, 160, 178]. It is clear that rupture of even a single BV will cause massive space occupying clot, not a thin film, and the bleeding will be at least partly subarachnoid [37]. The suggestion that BV are weak at their dural junction was derived from studies of four elderly patients [181]; in fact, this junction consists of a smooth muscle sphincter which controls cerebral venous outflow when intracranial pressure is increased, maintaining the patency of the cortical veins [7, 141, 166, 183].

There appear to be circumstances when large cortical veins may ooze blood. Cushing observed that subdural haemorrhage “may occur when too great strain has been put upon the vessels by the profound venous stasis of postpartum asphyxiation; just as in later months they may rupture under the passive congestion brought about by a paroxysm of whooping-cough or a severe convulsion” [33]. Imaging and pathological observation support the suggestion of venous leakage under tension; radiological

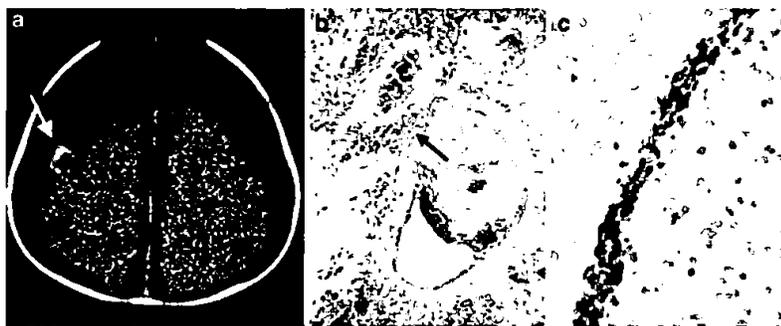


Fig. 5 Leakage of red cells across distended and thrombosed vein walls. **a** CT scan. A tiny flare of blood (*white arrow*) within a wide extra-axial fluid collection suggests leakage from a bridging vein. 5 months infant with cortical vein thrombosis. **b** Thrombosed surface vein with fresh bleeding into adjacent subarachnoid space (4×). **c** The

vein wall indicated by an *arrow* in **b** is stained for smooth muscle actin and shows red cells which appear to be escaping between the cells of the vein wall into the surrounding subarachnoid space. Three month baby with cortical vein thrombosis who died 7 days after head trauma

observations occasionally show small bleeds associated with BV crossing dilated extracerebral spaces and pathology of severely congested and thrombosed surface veins shows leakage of red cells across them into the subarachnoid space. In both circumstances, the bleeding is predominantly subarachnoid. (Fig. 5)

The dura

Intradural bleeding is common in the young infant and an almost universal finding at foetal and neonatal autopsy [28, 29, 59, 144, 149]. The anatomical and physiological basis for intradural bleeding in the infant has been discussed in detail [98]. In understanding the propensity for the infant dura to bleed, it must be borne in mind that the dura is not just a tough fibrous membrane providing physical support for the brain but it is the route of all venous outflow from the brain, via the dural sinuses.

The dura has two communicating vascular networks: the meningeal arteries, veins, and outermost periosteal plexus, which are superficially located, and the vascular plexus located between its periosteal and the meningeal leaflets, the remnant of a much more extensive network of the foetal dura [14]. The dense innervation of the dura is most abundant on the intradural sinuses and blood vessels. The dura also contains rounded fluid channels which may have a role in CSF uptake or monitoring [50, 120] and appears to be related both to age and to frequency and extent of intradural bleeding [148].

The dura at birth is very different from the dura after the end of the first year of life. At birth, the structure is of loosely arranged cells with a little collagen and the dural vascular plexuses and innervation are very much more extensive than in later life (unpublished observations); arachnoid granulations are not formed until about 7 months

of postnatal life [14, 118]. These continuing developmental features may all influence the predisposition of the young baby to dural haemorrhage in the first months of life.

The posterior falx and tentorium are frequent sites of bleeding in both the foetus and the young infant dying of natural causes [28, 29]. These are also the sites of the posterior interhemispheric haemorrhage, originally regarded as characteristic of SBS. This radiological sign is most likely to be due to intradural bleeding or congestion of the abundant venous sinuses which are part of the normal anatomy in this age group. It is impossible for either MRI or CT scans to distinguish between intradural and thin film subdural bleeding. Figure 6 illustrates the case of a baby who collapsed with brain swelling and febrile convulsions. Thin film interhemispheric haemorrhage was identified as subdural bleeding on scan but at autopsy the blood was entirely intrafalcine. The detailed anatomy of the infant dura questions the validity of the belief that posterior

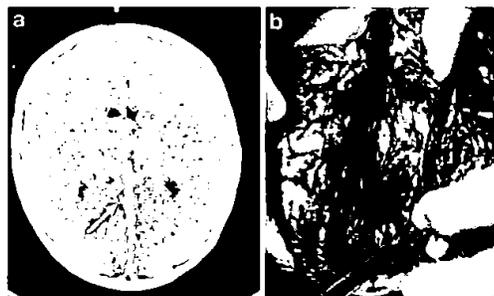


Fig. 6 Intrafalcine bleeding. **a** CT scan shows interhemispheric high signal which was interpreted as SDH. **b** Autopsy demonstrated all of the bleeding to be intradural in the posterior falx. Four months baby, sudden death with pyrexia and brain swelling. No evidence of trauma

interhemispheric bleeding is evidence of bridging vein rupture.

When it is extensive, intradural blood can almost always be seen seeping onto the subdural surface. Flecks of Perl's positive material, often extracellular and close to the walls of the sinuses may represent older, or birth-related bleeding (Fig. 3).

Subdural bleeding is often seen adjacent to the lateral recesses of the superior sagittal sinus at the vertex [170], the sites where arachnoid granulations will develop towards the end of the first year of life. The functions of arachnoid granulations remain unclear; their role in resorption of CSF under normal conditions had long been disputed [34]; there is ample evidence that CSF is resorbed from cranial and spinal nerve roots [80, 186]. Arachnoid granulations have mechanoreceptors and may monitor CSF pressure, or act as valves preventing reflux of blood from the sinus into the CSF compartment. As young infants have no arachnoid granulations, venous blood from the superior sagittal sinus or the lateral lacunae may reflux into the dura and seep into the subdural compartment [148, 175].

Healing subdural membrane

Dural bleeding promotes an inflammatory response that leads to development of a granulating membrane with a variable content of fibroblasts, macrophages, and wide thin-walled capillaries (Fig. 1c–f). Friede distinguished subdural neomembranes from granulation tissue elsewhere in the body on the basis of these distended capillaries and its looser structure [53]. The capillaries are far easier to see if endothelial markers are used, indeed such markers are necessary to determine not only the existence, but also the thickness and precise composition of a healing membrane, features which assist in assessing its age.

It is usual to see foci of bleeding of multiple ages in infant healing neomembranes, reflecting episodic rebleeding in the absence of trauma and leading to the “vicious circle” of healing which converts a recognisable reactive membrane to a fibrosed scar [53]. Fresh bleeding is an almost invariable finding at autopsy, even in babies who have been nursed on a ventilator for their past days or weeks, and is likely to be the result not of trauma, but of normal nursing or the swings of blood pressure and hypoxia which accompany brainstem death.

If sufficient, bleeding from a healing membrane will leak into, and mix with, older subdural fluid collections and effusions leading to a mixed density appearance on scans. There is a variable balance of influx and efflux as part of the natural evolution of subdural collections [75]; the number and severity of rebleeds will determine the rate of this process.

Healing subdural membranes of unknown cause are more common than generally recognised [131] and are the

most commonly encountered residuum of birth injury in SIDS autopsies [81]. Keeling warns that the “appearance should be commensurate with the age of the infant, i.e. it will be red brown and recognizable as a haematoma, probably 1–2 mm thick, for two possibly up to 4 weeks post partum” and “Later than that, brown staining of the dura is apparent. This may persist for several months” (Fig. 1). Ikeda [77] suggested that most infant SDH after minor trauma resulted from fresh bleeding into pre-existing subdural collections.

Ruptured intracranial vascular malformation

Cerebral vascular malformations occur in the brain and meninges of very young babies and may rupture and present with subdural haemorrhage, encephalopathy or the triad [106, 124], but the true frequency of the triad in association with aneurysmal rupture is impossible to assess because the eye examination is not usually described [15, 190].

Is hypoxia a cause of subdural haemorrhage?

An important and almost invariably overlooked part of the clinical history in babies presenting with the triad is a prolonged period of hypoxia, often 30 min or more between the baby being found collapsed and arriving in hospital and receiving advanced resuscitation. This sequence sets babies with the triad apart from cot death babies who are, by definition, found dead and have no pathology or intracranial bleeding. Prolonged hypoxia and resuscitation have been shown to be significantly associated with retinal haemorrhages [102] and may also explain the encephalopathy in babies with the triad. Experimental models of reperfusion injury confirm that longer periods of ischaemia cause greater small vessel damage and breakdown of the blood-brain barrier, exacerbated by resuscitation and reperfusion [97, 137].

Geddes proposed that in some infants with fatal head injury, the combination of severe hypoxia, brain swelling and raised central venous pressure is the cause of dural and retinal haemorrhage [59]. Geddes was not the first to make this observation; it had already been made by Cushing in 1905. There has been a tendency, notably in the Courts, to oversimplify this hypothesis to assume that hypoxia alone is a cause of subdural haemorrhage. This is misleading, as the physiological consequences of hypoxia are more complex. Subsequent research demonstrates an association between hypoxia and dural bleeding in young infants [28, 29]. Byard [16] found no SDH in hypoxic infants, but retrospective review of autopsy reports is unreliable in detecting small volume bleeds [22]. Hurley [74], in a

retrospective autopsy and imaging study, found only one subdural haemorrhage and two intradural bleeds in 47 babies up to 4 years of age. Data regarding duration of hypoxia and resuscitation were incomplete. Neurological outcome is known to be far worse in children suffering out-of-hospital compared with in-hospital cardiac arrests, probably due to prolonged hypoxia and less effective CPR in the former group. [100].

Birth-related SDH

A degree of subdural bleeding is extremely common after birth and seen on imaging in up to 46% of asymptomatic neonates after normal, instrumental and caesarean delivery [96, 132, 177]. More cases of SDH would be expected among symptomatic infants [24]. Two MRI studies have followed a total of just 27 babies with birth-related bleeding with repeated scans at 1–3 months of age. One baby developed further subdural bleeding [132, 177]. Due to the very small numbers used in these studies compared with the overall frequency of birth-related bleeding, meaningful interpretation is difficult and we have no good data on the natural history of birth-related SDH. It is obvious that most heal without any significant morbidity, although birth-related bleeding has been shown to be the cause of between 14 and 17% of infant chronic subdural haemorrhage [4, 69].

Studies of later onset infant subdural haemorrhage show that untreated small volume bleeds develop into chronic fluid collections. Loh [95] found chronic collections between 15 and 80 days after onset, the mean being 28 days. Hwang [75] described three cases of accidental subdural haemorrhage which resolved in CT scans only to reappear up to 111 days later. It seems likely that birth-related subdural haemorrhage will behave similarly.

Timing subdural bleeding

Dating healing subdural haemorrhage by pathology alone is difficult and cannot constitute reliable evidence of the timing of an injury. In clinical practice, it is important to take into account the entire clinical history and the other clinical and pathological findings. Several guidelines for timing the cellular reactions to subdural haemorrhage are published [92, 108, 114].

Clinical signs of chronic SDH

The neuropathologist needs to be aware that there may not always be a clinical history to indicate pre-existing

subdural haemorrhage. Chronic SDH can be extremely difficult to diagnose in infants and, unless specifically sought, the diagnosis is readily missed. Symptoms are non-specific and are sometimes purely systemic mimicking gastroenteritis, malnutrition or bronchopneumonia [68]; “Most often, the infant’s history includes failure to gain weight; refusal of feedings followed by frequent episodes of vomiting, some of which might be projectile; irritability; progressive enlargement of the head; and, ultimately, a seizure” [108].

Obstetric and birth records and brain scans should be reviewed. Head circumference charts are critical for the identification of extra-axial fluid and blood collections in life [184] and should be consulted in considering the possibility of pre-existing subdural haemorrhage.

Subdural hygroma/chronic subdural haemorrhage

The terminology of these entities is confused with no clear distinction between chronic subdural haemorrhagic collections, subdural hygromas and effusions. Subdural effusions may be xanthochromic or haemorrhagic and may evolve into frank subdural haematomas [19, 122]. Conversely, acute subdural haemorrhages may evolve into clear or xanthochromic protein-rich fluid collections or hygromas. The primary mechanism for the formation of hygromas remains unknown, it has been suggested that any pathologic condition at the dural border layer; fresh bleeding, lysis of pre-existing bleeding, inflammation or exudation from dural vessels can lead to effusion and fluid accumulation [48, 176].

Enlarged extra-axial spaces

Large fluid collections around the infant brain may be identified in otherwise normal babies and are usually self-limiting. Males outnumber females by 2–1. The causes are not known and include abnormalities in growth rates of the brain, the skull or the surrounding membranes, immaturity in the mechanisms of cerebrospinal fluid production and resorption, and old subdural bleeding. The many names, wide range of associated clinical findings and many aetiological hypotheses underscore the heterogeneity of the condition [60, 185]. Extra-axial fluid collections, whatever their cause, may predispose to SDH. Fresh bleeding into large extracerebral fluid collections after no, or only minor, trauma has been described [76, 107, 169]. Pittman [123] warned that acute SDH in the context of such a fluid collection around the brain could not be taken as evidence of abuse. A proposed mechanism for bleeding into extracerebral collections is leakage from over-stretched bridging

veins which cross them [121]. Evidence from imaging and microscopy suggests that large surface veins may leak when their walls are stretched. Figure 5 demonstrates red cells passing between the cells of a congested and thrombosed cortical vein wall into the subarachnoid space.

Subarachnoid haemorrhage (SAH)

In adults, the most common causes of SAH are trauma and ruptured aneurysm [43]. Hypoxia is a particularly frequent cause in the young infant, as are trauma and venous and sinus thrombosis.

