73278-1

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SONYA KRASKI
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SUPERIOR COURT OF WASHINGTON
FOR SNOHOMISH COUNTY

THE STATE OF WASHINGTON, Plaintiff, v. MICHAEL J. MORRIS, Defendant.	73278-1 No. 09-1-01071-9 ORDER TRANSFERRING MOTION FOR RELIEF FROM JUDGMENT (CLERK'S ACTION REQUIRED)

This matter came before the court pursuant to CrR 7.8(c)(2), for initial consideration of the defendant's Motion for Relief from Judgment. The court has considered the documents listed below. Being fully advised, the court hereby concludes and orders as follows:

I. CONCLUSIONS OF LAW

1. The defendant's motion is not time barred by RCW 10.73.090.

2. The defendant has not made a substantial showing that the defendant is entitled to relief.

3. Resolution of the defendant's motion will not require a factual hearing.

II. ORDER

1. Pursuant to CrR 7.8(c)(2), the defendant's Motion for Relief from Judgment is transferred to the Court of Appeals for consideration as a personal restraint petition.

2. The clerk of this court shall transmit copies of the following to the Court of Appeals:

a. This order;
b. The Defendant's Memorandum in support of CrR 7.8 Motion for Relief from Judgment or Order (sub No.219).
c. CrR 7.8 Motion for Relief from Judgment or Order (sub no. 221)
d. The State's Motion to Transfer Motion For Relief From Judgment (sub No. 226).
e. The Defendant's Response to State's Motion to Transfer Defendant's CrR 7.8 Motion to the Court of Appeals(sub No. 229).
f. The State's Reply to Response to State's Motion to Transfer Motion for New Trial Pursuant to CrR 7.8 (sub non. 230)
g. Motion and Declaration for Order Authorizing Expenditure of Public Funds (sub no. 22)
h. Order Authorizing Expenditure of Public Funds (sub no. 23)
i. Motion and Declaration for Order Authorizing Expenditure of Public Funds (sub no. 48)
j. Order Authorizing Expenditure of Public Funds (sub no. 49)
k. Response to State's Motion to Transfer Defendant's CrR 7.8 Motion to the Court of Appeals – Corrected (sub 231)

Entered this 18th day of December, 2014.

JUDGE Mary Beth Dingledy

Presented by:

KATHLEEN WEBBER 16040
Deputy Prosecuting Attorney

Copy Received:

M. FERNANDA TORRES 34587
Attorney for the Defendant

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CL17072892

SUPERIOR COURT OF WASHINGTON FOR SNOHOMISH COUNTY

STATE OF WASHINGTON, Plaintiff, vs. MICHAEL J. MORRIS, Defendant.	No. 09-1-01071-9 CrR 7.8 MOTION FOR RELIEF FROM JUDGMENT OR ORDER (Evidentiary Hearing and Oral Argument Requested) 73278-1

MOTION

Michael J. Morris, by and through his attorney, M. Fernanda Torres of the Innocence Project Northwest, moves this Court for an order vacating his conviction and granting a new trial based on Article I, Sections 3 and 22 of the Washington State Constitution, and the Sixth and Fourteenth Amendments of the United States Constitution.

This motion is brought pursuant to CrR 7.8(b) and RCW 10.73.100 and is based on the records and files therein, the following memorandum and appendices, as well as evidence and argument that may be presented at a hearing on this matter.

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221

Dated this 18th day of July 2014.

Respectfully submitted,

INNOCENCE PROJECT NORTHWEST CLINC

M. Fernanda Torres, WSBA No. 34587
Attorney for Michael Morris

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CL16692510

SUPERIOR COURT OF WASHINGTON FOR SNOHOMISH COUNTY

STATE OF WASHINGTON, Plaintiff, vs. MICHAEL J. MORRIS, Defendant.	No. 09-1-01071-9 MEMORANDUM IN SUPPORT OF CrR 7.8 MOTION FOR RELIEF FROM JUDGMENT OR ORDER

I. INTRODUCTION

Michael Morris' conviction rests upon flawed science. The State alleged that within a 10 to 15 minute time frame, Mr. Morris shook his infant child so violently that she suffered whiplash injuries typical of major trauma, such as a car accident. The State presented this conclusion, which is unsupported by evidence-based studies and refuted by epidemiology data, through its expert witness. Mr. Morris was denied his right to effective assistance of counsel because his trial attorney failed to competently challenge the State expert's opinion on causation, which was subject to exclusion or limitation under ER 702, and failed to expose the flaws in the State's case. Mr. Morris' due process rights were violated by the State's

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219

presentation of evidence it knew, or should have known was false and misleading. Mr. Morris respectfully requests that this Court grant him a new trial, or an evidentiary hearing.

II. JURISDICTION

Under CrR 7.8(b)(5), a court may grant relief from judgment for "any other reason [other than those specified in (b)(1)-(4)] justifying relief from the operation of the judgment." CrR 7.8 motion is timely. RCW 10.73.090 provides that a petition or motion for collateral attack on a judgment and sentence in a criminal case is timely if it is filed within one year after the judgment becomes final. The judgment in this case became final on July 19, 2013, the date the appellate court issued its mandate disposing of a timely direct appeal from the conviction. RCW 10.73.090(3)(b). *See* Mandate Letter (App. A).

This Court is the proper venue for this motion. Mr. Morris has presented substantial evidence to establish that his attorney was deficient and that his due process rights were violated, and that there is a reasonable probability the outcome of the trial would have been different but for counsel's errors, entitling him to relief. A trial court is not required to transfer the motion to the Court of Appeals for consideration as a personal restraint petition (PRP) if the court determines:

> [T]hat the motion is not barred by RCW 10.73.090 and *either*
>
> (i) the defendant has made a substantial showing that he or she is entitled to relief or
> (ii) resolution of the motion will require a factual hearing.

CrR 7.8(c)(2)(emphasis added).

Moreover, transfer to the Court of Appeals would unnecessarily waste both time and judicial resources. Since this motion contains a mixed question of law and fact, it is likely the appellate court will remand to the superior court for a reference hearing pursuant to RAP 16.11.

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To the extent that resolution of the motion requires settling a factual dispute, an evidentiary hearing will be required, in which case transfer under CrR 7.8(c)(2) is also improper.

III. STATEMENT OF THE CASE[1]

A. Medical Event and Opinion Leading to the Charges

On 5/29/09 at approximately 8:00 p.m., Michael Morris was feeding his six-week old daughter "A.M." a bottle when she began choking and vomiting. She arrived at Stevens Hospital at 8:20 p.m. and was very lethargic, with shallow and erratic breathing. Her fontanel was full, there was a small bruise under her chin, and a head CT scan showed a small subdural bleed. She was intubated and transferred to Harborview Medical Center ("HMC"). *See generally* 6/01/11 RP 17-23. (Dr. Borromeo testimony).

A.M. was stable at HMC and on May 31 she was seen by Dr. Kenneth Feldman, a child abuse specialist from Seattle Children's Hospital (SCH). 6/2/11 RP 131. Follow-up CT scans at HMC confirmed the small subdural bleed and possibly brain damage.[2] 6/2/11 RP 141-49. There were no skull fractures or scalp swelling, and the skeletal imaging was normal, but profuse bilateral retinal hemorrhages were noted. 6/2/11 RP 152; RP 135-36.

A.M. was transferred to SCH late in the day on May 31. RP 138. An MRI, done on June 1, confirmed the subdural bleeding. 6/1/11 RP 150. The MRI also showed hypoxic-ischemic changes in the brain, or damage from a lack of oxygen. 6/1/11 RP 148-49; 6/7/11 RP 403. Dr. Herlihy, an ophthalmologist, examined A.M. on June 1, and noted profuse bilateral retinal

[1] The trial was transcribed by two different court reporters and the volumes and not numbered sequentially. For clarity, references to the report of proceedings will be preceded by the date of the proceeding. A full copy of all the transcripts can be provided to the Court.

[2] The radiology reports do not note brain damage (typically detected through loss of gray/white differentiation and swelling), but Dr. Feldman and a defense expert in radiology, Dr. Barnes, did note possible loss of gray/white matter differentiation and swelling in the CT scans. 6/1/11 RP 138; 148 (Dr. Feldman); 6/7/11 RP 399-402 (Dr. Barnes).

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bleeding. RP 6/6/11 RP 295. At a follow-up appointment six weeks later, Dr. Herlihy noted a possible schisis cavity, or a split in the layers of the retina, in A.M.'s left eye.[3] RP 6/6/11 320.

Mr. Morris voluntarily met with the police on June 2, 2009. He gave a written statement, stipulated to by the parties under CrR 3.5. 2/2511 RP 2. In his statement and to the police, Mr. Morris explained that while he was feeding A.M., she spit up and stopped breathing. 6/2/11 RP 5. He shook her to get her to respond, and then shook her again harder for five seconds. 6/2/11 RP 41. When she was still not breathing, he took her in his arms and ran across the street as fast as he could. *Id.* at 39. He thought she might have been injured when he ran across the street with her head unsupported, or when he shook her to resuscitate her. *Id.*

Mr. Morris was arrested on June 9, 2009. *Id.* at 44-45. He was charged with assault of a child in the first degree and went to trial in May of 2011.

B. The State's Case

At trial, Dr. Feldman provided the official diagnosis of abusive head trauma. Other witnesses were: Dr. Borromeo (ER physician), Dr. Herlihy (ophthalmologist), Brittany Morris (A.M.'s mother), Cherlyn and Mike Orkiz (Morris family neighbors), Kim Krausz, Bianca Quesada and Jane Powers, (social workers), and Brier Police Department Officers Patrick Lee, Patrick Murphy and Kevin Kilpatrick. Dr. Feldman's and Dr. Herlihy's testimony is described here. Other pertinent facts are addressed in further detail in the relevant argument sections below.

C. Dr. Feldman's Diagnosis of Abuse

During the first day of his testimony, Dr. Feldman explained the medical findings, showing the jury pictures of the imaging done on A.M.'s head and body, and of her eyes and

[3] This is also referred to as retinoschisis.

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bruise under her chin. 6/2/11 RP X-X. He walked the jury through the subdural bleeding and explained the hypoxic-ischemic changes found in the MRI. RP X.

During the second day of testimony, Dr. Feldman testified as to causation and provided his official diagnosis. He explained that when trying to figure out the cause of A.M.'s injuries, he looked at the constellation of injuries—subdural and retinal hemorrhaging—together.[4] 6/3/11 RP 3. He explained that he focused on what causes both subdural and retinal hemorrhaging and excluded non-traumatic causes based on A.M.'s coagulation and metabolic testing. 6/3/11 RP 3-4. As for traumatic forces, Dr. Feldman testified that he looked for a force that had a whiplash component to it because "there's good studies that indicate that both the retinal bleeding and the subdural bleeding and the brain injury she sustained are typically the result of whiplash forces on the head, this sort of thing you might get in a motor vehicle whiplash event." 6/3/11 RP 5-6. He explained that the event here would be an inertial (rotational) event of the head moving in a rapid arc, versus a linear impact. *Id.* at 6.