Subpial haemorrhage

Subpial bleeding receives a little attention in the pathological literature and is not generally distinguished from subarachnoid haemorrhage. Larroche [89] considered the pathogenesis and clinical implications to be the same. Subpial bleeding can be mistaken both radiologically and pathologically for contusion and erroneously support a diagnosis of trauma.

Friede described subpial bleeding as representing 15% of perinatal intracranial haemorrhage [52]. He considered that the bleeding dissected through the superficial astrocytic foot processes, and was a variant of, subarachnoid bleeding due to respiratory distress syndrome. Lindenberg [94] described similar bleeding into the outer part of cortical layer I in babies under 5 months and Voigt [171] described it in adults. Superficial bleeding, assumed to be subpial or subarachnoid, is described in temporal pole haemorrhage (see below) [73, 89].

Subpial bleeding is macroscopically well circumscribed, and often seen at the edge of a gyrus. I have seen it in association with cortical vein thrombosis, beneath space occupying subdural haematoma and beneath fractures occurring during forceps delivery. Larroche [89] considered occlusion or compression of superficial veins as a potential mechanism. The cortical veins, unlike cortical arteries, have little or no leptomeningeal investment around them [188] and bleeding around their deep cortical tributaries can track directly into the subpial space. (see Figs. 11, 13).

Epidural haemorrhage: cranial and spinal

The skull bones develop within the outer mesenchymal layer which forms both the periosteum and the outer leaflet of the dura [98]. As the cranial dura is so intimately associated with the periosteum epidural bleeding is

uncommon except where there has been surgery or fracture. Old trauma may not be obvious and there may be no history; skull fractures are associated with normal delivery and low falls and may be asymptomatic in the neonate [41, 135]. Rarely, cranial epidural bleeding is seen beneath an intact skull bone and is considered to result from inbending of the pliable infant skull (ping-pong fracture) which tears off the periosteum in the absence of fracture.

The relationship of the spinal dura to the vertebral bones is quite different. There is a wide epidural space which contains fat and the epidural plexus. This extensive, valveless plexus communicates above with the cranial venous outflow and is responsible for cerebral venous return to the heart in the upright position [163]. It becomes massively congested when intracranial pressure is reduced or when intra-abdominal pressure is increased [156, 157, 179].

Spinal epidural bleeding has been described in infants who are thought to have suffered non-accidental injury and has been considered to be evidence of shaking [64, 117]. However, spinal epidural haemorrhage is common in infants dying from all causes and is not diagnostic of trauma [136], but is probably a response to physiological and pathological variations in intracranial pressure (Fig. 2d).

Retinal haemorrhage

Eisenbrey [45] was the first to suggest that retinal haemorrhages in a child under 4 years of age suggest abuse. Caffey [17] was prescient when he wrote “The retinal lesions caused by shaking will undoubtedly become valuable signs in the diagnosis of subclinical inapparent chronic subdural hematoma, and also become a productive screening test for the prevalence of whiplash dependent mental retardation and other types of so-called idiopathic brain damage”. Unilateral or bilateral retinal and vitreous haemorrhages, retinal folds and retinoschisis are indeed regarded as characteristic of Shaken Baby syndrome, and are estimated to be present in 65–90% of cases [40, 93, 134].

Vinchon [168] highlighted a pitfall in the use of RH in the diagnosis of abuse: “The importance of an RH for the diagnosis of child abuse is well established; however, the evaluation of its incidence in child abuse is almost impossible because the diagnosis of child abuse is in great part based on the presence of an RH, providing a circularity bias”. A further stumbling block in ascertaining the real significance of RH in abusive as compared to other forms of injury is that they may only be sought where abuse is suspected. In a study of SDH, ophthalmological opinion was sought in 94 of 106 cases of suspected NAHI but in

only a quarter of babies with SDH of any other cause [69]. In a meta-analysis of 1,283 children funduscopy was performed in 670 cases of suspected NAHI and only 328 cases with all other causes of brain injury [99].

All aspects of intraocular haemorrhage have been shown to occur without shaking [11, 127, 174]. A detailed, but yet unpublished, autopsy study found natural diseases to greatly outnumber inflicted injury in association with RH in infants under 1 year of age [87]. Thus, there is no diagnostic ocular neuropathology for SBS.

The pathologist must bear in mind that the ocular pathology resulting from the initial insult may be considerably modified by prolonged hypoxia, resuscitation, reperfusion, and a variable period of life support and must be interpreted in the context of the clinical findings closest to the time of injury. Clinical recognition of RH depends on the examination by an experienced physician using pupillary dilatation [168]; the timing of examination is crucial, as RH extend after initial injury [61].

A central issue is the mechanism of RH. The experimentally verified hydraulic theory is that retinal bleeding results from alterations in intracranial, intrathoracic and intra-abdominal pressure and blood pressure [146]. Muller and Deck [113] concluded that intraocular and optic nerve sheath haemorrhages result from the transmission of intracranial pressure into the optic nerve sheath and retinal venous hypertension. The alternative theory is that shaking causes vitreo-retinal traction, which tears the retina from its connections, disrupting the integrity of the blood vessels of the eye [93]. Ommaya [119] considered it biomechanically improbable that the levels of force generated by shaking would damage the eye directly and that a sudden rise of intracranial pressure is more likely to cause bleeding than the “shaken eye” hypothesis.

Observations of unilateral RH with ipsilateral intracranial haemorrhage or brain swelling indicate that RH may be due to the transmission of raised pressure along the optic nerve, potentially obstructing the central retinal vein [26, 61]. Pathology has not substantiated the theory of vitreo-

retinal traction during shaking, but implicates a secondary phenomenon due to raised intracranial pressure or venous stasis and leakage from retinal vessels [46]. This hypothesis is consistent with findings in the brain, whose capillary structure and physiology resembles that of the retina, where parenchymal bleeding tends to be associated with venous obstruction and tissue compression (see below and Fig. 15).

A statistically significant relationship between retinal and optic nerve sheath haemorrhage and reperfusion, cardiopulmonary resuscitation (CPR) and cerebral oedema has been demonstrated [102].

Encephalopathy

Brain swelling

The most common pathology encountered in babies with the triad is brain swelling which, together with congestion and neuronal death is regarded as consistent with hypoxic-ischaemic injury (HII) [57, 58, 117]. These findings are non-specific and may result from any insult leading to release of neurotransmitters and neuropeptides which promote a secondary cascade of vascular leakiness leading to brain swelling [129, 161]. Vasogenic oedema results from movement of water across capillary walls into the parenchyma of the brain. Cytotoxic oedema involves a shift of water from the extracellular to intracellular compartment and by itself does not result in a net increase in brain water content or swelling [101]. Brain swelling, which may take between 24 and 72 h to reach its maximum, can obstruct arterial inflow and lead to a perfusion failure and is the most important determinant of mortality and morbidity after head trauma.

Cerebellar tonsillar herniation can compress and distort the cervical cord and put tension on the nerve roots. Fragments of necrotic cerebellar cortex are often displaced around the spinal cord at all levels (Fig. 7).



Fig. 7 Cervical spinal cord damage due to compression by brain swelling and tonsillar herniation. Baby 20 months acute demyelinating encephalomyelitis, who died in hospital with severe brain swelling but without trauma. **a** The lower cervical spinal cord contains a central haemorrhagic, necrotic area just ventral to the

dorsal columns. The yellow/grey tissue around the cord and beneath the dura is displaced and fragmented cerebellar cortex. **b, c** Dorsal nerve roots from **b** upper and **c** lower cervical levels show axon swellings expressing β APP at the exit zone (10 \times)

Parenchymal bleeding

Parenchymal bleeding is uncommon in the infant brain, except in those with prolonged cerebral death who have been nursed on a ventilator. Focal perivascular haemorrhage is seen in compressed and distorted tissues of the herniated cerebellum, brainstem and medial temporal lobes in brain swelling. Perivascular bleeding elsewhere is uncommon and other causes should be sought. Parenchymal bleeding cannot be used as a surrogate for axonal injury as has been suggested [30]. Bleeding and axonal damage are independent of one another.

Hypoxic–ischaemic injury (HII)

Haemorrhage due to HII is usually minimal, perivascular and follows the pattern of neuronal necrosis, being the most common in the inferior olives and the cranial nerve nuclei. The features of infant HII have been described in detail [145].

Cortical vein thrombosis (CVST)

Subpial, cortical perivascular bleeding and bleeding in the immediate subcortical white matter are seen where cortical veins are compressed or thrombosed.

Diffuse intravascular coagulation (DIC)

The characteristic haemorrhages of DIC are typically round and centred on a damaged blood vessel in which a small amount of amorphous pink material may be seen.

Acute necrotising encephalopathy

This is a life-threatening complication of infection. Though rare, it is a significant differential diagnosis in a baby who collapses and dies soon after a short-pyrexial illness with no signs of injury. The bleeding is perivascular and characteristically in the tegmen of the pons and the thalamus.

Traumatic brain damage

Trauma causes brain damage in two distinct stages: primary mechanical tissue disruption and a complex secondary cascade which evolves over hours or days and is the primary target of therapy. Deformation and membrane depolarization lead to the activation of ion channels and disturbances in ionic fluxes which, if sustained, lead to oedema and secondary neurogenic inflammation.

In the vast majority of infants with the triad, hypoxic–ischaemic injury and oedema, rather than traumatic axonal injury, are the predominant cerebral pathologies [57, 58]. Axonal injury may cause immediate loss of consciousness [1]

but the variable pace of swelling means that the clinical manifestations of brain injury can be delayed. This is recognised in clinical practice as a “lucid interval” in which the infant may display only subtle and non-specific signs which a parent or carer may not recognise [5]; the potential for a lucid interval in SBS has recently been acknowledged [42]. Normal neurological examination and maintenance of consciousness do not preclude significant intracranial injury [142].

A number of genetically determined conditions may predispose children to severe brain swelling after minor trauma [85, 152].

Axonal injury

Axonal injury, identified histologically by axonal swellings, has been considered characteristic of trauma in the adult, but in infants it is far more commonly due to hypoxia, ischaemia or metabolic disturbance [36, 128]. Large axonal swellings may be identified in routine H&E stained sections, but are much more readily seen with the use of immunocytochemistry for β APP. The appearance of the axonal swellings does not identify their cause. In adults, their pattern and distribution may enable a diagnosis of diffuse traumatic axonal injury [56], but similar patterns have not been established in the infant brain. Geddes [58] described axonal swellings restricted to the corticospinal tracts in the brainstem and cervical nerve roots in a minority of infants considered to have NAHI, Oehmichen [117] and Johnson [79] were unable to distinguish traumatic axonal damage in the presence of hypoxic injury. An example of ischaemic axonal damage in a site where traumatic injury is characteristic is shown in Fig. 8.

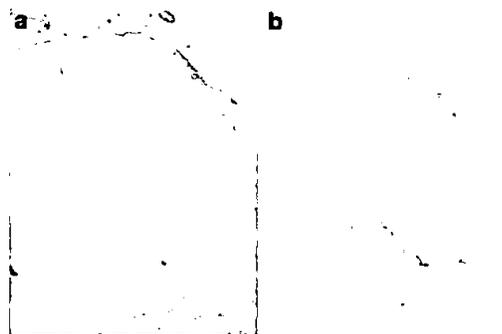


Fig. 8 “Geographical” axonal injury in the brainstem sections through the upper pons showing infarction in the superior cerebellar peduncle, a characteristic site for traumatic axonal injury in the adult. However, in this case, areas of ischaemic injury seen as tissue oedema and pallor in the H&E preparation (a) map precisely to the “geographical” pattern with bands of β APP positive axons sweeping around the areas of infarction in b (2 \times). 3-year-old male with multiple episodes of impact trauma



Fig. 9 Old brainstem axonal injury. **a** Clusters of axonal swellings are readily identified in the corticospinal tracts with H&E staining. **b** The majority of swellings are surrounded by CD68 positive cells processes indicating that they are at least 10 days old and may be much older. **c** The swellings are pale or granular with β APP. This plus

the macrophage reaction suggests that these swellings could be at least 2–4 weeks old (**a–c** $\times 10$). One month infant born with ventouse assistance at 36 weeks and died aged 25 days. There was an old skull fracture thought to be birth related

Axon swellings may develop very soon after injury, perhaps within 35 min [72]. Early swellings stain uniformly brown and persist for up to 10–14 days. After this, the staining fades or becomes granular then disappears, although some granular staining may be seen for up to 3 years after injury. A macrophage reaction around axonal swellings begins from 10 to 11 days after injury and persists for up to 5 months [25, 56] (Fig. 9). Routine use of a marker for microglia and macrophages such as CD68 together with β APP greatly assists in identifying subtle axonal injury.

Brainstem and cervical cord damage

This is the site that shaking must damage if it is to also cause SDH and an encephalopathy since it is the point where the head hinges on the neck [57, 64]. The reported incidence of neck injury in suspected abuse is between 2.5 and 71% [13, 111]. The more inclusive definition of spinal cord injury as “any cervical cord contusion, laceration, or transection; vertebral artery injury; nerve root avulsion/dorsal root ganglia hemorrhage; and meningeal hemorrhage (epidural, intradural, subdural, and/or subarachnoid)” may explain the high incidence in the latter study. Hadley [64] described 13 infants with no direct cranial trauma. 6 had autopsies, of whom 5 had epidural and/or subdural haemorrhage at the cervico-medullary junction and 4 had high cervical spinal contusions. Among infants thought to have suffered NAHI Geddes [57] described axonal injury localised to the corticospinal tracts of the caudal pons and the cervical spinal cord and/or dorsal nerve roots in 31%, Shannon [140] described damage to the cervical spinal cord and dorsal nerve roots in 7 of 11 cases and Oehmichen [117] identified focal axonal injury in 2 of 5 cases where the cervical spinal cord was examined.

The upper cervical cord is vulnerable to infarction in severe brain swelling; there is a watershed between the arterial supply descending from the vertebral system and the radicular vessels. When the brain becomes very swollen

the descending vessels are obstructed; this is not uncommon in “respirator brain” [92]. Figure 7 illustrates cervical cord and nerve root damage due to brain swelling and herniation in the absence of trauma.