According to Dr. Feldman, shaking "is a force that causes whiplashes to occur, [a]nd if, in the course of that, the baby's head struck against something" it results in very high levels of deceleration. *Id.* at 7. He testified one would not expect to see a skull fracture or scalp bruising if impact was against a soft surface. 6/3/11 RP 8. Regarding the level of force, Dr. Feldman testified that "it takes dramatic forces, very high levels of acceleration or deceleration to cause these injuries" and one would have to "exert a lot of force on a child that any reasonable person watching what was happening would think, oh, my God, what's happening to that baby?" 6/3/11 RP 9.

[4] The bruise was yellowing and was older. 6/2/11 RP 132; 6/3/11 RP 78-79. It was not attributed to abuse by Mr. Morris.

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Regarding the retinal hemorrhages, Dr. Feldman testified that the same whiplash forces that caused the subdural hemorrhages can also cause retinal hemorrhages. *Id.* at 10. He said that A.M. had profuse retinal hemorrhages, which could not be caused by a minor fall, and that the "evidence-based" literature shows that retinal hemorrhages and retinoschisis (splitting of the retina) are highly specific for abuse. *Id.* at 12-13.

Dr. Feldman testified that after going through his "differential diagnosis" and excluding different possibilities, he concluded A.M. suffered "abusive head trauma." *Id.* at 13. He said he did not know the specific mechanism that caused the injury, because he was not in the room when it happened. *Id.* at 16-17. Regarding the timing of the injuries, Dr. Feldman testified that it is very consistent that almost all of the children who suffer abusive head trauma have immediate symptoms of the injury. *Id.* at 18. He described a lucid interval as a period of getting better and maintained that a lucid interval was not really possible in this case—"it's not really the scenario we have here." *Id.*

D. Dr. Herlihy

Dr. Herlihy testified she ruled out underlying medical conditions and was left with severe head trauma. 6/7/11 RP 312. According to Dr. Herlihy, "there must have been some shearing injury, acceleration/deceleration force, something that would cause a shearing type of injury to tear blood vessels." *Id.* She did not see any schisis cavities during her initial examination, but in her subsequent exam saw some pigmentary changes in the macula suggesting there had been a schisis cavity there. *Id.* at 320. She conjectured that if the schisis cavity was there during before, it could have been obscured because there was so much blood. *Id.* at 321.

E. The Defense Case

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The defense introduced the testimony of two experts, Dr. Patrick Barnes and Dr. Stephen Gabaeff. Dr. Barnes, a pediatric radiologist, testified that he reviewed all of the provided brain imaging in this case as well as the x-ray studies. 6/7/11 RP 390. The CT scans and MRI showed a brain injury consistent with a lack of oxygen and/or blood flow and that there was bleeding between the brain and skull, and in the eyes. *Id.* at 390-91. He explained that based on the imaging, his differential diagnosis would be 1) lack of oxygen or blood flow to the baby's brain; 3) bleeding or clotting problem; 4) infection; 5) accidental or non-accidental trauma. *Id.* at 405. None of these things can be ruled out from the imaging. *Id.* at 401. As far as the timing of the injuries, Dr. Barnes testified that the brain injury could be a matter of hours old, up to a few days. *Id.* at 405. The subdural bleeding could be at least two to three days old and up to one week old. *Id.*

Dr. Gabaeff, an emergency room doctor, testified that in his opinion A.M. had viral meningitis. 6/8/11 RP 492. Her symptoms and the whole clinical picture was consistent with this infection, which may have caused her to choke during her feeding. *Id.* at 503-505. Viral meningitis can cause subdural bleeding and increased intracranial pressure (ICP). *Id.* at 633. In addition to infection, ICP, which can cause retinal hemorrhages, can also be caused by stroke, trauma, hypoxic ischemic encephalopathy, coagulation disorders, or cardiac arrest. *Id.* at 570-71; 637. Dr. Gabaeff also described the seven intubation attempts at SCH made between 10:05 p.m. and 10:36 p.m. on May 31, 2009 and the low oxygen saturation levels A.M. experienced during that timeframe. *Id.* at 534-540. He opined that A.M's intubation sequence caused the hypoxic-ischemic damage seen on the MRI. *Id.* at 551-552.

In rebuttal to the defense case, the State called Dr. John McGuire, who was the attending physician during the intubation process. 6/9/11 RP He described the same intubation

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sequence and said that the period of cumulative desaturation was five to eight minutes at the most, during which time A.M.'s vitals were monitored. *Id.* at 721-740. According to Dr. McGuire, the long-term impact of the intubation is hard to assess on a child who already had depressed neurological function. *Id.* at 740.

V. ARGUMENT

Having charged Mr. Morris with assault of a child in the first degree, the State had to prove beyond a reasonable doubt that: 1) On May 29, 2009, Mr. Morris intentionally assaulted A.M. and recklessly inflicted great bodily harm; 2) Mr. Morris was over eighteen years of age and A.M. less than three years of age, and; 3) the act occurred in the State of Washington. 6/10/11 RP 771. As the State pointed out, elements Nos. 2 and 3 were not in dispute. *Id.* The main issue was whether Mr. Morris intentionally assaulted A.M. while knowing of and disregarding a substantial risk of great bodily harm. *Id.*.

The State's case against Mr. Morris, on the disputed element, rested on the medical testimony of its witnesses, in particular Dr. Feldman, and on Mr. Morris' admission to shaking A.M.. Dr. Feldman's conclusion that A.M. suffered a traumatic head injury inflicted through "dramatic forces" immediately before she became symptomatic was the lynchpin of the State's case. Mr. Morris' allegedly incriminating admission that he shook A.M. fueled the State's theory of the case, since, according to Dr. Feldman, shaking possibly with impact, can cause A.M.'s injuries.

However, Dr. Feldman's determination of abuse is grossly unreliable and his testimony misleading, creating a false impression of the support for shaking that Dr. Feldman would have known is inaccurate, in violation of Mr. Morris' due process right to a fair trial. To the extent that preventing the introduction of unreliable testimony fell only on the shoulders of trial

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counsel, effective counsel would have ably challenged the admissibility of the testimony to begin with, and confronted Dr. Feldman at trial, to prevent the jury from relying on untrustworthy testimony. Instead, trial counsel's efforts fell short of constitutionally mandated standards of performance.

A. Legal Framework

i. Ineffective Assistance of Counsel

To prove ineffective assistance of counsel, a defendant must show that (1) defense counsel's representation was deficient; falling below an objective standard of reasonableness, and (2) the deficient performance prejudiced the defendant. *Strickland v. Washington,* 466 U.S. 668, 687, 104 S. Ct. 2052, 80 L. Ed.2d 674 (1984). 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,* 466 U.S. at 694. A reasonable probability is a probability sufficient to undermine confidence in the outcome. *Id.* Importantly, a defendant need *not* show that counsel's deficient conduct more likely than not altered the outcome of the case. *Id.* at 693. Instead, a defendant need only show that there is a reasonable probability that constitutionally effective counsel would have cast reasonable doubt on the State's case.

The prejudicial impact of counsel's errors must be considered cumulatively, rather than individually. *Strickland,* 466 U.S. at 695 (prejudice is met when there "is a reasonable probability that, but for counsel's unprofessional *errors*, the result of the proceeding would have been different.") (emphasis added).[5]

ii. Due Process

[5] *See also Williams v. Taylor,* 529 U.S. 362, 397, 120 S. Ct. 1495 (2000) (examining totality of the available mitigation evidence in assessing prejudice caused by defense counsel's failure to present evidence); *Harris v. Wood,* 64 F.3d 1432, 1438 (9th Cir. 1995) (counsel's numerous deficiencies, when considered cumulatively, established prejudice).

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It is well-established that a conviction that rests on false or misleading testimony violates due process. *See Giglio v. United States*, 405 U.S. 150 (1972); *Napue v. Illinois*, 360 U.S. 264 (1959). It does not matter whether the prosecutor intended or even knew that the testimony was false: "whether the nondisclosure [of the truth] was a result of negligence or design, it is the responsibility of the prosecutor." *Id.* at 154. Due process is also violated when State introduces misleading testimony. *Hayes v. Brown*, 399 F.3d 972, 984 (9th Cir. 2005).

If there is "any reasonable likelihood that the false testimony could have affected the judgment of the jury" the conviction must be set aside." *United States v. Agurs,* 427 U.S. 97, 103, 96 S.Ct. 2392 (1976)). Under this materiality standard, " '[t]he question is not whether the defendant would more likely than not have received a different verdict with the evidence, but whether in its absence he received a fair trial, understood as a trial resulting in a verdict worthy of confidence.'" *Hall v. Director of Corrections,* 343 F.3d 976, 983-84 (9th Cir.2003) (per curiam.) (quoting *Kyles v. Whitley,* 514 U.S. 419, 434, 115 S. Ct. 1555 (1995)).

B. Background on Abusive Head Trauma ("AHT") and Shaken Baby Syndrome ("SBS")

The history of "shaken baby syndrome" dates back to the early 1970's when two British doctors, Dr. Caffey and Dr. Guthkelch, hypothesized that whiplash forces from shaking could account for the subdural bleeding sometimes seen in babies who collapsed and otherwise had no external signs of trauma. *See* A.N. Guthkelch, *Problems of Infant Retino-Dural Hemorrhage With Minimal External Injury,* 12 Hous. J. Health L. & Policy 201 (2012) (discussing the history and the original hypothesis) (App. B). This evolved into SBS, which is diagnosed when subdural hemorrhaging and/or retinal hemorrhaging and/or brain damage (and/or other findings, such as fractures) are present and there is no known history trauma such as a car accident or major fall. *See* Ann-Christine Duhaime et al., *The shaken baby syndrome, a*

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clinical, pathological and biomechanical study, 66 J. Neurosurg. 409 (1987) (hereinafter Duhaime 1987) (App. C).

Under the SBS hypothesis, the whiplash forces (or "rotational") forces generated by violent shaking result in the traumatic tearing of veins carrying blood between the brain and the large veins in the dura, the outermost layer of the meninges that envelop the brain.[6] *See* A.K. Ommaya et al., *Biomechanics and Neuropathology of Adult and Paediatric Head Injury*, 16 Br. J. Neurosurgery 220, 226, 232-33 (2002) (hereinafter Ommaya 2002) (App. D). The tearing of these veins leads to subdural bleeding, or bleeding between the dura and the arachnoid maters. *Id.* at 227. Similarly, SBS attributes the retinal bleeding to the traumatic tearing of retinal blood vessels and the brain damage to traumatic tearing of the axons (nerve fibers) that connect nerve cells throughout the brain.[7] *Id.* In other words, the SBS hypothesis assumes that these medical findings are traumatic in origin and that violent shaking caused the trauma. *Id.* It further assumes the immediate onset of symptoms upon infliction of the injuries. *Id. See also generally* 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) (discussing the history of and basis for the SBS/AHT hypothesis).

In 2009, the American Academy of Pediatrics (AAP) issued a policy statement encouraging doctors to use the term "Abusive Head Trauma" instead of "Shaken Baby Syndrome" because the "[t]he use of broad medical terminology that is inclusive of all mechanisms of injury, including shaking, is required." *See* State's Exh. 30 at 1409 (attached here as App. E). As the position paper explains, the change in terminology was prompted by

[6] Dr. Feldman referred to these bridging veins during his testimony: "We've got these veins that then leave from the brain to the sinus that run along the top of the brain... [I]f the brain moves too far—they're anchored to the brain. They're anchored to the sinuses. They'll get stretched and they'll get torn." 6/2/11 RP 46.