Axonal injury in the lower brainstem appears, perhaps surprisingly, to be compatible with survival. Geddes [58] identified swellings of several days old in one case. I have seen old axonal swellings in the brainstem of four babies who survived between 4 days and 4 months before death. Two had suffered traumatic delivery and two had been the subjects of witnessed head trauma (Fig. 9). The clinical effects of this axonal injury are unknown. This part of the brain contains centres controlling vital functions and the reflexes governing breathing and swallowing are still developing in the first year of life. While brainstem axonal injury is clearly survivable, it is likely to make the baby vulnerable and less able to recover from life-threatening events than normal babies, as has been suggested in SIDS [83, 84]. Gliosis and smallness of the brainstem is virtually universal in premature babies with white matter disease [172]. A history of prematurity is not uncommon in babies presenting with the triad and any examination of the brain of a baby dying suddenly, must pay meticulous attention to the possibility of brainstem injury and gliosis as a factor in collapse.

Spinal nerve root pathology

Axonal swellings in spinal nerve roots are sometimes said to represent independent evidence of trauma, either due to shaking and hyperflexion of the neck, or as an indication of direct spinal trauma [103]. However, spinal nerve root swelling has been identified where death is due to natural causes with neither a history nor evidence of trauma [151]. There are no published studies to assist in distinguishing traumatic from other causes of axonal swellings in spinal nerve roots.

Spinal nerve roots are the sites of CSF resorption and surrounded by a dense, valveless vascular plexus



Fig. 10 Spinal nerve root bleeding. **a** Intradural bleeding is seen in the spinal dura and extending out into the nerve root sheath at several sites (*arrows*) close to a dorsal root ganglion. There is fresh intraneural bleeding. An *asterisk* marks the site of bleeding into the spinal dural border layers. Note the very vascular and congested epidural fat. (2 \times). **b** At higher power bleeding is seen into the nerve root sheath as well as within and around the nerve roots themselves (*arrows*) (4 \times). Male 4 months who died after prolonged seizures. There was no evidence of proximate trauma

[164, 187] and so are prone to bleeding, usually as they cross the dura. Spinal subdural haemorrhage can track out along nerve roots beyond the dorsal root ganglia. Further, intraneural bleeding is seen when there has been venous congestion due to raised intrathoracic pressures during resuscitation and ventilation (Fig. 10).

Cerebral contusions/contusional tears

Superficial cortical contusions as seen in adults are not seen in pathological studies of infants, but subcortical contusions (or contusional tears) have been described in infants under 5 months of age. They are rare; Geddes described only 4 in 53 infants, and Oehmichen did not describe any in 18 babies thought to have been abused [57, 117].

Lindenberg originally described contusional tears as clean-walled cysts found just beneath an intact cortex, usually frontal and bilateral, which “could hardly be differentiated from artefact except for some bleeding into the defect and occasionally its margins” [94]. He described microglial hypertrophy but very rare macrophages, and no vascular or oligodendrocyte proliferation. In particular he, and others since, failed to identify axonal injury in relation to subcortical clefts; the axons “simply terminated at the margins”. [20, 173]. Of note, Lindenberg recorded that “The brains of those infants who died shortly after the injury were markedly swollen”. These clefts have similar pathological characteristics to “subcortical leucomalacia” which is associated with brain swelling in the young infant and is not specifically associated with trauma [150].

In a radiological study Jaspán [78] suggested that subcortical contusions are pathognomonic of shaking. The proposed mechanism, gliding of the grey matter over the white matter, defies all known anatomy of the cortex. More recently, this group described subcortical cysts in young infants which were thought to be the result of birth injury. None had any objective evidence of birth or inflicted trauma [6].

Figure 11 illustrates subcortical “contusions” beneath parietal bone fractures. The relative preservation of the cortex indicates that the bleeding beneath it was not due to the direct mechanical forces associated with fracture. Rather, the pattern of bleeding resembles that seen in obstruction of cortical venous drainage (see Figs. 12, 13) and suggests that the cause may have been transient obstruction of the superior sagittal sinus during delivery [126, 159].

Temporal lobe haemorrhage

Bleeding in the temporal pole is sometimes seen in babies with the triad, mistakenly diagnosed as contusion and ascribed to trauma. Superficial (subarachnoid, subpial and subcortical) bleeding over one or both temporal lobes is described in neonates with seizures and apnoea [71, 73, 143]. There is not usually an obvious history of birth trauma, although scalp swelling is sometimes described.

The pathophysiology of this haemorrhage has not been explained but appears to be venous in origin. Veins of the anterior temporal lobe drain into the small and variable sphenoparietal sinuses which connect with the anterior temporal diploic veins [162]. The diploic veins are delicate and superficial and are particularly vulnerable to compression before the outer table of the skull develops at about 5 years of age [67]. Larroche [89] described temporal lobe subarachnoid bleeding in 33 neonates without evidence of trauma and concluded that the pathology was likely to be the result of venous hypertension. Figure 13 illustrates examples of temporal lobe bleeding with radiological correlation.

Cortical vein and/or sinus thrombosis (CVST)

Superficial cortical vein and/or sinus thrombosis (CVST) are discussed together. They are, in my experience, one of the most frequently overlooked pathologies, clinically and pathologically, in babies with the triad.

Radiological studies show extraparenchymal bleeding, including subdural, subarachnoid and subpial haemorrhage and subdural effusion in association with CVST [8, 44, 68, 130]. Parenchymal damage is usually venous infarction which may become haemorrhagic [109, 159]. Subcortical bleeding may be confused with traumatic

Fig. 11 Subcortical and subpial bleeding. **a** Parietal bone fractures (*arrow*) in a term infant who died 5 days after birth by emergency caesarean section with forceps lift-out. **b** The surface of the fixed brain showing congested veins with sharply defined surrounding bleeding which tends to be seen on the edges of sulci. Histology confirmed subpial bleeding. **c** CT scan of this baby shows multiple patches of superficial high signal which was described as “shearing injury”. No tissue shearing was found histologically, there were no axonal swellings. **d** Coronal slice through the fixed brain shows perivascular haemorrhages and blood filled cysts in the immediate subcortical white matter. The overlying cortex, which was beneath the skull fractures, is intact

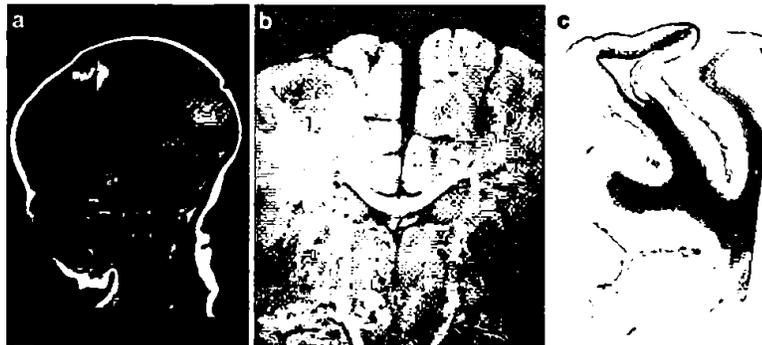
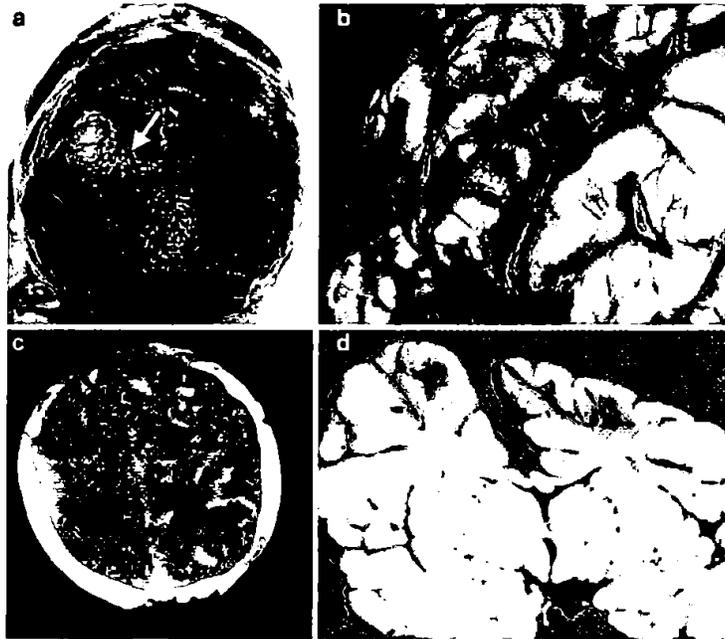


Fig. 12 Subcortical clefts. **a** CT scan. Lateral view showing a fluid containing cyst in the immediate subcortical white matter. Infant 28 days old with pneumonia and no evidence of trauma. **b** The baby died a year later. Residual collapsed clefts are seen in the parasagittal subcortical white matter bilaterally. The cortex is almost completely

spared. **c** Male 3 months. This twin boy had a history of poor head control since the neonatal period. A section of frontal lobe stained with GFAP shows parasagittal subcortical clefts with the overlying cortex intact but focally thin and gliotic

shear injuries and misdiagnosed as “cortical contusions” [8] (see Fig. 11c).

Parietal veins are commonly thrombosed as these veins turn at an acute angle and pass into a dural sleeve for some distance prior to entering the sinus. Compression of the superior sagittal sinus by the upper occipital bone close to the posterior fontanelle has been associated with the development of CVST [159]. Reduced flow in the superficial veins or

sinuses causes damage in the immediate subcortical white matter which is the watershed of the deep and superficial venous systems. This leads to local oedema which can be transient and reversible, or to venous infarction with or without haemorrhage [126]. Bleeding around small cortical veins may track into the subpial areas [171] (Fig. 13e).

There is a striking male predominance (up to 75%) in infant CVST [10, 182]. Clinical diagnosis is difficult in

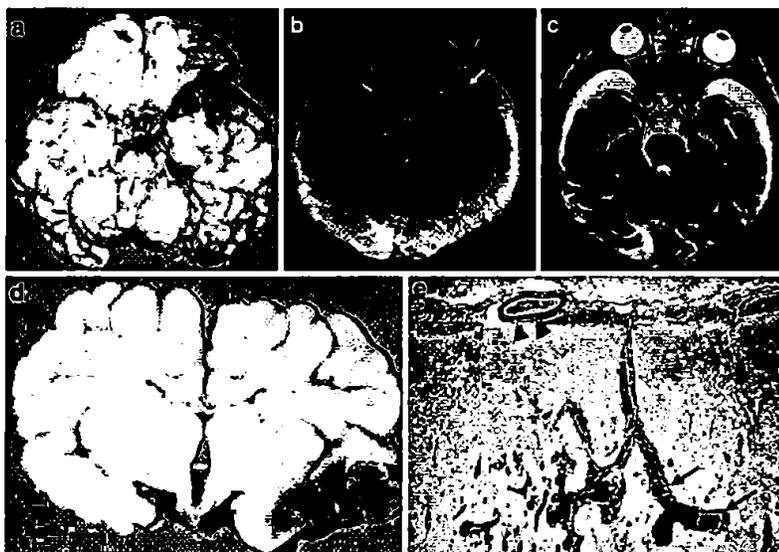


Fig. 13 Temporal lobe haemorrhage. **a** Surface bleeding over the left temporal pole. A corresponding coronal slice of the fixed brain is seen in **d**. **b**, **c** Radiological appearance of temporal lobe haemorrhage in a 5-month infant with thrombosis of the superficial middle temporal vein. **b** T2*/GRE (haem sensitive sequence) shows a thrombosed vein (*arrow*). **c** T2 shows fluid/oedema in the immediate subcortical white matter (*arrow*), the cortex apparently intact. **d** Bleeding is seen in a

very fine subpial layer as well as in the immediate subcortical white matter; the cortex appears intact. **e** Subpial bleeding in a sulcus beneath a space-occupying subdural haemorrhage. *Arrowheads* indicate where the pia is lifted off by a thin fresh surface bleed. There is blood around the venous tributaries in the deep cortical levels and extending up towards the surface (*arrows*) (Reticulin 4 \times). Female of 20 months with head impact due to a fall

infants; at least 10% of babies are asymptomatic, and others have non-specific presentations including depressed consciousness, lethargy, poor feeding, vomiting or seizures [139, 165].

Venous thrombosis is associated with a number of common illnesses. 75% have infections, 33% prothrombotic disorders and 4% recent head trauma [139]. Neonates have additional risk factors, including asphyxia, complicated delivery and altered sinus flow during skull moulding [10, 180].

Timing of intravascular clot by pathology alone is difficult and reliable, but may assist in understanding the totality of a case by relating the pathology to the clinical evidence. Old organising cortical vein thrombosis and associated subcortical damage is shown in Fig. 14. Histological criteria for timing, derived from studies of adult and animal CVST, have been published [49, 115, 116, 138].

Respirator brain

Not infrequently a baby is kept alive on a ventilator for several days after the brain has become severely swollen and is no longer receiving an adequate blood supply. There is little inflammatory response in the brain due to the

absence of circulation. However, there may be reactive change around the spinal cord and at the vascular watershed in the cervical spinal cord where central necrosis can be mistaken for traumatic damage [92]. The dural blood supply is preserved and timing of the pathology here may be more reliable than in the brain where lack of a blood supply makes timing impossible as the normal sequence of cellular processes is inactive.

Conditions which may present with the triad

The list of conditions which may cause an infant to develop the triad is exhaustive. Below are brief notes on the most common causes of the triad which I have encountered in my own clinical diagnostic and forensic practices. Many others are discussed elsewhere [8, 51].

Chronic subdural haemorrhage

The majority of babies with the triad, perhaps 70–80% have chronic SDH. In the absence of any recent or remote evidence of trauma the question of residual consequences of birth related bleeding must be considered.