[7] This traumatic tearing of the axons can also be referenced as diffuse axonal injury ("DAI") or traumatic axonal injury ("TAI).

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studies showing the importance of impact in causing an acceleration/deceleration injury, and advised that shaking is a subset of AHT. *Id.* at 1409-10. As is clear from the position paper and ongoing research, while the term AHT is broader—and "less mechanistic" according to the AAP—the underpinnings of AHT are the same as SBS. Namely, that subdural hematoma ("SDH"), retinal hemorrhages ("RH") and axonal injury are traumatic in origin and, in the absence of known trauma, highly specific for abuse.

The origin of the necessity of impact to the SBS/AHT hypothesis can be traced back to the Duhaime 1987 study, finding that the force generated by shaking was well-below established head injury criteria and was one-fiftieth the force generated by impact. Duhaime 1987 at 409. Subsequent biomechanical research has again shown that shaking alone does not produce the force necessary to cause traumatic head injury, and that violent shaking would cause neck injuries before causing the traumatic injuries associated with SBS. This research includes:

- John Lloyd et al, *Biomechanical Evaluation of Head Kinematics During Infant Shaking Versus Pediatric Activities of Daily Living*, Journal of Forensic Biomechanics (2011) (App. F) (angular acceleration of the head during aggressive shaking of a biofidelic mannequin is statistically indistinguishable from angular head kinematics experienced by a 7-month-old infant fervently playing in a jumparoo, and angular accelerations fall 84% below the scientifically accepted biomechanical threshold for bridging-vein rupture).
- Faris A. Bandak, *Shaken Baby Syndrome: A Biomechanics Analysis of Injury Mechanisms*, 151 Forensic Sci. Int. 71 (2005) (App. G) (infants subjected to the levels of rotational velocity and acceleration called for in the SBS literature would experience forces on the infant neck far exceeding the limits for structural failure of the cervical spine).
- Michael T. Prange et al., *Anthropomorphic Simulations of Falls, Shakes, and Inflicted Impacts in Infants*, 99 J. Neurosurg. 143, 143 (2003) (App. H) (hereinafter Prange 2003) (inflicted impacts against hard surfaces are more likely to be associated with inertial brain injuries than falls from a height less than 1.5 m or from shaking; impact against soft surface did not reach levels high enough to cause subdural bleeding or axonal damage)

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C. The Controversy

Challenges to the validity of the medical underpinnings of SBS/AHT have been ongoing for years. Although it entails somewhat complex medical issues, the SBS/AHT controversy revolves primarily around the following issues: 1) A study by Dr. Geddes *et al.*, showing non-traumatic origin of the brain damage found in allegedly abused children; 2) a growing list of alternative causes for the medical findings attributed to abuse under SBS/AHT, including birth trauma, genetic abnormalities, infection, childhood stroke, re-bleeds of subdural hematomas, and accidental falls; 3) the flawed circularity of research supporting SBS/AHT; 4) biomechanics; and 5) the possibility of "lucid intervals."

Legal cases, the medical literature and secondary sources illustrate the development of the medical knowledge base. *See* App. I. In *Del Prete v. Thompson*, --- F.Supp.2d --- (2014), the court engaged in a detailed description of the competing views of SBS/AHT, as well as the areas in which there is now agreement among the scientific community. The *Del Prete* court granted relief after holding an evidentiary hearing to evaluate Del Prete's actual innocence claim. The Court made two important conclusions relevant to Mr. Morris' case.

First, *Del Prete* concluded a defense expert was not biased simply because she disagreed with the SBS hypothesis:

> [Disagreement with the "shaken baby syndrome" hypothesis is hardly a sign of bias. One of respondent's own experts, Dr. Jenny, conceded that a chapter in her child abuse text (which she characterized as one of the best in a text that she considers the authoritative text in the field) makes it clear that the evidence base for the hypothesis that shaking alone can cause injury of the sort that I.Z. suffered consists exclusively of perpetrator admissions, the circumstances of which Jenny conceded she was unaware. A reasonable person—including a reasonable medical professional like Dr. Teas—could find this to be unscientific and thus unsupportable. Furthermore, respondent's expert in biomechanical engineering said that it is not yet possible for that science to establish an injury threshold for head injuries in infants. And it appears from the evidence at the hearing that the mechanism by which shaking purportedly causes these sorts of

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> injuries is as yet unclear, assuming it exists at all. All of this, given what one of Del Prete's experts referred to as a renewed emphasis on "evidence-based medicine," makes it less than surprising, and certainly not a sign of bias, that a medical professional like Teas would decline to adopt the causation and related testimony offered by Dr. Flaherty at Del Prete's trial and several of respondent's experts who testified before this Court, or the hypothesis underlying that testimony.

Del Prete, --- F.Supp.2d at 40.

Second, addressing lucid intervals, the Court concluded that:

> One way or another . . . the evidence regarding lucid intervals directly undercuts the prosecution's theory at Del Prete's criminal trial. At that trial, Dr. Flaherty testified that because I.Z. was conscious and responsive on the morning of December 27, she must have been neurologically intact at that time. She concluded from this that I.Z.'s collapse had to have been the result of abusive head trauma inflicted later that day, during a period when Del Prete was her only caregiver. This conclusion is unsupportable, given the testimony regarding lucid intervals. Indeed, Dr. Jenny went one step further, stating directly that one can no longer assume that the last caregiver with an infant who dies of abusive head trauma must have been the perpetrator.

Id. at 43.

Both of these issues—causation and lucid intervals—were, as explored below, critical issues at Mr. Morris' trial.

D. The State made the Case for Shaking

The State's theory was that Mr. Morris shook six-week old A.M. violently. *See* 6/10/11 RP 772.[8] Pretrial, defense counsel requested a *Frye* hearing, and the State responded by questioning whether a *Frye* hearing was necessary. *See* App. J. The defense filed a responsive brief (App. K), but ultimately a hearing was never held. Instead, the issue was settled when the parties stipulated to the fact that no one would use the term "shaken baby syndrome." 5/27/11 RP 61-64. When litigating this issue, the State clearly articulated its shaking theory. In its first

[8] Although the State qualified its theory with caveats about the lack of certainty as to the mechanism and used the cloak of AHT, the record clearly reflects what the State was relying on and what it invited the jury to conclude was that violent shaking, possibly with impact, caused AM's injuries.

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brief, the State said: "[I]n this case the defendant admitted to shaking the victim. Dr. Feldman will simply testify that shaking can cause the injuries presented. He will also testify regarding the degree of force necessary to cause the injuries." App. J at 5. In a reply brief, the State argued:

> This case is fundamentally different than Edmunds and the cases that are the focus of the law review article. The Defendant in this case admitted to holding the victim straight out from his chest and then shaking her for at least five seconds. He then admitted to shaking her again for another five seconds. Given the Defendant's admissions, there is no question at trial regarding whether or not he shook the victim. The question for the expert(s) is simply whether or not such shaking could cause the injuries in this case.

App. L at 3 (emphasis in original).

At trial, Officer Murphy testified Mr. Morris told him he shook A.M. twice after she stopped breathing and Mr. Morris' written statement was admitted. 6/2/11 RP 40-41; State's Exh. No. 4 (attached here as App. M). The State painted Mr. Morris' statement as incriminating, arguing Mr. Morris did not initially mention the shaking during questioning because he did not want Officer Murphy to know about it. 6/10/11 RP 778.

The State also relied on a text message from Mr. Morris to Brittany Morris saying "I did shake her after she stopped breathing brit. She would not respond to me am i a bad parent im so sorry baby i forgot this is all my fault." *See* 5/27/11 RP 65-72 (pretrial motion addressing the State's proffered use of the text message); State's Exh. No. 2 (attached here as App. N). Similar to the argument about the custodial statement, the State argued that he sent the text message after he had time to think about it and come up with a plan.[9] 6/10/11 RP 763.

On direct, Dr. Feldman explained that:

[9] Under this view of Mr. Morris' statements, where the proof that they are incriminating rests on the fact that he tried to hide the shaking, it is unclear how Mr. Morris benefits from a "plan" that reveals the very thing he is allegedly trying to hide.

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> There's a lot of debate whether shaking is possible to cause these injuries, but that is a force that causes whiplashes to occur. And if, in the course of that, the baby's head struck against something, we get very high levels of acceleration -- or deceleration, it would be. So there's a variety of things that potentially could develop forces like that.

6/3/11 RP 7. In closing the State explicitly made the case for shaking: "[W]e know from Dr. Feldman's testimony, even if it was just shaking, we're not talking about this. We're not talking about this.[10] *What he said is violent shaking*. Possibly joined with soft impact on a soft surface." 6/10/11 RP 772 (emphasis added). Thus, the State's theory was simple: Michael Morris violently shook A.M., "possibly" with impact on a soft surface.

Because there was no evidence of skull fractures, scalp swelling or scalp bruising (RP 6/3/11 RP 7), the argument that hard impact was involved was effectively foreclosed. Thus, the only feasible hypothesis was the "possibility" of impact on a soft surface. To bolster the entirely speculative claim of impact, the State elicited testimony from Dr. Feldman that since there was no autopsy and examination of the underside of the scalp, scalp bruising may be missed. *See* RP 6/3/11 RP 7.

E. Dr. Feldman's Unreliable Testimony was Inadmissible Under *Frye*[11] and ER 702

i. Dr. Feldman's expert opinion on the issue of causation was subject to a reliability analysis under ER 702

In Washington, the admissibility of expert testimony is governed by ER 702. The *Frye* test is an additional tool used by judges when proffered evidence is based upon novel theories and novel techniques or methods. *Anderson v. Akzo Nobel Coatings, Inc.*, 172 Wn. 2d 593, 606, 260 P.3d 857, 863-64 (2011). Thus, even if a *Frye* hearing is not necessary, expert testimony still has to be admissible under ER 702 and the evidence rules in general.

[10] Presumably gesticulating shaking actions for the jury.

[11] *Frye v. United States*, 293 F. 1013, 34 A.L.R. 145 (D.C.Cir.1923)).

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The court may permit "a witness qualified as an expert" to provide an opinion regarding "scientific, technical, or other specialized knowledge" if such testimony "will assist the trier of fact." ER 702. Admissibility under ER 702 requires a two-step inquiry: 1) whether the witness qualifies as an expert and 2) whether the expert testimony would be helpful to the trier of fact. *Reese v. Stroh*, 128 Wn.2d 300, 306, 907 P.2d 282, 285 (1995). Under ER 702, questions related to reliability may be considered by the court under the "helpfulness to the jury" standard of admissibility. *In re Det. of Halgren*, 124 Wn. App. 206, 219-20, 98 P.3d 1206, 1213 (2004) *aff'd*, 156 Wn.2d 795, 132 P.3d 714 (2006) (citing *State v. Copeland*, 130 Wn.2d 244, 270, 922 P.2d 1304 (1996); *State v. Janes*, 121 Wn.2d 220, 235–36, 850 P.2d 495 (1993)). In general, unreliable testimony does not assist the trier of fact. *Lakey v. Puget Sound Energy*, Inc., 176 Wn.2d 909, 918, 296 P.3d 860, 865 (2013) (excluding expert testimony on the casual link between electromagnetic fields and leukemia under ER 702 where the expert's flawed application of epidemiology rendered his conclusions unreliable).