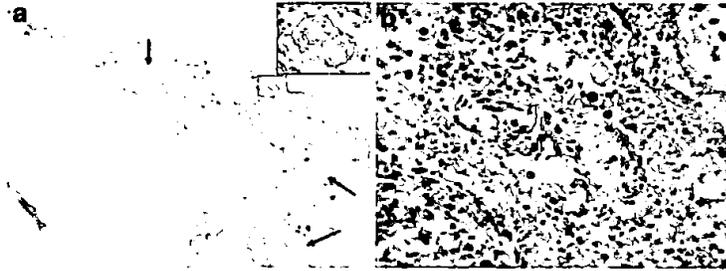


Fig. 14 Old cortical vein thrombosis. A cleft in the parasagittal cortex contains fresh blood beneath a residual band of thinned and gliotic cortex (*arrows*). The overlying leptomeninges are thickened

and cellular with many pigment-containing macrophages. Recanalised vessels are seen within them. An example in the box is seen in a, another in b. (H&E a 2 \times , b 10 \times)

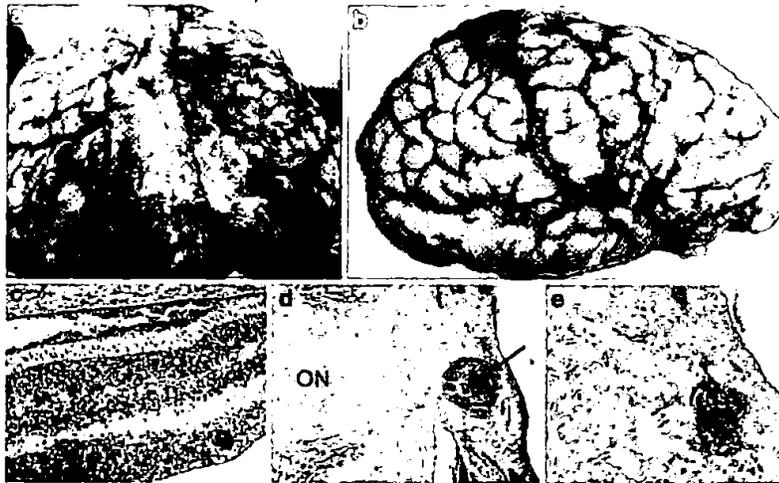


Fig. 15 Cortical vein and sinus thrombosis. Male infant aged 4 weeks who collapsed and became floppy in a public park. Subdural bleeding was diagnosed on CT scan. **a** The autopsy showed dural sinus thrombosis. There is patchy bleeding over the surface of the brain related to thrombosed cortical veins. **b** Fixed brain: the right superficial middle (anastomatic) cerebral vein is thrombosed. This

baby also had retinal haemorrhage which was due to central retinal vein thrombosis. **c** Haemorrhage at all levels of the retina (H&E 4 \times). **d** There is thrombus in the central retinal vein in the optic nerve head (*arrow*). *ON* optic nerve (Masson's trichrome). **e** CD31 staining shows organisation and early recanalisation of the central retinal vein (d, e 2 \times)

Accidental falls

There is evidence that even low level falls may cause intracranial damage in the infant. Skull fractures may be asymptomatic and symptoms non-specific. The carer's account should be considered "The clinical history is perhaps the most important clinical tool available to the clinician and to reject the carer's version of events in favour of another requires the highest possible level of medical evidence. After all, the Doctor is effectively accusing the carer of lying" [54].

Resuscitated SIDS

The difference between SIDS and SBS may be due to the long period of hypoxia and subsequent resuscitation that most SBS babies experience. Both share demographic factors such as age, male predominance and mild illness prior to the presentation. Certain clinical circumstances are particularly common in babies with the triad for example:

- *Aspiration* of stomach contents or pooled secretions have been implicated in SIDS through activation of the oxygen conserving reflexes such as the laryngeal

chemo reflex which are particularly powerful in infants. [62, 83]. In many cases, a parent gives a detailed account of a baby collapsing after or during a feed, regurgitating vomit and milk. The story is accurately repeated in multiple interviews. There is often a history of feeding difficulties and “reflux” and most of these babies have a chronic subdural haemorrhage.

- *Premature* babies are likely to have brainstem compromise and may not be able to overcome events that a normal baby can. The vulnerability of even mildly premature babies has been stressed [12].
- *Vaccination* Infants may collapse with the triad in the days following immunisations, possibly due to a pyrexial response triggering seizures.

Cortical vein and sinus thrombosis

These conditions are frequent but are underdiagnosed both clinically and pathologically in infants. The surface veins and dural sinuses must *always* be examined.

Inflicted injury

Many babies suffer inflicted trauma which causes the triad. In the absence of clinical or pathological evidence of trauma, it is beyond the ability or expertise of the neuropathologist to make this diagnosis, which is a matter for the legal authorities.

Vitamin D deficiency

There is a newly recognised epidemic of Vitamin D deficiency among pregnant women. Experimental evidence indicates that in addition to the classical bone lesions, brain growth and immune function may be compromised [88]. Complications of Vitamin D deficiency including tetany, seizures and cardiac failure can lead to collapse with brain swelling and presentation with the triad.

Second impact syndrome

This syndrome describes acute hemispheric swelling beneath a thin film of subdural bleeding of heterogeneous appearance after a second head injury, often very mild, occurring days or weeks after a first [21, 112]. Most patients are adolescents but the similarities to infants with SDH who may have suffered non-accidental trauma were noted by Cantu [21]. Careful review of the clinical history often discloses an impact in the days or weeks prior to collapse from which the baby apparently recovered and which may not have been taken into account on admission.

More research is needed to define whether this syndrome may underlie the triad in some infants.

Aneurysm rupture

Intracranial vascular malformations can and do rupture in infants and cause the triad.

Rare genetic conditions

Many infant deaths have underlying genetic conditions. Disorders of cardiac rhythm, coagulation or osteogenesis are the most likely to lead to being confused with abusive injury.

Conclusion

Neuropathologists have the benefit of detailed study of the empirical evidence offered by the tissues. A pragmatic analysis of this evidence remains the cornerstone of the clinical and forensic diagnosis.

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References

1. Adams JH, Graham DI, Murray LS, Scott G (1982) Diffuse axonal injury due to nonmissile head injury in humans: an analysis of 45 cases. *Ann Neurol* 12:557–563
2. Adamsbaum C, Grabar S, Mejean N, Rey-Salmon C (2010) Abusive head trauma: judicial admissions highlight violent and repetitive shaking. *Pediatrics* 126:546–555
3. Aoki N, Masuzawa H (1984) Infantile acute subdural hematoma. Clinical analysis of 26 cases. *J Neurosurg* 61:273–280
4. Aoki N (1990) Chronic subdural hematoma in infancy. Clinical analysis of 30 cases in the CT era. *J Neurosurg* 73:201–205
5. Arbogast KB, Margulies SS, Christian CW (2005) Initial neurologic presentation in young children sustaining inflicted and unintentional fatal head injuries. *Pediatrics* 116:180–184
6. Au-Yong IT, Wardle SP, McConachie NS, Jaspan T (2009) Isolated cerebral cortical tears in children: aetiology, characterisation and differentiation from non-accidental head injury. *Br J Radiol* 82:735–741
7. Auer LM, Ishiyama N, Hodde KC, Kleinert R, Pucher R (1987) Effect of intracranial pressure on bridging veins in rats. *J Neurosurg* 67:263–268
8. Barnes PD (2011) Imaging of nonaccidental injury and the mimics: issues and controversies in the era of evidence-based medicine. *Radiol Clin North Am* 49:205–229

9. Bell JE (2005) The Neuropathology of non-accidental head injury. In: Minns R (ed) Shaking and other non-accidental head injuries in children. Mac Keith, London
10. Berfelo FJ, Kersbergen KJ, van Ommen CH, Govaert P, van Straaten HL, Poll-The BT, van Wezel-Meijler G, Vermeulen RJ, Groenendaal F, de Vries LS, de Haan TR (2010) Neonatal cerebral sinovenous thrombosis from symptom to outcome. *Stroke* 41:1382–1388
11. Bhardwaj G, Chowdhury V, Jacobs MB, Moran KT, Martin FJ, Coroneo MT (2010) A systematic review of the diagnostic accuracy of ocular signs in pediatric abusive head trauma. *Ophthalmology* 117:983–992, e917
12. Boyle JD, Boyle EM (2011) Born just a few weeks early: does it matter? *Arch Dis Child Fetal Neonatal Ed*
13. Brennan LK, Rubin D, Christian CW, Duhaime AC, Mirchandani HG, Rorke-Adams LB (2009) Neck injuries in young pediatric homicide victims. *J Neurosurg Pediatr* 3:232–239
14. Browder J, Kaplan HA, Krieger AJ (1975) Venous lakes in the suboccipital dura mater and falx cerebelli of infants: surgical significance. *Surg Neurol* 4:53–55
15. Buis DR, van Ouwkerk WJ, Takahata H, Vandertop WP (2006) Intracranial aneurysms in children under 1 year of age: a systematic review of the literature. *Childs Nerv Syst* 22:1395–1409
16. Byard RW, Blumbers P, Rutty G, Spherhake J, Banner J, Krous HF (2007) Lack of evidence for a causal relationship between hypoxic-ischemic encephalopathy and subdural hemorrhage in fetal life, infancy, and early childhood. *Pediatr Dev Pathol* 10:348–350
17. Caffey J (1972) On the theory and practice of shaking infants. Its potential residual effects of permanent brain damage and mental retardation. *Am J Dis Child* 124:161–169
18. Caffey J (1974) 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* 54:396–403
19. Caldarelli M, Di RC, Romani R (2002) Surgical treatment of chronic subdural hygromas in infants and children. *Acta Neurochir (Wien)* 144:581–588
20. Calder JM, Hill I, Scholtz CL (1984) Primary brain trauma in non-accidental injury. *J ClinPathol* 37:1095–1100
21. Cantu RC, Gean AD (2010) Second-impact syndrome and a small subdural hematoma: an uncommon catastrophic result of repetitive head injury with a characteristic imaging appearance. *J Neurotrauma* 27:1557–1564
22. Case ME, Graham MA, Handy TC, Jentzen JM, Monteleone JA (2001) Position paper on fatal abusive head injuries in infants and young children. *Am J Forensic Med Pathol* 22:112–122
23. Case ME (2007) Abusive head injuries in infants and young children. *Leg Med (Tokyo)* 9:83–87
24. Chamaanvanakij S, Rollins N, Perlman JM (2002) Subdural hematoma in term infants. *Pediatr Neurol* 26:301–304
25. Chen XH, Johnson VE, Uryu K, Trojanowski JQ, Smith DH (2009) A lack of amyloid beta plaques despite persistent accumulation of amyloid beta in axons of long-term survivors of traumatic brain injury. *Brain Pathol* 19:214–223
26. Christian CW, Taylor AA, Hertle RW, Duhaime AC (1999) Retinal hemorrhages caused by accidental household trauma. *J Pediatr* 135:125–127
27. Christian CW, Block R (2009) Abusive head trauma in infants and children. *Pediatrics* 123:1409–1411
28. Cohen MC, Scheimberg I (2008) Evidence of occurrence of intradural and subdural hemorrhage in the perinatal and neonatal period in the context of hypoxic ischemic encephalopathy. An observational study from two referral institutions in the United Kingdom. *Pediatr Dev Pathol* 1
29. Cohen MC, Sprigg A, Whitby EH (2010) Subdural hemorrhage, intradural hemorrhage and hypoxia in the pediatric and perinatal post mortem: are they related? An observational study combining the use of post mortem pathology and magnetic resonance imaging. *Forensic SciInt* 200:100–107
30. Colbert CA, Holshouser BA, Aaen GS, Sheridan C, Oyoyo U, Kido D, Ashwal S (2010) Value of cerebral microhemorrhages detected with susceptibility-weighted MR Imaging for prediction of long-term outcome in children with nonaccidental trauma. *Radiology* 256:898–905
31. Cory CZ, Jones BM (2003) Can shaking alone cause fatal brain injury? A biomechanical assessment of the Duhaime shaken baby syndrome model. *Med Sci Law* 43:317–333
32. Crown Prosecution S (2011) Non-accidental head injury (nahi, formerly referred to as shaken baby syndrome (SBS))—prosecution approach. http://www.cps.gov.uk/legal/l_to_o/non_accidental_head_injury_cases/
33. Cushing H, In Goodrich JT (2000) Reprint of “concerning surgical intervention for the intracranial hemorrhages of the new-born” by Harvey Cushing, M.D. 1905. *Childs Nerv Syst* 16:484–492
34. Dandy W (1929) Where is cerebrospinal fluid absorbed? *J Am Med Assoc*
35. Dias MS (2010) The case for shaking. In: Jenny C (ed) Child abuse and neglect. Elsevier Saunders
36. Dolinak D, Smith C, Graham DJ (2000) Hypoglycaemia is a cause of axonal injury. *Neuropathol Appl Neurobiol* 26:448–453
37. Duhaime AC, Gennarelli TA, Thibault LE, Bruce DA, Margulies SS, Wiser R (1987) The shaken baby syndrome. A clinical, pathological, and biomechanical study. *J Neurosurg* 66:409–415
38. Duhaime AC, Gennarelli TA, Sutton LN, Schut L (1988) “Shaken baby syndrome”: A misnomer? *J Pediatr Neurosci* 4:77–86
39. Duhaime AC, Alario AJ, Lewander WJ, Schut L, Sutton LN, Seidl TS, Nudelman S, Budenz D, Hertle R, Tsiaras W (1992) Head injury in very young children: mechanisms, injury types, and ophthalmologic findings in 100 hospitalized patients younger than 2 years of age. *Pediatrics* 90:179–185
40. Duhaime AC, Christian CW, Rorke LB, Zimmerman RA (1998) Nonaccidental head injury in infants—the “shaken-baby syndrome”. *N Engl J Med* 338:1822–1829
41. Dupuis O, Silveira R, Dupont C, Mottolose C, Kahn P, Dittmar A, Rudigoz RC (2005) Comparison of “instrument-associated” and “spontaneous” obstetric depressed skull fractures in a cohort of 68 neonates 2. *Am J Obstet Gynecol* 192:165–170
42. Ebbs Justice D (2011) Report to the attorney general: Shaken baby death review. Ontario
43. Edlow JA, Caplan LR (2000) Avoiding pitfalls in the diagnosis of subarachnoid hemorrhage. *N Engl J Med* 342:29–36
44. Eichler F, Krishnamoorthy K, Grant PE (2007) Magnetic resonance imaging evaluation of possible neonatal sinovenous thrombosis. *Pediatr Neurol* 37:317–323
45. Eisenbrey AB (1979) Retinal hemorrhage in the battered child. *Childs Brain* 5:40–44
46. Emerson MV, Jakobs E, Green WR (2007) Ocular autopsy and histopathologic features of child abuse. *Ophthalmology* 114:1384–1394
47. Ewing-Cobbs L, Prasad M, Kramer L, Louis PT, Baumgartner J, Fletcher JM, Alpert B (2000) Acute neuroanatomic findings in young children with inflicted or noninflicted traumatic brain injury. *Childs Nerv Syst* 16:25–33
48. Feng JF, Jiang JY, Bao YH, Liang YM, Pan YH (2008) Traumatic subdural effusion evolves into chronic subdural hematoma: Two stages of the same inflammatory reaction? *Med Hypotheses* 70:1147–1149
49. Fineschi V, Turillazzi E, Neri M, Pomara C, Riezzo I (2009) Histological age determination of venous thrombosis:

- a neglected forensic task in fatal pulmonary thrombo-embolism. *Forensic Sci Int* 186:22–28
50. Fox RJ (1996) Anatomic details of intradural channels in the parasagittal dura: a possible pathway for flow of cerebrospinal fluid. *Neurosurgery* 39:84–91
 51. Frasier L et al. (2006) In: Frasier L (ed) *Abusive head trauma in infants and children*, G W Medical
 52. Friede RL (1972) Subpial hemorrhage in infants. *J Neuropathol Exp Neurol* 31:548–556
 53. Friede RL (1989) Subdural haematomas, hygromas and effusions. In: Friede RL (ed) *Developmental neuropathology*. Springer, Göttingen, pp 198–208
 54. Gage LJ, Harris RV et al (2005) EWCA. *Crim* 1980
 55. Gardner HB (2007) A witnessed short fall mimicking presumed shaken baby syndrome (inflicted childhood neurotrauma). *Pediatr Neurosurg* 43:433–435
 56. Geddes JF, Vowles GH, Beer TW, Ellison DW (1997) The diagnosis of diffuse axonal injury: implications for forensic practice. *Neuropathol Appl Neurobiol* 23:339–347
 57. Geddes JF, Hackshaw AK, Vowles GH, Nickols CD, Whitwell HL (2001) Neuropathology of inflicted head injury in children. I. Patterns of brain damage. *Brain* 124:1290–1298
 58. Geddes JF, Vowles GH, Hackshaw AK, Nickols CD, Scott IS, Whitwell HL (2001) Neuropathology of inflicted head injury in children. II. Microscopic brain injury in infants. *Brain* 124:1299–1306
 59. Geddes JF, Tasker RC, Hackshaw AK, Nickols CD, Adams GG, Whitwell HL, Scheinberg I (2003) Dural haemorrhage in non-traumatic infant deaths: does it explain the bleeding in 'shaken baby syndrome?'. *Neuropathol Appl Neurobiol* 29:14–22
 60. Ghosh PS, Ghosh D (2011) Subdural hematoma in infants without accidental or nonaccidental injury: benign external hydrocephalus, a risk factor. *Clin Pediatr*
 61. Gilles EE, McGregor ML, Levy-Clarke G (2003) Retinal hemorrhage asymmetry in inflicted head injury: a clue to pathogenesis? *J Pediatr* 143:494–499
 62. Gorini C, Philbin K, Buteman R, Mendelowitz D (2010) Endogenous inhibition of the trigeminally evoked neurotransmission to cardiac vagal neurons by muscarinic acetylcholine receptors. *J Neurophysiol* 104:1841–1848
 63. Guthkelch AN (1971) Infantile subdural haematoma and its relationship to whiplash injuries. *Br Med J* 2:430–431
 64. Hadley MN, Sonntag VK, Rekate HL, Murphy A (1989) The infant whiplash-shake injury syndrome: a clinical and pathological study. *Neurosurgery* 24:536–540
 65. Haines DE, Harkey HL, al Mefty O (1993) The "subdural" space: a new look at an outdated concept. *Neurosurgery* 32:111–120
 66. Hall JR, Reyes HM, Horvat M, Meller JL, Stein R (1989) The mortality of childhood falls. *J Trauma* 29:1273–1275
 67. Hershkovitz I, Greenwald C, Rothschild BM, Latimer B, Dutour O, Jellema LM, Wish-Baratz S, Pap I, Leonetti G (1999) The elusive diploic veins: anthropological and anatomical perspective. *Am J Phys Anthropol* 108:345–358
 68. Herzberger E, Rotem Y, Braham J (1956) Remarks on thirty-three cases of subdural effusion in infancy. *Arch Dis Child* 31:44–50
 69. Hobbs C, Childs AM, Wynne J, Livingston J, Seal A (2005) Subdural haematoma and effusion in infancy: an epidemiological study. *Arch Dis Child* 90:952–955
 70. Holbourn A (1943) Mechanics of head injuries. *The Lancet*:438
 71. Hoogstraate SR, Lequin MH, Huysman MA, Ahmed S, Govaert PP (2009) Apnoea in relation to neonatal temporal lobe haemorrhage. *Eur J Paediatr Neurol* 13:356–361
 72. Hortobagyi T, Wise S, Hunt N, Cary N, Djurovic V, Fegan-Earl A, Shorrock K, Rouse D, Al Sarraj S (2007) Traumatic axonal damage in the brain can be detected using beta-APP immunohistochemistry within 35 min after head injury to human adults 1. *Neuropathol Appl Neurobiol* 33:226–237
 73. Huang AH, Robertson RL (2004) Spontaneous superficial parenchymal and leptomeningeal hemorrhage in term neonates. *AJNR Am J Neuroradiol* 25:469–475
 74. Hurley M, Dineen R, Padfield CJ, Wilson S, Stephenson T, Vyas H, McConachie N, Jaspan T (2010) Is there a causal relationship between the hypoxia-ischaemia associated with cardiorespiratory arrest and subdural haematomas? An observational study. *Br J Radiol* 83:736–743
 75. Hwang SK, Kim SL (2000) Infantile head injury, with special reference to the development of chronic subdural hematoma. *Childs Nerv Syst* 16:590–594
 76. Hymel KP, Jenny C, Block RW (2002) Intracranial hemorrhage and rebleeding in suspected victims of abusive head trauma: addressing the forensic controversies. *Child Maltreat* 7:329–348
 77. Ikeda A, Sato O, Tsugane R, Shibuya N, Yamamoto I, Shimoda M (1987) Infantile acute subdural hematoma. *Childs Nerv Syst* 3:19–22
 78. Jaspan T, Narborough G, Punt JA, Lowe J (1992) Cerebral contusional tears as a marker of child abuse—detection by cranial sonography. *Pediatr Radiol* 22:237–245
 79. Johnson MW, Stoll L, Rubio A, Troncoso J, Pletnikova O, Fowler DR, Li L (2011) Axonal injury in young pediatric head trauma: a comparison study of beta-amyloid precursor protein (beta-APP) immunohistochemical staining in traumatic and nontraumatic deaths*. *J Forensic Sci*
 80. Johnston M, Armstrong D, Koh L (2007) Possible role of the cavernous sinus veins in cerebrospinal fluid absorption. *Cerebrospinal Fluid Res* 4:3
 81. Keeling J (2009) *Paediatric forensic medicine and pathology*. Edward Arnold
 82. Keeling JW (1993) *Fetal and neonatal pathology*. Springer, London
 83. Kinney HC, Thach BT (2009) The sudden infant death syndrome. *N Engl J Med* 361:795–805
 84. Kinney HC, Broadbelt KG, Haynes RL, Rognum IJ, Paterson DS (2011) The serotonergic anatomy of the developing human medulla oblongata: implications for pediatric disorders of homeostasis. *J Chem Neuroanat* 41:182–199
 85. Kors EE, Terwindt GM, Vermeulen FL, Fitzsimons RB, Jardine PE, Heywood P, Love S, van den Maagdenberg AM, Haan J, Frants RR, Ferrari MD (2001) Delayed cerebral edema and fatal coma after minor head trauma: role of the CACNA1A calcium channel subunit gene and relationship with familial hemiplegic migraine. *Ann Neurol* 49:753–760
 86. Koumellis P, McConachie NS, Jaspan T (2008) Spinal subdural hematomas in children with non accidental head injury. *Arch Dis Child*
 87. Lantz PE, Stanton CA (2006) Postmortem detection and evaluation of retinal hemorrhages. *Am Acad Forensic Sci* 271
 88. Lapillonne A (2010) Vitamin D deficiency during pregnancy may impair maternal and fetal outcomes. *Med Hypotheses* 74:71–75
 89. Larroche J-C (1977) Lesions of haemorrhagic type, mainly venous. *Developmental pathology of the neonate*. Excerpta Medica, Amsterdam, pp 355–398
 90. Leach JL, Fortuna RB, Jones BV, Gaskill-Shiple MF (2006) Imaging of cerebral venous thrombosis: current techniques, spectrum of findings, and diagnostic pitfalls. *Radiographics: a review publication of the Radiological Society of North America, Inc* 26(Suppl 1):S19–S41; discussion S42–13
 91. Leestma JE (2005) Case analysis of brain-injured admittedly shaken infants: 54 cases, 1969–2001. *Am J Forensic Med Pathol* 26:199–212
 92. Leestma JE (2009) *Forensic neuropathology*. CRC Press, Taylor and Francis Group

93. Levin AV, Forbes B, Alexander R, Jenny C (2010) Information statement. Abusive head trauma/shaken baby syndrome. American Academy of Ophthalmology, San Francisco
94. Lindenberg R, Freytag E (1969) Morphology of brain lesions from blunt trauma in early infancy. *Arch Pathol* 87:298–305
95. Loh JK, Lin CL, Kwan AL, Howng SL (2002) Acute subdural hematoma in infancy. *Surg Neurol* 58:218–224
96. Looney CB, Smith JK, Merck LH, Wolfe HM, Chescheir NC, Hamer RM, Gilmore JH (2007) Intracranial hemorrhage in asymptomatic neonates: prevalence on MR images and relationship to obstetric and neonatal risk factors. *Radiology* 242:535–541
97. Lu H, Zhao J, Li M, Cheng Y, Li Y, You X, Zhao Y (2010) Microvessel changes after post-ischemic benign and malignant hyperemia: experimental study in rats. *BMC Neurol* 10:24
98. Mack J, Squier W, Eastman JT (2009) Anatomy and development of the meninges: implications for subdural collections and CSF circulation. *Pediatr Radiol* 39:200–210
99. Maguire S, Pickerd N, Farewell D, Mann M, Tempest V, Kemp AM (2009) Which clinical features distinguish inflicted from non-inflicted brain injury? A systematic review. *Arch Dis Child* 94:860–867
100. Manolic MD, Kochanek PM, Fink EL, Clark RS (2009) Post-cardiac arrest syndrome: focus on the brain. *Curr Opin Pediatr* 21:745–750
101. Marmarou A (2007) A review of progress in understanding the pathophysiology and treatment of brain edema. *Neurosurg Focus* 22:E1
102. Matshes E (2010) Retinal and optic nerve sheath haemorrhages are not pathognomonic of abusive head injury. Abstract American Association of Forensic Sciences Conference
103. Matshes E, Rea E (2011) Shaken infants die of neck trauma, not brain trauma. *Acad Forensic Pathol* 1:b–k
104. Maxeiner H (1997) Detection of ruptured cerebral bridging veins at autopsy. *Forensic SciInt* 89:103–110
105. Maxeiner H (2001) Demonstration and interpretation of bridging vein ruptures in cases of infantile subdural bleedings. *J Forensic Sci* 46:85–93
106. McLellan NJ, Prasad R, Punt J (1986) Spontaneous subhyaloid and retinal haemorrhages in an infant. *Arch Dis Child* 61:1130–1132
107. McNeely PD, Atkinson JD, Saigal G, O’Gorman AM, Farmer JP (2006) Subdural hematomas in infants with benign enlargement of the subarachnoid spaces are not pathognomonic for child abuse. *AJNR Am J Neuroradiol* 27:1725–1728
108. Menkes JH, Sarnat HB, Maria BL (2006) *Child neurology*, 7th edn
109. Moharir MD, Shroff M, Stephens D, Pontigon AM, Chan A, MacGregor D, Mikulis D, Adams M, DeVeber G (2010) Anticoagulants in pediatric cerebral sinovenous thrombosis: a safety and outcome study. *Ann Neurol* 67:590–599
110. Mokri B, Parisi JE, Scheithauer BW, Piepgras DG, Miller GM (1995) Meningeal biopsy in intracranial hypotension: meningeal enhancement on MRI. *Neurology* 45:1801–1807
111. Molina DK, Clarkson A, Farley KL, Farley NJ (2011) A review of blunt force injury homicides of children aged 0 to 5 years in Bexar County, Texas, from 1988 to 2009. *Am J Forensic Med Pathol*
112. Mori T, Katayama Y, Kawamata T (2006) Acute hemispheric swelling associated with thin subdural hematomas: pathophysiology of repetitive head injury in sports. *Acta Neurochir Suppl* 96:40–43
113. Muller PJ, Deck JH (1974) Intraocular and optic nerve sheath hemorrhage in cases of sudden intracranial hypertension. *J Neurosurg* 41:160–166
114. Munro D, Merritt HH (1936) Surgical pathology of subdural hematoma. Based on a study of one hundred and five cases. *Arch Neurol Psychiatry* 35:64–78
115. Nosaka M, Ishida Y, Kimura A, Kondo T (2009) Time-dependent appearance of intrathrombus neutrophils and macrophages in a stasis-induced deep vein thrombosis model and its application to thrombus age determination. *Int J Legal Med* 123:235–240
116. Nosaka M, Ishida Y, Kimura A, Kondo T (2010) Time-dependent organic changes of intravenous thrombi in stasis-induced deep vein thrombosis model and its application to thrombus age determination. *Forensic SciInt* 195:143–147
117. Oehmichen M, Schleiss D, Pedal I, Saierus KS, Gerling I, Meissner C (2008) Shaken baby syndrome: re-examination of diffuse axonal injury as cause of death. *Acta Neuropathol* 116:317–329
118. Oi S, Di Rocco C (2006) Proposal of “evolution theory in cerebrospinal fluid dynamics” and minor pathway hydrocephalus in developing immature brain. *Childs Nerv Syst* 22:662–669
119. Ommaya AK, Goldsmith W, Thibault L (2002) Biomechanics and neuropathology of adult and paediatric head injury. *Br J Neurosurg* 16:220–242
120. Papaiconomou C, Zakharov A, Azizi N, Djenic J, Johnston M (2004) Reassessment of the pathways responsible for cerebrospinal fluid absorption in the neonate. *Childs Nerv Syst* 20:29–36
121. Papasian NC, Frim DM (2000) A theoretical model of benign external hydrocephalus that predicts a predisposition towards extra-axial hemorrhage after minor head trauma. *Pediatr Neurosurg* 33:188–193
122. Park CK, Choi KH, Kim MC, Kang JK, Choi CR (1994) Spontaneous evolution of posttraumatic subdural hygroma into chronic subdural haematoma. *Acta Neurochir (Wien)* 127:41–47
123. Pitman T (2003) Significance of a subdural hematoma in a child with external hydrocephalus. *Pediatr Neurosurg* 39:57–59
124. Plunkett J (1999) Sudden death in an infant caused by rupture of a basilar artery aneurysm. *Am J Forensic Med Pathol* 20:211–214
125. Prange MT, Coats B, Duhaime AC, Margulies SS (2003) Anthropomorphic simulations of falls, shakes, and inflicted impacts in infants. *J Neurosurg* 99:143–150
126. Ramenghi LA, Govaert P, Fumagalli M, Bassi L, Mosca F (2009) Neonatal cerebral sinovenous thrombosis. *Semin Fetal Neonatal Med* 14:278–283
127. Reddie IC, Bhardwaj G, Dauber SL, Jacobs MB, Moran KT (2010) Bilateral retinoschisis in a 2-year-old following a three-storey fall. *Eye (Lond)* 24:1426–1427
128. Reichard RR, Smith C, Graham DI (2005) The significance of beta-APP immunoreactivity in forensic practice. *Neuropathol Appl Neurobiol* 31:304–313
129. Richardson JD, Vasko MR (2002) Cellular mechanisms of neurogenic inflammation. *J Pharmacol Exp Ther* 302:839–845
130. Roach ES, Golomb MR, Adams R, Biller J, Daniels S, DeVeber G, Ferrero D, Jones BV, Kirkham FJ, Scott RM, Smith ER (2008) Management of stroke in infants and children: a scientific statement from a Special Writing Group of the American Heart Association Stroke Council and the Council on Cardiovascular Disease in the Young. *Stroke* 39:2644–2691
131. Rogers CB, Itabashi HH, Tomiyasu U, Heuser ET (1998) Subdural neomembranes and sudden infant death syndrome. *J Forensic Sci* 43:375–376
132. Rooks VJ, Eaton JP, Ruess L, Petermann GW, Keck-Wherley J, Pedersen RC (2008) Prevalence and Evolution of Intracranial Hemorrhage in Asymptomatic Term Infants. *AJNR Am J Neuroradiol*
133. Roth S, Raul JS, Ludes B, Willinger R (2007) Finite element analysis of impact and shaking inflicted to a child. *Int J Legal Med* 121:223–228
134. Royal College of P (2009) Report of a meeting on the pathology of traumatic head injury in children
135. Ruddick C, Ward Platt MP, De San LC (2009) Head trauma outcomes of verifiable falls in newborn babies. *Arch Dis Child*