By the time of trial, trial counsel would have been well aware that Dr. Feldman would be providing an expert opinion regarding the cause of A.M.'s injuries. In fact, in the briefing regarding a Frye hearing, counsel argued that the medical testimony was inadmissible because the expert's opinion and *causation* did not meet the generally accepted thinking of the medical community. App. K at 3-4 (emphasis added). However, once an agreement was reached that the term "shaken baby syndrome" would not be used, the request for a *Frye* hearing was dropped.

ii. Dr. Feldman's opinion is unreliable because his diagnosis ruled in a cause incapable of causing the injuries at issue

As the State described it, Dr. Feldman explained how A.M. came to be injured: Violent shaking. 6/10/11 RP 772; *see also* 6/3/11 RP 7; 23-25; 27-28; 102-108; 117 (discussing shaking

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as the mechanism of injury). Such causation testimony, of course, is based on the biomechanical premise that shaking *can* cause A.M.'s injuries.

A critical part of reliability is ensuring that the potential cause can in fact cause the injury claimed. *Bowers v. Norfolk S. Corp.*, 537 F.Supp.2d 1343, 1360 (M.D. Ga. 2007) *aff'd*, 300 Fed.Appx. 700 (11th Cir. 2008) (citing *Clausen v. M/V NEW CARISSA*, 339 F.3d 1049, 1057-58 (9th Cir. 2003); *Heller v. Shaw Indus., Inc.*, 167 F.3d 146, 155 (3d Cir.1999); *McClain v. Metabolife Int'l, Inc.*, 401 F.3d 1233, 1252 (11th Cir. 2005)). As the *Clausen* court points out, "expert testimony that rules in a potential cause that is *not* so capable is not reliable." *Clausen*, 339 F.3d at 1058 (emphasis in original).

As described in Section IV(B), it has been repeatedly shown that shaking does not achieve the level of force necessary to inflict the injuries at issue. Since Duhaime 1987, follow-up studies have failed to rule in shaking alone as a potential cause of the injuries attributed to SBS. Of note is a follow-up study conducted by Dr. Prange (along with Dr. Duhaime) in 2003, which measured the rotational velocities experienced by the head of a surrogate dummy during shakes, inflicted impacts, and short-distance falls. Prange 2003 at 144. Specifically for shaking, the researchers measured 61 sequences of shaking *with impact* against three different surfaces, including foam from a crib mattress. They concluded:

> Vigorous shakes of this infant model produced rotational responses similar to those resulting from minor falls, but inflicted impacts produced responses that were significantly higher than even a 1.5-m fall onto concrete. Because larger accelerations are associated with an increasing likelihood of injury, the findings indicate that inflicted impacts *against hard surfaces* are more likely to be associated with inertial brain injuries than falls from a height less than 1.5 m or *from shaking*.

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Prange 2003 at 143 (emphasis added). Thus, the study confirmed the 1987 Duhaime results and cast further doubt on shaking as a mechanism of injury.[12]

Critically, the Prange 2003 study directly undermines Dr. Feldman's biomechanical conjecture that the reason there was no external bruising or injuries is because impact against a soft surface would increase the forces of deceleration while at the same time spreading the force, such that bruising would not necessarily be expected. *See* 6/3/11 RP 3. Prange 2003 found that shaking with impact against foam from a crib mattress—a soft surface—did not reach levels high enough to cause SDHs or primary TAIs (traumatic axonal injury) in an infant. *See* Prange 2003 at 145; 148; 149.[13]

Dr. Feldman ruled in shaking and added, as a possibility, impact against a soft surface. But, according to biomechanical research, the shaking cannot be ruled in as a cause, even with impact against a soft surface. It is possible that shaking followed by impact on a soft surface does not leave bruises; but, according to Dr. Feldman's colleagues, it also would not cause the traumatic head findings of AHT. *Id.* Thus, the very conclusion the State asked the jury to reach based on Dr. Feldman's causation opinion is not supported by biomechanics.

That Dr. Feldman arrived at his conclusion after doing a "differential diagnosis" (6/3/11 RP 13) does not render the conclusion reliable. As courts considering the reliability of expert testimony have cautioned:

> [S]imply claiming that an expert used the "differential diagnosis" method is not some incantation that opens the *Daubert* gate to allow an expert's opinions to be

[12] In response to an inquiry in a medical journal asking why invoke shaking at all given the results of both the 1987 and 2003 studies, the study's authors agree that "we cannot yet answer if shaking can cause intracranial injury in infants." *See* App. H.

[13] The Prange authors did caution that their dummy was not biofidelic—one of the criticisms of the biomechanical models advanced by Dr. Feldman (see 6/3/11 RP 27-28). But, the authors felt this would result in an *overestimation* of both the response measured and of the rotational motions experienced, thus the results still provide the upper boundary for the measured head response to shaking, inflicted impact, and falls. Prange 2003 at 147-148 (emphasis added).

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> admitted at trial. Indeed, it can easily Amount to nothing more than medico-legal sophistry used in an attempt to avoid the Court's reliability analysis.

Bowers v. Norfolk S. Corp., 537 F.Supp.2d 1343, 1360 (M.D. Ga. 2007) *aff'd*, 300 Fed.Appx. 700 (11th Cir. 2008). Stated differently, the "differential diagnosis" approach has to be reliably applied and the analysis in *Bowers* is instructive.[14]

In *Bowers*, a plaintiff suing the railroad for injuries allegedly caused while operating a train offered an orthopedist's testimony that excessive vibration caused the plaintiff's back and neck injuries. The defendant objected to the reliability of the expert's causation opinions, to which the plaintiff responded by arguing that the expert used a "differential diagnosis" approach. *Id.* at 1352. Accordingly, the Court explored at length the specific issue of a differential diagnosis, first distinguishing between "differential diagnosis"—diagnosis of the disease—and "differential etiology"—the investigation and reasoning the leads to the determination of external causation, and noted that the latter more aptly described the expert's approach. *Id.* at 1361. But whereas the differential diagnosis approach has inherent reliability, the differential etiology approach does not. *Id.* Looking at the substance of the expert's analysis, rather than just the label, the *Bowers* court ruled the expert testimony was unreliable and therefore inadmissible. *Id. at 1362.*

Here, as in *Bowers*, the "differential diagnosis" advanced by Dr. Feldman is more than a diagnosis of the disease, since it encompasses the manner of injury. AHT not only detects medical findings—retinal and subdural bleeding in this case—but asserts the etiology of the

[14] Under *Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579, 113 S.Ct. 2786 (1993), Fed. R. Evid. 702 imposes on courts a duty to ensure expert opinions are reliable. Thus, an analysis of reliability of expert testimony is instructive here, where Washington's ER 702 also requires reliability in order to be helpful to the trier of fact.

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findings to be abusive trauma.[15] Thus, it was important to identify Dr. Feldman's diagnosis for what it was—a causation determination—that ruled in a potential cause of A.M.'s injuries that has been disproved.

The Washington Supreme Court explained in *Lakey* that testimony based on novel scientific methodology is excluded under *Frye*, until a scientific consensus decides the methodology is reliable;[16] and ER 702 excludes testimony where the expert fails to adhere to that reliable methodology. *Lakey*, 176 Wn.2d at 918-19. Unreliably applying disproved or unsupported bio-mechanical assertions to arrive at his diagnosis renders Dr. Feldman's methodology defective, no matter how certain of the conclusion he may be. As in *Bowers*, the *ipse dixit* of his testimony does not render it reliable. *Id.* at 1355. Accordingly, Dr. Feldman's testimony is not helpful to the jury and should have been excluded.

iii. Dr. Feldman's opinion is unreliable because the current knowledge base does not support making definitive conclusions on causation

Since the 2003 Prange study, injury thresholds and how infant tissue responds to repetitive forces still have not been established. Thus, a leading child abuse book concludes that more research is needed to provide a comprehensive answer to the question "what mechanisms cause what injuries in children of what age." *See* Susan Marguiles and Brittany Coates,

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[15] For a specific discussion of this issue, *see* A.N. Guthkelch, 12 Hous. J. Health L. & Policy at 202 (suggesting that that the appellation be changed to describe the medical findings only so that the issue of causation can be investigated objectively) (App. B).

[16] Where there is no longer continued acceptance, a *Frye* hearing is necessary. *See State v. Cauthron*, 120 Wn.2d 879, 890, 846 P.2d 502 (1993), *overruled in part on other grounds by State v. Buckner*, 133 Wn.2d 63, 941 P.2d 667 (1997). The field of biomechanics being a critical component of the diagnosis, the lack of continued acceptance in that community requires a *Frye* hearing where the State expert is offering testimony on the type and level of force inflicted, testimony that rests squarely on biomechanics. Thus, counsel had a legitimate basis to request a *Frye* hearing based on the biomechanical aspect of the case. Pursuant to a *Frye* hearing or motion, counsel could have established that the biomechanical foundation of the State's shaking theory is no longer accepted, primarily because it has never been proved despite repeated studies. Accordingly, and at a minimum, Dr. Feldman's biomechanical assertions would have been excluded or appropriately limited to those grounded in the current knowledge-base and admissible under ER 401-403.

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Biomechanics of Head Trauma in Infants and Young Children, in Child Abuse and Neglect, Diagnosis, Treatment and Evidence 359, 361 (Carole Jenny, ed., 2011) (App. O); *See also Del Prete*, --- F.Supp.2d at 19 (summarizing the testimony of the State's biomechanical expert as follows: The science of biomechanics cannot determine that cause of [victim's] injuries and cannot yet determine the threshold necessary to produce head injuries in infants; there is no reliable well-accepted injury threshold for head injury to an infant as a result of rotational acceleration (*i.e.* shaking back and forth) and for neck injury in an infant). As the *Del Prete* court concluded, the claim that the science of biomechanics is not yet able to establish an injury threshold in this area "provides a newfound basis for skepticism about causation and mechanism testimony." *Id.* at 41.

Significantly, Dr. Feldman well knows that injury thresholds have not been established. Dr. Feldman testified that biomechanical studies are gradually getting better, "but, as yet, there is no study that defines how infant brain tissue responds to repetitive sheer forces back and forth." 6/3/11 RP 27-28. Later, when discussing Duhaime 1987, Dr. Feldman reiterates that there is a lack of reliable scientific information: "It was a landmark early study that has subsequently been refined and re-refined but still isn't in any of the studies to the point where it reliably tells us what happens in infants." 6/3/11 RP 44.

These statements, made in the context of explaining why the biomechanical studies concluding shaking is not a viable mechanism of injury are not reliable, acknowledge the lack of information necessary to render a valid, grounded opinion on causation. Nonetheless, Dr. Feldman still claims to know that the trauma sustained in this case was abusive. Stated differently, Dr. Feldman agrees the biomechanical data is too unreliable to make definitive

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claims about the mechanism of injury, but only takes this into consideration when questioning the challenges to shaking.

However, the unreliability works both ways. Taking as true Dr. Feldman's concerns, what is also true is that not knowing how infant tissue responds to repetitive sheer forces back and forth (*i.e.* shaking) means we do not know a key premise of his opinion: It takes very dramatic forces. If we do not have the information that "reliably tells us what happens in infants," Dr Feldman's unequivocal conclusion that abuse—forceful shaking that any reasonable person would know is excessive, possibly with impact against a soft surface—caused the injuries is baseless. *See* 6/3/11 RP 9.

The imprecise nature of Dr. Feldman's testimony illustrates the lack of a basis. Other than claiming that it takes "dramatic forces" or "very high levels of acceleration or deceleration," there is no specific information about the amount of forces that are harmful, over what amount of time, the number of shakes (or whiplash events), or even any information to gauge what is considered "very high" levels. Presumably, Dr. Feldman does not have this information, since there is no reliable, well-accepted injury threshold for rotational forces.

The injury threshold and level of force being inherent components to determining causation, the lack of reliable information renders the testimony inadmissible. "[I]t is well established that conclusory or speculative expert opinions lacking an adequate foundation will not be admitted." *Safeco Ins. Co. v. McGrath*, 63 Wn. App. 170, 177, 817 P.2d 861, 865 (1991). Without an adequate foundation, the testimony is not probative on the issue at hand and is not helpful. *See State v. Riker*, 123 Wn.2d 351, 364, 869 P.2d 43, 50 (1994) (the helpfulness test subsumes a relevancy analysis, under which the court considers the state of knowledge presently existing about the subject of the proposed testimony and the facts of the case).

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Imprecise and vague testimony is not helpful, since it does not help the jury determine whether the expectation that A.M.'s injuries are the result of abusive shaking (possibly with impact) are "a virtual certainty, a strong probability, a more likely than not possibility, or a mere possibility." *See Bowers*, 537 F.Supp.2d at 1359 (*quoting Frazier*, 387 F.3d at 1266)). Indeed, it is unacceptable and unfair for the State, in a criminal case requiring proof beyond a reasonable doubt, to present and rely on testimony that would not pass muster in a medical malpractice case, where it is well-established that a finding of proximate cause must be based on more than speculation, conjecture or mere possibility. *See Reese v. Stroh*, 128 Wn.2d 300, 309, 907 P.2d 282, 287 (1995).

iv. Dr. Feldman's opinion is unreliable because the evidence-base for shaking is admittedly weak and non-scientific

The weakness of the evidence base for shaking was acknowledged, even by SBS/AHT proponents, as early as 2002. At an AAP conference addressing inflicted childhood neurotrauma, the following introductory remarks were made:

> 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.

See Findley et al., 12 Hous. J. Health L. & Pol'y at 234, fn. 83.

Almost 10 years after the NIH conference and the Donohoe review of literature discussed below in Section IV(H)(i), the basic criticism that the evidence base is flawed has not changed. A leading child abuse textbook, for which Dr. Feldman authored a chapter, acknowledges that "nobody has yet marshaled a coherent and comprehensive argument in support of shaking as a causal mechanism for abusive head injury." Mark S. Dias, *The Case for*

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Shaking, in Child Abuse and Neglect, Diagnosis, Treatment and Evidence 362, 368 (Carole Jenny, ed., 2011) (App. P).

As Dr. Dias makes clear, "the evidence base for shaking *is* confessions." *See Id.* at 370. (emphasis in original). But, confessional studies are problematic, including for the reason that the studies themselves reveal the "confessions" do not always match the objective medical findings. *See* Suzanne P. Starling *et al., Analysis of Perpetrator Admissions to Inflicted Traumatic Brain Injury in Children*, 158 Archives Pediatric & Adolescent Med. 454 (2004) (noting that twelve percent of "shaking only" confessions did not match the medical findings).[17] If the confessions are only "partially legitimate" (Dr. Feldman's words at 6/3/11 RP 29), they do not provide an accurate basis to reliably identify the cause of injury.

Relying on perpetrator admissions to make scientific claims about causation is dubious, at best. Perpetrator admissions are hardly scientific and are a known source of wrongful convictions. *See* Innocence Project, "False Confessions"[18] (innocent defendants made incriminating statements, delivered outright confessions or pled guilty in about 30% of DNA exoneration cases). *See also Del Prete*, ---F.Supp.2d at 40 (finding it reasonable to believe that the SBS hypothesis is unscientific and unsupportable where the evidence base consists exclusively of perpetrator admissions). Courts considering confessions in SBS/AHT cases have noted that the unique circumstances of abuse cases make the nature of alleged confessions particularly hard to assess and their reliability doubtful. *See Aleman v. Vill. of Hanover Park*, 662 F.3d 897, 906-907 (7th Cir. 2011) (discussing at length the unreliability of alleged confessions in child abuse cases where the suspect is told that the only possible cause of the

[17] The study by Starling was referenced by Dr. Feldman as supporting his conclusion that abused children exhibit immediate symptoms. It did not come out that this study, like Vinchon 2010, is based on confessions. *See* 6/3/11 RP 116-117.

[18] http:// www.innocenceproject.org/understand/False-Confessions.php (last visited July 15, 2014)

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injuries is abusive shaking; "if a question has only two answers—A and B—and you tell the respondent that the answer is not A, and he has no basis for doubting you, then he is compelled by logic to "confess" that the answer is B."); *People v. Thomas*, 22 N.Y.3d 629, 646, 985 N.Y.S.2d 193, 202 (2014) (Excluding proffered confession as involuntary and noting that "defendant's admissions were not necessarily rendered more probably true by the medical findings of the treating physicians;" instead, the similarity between the findings and the confession "can be understood as a congruence forged by the interrogation.").

Confessional literature does not fix the problem, acknowledged by SBS/AHT community, that support for shaking as a causal mechanism is lacking. Without adequate support, Dr. Feldman's causation testimony is unfounded and the State's reliance on it to advance its theory of shaking is unreasonable. This case itself illustrates the problem with relying on confessions. According to the State, Mr. Morris confessed to shaking his daughter. While he did admit to resuscitative shaking, the "confession" is disputed. This case would now, however, be used as a confirmed case of abuse based on a confession and a conviction, which in turn relied on other judicial confessions. Thus, the SBS/AHT hypothesis gains entrenchment through legal fiat rather than through scientific investigation.

F. Trial Counsel's Failure to Challenge the Admissibility of Dr. Feldman's Opinion Was Deficient Performance

Trial counsel's failure to ensure the proffered expert testimony was sound and admissible under ER 702, even though "ER 702 has independent force and effect" and "has a significant role in admissibility of scientific evidence aside from *Frye*" (*State v. Copeland*, 130 Wn.2d at 259) was deficient and prejudiced Mr. Morris. It was crucial for trial counsel to ensure that the substance of the opinion testimony on the critical issue of causation was

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reliable, regardless of the official terminology. The focus on terminology versus substance was misguided.

"Counsel 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). Trial counsel's failure to know the biomechanical literature and the history of SBS/AHT hypothesis, confirming that the evidence-base for shaking is weak and now anchored in confessions only, is inexcusable. The State made it abundantly clear they were relying on shaking. Counsel was concerned about the causation aspect of the case, yet, unreasonably, trial counsel was appeased once the State agreed not to use the term "shaken baby syndrome."

Not investigating or presenting beneficial evidence falls below acceptable performance standards where the failure to do either is not the result of *reasonable* judgment.[19] Similarly, not subjecting Dr. Feldman's opinion to the admissibility threshold of the applicable evidentiary rules simply because the State agreed not to say "shaken baby syndrome" is not reasonable judgment. Trial counsel should have moved to exclude Dr. Feldman's causation

[19] *See, e.g., In re Brett*, 142 Wn.2d 868, 881-82, 16 P.3d 601 (1998) (finding counsel's performance defective where he knew or had reason to know of defendant's relevant medical and mental problems but failed to conduct a reasonable investigation that would have uncovered available medical evidence); *Alcala v. Woodford*, 334 F.3d 862, 870-71 (9th Cir. 2003) (finding that a competent attorney would have introduced evidence of an alibi witness and documents corroborating another alibi witness unless he did not know about the evidence or had a sound reason for not doing so, and declining to manufacture a reason for counsel); *Avila v. Galaza*, 297 F.3d 911, 918-19 (9th Cir. 2002 (finding counsel's performance defective where he failed to adequately investigate or present available evidence that defendant's brother was the shooter); *Caro v. Calderon*, 165 F.3d 1223, 1226-27 (9th Cir. 1998) (finding counsel's performance during penalty phase ineffective where he failed to consult a neurologist or toxicologist on the effects of chemical poisoning, a potentially mitigating factor, and failed to provide the four experts that did examine the defendant with the information necessary to do an accurate neurological evaluation even though he was aware of defendant's exposure to neurotoxicants); *Turner v. Duncan*, 158 F.3d 449, 456-57 (9th Cir. 1998) (holding that counsel was defective when he failed to investigate further and present psychiatric evidence that corroborated his client's defense, even though the previous attorney had already arranged for a psychiatric evaluation and obtained a helpful report).

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testimony—at a minimum, his biomechanical claims—which under ER 702 and the applicable caselaw was not admissible, as described above.

The State relied heavily on the causation testimony of Dr. Feldman to prove its case. In fact, without establishing causation, the State could not prove that Mr. Morris intentionally assaulted A.M. and recklessly injured her. Thus, there is a reasonable probability that but for counsel's failure to challenge the admissibility of the testimony, the outcome of the trial would have been different. *Strickland*, 466 U.S. at 695.

G. The State and Dr. Feldman Had a Duty to Present Accurate Information

It was incumbent upon the State, and Dr. Feldman, to present accurate information. *See Mohil v. Glick*, 842 F.Supp.2d 1072, 1077 (N.D. Ill. 2012) (doctors reporting and investigating suspected child abuse are state actors for purposes of § 1983 claim); *Billups v. Penn State Milton S. Hershey Med. Ctr.*, 910 F.Supp.2d 745, 757 (M.D. Pa. 2012) (finding sufficient facts to establish reasonable likelihood that child abuse doctors will qualify as state actors in § 1983 claim).

As explained in Section IV (B) & (E), the uncertainty about shaking as a mechanism is well-established. Dr. Feldman was almost certainly aware that the Prange 2003 study does not support shaking with soft impact as a cause (and that rest of the biomechanical literature does not either), and that the evidence base is anchored in confessional studies. To the degree Dr. Feldman's testimony created a false impression of the state of science and research purportedly supporting the State's theory, the State's conviction was obtained in violation of Mr. Morris' due process right to a fair trial. *Hayes v. Brown*, 399 F.3d at 978. That the State made its case against Mr. Morris by officially claiming AHT, so as to fall within the cloak of a recognized

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diagnosis, where in fact the actual theory was one not supported by the current medical thinking, further taints the State's actions.

H. The unreliability of Dr. Feldman's conclusions is further illustrated by epidemiological data and A.M.'s post-trial head Injury

i. Lack of epidemiological support

As the history of the SBS/AHT hypothesis and this case make clear, the presence of RH and SDH are suspicious for abuse in the absence of a known event that also explains the findings, such as a car accident or major fall. Dr. Feldman explained that "[w]henever we have obvious trauma but no history of trauma, we have to get concerned about what happened" and "having both [RH and SDH] at the same time and having the profuse retinal hemorrhages and the retinoschisis are highly specific for abusive head injury." 6/3/11 RP 12, 16. Stated differently, RH and SDH are good proxies for abuse. This is supported, according to Dr. Feldman by the literature, such as the Vinchon 2010 study.