136. Rutty GN, Squier WM, Padfield CJ (2005) Epidural haemorrhage of the cervical spinal cord: a post-mortem artefact? *Neuropathol Appl Neurobiol* 31:247–257
137. Safur P, Behringer W, Bottiger BW, Sterz F (2002) Cerebral resuscitation potentials for cardiac arrest. *Crit Care Med* 30:S140–S144
138. Saukko P, Knight B (2004) *Knight's forensic pathology*. Arnold, London
139. Sebire G, Tabarki B, Saunders DE, Leroy I, Liesner R, Saint-Martin C, Husson B, Williams AN, Wade A, Kirkham FJ (2005) Cerebral venous sinus thrombosis in children: risk factors, presentation, diagnosis and outcome. *Brain* 128:477–489
140. Shannon P, Smith CR, Deck J, Ang LC, Ho M, Becker L (1998) Axonal injury and the neuropathology of shaken baby syndrome. *Acta Neuropathol (Berl)* 95:625–631
141. Si Z, Luan L, Kong D, Zhao G, Wang H, Zhang K, Yu T, Pang Q (2008) MRI-based investigation on outflow segment of cerebral venous system under increased ICP condition. *Eur J Med Res* 13:121–126
142. Simon B, Letourneau P, Vitorino E, McCall J (2001) Pediatric minor head trauma: indications for computed tomographic scanning revisited. *J Trauma* 51:231–237
143. Slaughter L, Egelhoff J, Balmakund T (2009) Neurologic outcome in neonatal temporal lobe hemorrhagic venous infarcts. *J Child Neurol* 24:1236–1242
144. Smith C, Bell JE, Keeling JW, Risden RA (2003) Dural haemorrhage in nontraumatic infant deaths: does it explain the bleeding in 'shaken baby syndrome'? Geddes JE et al. A response. *Neuropathol Appl Neurobiol* 29:411–412
145. Smith CS, M (2007) Acquired diseases of the nervous system. In: Keeling JYK, T (ed) *Fetal and neonatal pathology*. Springer, London
146. Smith D (1957) Preretinal and optic nerve sheath hemorrhage: pathological and experimental aspects in subarachnoid hemorrhage. *Trans Am Acad Ophthalmol Otolaryngol* 61:201–211
147. Squier W (2008) Shaken baby syndrome: the quest for evidence. *Dev Med Child Neurol* 50:10–14
148. Squier W, Lindberg E, Mack J, Darby S (2009) Demonstration of fluid channels in human dura and their relationship to age and intradural bleeding. *Childs Nerv Syst* 25:925–931
149. Squier W, Mack J (2009) The neuropathology of infant subdural haemorrhage. *Forensic Sci Int* 187:6–13
150. Squier W, Austin T, Anslow P, Weller RO (2011) Infant subcortical cystic leucomalacia: a distinct pathological entity resulting from impaired fluid handling. *Early Hum Dev*
151. Squier W, Scheimberg I, Smith C (2011) Spinal nerve root beta-APP staining in infants is not a reliable indicator of trauma. *Forensic Sci Int*
152. Stam AH, Luijckx GJ, Poll-The BT, Ginjaar IB, Frants RR, Haan J, Ferrari MD, Terwindt GM, van den Maagdenberg AM (2009) Early seizures and cerebral oedema after trivial head trauma associated with the CACNA1A S218L mutation. *J Neurol Neurosurg Psychiatry* 80:1125–1129
153. Starling SP, Patel S, Burke BL, Sirotiak AP, Stronks S, Rosquist P (2004) Analysis of perpetrator admissions to inflicted traumatic brain injury in children. *Arch Pediatr Adolesc Med* 158:454–458
154. Stein KM, Ruf K, Ganten MK, Mutter R (2006) Representation of cerebral bridging veins in infants by postmortem computed tomography. *Forensic Sci Int* 163:93–101
155. Stroobandt G, Evrard P, Latre C (1978) Pathogenesis of persistent subdural effusions in infants (author's transl). *Neuro Chirurgie* 24:47–51
156. Su CS, Lan MY, Chang YY, Lin WC, Liu KT (2009) Clinical features, neuroimaging and treatment of spontaneous intracranial hypotension and magnetic resonance imaging evidence of blind epidural blood patch. *Eur Neurol* 61:301–307
157. Takiguchi T, Yamaguchi S, Tezuka M, Furukawa N, Kitajima T (2006) Compression of the subarachnoid space by the engorged epidural venous plexus in pregnant women. *Anesthesiology* 105:848–851
158. Talvik I, Alexander RC, Talvik T (2008) Shaken baby syndrome and a baby's cry. *Acta Paediatr* 97:782–785
159. Tan M, Deveber G, Shroff M, Moharir M, Pontigon AM, Widjaja E, Kirton A (2011) Sagittal sinus compression is associated with neonatal cerebral sinovenous thrombosis. *Pediatrics* 128:e429–e435
160. Taylor GA (1992) Intracranial venous system in the newborn. *Radiology* 183
161. Thornton E, Ziebell JM, Leonard AV, Vink R (2010) Kinin receptor antagonists as potential neuroprotective agents in central nervous system injury. *Molecules* 15:6598–6618
162. Tubbs RS, Salter EG, Wellons JC, III, Blount JP, Oakes WJ (2007) The sphenoparietal sinus. *Neurosurgery* 60:ONS9–12
163. Valdeuza JM, von Munster T, Hoffman O, Schreiber S, Einhaupl KM (2000) Postural dependency of the cerebral venous outflow. *Lancet* 355:200–201
164. van der Kuip M, Hoogland PV, Groen RJ (1999) Human radicular veins: regulation of venous reflux in the absence of valves. *Anat Rec* 254:173–180
165. Vieira JP, Luis C, Monteiro JP, Temudo T, Campos MM, Quintas S, Nunes S (2010) Cerebral sinovenous thrombosis in children: clinical presentation and extension, localization and recanalization of thrombosis. *Eur J Paediatr Neurol* 14:80–85
166. Vignes JR, Dagain A, Guerin J, Liguoro D (2007) A hypothesis of cerebral venous system regulation based on a study of the junction between the cortical bridging veins and the superior sagittal sinus. Laboratory investigation. *J Neurosurg* 107:1205–1210
167. Vinchon M, Noizet O, Defoort-Dhellemmes S, Soto-Ares G, Dhellemmes P (2002) Infantile subdural hematomas due to traffic accidents. *Pediatr Neurosurg* 37:245–253
168. Vinchon M, Defoort-Dhellemmes S, Desurmont M, Dhellemmes P (2005) Accidental and nonaccidental head injuries in infants: a prospective study. *J Neurosurg* 102:380–384
169. Vinchon M, Delestret I, Defoort-Dhellemmes S, Desurmont M, Noule N (2010) Subdural hematoma in infants: can it occur spontaneously? Data from a prospective series and critical review of the literature. *Childs Nerv Syst* 26:1195–1205
170. Vinchon M, Desurmont M, Soto-Ares G, De Foort-Dhellemmes S (2010) Natural history of traumatic meningeal bleeding in infants: semiquantitative analysis of serial CT scans in corroborated cases. *Childs Nerv Syst* 26:755–762
171. Voigt GE, Lowenhielm CG, Ljung CB (1977) Rotational cerebral injuries near the superior margin of the brain. *Acta Neuropathol* 39:201–209
172. Volpe JJ (2009) Brain injury in premature infants: a complex amalgam of destructive and developmental disturbances. *Lancet Neurol* 8:110–124
173. Vowles GH, Scholtz CL, Cameron JM (1987) Diffuse axonal injury in early infancy. *J Clin Pathol* 40:185–189
174. Watts P, Obi E (2008) Retinal folds and retinoschisis in accidental and non-accidental head injury. *Eye (Lond)* 22:1514–1516
175. Welch K, Friedman V (1960) The cerebrospinal fluid valves. *Brain* 83:454–469
176. Wells RG, Sty JR (2003) Traumatic low attenuation subdural fluid collections in children younger than 3 years. *Arch Pediatr Adolesc Med* 157:1005–1010
177. Whitby EH, Griffiths PD, Rutter S, Smith MF, Sprigg A, Oha-dike P, Davies NP, Rigby AS, Paley MN (2004) Frequency and natural history of subdural haemorrhages in babies and relation to obstetric factors. *Lancet* 363:846–851

178. Wintermark M, Lepori D, Cotting J, Roulet E, van Melle G, Meuli R, Maeder P, Regli L, Verdun FR, Deonna T, Schnyder P, Gudinchet F (2004) Brain perfusion in children: evolution with age assessed by quantitative perfusion computed tomography. *Pediatrics* 113:1642–1652
179. Wolfe SQ, Bhatia S, Green B, Ragheb J (2007) Engorged epidural venous plexus and cervical myelopathy due to cerebrospinal fluid overdrainage: a rare complication of ventricular shunts. Case report. *J Neurosurg* 106:227–231
180. Wu YW, Miller SP, Chin K, Collins AE, Lomeli SC, Chuang NA, Barkovich AJ, Ferriero DM (2002) Multiple risk factors in neonatal sinovenous thrombosis. *Neurology* 59:438–440
181. Yamashima T, Friede RL (1984) Why do bridging veins rupture into the virtual subdural space? *J Neurol Neurosurg Psychiatry* 47:121–127
182. Yang JY, Chan AK, Callen DJ, Paes BA (2010) Neonatal cerebral sinovenous thrombosis: sifting the evidence for a diagnostic plan and treatment strategy. *Pediatrics* 126:e693–e700
183. Yu Y, Chen J, Si Z, Zhao G, Xu S, Wang G, Ding F, Luan L, Wu L, Pang Q (2010) The hemodynamic response of the cerebral bridging veins to changes in ICP. *Neuro Crit Care* 12:117–123
184. Zahl SM, Wester K (2008) Routine measurement of head circumference as a tool for detecting intracranial expansion in infants: what is the gain? A nationwide survey. *Pediatrics* 121:e416–e420
185. Zahl SM, Egge A, Helseth E, Wester K (2011) Benign external hydrocephalus: a review, with emphasis on management. *Neurosurg Rev*
186. Zenker W, Bankoul S, Braun JS (1994) Morphological indications for considerable diffuse reabsorption of cerebrospinal fluid in spinal meninges particularly in the areas of meningeal funnels. An electron microscopical study including tracing experiments in rats. *Anat Embryol (Berl)* 189:243–258
187. Zenker W, Kubik S (1996) Brain cooling in humans—anatomical considerations. *Anat Embryol (Berl)* 193:1–13
188. Zhang ET, Inman CB, Weller RO (1990) Interrelationships of the pia mater and the perivascular (Virchow-Robin) spaces in the human cerebrum. *J Anat* 170:111–123
189. Zimmerman RA, Bilaniuk LT, Bruce D, Schut L, Uzzell B, Goldberg HI (1979) Computed tomography of craniocerebral injury in the abused child. *Radiology* 130:687–690
190. Zuccaro G, Arganaraz R, Villasante F, Ceciliano A (2010) Neurosurgical vascular malformations in children under 1 year of age. *Childs Nerv Syst* 26:1381–1394

Exhibit II

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Using US Data to Estimate the Incidence of Serious Physical Abuse in Children

John M. Leventhal, Kimberly D. Martin and Julie R. Gaither

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Using US Data to Estimate the Incidence of Serious Physical Abuse in Children

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KEY WORDS

abuse, epidemiology-injury, injury severity

ABBREVIATIONS

CI—confidence interval

CPS—Child Protective Services

E-codes—external cause of injury codes

HCUP—Healthcare Costs and Utilization Project

ICD-9-CM—International Classification of Diseases, Ninth Revision, Clinical Modification

KID—Kids' Inpatient Database

NCANDS—National Child Abuse and Neglect Data System

NIS—National Incidence Study

TBI—traumatic brain injury

Dr Leventhal conceived of the project, reviewed the data, drafted the manuscript and prepared revisions of the manuscript, and had access to all of the data; Ms Gaither conducted the analyses and provided feedback on drafts of the manuscript, approved the submissions of the manuscript, and had access to all of the data; and Dr Martin assisted with the analyses, provided feedback on the drafts of the manuscript, and approved the submissions of the manuscript.