A study of hospital data looking at these proxies for abuse, separately and together, establishes that, in fact, SDH and RH are very poor indicators of abuse. Dr. Michael Freeman, a forensic doctor and epidemiologist, analyzed the Kids' Inpatient Database (KID), a vast, national database of pediatric hospital stays, for the years 2000-2009. *See* App. Q (Report of Dr. Michael Freeman). Specifically, Dr. Freeman selected from the database every child age 3 or less who had a diagnosis of RH or SDH or retinoschisis and determined the rate of diagnosed child abuse amongst those with and without additional diagnoses of trauma/or injury (such as A.M.). *See Id.* at 7. Examining the rate of child abuse for children with only SDH and RH, such as A.M., and the rate of child abuse for kids who also had RH and SDH, but, unlike A.M., had additional findings of trauma or injury, allows one to arrive at the true positive rate of the test for abuse employed by Dr. Feldman. *See Id.* at 6-7.

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The results make clear that the presence of RH and SDH is not a very reliable indicator of abuse. As explained by Dr. Freeman, when both conditions, RH and SDH, are present and there are no additional findings of traumatic injury (as was the case here), there is less than a 20% probability of abuse. When RH and SDH are present, and there are additional findings of traumatic injury, the probability increases, to more than 50%. *Id.* at 8. Adding the diagnosis of retinoschisis did not increase the reliability of RH and SDH as proxies for abuse. *Id.*

That these numbers look very different from the data Dr. Feldman discusses can be understood by what is a common error of conditional probability. *See* App. R (Michael D. Freeman et al., *Forensic Epidemiology: A systematic approach to probabilistic determinations in disputed matters*, 5 J. For. Legal Med. 15, 281-90 (2008) (discussing statistical fallacies and other probability errors common in causation determinations). Stated simply, the error happens when one confuses, for example, the chance that a person speaks Spanish if she is from Spain, with the chance that a person is from Spain if she speaks Spanish.

Applied here, the error arises when one confuses the probability that an abused child has RH and SDH, with the probability that a child with RH and SDH has been abused. The studies Dr. Feldman relies on, which are only looking at cases of abuse, have answered, potentially, the former question, but not the latter. Dr. Freeman's analysis, showing the true positive rate of SDH and RH as indicators for abuse, and by corollary the false positive rate of the test (82%), confirms the unreliability of Dr. Feldman's conclusion that A.M. was the victim of AHT. Indeed, the fallibility of his conclusion presents an intolerable risk that Mr. Morris was wrongfully accused and convicted.

ii. A subsequent unexplained head injury assumed to be abusive

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Shortly after sentencing, A.M. suffered another closed-head injury on July 17, 2011 while with an 11-year old babysitter in Missouri. Trial counsel brought this to the Court's attention, pointing out that the doctors in Missouri believed there had been abusive shaking going on for some time. *See* App. S at 2 (Defense Memorandum for a New Trial). On August 2, 2011, the State appraised the defense that the child abuse specialist at the Missouri hospital believed the injuries were the result of abusive trauma, with a bruise suggesting impact; but at the same time that he doubted the babysitter could inflict the injury given the severity. *See* 8/2/11 E-mail correspondence (App. T). The medical records reflect the medical findings were subdural and retinal hemorrhaging and major brain swelling; the police reports reflect that abuse remained the conclusion despite being unable to locate a "guilty party." *See* App. U (excerpt from Missouri medical records); App. V (Poplar Bluff Police Department Supplemental Narrative Report dated February 23, 2012).

Abuse was immediately suspected by the doctors in Missouri given the constellation of injuries, while at the same time it was decided that abuse seemed implausible under the circumstances. Since abuse is, admittedly, an unsatisfactory explanation for the injuries, it is unclear why abuse allegedly producing immediate symptoms remained the sole consideration. It would appear that like Dr. Feldman, the doctors in Missouri are, erroneously, assuming the presence of the same medical findings are reliable indicators for abuse. Petitioner is currently trying to obtain review of all the medical records from Missouri by a medical expert.

1. <u>The State's Case Relied on Misleading Evidence that Trial Counsel Failed to Effectively Confront</u>

Where it was trial counsel's responsibility to expose the flaws in the State's case, effective assistance of counsel, to which Mr. Morris is entitled, was necessary. *See Perry v. New Hampshire*, 132 S.Ct. 716, 723, 181 L.Ed.2d 694 (2012) (effective assistance of trial

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counsel is one of the protections afforded defendants against evidence of questionable reliability).

i. The impression that the evidence-base in support of SBS/AHT is strong is misleading

After making a broad reference to studies regarding whiplash forces on the head, Dr. Feldman made the conclusory remark that shaking is a force that causes traumatic whiplash forces to occur.[20] Thereafter, the well-grounded case in support of shaking was not made, and could not have been made given the current knowledge base. Instead, the State focused on pre-empting the criticism of shaking by what the State referred to as the "skeptics." 6/3/11 RP 103; 108.

Specifically, Dr. Feldman said: "Well, there's skepticism of whether shaking can do it. The commonest article they rely on there is one by Donohoe[21] that he claims is an evidence-based review of the literature...[but] he violated all principles of doing an evidence-based review." 6/3/11 RP 25. According to Dr. Feldman, Donohoe only searched up to 1999, he did not have criteria for selecting which articles had "adequate quality" to be included in the review, and he inappropriately applied evidence-based standards. *Id.* at 26.

Trial counsel never pointed out that: 1) Dr. Donohoe explains why he's only searching up to 1999 (because that was the timeframe before evidence-based standards were widely accepted); 2) that the whole point of the review is to assess the quality of the articles, so pre-selecting them based on quality is redundant and avoids the whole exercise; and 3) that Dr.

[20] It is not disputed that major events can result in severe whiplash forces. The issue here, however, is whether the specific mechanism—shaking—advanced by the State is one of those major events. Stated differently, the relevant question is whether the whiplash forces generated by shaking are high enough to cause the specific injuries Dr. Feldman relies on, retinal and subdural bleeding.

[21] *See* Mark Donohoe, *Evidence-Based Medicine and Shaken Baby Syndrome Part I: Literature Review*, 1966-1998, 24 A.M.. J. Forensic Med. Pathology 239, 241 (2003) (hereinafter Donohoe 2003) (Appendix W).

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Donohoe did not conclude that Level-I evidence (randomized control trials) is the only appropriate standard; instead, he accurately pointed that none of the evidence reaches the highest level of evidence-based medicine (controlled trials) and that most of the literature achieved only the lowest level IV—opinions based on clinical experience and descriptive reports. Donohoe 2003 at 239-241.

More importantly, Dr. Feldman would have known, and trial counsel could have pointed out, that the overall point of Donohoe's review still stands: The evidence base for shaking is weak. Donohoe's conclusion that the evidence is largely Level-IV studies and that it suffers from circularity flaws is accepted, albeit not happily. The problem of circularity, described by Donohoe as "the obvious logical error of selecting cases by the presence of the very clinical findings and test results they seek to validate as diagnostic" still remains. Donohoe 2003 at 239.

Dr. Vinchon's study,[22] the very study relied on by Dr. Feldman, explains the circularity as a major problem:

> The hallmarks of shaken baby syndrome (SBS) are subdural hematomas (SDH), encephalopathy, and retinal hemorrhage (RH). Since in most series the diagnosis of IHI [inflicted head trauma] is based on the presence of these features, evaluation of the diagnostic value of these criteria is flawed by a bias of circularity.

Vinchon 2010 at 637-638 (citations omitted). This is why Dr. Vinchon did a study where cases were selected as being abusive based not on the medical findings; instead confessions were used as the proxy to "confirm" abuse. *See Id.* at 642. Where the research makes clear that

[22] 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) (hereinafter Vinchon 2010) (App. X).

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Donohoe's concerns with the evidence base are legitimate and acknowledged, Dr. Feldman's testimony that the "skeptics" rely primarily on a flawed literature review is grossly misleading.

Dr. Feldman further bolstered the case for SBS/AHT with the Vinchon 2010 study, describing the results as follows:

> In every kid who had intracranial bleeding, the subdural hemorrhage, who had profuse retinal hemorrhages and who lacked surface evidence of head trauma, no skull fracture, no swollen scalp, had suffered abusive head injury. That's exactly the combination we're dealing with here.

6/3/11 RP 16. Since the basis of Dr. Feldman's opinion is never explored meaningfully, what is not mentioned is that the confessions relied on by the study's authors came from judicial sources; the authors assumed the information could be trusted; they had limited data on the details of the confession, and; they had limited data on the mechanism of abuse. *See* Vinchon 2010 at 642.

Several important points flow from the above. It is not known: 1) what was considered a confession and whether, for example, confessions to resuscitative shaking were included; 2) whether the confessions were spontaneous or came about after being confronted by allegations of abuse and claims that the medical findings were consistent with abuse; 3) whether the confessions were disputed, and; 4) whether, with limited data on the mechanism of abuse, the abuse was shaking only, shaking with impact, or something else. Overall, without sufficient information about the underlying data, there is no way to assess whether the study Dr. Feldman relies on is itself reliable.

Later, Dr. Feldman testified the Bhardwaj study concluded that retinal hemorrhages are specific for head injury 95% of the time and *that retinoschisis occurs in no other situation but*

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abusive head trauma.[23] 6/3/11 RP 12-13 (emphasis added). However, as Dr. Feldman knew and trial counsel could have pointed out, in the Bhardwaj 2009 study, which was a systemic review of 20 studies, the authors themselves caution that there was the potential for circular bias in all but four of the studies. *See* Bhardwaj 2009 at 985. Regarding retinoschisis, the authors did observe that retinoschisis was only seen in AHT, as Dr. Feldman asserts. But given that the review only looked at studies that included children diagnosed with AHT (*Id.* at 984), this is hardly surprising. The authors themselves conclude that the specificity of retinoschisis for AHT is undetermined: "Further studies, which accurately and extensively document retinal findings in a wide variety of conditions, are required to better estimate the specificity of these signs for AHT." *Id.* at 990.

Since the case against Mr. Morris rested on the State's ability to convince the jury that shaking was a plausible mechanism, counsel could and should have shown that the doubt about shaking is not held by a minority of "skeptics." It is grounded in valid criticism of the evidence-base, and is the reason studies, such as Vinchon 2010, rely on confessions. Not only would this have substantially weakened Dr. Feldman's seemingly unchallengeable assertions, but it would have bolstered Mr. Morris' own experts, who instead appeared as experts with outlier opinions.

The fact that the defense put on experts does not minimize the prejudice to Mr. Morris from counsel's failure to effectively confront Dr. Feldman. None of the biomechanical or other literature that counters Dr. Feldman's claims was explained or explored through the defense experts. Dr. Barnes made a broad reference to medical studies, noting that in the last decade we have learned that injuries always thought to be abuse can be caused by accident or natural

[23] Dr. Feldman does not give the study's author during direct, but based on the summary of the study's conclusions and cross-examination, it appears to be the Bhardwaj study. *See* Gaurav Bhardwaj et al., *A Systematic Review of the Diagnostic Accuracy of Ocular Signs in Pediatric Abusive Head Trauma*, 117 Ophthalmology 983, 985 (2010) (App. Y).