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WHAT'S KNOWN ON THIS SUBJECT: Limited data exist about the frequency and incidence of serious injuries due to physical abuse of children. Data from Child Protective Services, which are published yearly, do not have information about severity.

WHAT THIS STUDY ADDS: This is the first study to provide US estimates on the occurrence of serious injuries due to physical abuse. The incidence was highest in infants on Medicaid. Such data can be used to track changes due to prevention.

abstract

BACKGROUND: There are limited data on the epidemiology of serious injuries due to physical abuse of children.

METHODS: We used the 2006 Kids' Inpatient Database to estimate the incidence of hospitalizations due to serious physical abuse among children <18 years of age. Abuse was defined by using International Classification of Diseases, Ninth Revision, Clinical Modification codes for injuries (800–959) and for physical abuse (995.50, 995.54, 995.55, or 995.59), selected assault codes (E960-966, 968), or child battering (E967). We examined demographic characteristics, mean costs, and length of stay in 3 groups of hospitalized children: abusive injuries, nonabusive injuries, and all other reasons for hospitalization. Incidence was calculated using the weighted number of cases of physical abuse and the number of children at risk based on 2006 intercensal data.

RESULTS: The weighted number of cases due to abuse was 4569; the incidence was 6.2 (95% confidence interval [CI]: 5.5–6.9) per 100 000 children <18 years of age. The incidence was highest in children <1 year of age (58.2 per 100 000; 95% CI: 51.0–65.3) and even higher in infants covered by Medicaid (133.1 per 100 000; 95% CI: 115.2–151.0 [or 1 in 752 infants]). Overall, there were 300 children who died in the hospital due to physical abuse.

CONCLUSIONS: This is the first study to provide national US data on the occurrence of serious injuries due to physical abuse in hospitalized children. Data from the 2006 Kids' Inpatient Database on hospitalizations due to serious physical abuse can be used to track trends over time and the effects of prevention programs on serious physical abuse. *Pediatrics* 2012;129:1–7

In the United States, 2 approaches have been used to ascertain the national occurrence of child maltreatment. The first has counted cases of suspected maltreatment reported to state or local Child Protective Services (CPS). Since 1990, state-level and national data have been prepared by the National Child Abuse and Neglect Data System (NCANDS).¹ Data include the number of reported and substantiated cases, the types of maltreatment (eg, physical abuse, sexual abuse, or neglect), and the number of deaths due to maltreatment.

The second approach has used the National Incidence Studies (NISs), which are congressionally mandated studies aimed at providing national estimates of the incidence of child maltreatment. Four studies have been conducted (1979–1980, 1986–1987, 1993–1994, and 2005–2006).² In each, professionals in selected counties throughout the country were trained to provide case surveillance over two 3-month periods during the year. Counted cases included those reported to CPS and those identified by professionals but not reported. In each NIS, the number of cases of maltreatment was greater than the number from the comparable year of NCANDS.

Although cases of physical abuse are counted in both approaches to collecting national data, neither approach can specifically identify cases of serious physical abuse, such as children who are hospitalized with head injuries, fractures, or burns due to abuse. Knowing the number of children with serious injuries from physical abuse would provide an important estimate of the burden of the problem, and the number of such injuries could be tracked over time to determine whether prevention programs are able to reduce these serious injuries.

Therefore, to estimate the number of children in the United States with serious injuries due to physical abuse, we

used a national dataset of hospitalized children. We used diagnostic codes to identify children with injuries and child abuse or assault codes to identify those injuries due to physical abuse. Demographic characteristics of the abused children were compared with those of children hospitalized with injuries not due to abuse and with all other causes of hospitalization. In addition, we sought to determine the estimated costs of hospitalizations and length of stay due to physical abuse and the estimated number of in-hospital deaths due to physical abuse.

METHODS

We used the 2006 Kids' Inpatient Database (KID), which was prepared by the Healthcare Cost and Utilization Project (HCUP).³ The KID is a weighted US sample of discharged patients from all nonrehabilitation hospitals in HCUP's State Inpatient Databases. Systematic random sampling is used to select 10% of uncomplicated in-hospital births and 80% of complicated in-hospital births and other pediatric cases. To obtain national estimates, weighting takes into account 6 hospital characteristics (ownership/control, bed size, teaching status, rural/urban location, US region, and freestanding children's hospitals). The sampling frame for the 2006 KID includes 3739 hospitals from 38 states, which represent 88.8% of the US population in 2006.

For each hospital discharge in the KID, there is information about demographics, payment, hospital type, diagnoses, and external cause of injury (E-codes) based on the *International Classification of Diseases, Ninth Revision, Clinical Modification* (ICD-9-CM) and disposition (eg, discharged or died). Because most CPS agencies in the country accept reports on children up to the age of 18, we defined a child as a patient who was <18 years of age at the time of discharge from the hospital.

We excluded cases where age was missing and all in-hospital births. Physical abuse was defined in 1 of the following 4 ways:

1. An ICD-9-CM code for an injury (800–959) and either an ICD-9-CM code for child abuse (995.50, 0.54, 0.55, or 0.59) or an E-code for the identified perpetrator of child abuse (E967). We excluded injuries coded as late effects (905–909) so as not to include children who were being hospitalized for the nonacute consequences of an acute injury (eg, late effect of a burn).
2. An ICD-9-CM code for an injury and an E-code for assault (E960–966, 968). Because these E-codes for assault are not specific for child abuse and can be used when a child is hurt in a fight with peers or older persons or the child is shot with a gun on the street, for example, we used the following stepwise approach to derive the eligible cases identified in this category: (1) we excluded injuries that were coded as occurring in the following places: farms, recreation or sports sites, industrial places, public buildings, streets, or residential facilities (E849.1–0.6); (2) we excluded injuries caused by air guns (E968.6) or firearms (E965); (3) we included injuries that were coded as occurring in the home (E849.0); and (4) for children >8 years of age, we excluded cases that did not provide an E-code for the location. This last restriction was used to avoid including children where no information was coded about where the injury had occurred and thus the injury may have been due to an assault, as opposed to abuse from a caretaker.
3. An ICD-9-CM code for child abuse or the E-code 967 (identified perpetrator of abuse) and at least 1 ICD-9-CM code that was consistent with

abuse (eg, retinal hemorrhages or anoxic brain damage). These children had no specific diagnostic code of an injury (800–959) but were included because of the specific child abuse code.

4. The ICD-9-CM code 995.55, which is the code for “shaken infant syndrome,” and no specific injury code.

We grouped the children with specific injury codes into the following types of injuries (Table 1): fractures, traumatic brain injuries (TBIs) (excluding isolated skull fractures), abdominal injuries, burns, skin injuries/open wounds, and other types of injuries.*

Data on length of stay, charges for the hospitalization, and whether the child died during the hospitalization are provided in the KID. Demographic data available included the child's age, gender, race, and source of payment for the hospitalization (grouped as Medicaid, private, self, and other). To calculate costs, we followed the HCUP recommendations⁵ and used the hospital-specific cost-to-charge ratio, which was available in 78% of the hospitals, and the weighted group average when the hospital specific cost-to-charge ratio was not available.

We used the weightings provided in the KID to calculate the number of physically abused children in the United States. To calculate the incidence of physically abused children in 2006, we used this number as the numerator. The denominator was based on the number of children nationally in the age group in 2006; this number was

obtained from the 2006 intercensal estimates.⁶ Confidence intervals (CIs) were calculated by using the Taylor Series in SAS Version 9.2 (SAS Institute, Inc, Cary, NC). We used an identical approach to determine the number of children who died during the hospitalization in the abused group and the in-hospital mortality due to abuse.

We compared the cases in the abused group with 2 groups: (1) cases with injury codes but not classified as abuse (called “nonabusive injures”) and (2) all other acute care hospitalizations in the dataset. We used χ^2 or analysis of variance to compare the demographic characteristics, the percent of deaths, costs, and length of stay.

Because of the association of physical abuse and poverty, the incidence of serious injuries due to abuse was calculated in children who were on Medicaid and compared with the incidence in children with other types of health insurance. To obtain estimates of children on Medicaid in 2006, we used the Health Insurance Historical Tables prepared by the US Census Bureau.⁷

The study was considered exempt from approval by the Yale Medical School institutional review board.

RESULTS

In 2006, the weighted number of children hospitalized with serious injuries due to physical abuse was 4569. Table 2 shows the demographic characteristics in the abuse group compared with children with nonabusive injuries and other causes of hospitalization. There

were statistically significant differences ($P < .0001$) among the 3 groups for gender, race/ethnicity, and medical insurance. The most striking difference between the cases of abuse and the other 2 groups concerned the type of medical insurance: 71.6% of abuse cases were on Medicaid compared with 36.6% of cases with nonabusive injuries and 48.5% of cases with other hospitalization causes.

The mean length of stay for the children in the abuse group (7.4 days; 95% CI: 6.9–8.0) was significantly longer ($P < .0001$) than for children with nonabusive injuries (3.9 days; 95% CI: 3.8–4.1) or with other hospitalizations (4.5 days; 95% CI: 4.4–4.6). Also, mean hospital costs were statistically significantly higher ($P < .0001$) in the abuse group (\$16 058; 95% CI: \$14 644–\$17 473) versus the 2 other groups (\$9550 [95% CI: \$9084–\$10 017] for the nonabusive injury group and \$7964 [95% CI: \$7516–\$8411] for the other hospitalization group). The national costs for hospitalization of abused children were ~\$73.8 million.

The yearly incidence of hospitalization of children with serious physical abuse was 6.2 (95% CI: 5.5–6.9) per 100 000 children <18 years of age. Figure 1 shows the incidence of cases for each year of life, and Table 3 shows the results for 3 age groups. The incidence was highest in the first year of life: 58.2 (95% CI: 51.0–65.3).

When the incidence was calculated for the estimated number of children on Medicaid in 2006, the rate was 16.3 per 100 000 children <18 years of age (95% CI: 14.2–18.4) compared with 2.4 (95% CI: 2.1–2.7) for all other children not covered by Medicaid (Table 3). In each age group, the incidence in children covered by Medicaid was about 6 times greater compared with all other children not covered by Medicaid.

The weighted number of children who died during the hospitalization from

TABLE 1 Types of Injuries Based on ICD-9-CM Codes

Injury	ICD-9-CM Codes
Fracture	800–829
TBI	800.1–800.4; 800.6–800.9; 801.1–801.4; 801.6–801.8; 803.1–803.4; 803.6–803.9; 804.1–804.4; 804.6–804.9; 850–854
Abdominal	863–869; 902
Burn	940–949
Skin/open wound	870–897; 910–924
Other*	830–839; 840–848; 860–882; 900–901; 903–904; 925–929; 930–939; 950–957; 959

* For example, 881.21 = contusion of lung without mention of open wound into thorax.

TABLE 2 Demographic Characteristics by Percentage in Each of the 3 Hospitalized Groups: Abuse, Nonabusive Injuries, and Other Causes of Hospitalization

	Abuse (n = 4569)	Nonabusive Injuries (n = 189 414)	Other Causes of Hospitalization (n = 2 291 632)	P
Gender				<.0001
Male	58.8	65.0	49.8	
Race/ethnicity				<.0001
White	45.3	56.3	49.8	
African American	25.5	15.6	17.1	
Hispanic	19.6	19.8	24.3	
Other	9.5	8.3	8.8	
Medical insurance				<.0001
Medicaid	71.6	36.6	48.5	
Private/HMO	18.5	51.8	43.1	
Self-pay	4.5	6.3	3.9	
Other	5.4	5.3	4.5	

n = weighted number in group. Data were missing for gender, 16 353; race/ethnicity, 404 178; and medical insurance, 2299. HMO, health maintenance organization.

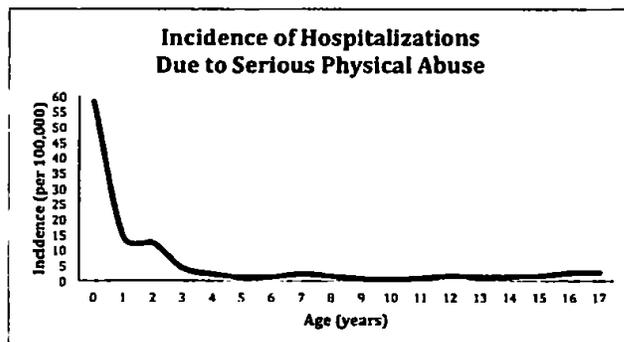


FIGURE 1 Incidence of hospitalizations due to serious physical abuse.

injuries due to physical abuse was 300. This number represents 6.6% of the abused group compared with 0.9% of the group with nonabusive injuries and 0.5% of the group with other causes of hospitalization ($P < .0001$). The overall in-hospital mortality rate due to abuse was 0.41 (95% CI: 0.32–0.49) per 100 000 children <18 years of age. As shown in Fig 2, the in-hospital mortality rate due to abuse for each year of life shows a similar pattern to the incidence of hospitalizations due to physical abuse. The incidence was 4.09 (95% CI: 3.11–5.07) deaths per 100 000 children <1 year old, was about 1.0 per 100 000 children for each of the second and third years of life, and was lowest in school-age children and adolescents.

DISCUSSION

In this study, the first to use the KID to estimate the number of children with serious abusive injuries resulting in hospitalizations, we found 4569 children nationally in 2006; 300 of these children (6.6%) died because of their abuse. Not surprisingly, the incidence was highest for children during the first year of life (58.2 per 100 000 children) and was substantially higher for children covered by Medicaid compared with all other children. The estimated national cost for the hospitalizations due to serious abusive injuries was \$73.8 million.

We included in our definition of “serious abuse” any child who was admitted

to the hospital with an injury that was coded as abuse (or assault in younger children). Such children included a 3-month-old with multiple bruises due to abuse (who was admitted for safety and further evaluation) and a 3-month-old with life-threatening abusive head trauma. Our definition did not include children who were admitted with injuries suspicious for abuse but eventually were diagnosed as having nonabusive injuries. These children, regardless of the severity of the injuries, would not receive an ICD-9-CM code for abuse or assault and, therefore, would not be counted as having an injury caused by abuse.

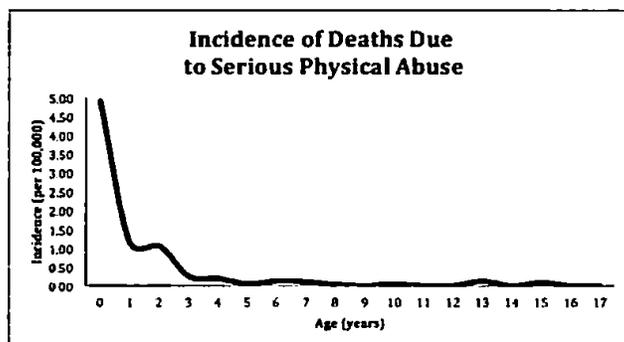
Previous estimates of the number of physically abused children in the United States have been based on data from the yearly NCANDS reports or the periodic NISs, but no information is provided about the seriousness of the injuries or the number of hospitalizations due to abusive injuries. In 2006, based on the NCANDS data, there were 142 000 cases of substantiated physical abuse.¹ In the latest NIS, which collected data in 2005–2006 from CPS and mandated reporters in selected regions of the country, there were 323 000 cases of physical abuse with at least a moderate injury.² Moderate injuries were defined as physical injuries, such as bruises, that lasted ≥ 48 hours.

These 2 approaches capture many more children who have been physically abused compared with those children hospitalized because of abuse and identified in the 2006 KID. By using the higher estimate of physically abused cases from NIS-4 ($N = 323 000$), the results from the KID show that about 1.4% of physically abused children were hospitalized in 2006.

One other national US study of maltreated children used the HCUP 2005 Nationwide Inpatient Sample, which includes a 20% sample of hospitalizations

TABLE 3 Incidence of Hospitalization of Children With Serious Injuries Due to Abuse Incidence per 100 000 Children and 95% CIs

Age Group, y	Overall	Children Covered by Medicaid	Children Not Covered by Medicaid
0–18	6.2 (5.5–6.9)	16.3 (14.2–18.4)	2.4 (2.1–2.7)
0–5	28.4 (24.9–32.0)	64.2 (55.3–73.2)	10.7 (9.2–12.2)
0–1	58.2 (51.0–65.3)	133.1 (115.2–151.0)	21.8 (18.2–25.5)

**FIGURE 2**
Incidence of deaths due to serious physical abuse.

from acute care community hospitals. In this study reported by the Agency for Healthcare Research and Quality, there were 6700 hospitalized cases associated with maltreatment.⁸ When cases of neglect and sexual abuse are removed from the total, there were 5290 hospitalizations due to physical abuse. Not surprisingly, this number is similar to our result using the 2006 KID, because the sample for the KID is drawn from the same population of community hospitals as the Nationwide Inpatient Sample. The KID provides more precise incidence estimates since it includes an 80% sample of pediatric discharges, while the Nationwide Inpatient Sample includes a 20% sample. The estimated costs of about \$74 million due to the hospitalizations for abuse are similar to the results using the 2005 Nationwide Inpatient Sample, Statistical Brief,⁸ but markedly different from the often quoted figure from Prevent Child Abuse America of \$6.6 billion (in 2007 dollars).⁹ The latter figure was based on the NIS-3 estimate of 565 000 maltreated children and the

assumption that 50% of these children were hospitalized. As shown in our study, the percentage of physically abused children who are hospitalized in 1 year is much closer to 1.5% than to 50%.

We found the incidence of hospitalization due to serious abuse in the first year of life to be 58.2 per 100 000 children in this age group. This rate is almost twice the rate for abusive head trauma of about 30 per 100 000 infants.¹⁰ Interventions aimed at the prevention of abusive head trauma might consider broadening the message to include other types of abusive injuries in children. This broadening of the message would mean that the emphasis of the message changes from "not shaking" to "not hurting" an infant.

Two other studies have examined the incidence of serious abusive injuries. Gessner et al¹¹ linked birth certificate, CPS, and hospital data over a 7-year period (1994–2000) in Alaska and found that the incidence of physical abuse in infants leading to hospitalization and/or death was 100 per 100 000

births. These authors note that the rates of physical abuse in Alaska were higher than those in other states. In a study using a different methodology, Sibert et al¹² determined the incidence of severe physical abuse in Wales by using a prospective case-surveillance system to collect data from April 1996 to March 1998 about serious physical abuse in children from birth to age 14. In this study, the incidence in the first year of life was 54 per 100 000, which is remarkably similar to our rate of 58 per 100 000.

In our study, children covered by Medicaid had rates of serious abuse about 6 times higher than those not on Medicaid. In the first year of life, the incidence for Medicaid-covered children was 133 per 100 000 compared with 22 for all other children. Thus, 1 in 752 children who are covered by Medicaid were hospitalized due to serious physical abuse in the first year of life. This very high rate speaks to the importance of poverty as a major risk factor for serious abuse and for the need to prevent these serious injuries.¹³ We also found less dramatic differences by gender and race, although the number of missing data for race was substantial.

It is important to note that the incidence of serious abuse in the first year of life is higher than the rate of sudden infant death syndrome, which is about 50 per 100 000 births.¹⁴ The national "Back to Sleep" campaign has successfully reduced the incidence of sudden infant death syndrome from about 100 per 100 000 to its current rate. Perhaps a national campaign to reduce serious abusive injuries would have similar success at decreasing abusive injuries in infants.

Our study has at least 4 limitations. First, the KID only includes hospitalized children, so children with serious abusive injuries who die before hospitalization or who are never hospitalized are not counted. Thus, our estimate of

abused children with serious injuries is clearly an underestimate of the problem. In Keenan et al's prospective study of traumatic brain injuries in children <2 years of age,¹⁵ 8.8% of children diagnosed with abusive head injuries died prior to hospitalization and the diagnosis was made by the medical examiner (H.T. Kennan, MDCM, MPH personal communication, 2010). A larger component of the underestimation of serious injuries due to abuse would occur when abused children with serious injuries are not hospitalized. While it is unlikely that children identified with abusive head or abdominal injuries would not be hospitalized, some children with abusive fractures are managed without hospitalization. For example, in a study by Leventhal and colleagues of fractures in 672 children <3 years of age who were evaluated at Yale-New Haven Hospital, 15% of children <12 months of age and 29% of those aged 12–23 months with fractures due to abuse were not hospitalized.¹⁶

Second, the KID counts hospitalizations during a specific year, not children. A few children may have been hospitalized twice in the same year for acute injuries due to serious physical abuse.

To help ensure that we did not count the same child twice, we excluded hospitalizations that included codes for the late effects or complications of an injury.

Third, since we relied on ICD-9-CM codes to diagnose abuse, we have no information on the accuracy of the diagnosis of abuse made by the physicians who cared for the child. A previous study has shown variability in the likelihood of abuse when child abuse physicians review the same case.¹⁷ There also are data to suggest that physicians overdiagnose abuse in minority children and underdiagnose in white children.¹⁸

Fourth, there have been concerns that child abuse codes may not be used accurately when physicians make the diagnosis of abuse. Unfortunately, there is limited research to address this question. For example, Winn et al¹⁹ showed that the use of the child abuse and assault codes may underestimate injuries due to abuse. To the extent that this concern is true, our results may underestimate the number of hospitalizations due to physical abuse. On the other hand, Ellingson et al¹⁰ showed that the use of these codes in the 1997, 2000, and 2003 KID

provided estimates of the incidence of abusive head injuries during the first year of life that were comparable to those from prospective studies. Thus, the estimates of hospitalized children due to abuse are likely not substantially underestimated due to the failure to use appropriate coding.

Data from the KID can be used in future studies to track changes in the incidence of serious injuries due to physical abuse over time. Such studies might examine the effects of a large child abuse prevention program or the effects of an economic downturn.²⁰

CONCLUSIONS

In 2006, there were ~4600 children hospitalized with serious injuries due to physical abuse. The incidence was highest in children <1 year of age (58.2 per 100 000 infants) and about 6 times higher in children on Medicaid compared with all other children not on Medicaid. The overall cost of hospitalization was \$73.8 million. Data from the KID on hospitalizations due to serious abuse should be useful in examining trends over time and in studying the effects of large-scale prevention programs.

REFERENCES

1. US Department of Health and Human Services, Administration on Children, Youth and Families. *Child Maltreatment 2006*. Washington, DC: US Government Printing Office; 2008
2. Sedlak AJ, Mettenberg J, Basena M, et al. *Fourth National Incidence Study of Child Abuse and Neglect (NIS-4): Report to Congress*. Washington, DC: US Department of Health and Human Services, Administration for Children and Families; 2010
3. Agency for Healthcare Research and Quality. Overview of the Kids' Inpatient Database (KID). Available at: www.hcup-us.ahrq.gov/kidoverview.jsp. Accessed October 1, 2008
4. National Center for Health Statistics. *International Classification of Diseases, Ninth Revision, Clinical Modification*. Hyattsville, MD: National Center for Health Statistics; 1999
5. Cost-to Charge Ratio Files. 2006 Kids' User Guide. Available at: www.hcup-us.ahrq.gov/db/state/costtocharge.jsp#how. Accessed December 14, 2010
6. US Census Bureau. Population estimates. Available at: www.census.gov/popest/states/files/SC-EST2008-AGESEX_RES.csv. Accessed December 8, 2008
7. U.S. Census Bureau. Health Insurance Coverage Status and Type of Coverage—Children Under 18 by Age: 1999 to 2009. Available at: www.census.gov/hhes/www/hlthins/data/historical/index.html. Accessed March 2, 2011
8. Russo CA, Hambrick MM, Owens PL. Hospital stays related to child maltreatment. *Statistical Brief #49*, Agency for Health Care Research and Quality; 2008. Available at: www.hcup-us.ahrq.gov/reports/statbriefs/sb49.jsp. Accessed September 7, 2010
9. Wang C, Holton J. Total Estimated Cost of Child Abuse and Neglect in the United States. Chicago, IL: Prevent Child Abuse America; 2007. Available at: http://member.preventchildabuse.org/site/DocServer/cost_analysis.pdf?docID=144. Accessed September 8, 2010
10. Ellingson KD, Leventhal JM, Weiss HB. Using hospital discharge data to track inflicted traumatic brain injury. *Am J Prev Med*. 2008;34(suppl 4):S157–S162
11. Gessner BD, Moore M, Hamilton B, Muth PT. The incidence of infant physical abuse in Alaska. *Child Abuse Negl*. 2004;28(1):9–23

12. Sibert JR, Payne EH, Kemp AM, et al. The incidence of severe physical child abuse in Wales. *Child Abuse Negl*. 2002;26(3):267-276
13. Berger LM. Income, family characteristics and family violence toward children. *Child Abuse Negl*. 2005;29(2):107-133
14. Hauck FR, Tanabe KO. International trends in sudden infant death syndrome: stabilization of rates requires further action. *Pediatrics*. 2008;122(3):660-666
15. Keenan HT, Runyan DK, Marshall SW, Nocera MA, Merten DF, Sinal SH. A population-based study of inflicted traumatic brain injury in young children. *JAMA*. 2003;290(5):621-626
16. Leventhal JM, Larson IA, Abdoo D, et al. Are abusive fractures in young children becoming less common? Changes over 24 years. *Child Abuse Negl*. 2007;31(3):311-322
17. Lindberg DM, Lindsell CJ, Shapiro RA. Variability in expert assessments of child physical abuse likelihood. *Pediatrics*. 2008;121(4). Available at: www.pediatrics.org/cgi/content/full/121/4/e945
18. Lane WG, Rubin DM, Monteith R, Christian CW. Racial differences in the evaluation of pediatric fractures for physical abuse. *JAMA*. 2002;288(13):1603-1609
19. Winn DG, Agran PF, Anderson CL. Sensitivity of hospitals' E-coded data in identifying causes of children's violence-related injuries. *Public Health Rep*. 1995;110(3):277-281
20. Berger RP, Fromkin JB, Stutz H, et al. Abusive head trauma during a time of increased unemployment: a multicenter analysis. *Pediatrics*. 2011;128(4):637-643

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