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causes. *See* 6/7/11 RP 383-384. Dr. Gabaeff was objected to and the objection sustained when he began to discuss biomechanics (6/8/11 RP 571-72), and the only study discussed was the Bandak 2005 study, which Dr. Feldman discredited during his testimony as being incorrect. *See* 6/3/11 RP 22-24.[24]

ii. The claim that the timing of the injuries could be precisely timed is misleading

Brittany Morris testified that on May 29, 2009, she took her older daughter, TM, to nearby Stevens Hospital because TM was not feeling well. 6/1/11 RP 155-156. Mr. Morris stayed behind with A.M., and when she left, Mr. Morris was sitting on the couch with A.M., feeding her. *Id.* at 157. She was filling out paperwork at the counter when she received a call from Ms. Orkiz that Mr. Orkiz was the way to hospital with Mr. Morris and A.M... *Id.* at 158-59. She had not been gone longer than fifteen minutes. *Id.* at 188.

Since Mr. Morris was with A.M. for only 10-15 minutes before she collapsed, the question of the timing of the alleged injuries was critical at trial.[25] In order to prove beyond a reasonable doubt that it was Mr. Morris who assaulted A.M. the State had to prove that A.M.'s injuries were inflicted immediately before she collapsed, during a narrow 10 to 15 minute window of time.

To time the alleged infliction of abuse, Dr. Feldman testified that "[i]t's very consistent that almost all of the kids have immediate symptoms of the injury." 6/3/11 RP 18. He further explained that there should be some behavioral alteration in the child at the time of the event,

[24] The Bandak study has been criticized, including by Dr. Feldman, as containing erroneous calculations and using inappropriate injury thresholds. Dr. Bandak has stood by his conclusions and answered the criticism, agreeing that the neck forces obtained in his study are high but not because of numerical error, but because that is what results when the described SBS accelerations are applied to the infant head for the time duration of shaking; lower injury thresholds for the neck proposed by the critics stem from studies that would also have rotational forces too low under the SBS hypothesis. *See* Faris A. Bandak, *Response to the Letter to the Editor*, 164 Forensic Sci. Int. 282-283 (2006) (App. G). Counsel did not point this out.

[25] This assumes, of course, that the findings are inflicted to begin with and not caused by another process, such as disease.

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and that it is possible that A.M. vomited and choked, but this would have happened after the head injury. *Id.* at 17. He conceded that lucid intervals can happen, but explained that if there is a lucid interval, you would expect the child to exhibit symptoms after the event, not act "perfectly normal." *Id.* at 10.

Trial counsel discussed the possibility of lucid intervals, using a study Dr. Feldman pointed her to, a study looking at cases of abuse confirmed to be such through confessions. 6/3/11 RP 94-95.[26] Dr. Feldman minimized the results of the study, pointing out that most of the babies were put immediately to sleep after being shaken such that the perpetrators were not able to observe whether or not there were symptoms. *Id.* at 97-98. Other than Dr. Feldman mentioning studies by Starling and Sirotnak in redirect, no other studies were discussed. See 6/3/11 116-117.

But, the issue of lucid intervals is not disputed and it is acknowledged that a child may be normal or near normal before developing severe symptoms or collapsing. As early as 1998, there was a study showing periods of lucidity of at least 24 hours in a majority of the cases studied, and periods of lucidity of more than 24 hours between the trauma and the collapse in approximately 25% of the shaking/impact cases and blunt injury cases. M.G.F. Gilliland, *Interval Duration Between Injury and Severe Symptoms in Non-accidental Head Trauma in Infants and Young Children*, 43 J. Forensic Sci. 723, 723 (1998) (App. Z). Due to the results, the article cautions that: "Enough variability in the interval between injury and the time of severe symptoms or presentation for medical care in fatally injured children exists *to warrant circumspection* in describing such an interval for investigators or triers of fact." *Id.* at 724 (emphasis added).

[26] Catherine Adamsbaum et al., *Abusive Head Trauma: Judicial Admissions Highlight Violent and Repetitive Shaking*, 126 Pediatrics 546 (2010)

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In 2005, the journal of the American Academy of Pediatrics journal acknowledged lucid intervals. *See* 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 toddler may sustain a fatal head injury yet present to hospital clinicians as lucid before death) (App. AA)

Given this research, a leading child abuse textbook cautions that: "Severe and *delayed* acute clinical deterioration is possible after accidental or inflicted childhood neurotrauma." Kent Hymel et al., *Abusive Head Trauma*, in Child Abuse and Neglect, Diagnosis, Treatment and Evidence 249, 350 (Carole Jenny ed., 2011) (emphasis in original) (internal citations omitted) (App. BB). As part of this very same community, Dr. Feldman was almost certainly aware of the advice that caution is warranted when making timing determinations. More importantly, he would have been well aware that claiming to reliably pinpoint the injury *to within 10-15 minutes* is not anchored to the knowledge base.

Trial counsel made a Motion to Dismiss pursuant to *State v. Green*, arguing that the evidence was insufficient to convict given lucid intervals. In her memorandum, she stated:

> Dr. Feldman's exact testimony with regards to the possibility of lucid interval in the case of A.M. indicates that it is 'highly unlikely' such a thing occurred. This doesn't rise to the level required to allow this case to proceed. Again the standard in Green is whether any reasonable trier of fact can find all the elements *beyond a reasonable doubt* . . . Even. Dr. Feldman himself cannot definitively say that the harm occurred in the 10 minutes in which Mr. Morris was alone with A.M.

See App. BB at 2 (internal citations omitted).[27] But as is clear from the testimony, Dr. Feldman did claim to know the harm happened when A.M. was with Mr. Morris, and the testimony that a lucid interval is highly unlikely supports this claim.

[27] Responding to the motion, the State again made it clear that shaking was the theory of the case: "Mr. Morris admitted in a text to shaking the child. He admitted to the officer that he shook the child twice for periods of five

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Had trial counsel used the studies available on this issue rather than just rely on the article Dr. Feldman himself pointed her to, she could have established through Dr. Feldman the facts supporting a motion to dismiss: That he could not state precisely when the injury occurred. Medical testimony that cannot precisely time an injury where the window of time was only 10-15 minutes does not support an inference beyond a reasonable doubt that the cause of A.M.'s occurred during the period she was alone with Mr. Morris, as required under *State v. Green*, 94 Wn.2d 216, 616 P.2d 628 (1980). At a minimum, she could have argued to the jury that Dr. Feldman's certitude that the injury happened under Mr. Morris' care is unreasonable and well outside what current medical knowledge supports. *See Del Prete*, ---F.Supp.2d at 42 (noting that child abuse expert Dr. Jenny conceded that "given current medical thinking on this issue, one can no longer say automatically that the last person with the infant was responsible for abuse.")

V. CONCLUSION

For all the foregoing reasons, Mr. Morris respectfully requests that this Court vacate his convictions and grant him a new trial pursuant to CrR 7.8(b). Alternatively, if further facts are necessary to resolve the motion, Mr. Morris requests that an evidentiary hearing be held.

Respectfully submitted this 18th day of July 2014

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seconds each, which is consistent with both the testimony of Dr. Feldman and Dr. Herlihy with regards to acceleration and deceleration forces causing the injuries -- their findings." 6/6/11 RP 367.

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CERTIFICATE OF SERVICE

I certify that I mailed a copy of the foregoing Motion and Memorandum, postage prepaid, on July 18, 2014 to

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APPENDIX INDEX

Appendix A	Mandate Letter
Appendix B	A.N. Guthkelch, *Problems of Infant Retino-Dural Hemorrhage With Minimal External Injury*, 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, *Biomechanical Evaluation of Head Kinematics During Infant Shaking Versus Pediatric Activities of Daily Living*, 2011
Appendix G	Faris A. Bandak, *Shaken Baby Syndrome: A Biomechanics Analysis of Injury Mechanisms*, 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, *Biomechanics of Head Trauma in Infants and Young Children*, in Child Abuse and Neglect, Diagnosis, Treatment and Evidence, 2011
Appendix P	Mark S. Dias, *The Case for Shaking*, in Child Abuse and Neglect, Diagnosis, Treatment and Evidence, 2011
Appendix Q	Report of Dr. Michael Freeman
Appendix R	Michael D. Freeman et al., *Forensic Epidemiology: A systematic approach to probabilistic determinations in disputed matters*, 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., *A Systematic Review of the Diagnostic Accuracy of Ocular Signs in Pediatric Abusive Head Trauma*, 2010
Appendix Z	M.G.F. Gilliland, *Interval Duration Between Injury and Severe Symptoms in Non-accidental Head Trauma in Infants and Young Children*, 1998
Appendix AA	Kristy B. Arbogast et al., *Initial Neurologic Presentation in Young Children Sustaining Inflicted and Unintentional Fatal Head Injuries*, 2005
Appendix BB	Kent Hymel et al., *Abusive Head Trauma*, in Child Abuse and Neglect, Diagnosis, Treatment and Evidence, 2011
Appendix CC	Trial Memorandum in Support of Defendant's Motion to Dismiss Pursuant to *State v. Green*

Appendix A

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

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

FILED
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SONYA KRASKI
COUNTY CLERK
SNOHOMISH CO. WASH

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

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

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

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

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

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

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58

209

Appendix B

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PROBLEMS OF INFANT RETINO-DURAL HEMORRHAGE WITH MINIMAL EXTERNAL INJURY

A.N. Guthkelch*

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

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

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

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

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

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

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

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

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

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

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

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

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

3 A.C. Duhaime et al., *Head Injury in Very Young Children: Mechanisms, Injury Types, and Ophthalmologic Findings in 100 Hospitalized Patients Younger Than 2 Years of Age*, 90 Pediatrics 184 (1992).

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

5 *Id.* at 8-10.

6 *Id.*

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

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

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

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

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8 *Supra* note 4 at 7.

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

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

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

10 *Id.* at 233.

11 *Id.* at 236.

12 *Id.* at 234.

13 *Id.* at 236.

14 *Id.* at 234.

15 *Id.* at 235.

16 *Supra* note 1 at 431.

17 *Id.* at 430.

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

Appendix C

J Neurosurg 66:409–415, 1987

The shaken baby syndrome

A clinical, pathological, and biomechanical study

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Division of Neurosurgery and Department of Bioengineering, University of Pennsylvania, Philadelphia, Pennsylvania

✓ Because a history of shaking is often lacking in the so-called "shaken baby syndrome," diagnosis is usually based on a constellation of clinical and radiographic findings. Forty-eight cases of infants and young children with this diagnosis seen between 1978 and 1985 at the Children's Hospital of Philadelphia were reviewed. All patients had a presenting history thought to be suspicious for child abuse, and either retinal hemorrhages with subdural or subarachnoid hemorrhages or a computerized tomography scan showing subdural or subarachnoid hemorrhages with interhemispheric blood. The physical examination and presence of associated trauma were analyzed; autopsy findings for the 13 fatalities were reviewed. All fatal cases had signs of blunt impact to the head, although in more than half of them these findings were noted only at autopsy. All deaths were associated with uncontrollably increased intracranial pressure.

Models of 1-month-old infants with various neck and skull parameters were instrumented with accelerometers and shaken and impacted against padded or unpadded surfaces. Angular accelerations for shakes were smaller than those for impacts by a factor of 50. All shakes fell below injury thresholds established for subhuman primates scaled for the same brain mass, while impacts spanned concussion, subdural hematoma, and diffuse axonal injury ranges. It was concluded that severe head injuries commonly diagnosed as shaking injuries require impact to occur and that shaking alone in an otherwise normal baby is unlikely to cause the shaken baby syndrome.

KEY WORDS • shaken baby syndrome • head injury • child abuse

THE term "whiplash shaken baby syndrome" was coined by Caffey[3] to describe a clinicopathological entity occurring in infants characterized by retinal hemorrhages, subdural and/or subarachnoid hemorrhages, and minimal or absent signs of external trauma. Because a nursemaid admitted that she had held several such children by the arms or trunk and shaken them, the mechanism of injury was presumed to be a whiplash-type motion of the head, resulting in tearing of the bridging veins. Such an injury was believed to be frequently associated with fatalities in infantile child abuse and has been postulated as a cause of developmental delay in survivors.[4,15]

While the term "shaken baby syndrome" has become well entrenched in the literature of child abuse, it is characteristic of the syndrome that a history of shaking in such cases is usually lacking.[12] Shaking is often assumed, therefore, on the basis of a constellation of clinical findings and on the computerized tomography (CT) picture of subarachnoid and subdural hematomas, particularly in the posterior interhemispheric fissure.[17] Because of the ambiguous circumstances of such injuries, medicolegal questions are particularly troublesome, and the neurosurgeon is often consulted to give an opinion as to whether the findings are consistent with child abuse or accidental injury.

This paper reviews all cases of the shaken baby syndrome seen at the Children's Hospital of Philadelphia (CHOP) between January, 1978, and March, 1985. To better study the mechanism of injury, autopsy results in all fatal cases were reviewed, and the biomechanics of this injury were studied in a series of infant models. Based on these observations, we believe that shaking alone does not produce the shaken baby syndrome.

Clinical Studies

Clinical Material and Methods

All reports submitted to the Suspected Child Abuse and Neglect team were reviewed. Since house officers

TABLE 1

Initial clinical criteria for diagnosis of shaken baby syndrome

Diagnosis*	Cases		No. of Deaths
	No.	Percent	
retinal hemorrhage + SAH or SDH	29	60	5
retinal hemorrhage + SAH & SDH	10	21	5
bilateral chronic SDH	3	6	0
SAH &/or SDH & interhemispheric blood on CT	6	13	3
total	48	100	13

* SAH = subarachnoid hemorrhage; SDH = subdural hemorrhage; CT = computerized tomography.

TABLE 2

Best history in 48 cases of shaken baby syndrome

Etiology	Cases	
	No.	Percent
shaking only	1	2
fall or accidental blunt trauma	15	31
strike or fall plus shaking	10	21
strike only	3	6
trauma or shaking denied, caretakers in attendance	8	17
history unknown, caretakers not in attendance	10	21
cardiopulmonary resuscitation	1	2

TABLE 3

Trauma associated with shaken baby syndrome in 48 cases

Associated Trauma	Cases	
	No.	Percent
no evidence of blunt impact to head	18	37.5
no extracranial trauma	12	25.0
additional extracranial trauma	6	12.5
acute	3	6.25
old trauma only	3	6.25
evidence of blunt impact to head	30	62.5
skull fractures	12	25.0
cranial soft-tissue contusions	18	37.5
additional extracranial trauma	18	37.5
acute	15	31.25
old trauma only	3	6.25

and emergency room personnel are well trained in recognizing the clinical manifestations associated with this syndrome, it is considered that essentially all cases seen at CHOP are reported to this group.

Suspicion of shaking was based on history, clinical findings, and CT data. All subjects met the following criteria: presence of retinal hemorrhages with subdural and/or subarachnoid hemorrhages, bilateral chronic subdural hematomas, or a CT scan showing subdural or subarachnoid hemorrhages with interhemispheric blood. In addition, all patients were judged to have histories suggestive of child abuse or neglect; well-documented, witnessed accidental trauma was excluded. Histories were obtained from several interviews with caretakers by physicians, social workers, and in some cases law enforcement agents. Caretakers were routinely asked specifically about shaking.

Associated trauma data were obtained from physical examination, skull radiographs, CT scans, and skeletal surveys. All fatal cases were examined by the Philadelphia Medical Examiner, and pathology data were obtained from that office.

Results

Fifty-seven patients with suspected shake injury were identified. Of these, detailed clinical information was available in 48 cases. These patients ranged in age from 1 month to 2 years (mean 7.85 months). Thirty-one patients were male (65%). There were 13 fatalities (27%). Initial clinical criteria for diagnosis of the shaken baby syndrome are listed in Table 1. Thirty-nine patients (81%) had retinal hemorrhages plus subarachnoid and/or subdural hemorrhages. The remainder had bilateral chronic subdural hematomas (6%) or the above-mentioned CT findings without retinal hemorrhages (13%).

The most common presenting complaints were lethargy, breathing difficulty, irritability, poor feeding, and seizures. Best history is listed in Table 2; the most common histories were accidental blunt trauma (usually a fall) in 15 (31%) and blunt trauma plus shaking in 10 (21%); trauma and shaking were denied in eight (17%). In three cases (6%) the child was struck by the caretaker. In eight additional cases the history was unknown, usually because the child was left alone or with a babysitter. There were two cases (4%) with no history to explain the present findings, but both children were known to have been abused previously or subsequently. One case was associated with cardiopulmonary resuscitation (2%). In only one case was a history of shaking alone obtained; this child was reportedly shaken when she appeared to have difficulty in breathing associated with a respiratory infection.

Associated trauma observed clinically, radiographically, or at autopsy is listed in Table 3. The presence of scalp contusion, subgaleal or subperiosteal hemorrhage, and/or skull fracture was considered evidence of blunt impact to the head. Twelve cases (25%) had intracranial findings associated with the shaken baby syndrome alone, with no findings of associated blunt trauma to the head and no extracranial trauma. Six additional cases (13%) had the syndrome without signs of blunt head trauma but did have associated extracranial trauma. Thirty cases (63%) had findings of blunt impact to the head in addition to the intracranial findings of the shaken baby syndrome. Of these, 12 (25%) had skull fractures and 18 (38%) had significant cranial soft-tissue contusions. Most of the fractures were in the occipital or parieto-occipital region.

Clinical history, physical findings, hospital course, intracranial pressure (ICP, when measured), and pathological findings of the 13 fatalities are listed in Tables

TABLE 4

*Clinical and pathological findings in 13 fatal cases of shaken baby syndrome**

Factor	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6	Case 7	Case 8	Case 9	Case 10	Case 11	Case 12	Case 13
age (mos)	24	7	3	22	11	9	8	5	10	13	24	4	19
sex	F	M	M	M	F	F	F	M	F	M	M	M	F
history													
fall or hit	+		+	+	+			+	+	+	+	+	+
shaking			+								+		+
trauma denied													
unknown		+				+	+						
initial examination													
unresponsive	+	+	+	+	+	+	+	+	+	+	+	+	+
retinal hemorrhages	+	+		+			+	+	+	+	+	+	
cranial impact	+			+						+	+	+	+
extracranial trauma	+			+	+						+	+	
intracranial pressure	↑↑	NM	↑↑	↑↑	↑↑	NM	↑↑	NM	↑↑	NM	↑↑	↑↑	↑↑
survival time (days)	2	2	7	2	3	2	2	1	1	1	4	1	1
pathology													
cranial contusions	+	+	+	+	+	+	+	+	+	+	+	+	+
skull fracture(s)	++			++	++							+	+
subdural hematoma	+	+	+	+	+	+	+	+	+	+	+	+	+
subarachnoid hemorrhage	+	+	+	+	+	+	+	+	+	+	+	+	+
hemispheric contusions	+	+	+			+		+					+
white matter tears	+					+			+				+
diffuse brain swelling	+	+	+	+	+	+	+	+	+	+	+	+	+

* ↑↑ = increased; NM = not measured; + = factor present; ++ = severe.

4 and 5. Mean age in this group was 12.23 months; 54% were male. All of these children arrived at the hospital in an essentially unresponsive state, and all died from the effects of uncontrollably increased ICP associated with massive brain swelling. In only one case was a subdural hematoma thought to be of significant size to warrant surgical intervention, and drainage was ineffective in controlling elevated ICP.

Pathological examination showed that all of the children who died had evidence of blunt head trauma. Eight had soft-tissue contusions and five had contusions and skull fractures. In seven cases, however, impact findings were noted only at autopsy, and had not been apparent prior to death. All fatal cases had subdural and subarachnoid bleeding. Focal cerebral contusions and lacerations occurred in six. Microscopic examination was performed in three cases and showed corpus callosum hemorrhages, cortical laminar necrosis, or white matter hemorrhages. All children had diffuse and usually massive brain swelling.

Biomechanical Studies

Whole Infant Models

To test the hypothesis that infants are particularly susceptible to injury from shaking because of a relatively large head and weak neck, we constructed models of 1-month-old infants that were implanted with an accelerometer to measure the results of shaking or impact manipulations. Since the mechanical properties of the infant neck have not been studied, three models were built with different neck structures in order to include the range of limiting conditions that might exist in the live infant. Both a fixed center of rotation with zero resistance (hinge model) and moving centers of rotation with low and moderate resistance (rubber neck models) were tested.

Experimental Methods

The heads and bodies of the models were adapted from Just Born dolls. Head circumference was 36 cm, coronal width was 10 cm, anteroposterior diameter was 10.75 cm, and height from vertex to base (calculated from a line drawn from chin to caudal occiput) was 9.0 cm; values were comparable to human infants. Brain weight for an infant of this size was assumed to be 500 gm.[1] The ideal weight of the head was estimated by balance-weight measurements of several infants with an average age of 1 month, and was 770 to 870 gm. The heads of the models were tightly filled with cotton, with water added until the desired weight range was reached. The water was absorbed by the cotton and distributed so that no sloshing of the contents occurred. The heads were reweighed after neck insertion and sealing and at the end of all experiments.

Neck length from the skull base to the T-1 vertebra

TABLE 5

Summary of findings in 13 fatal cases of shaken baby syndrome

Factor	Finding
age (mos)	
mean	12.23
range	3–24
sex M/F	7/6
history	
fall or hit (three with shaking)	10
unknown	3
initial examination	
unresponsive	13
retinal hemorrhages	9
cranial impact	6
extracranial trauma	5
intracranial pressure	
measured, unable to control	9
not measured	4
survival time (days)	
range	1–7
mean	2.2
pathology	
cranial contusions	13
skull fractures(s)	5
subdural hematomas (one requiring surgery)	13
subarachnoid hemorrhage	13
unilateral	3
diffuse	3
multifocal	7
hemispheric contusions	6
diffuse, multiple	3
focal, coup-contrecoup	3
white matter tears	4
gross	2
microscopic	2
diffuse brain swelling (11 with herniation evident)	13

TABLE 6

Mean acceleration and time course of shakes and impacts in all models

Manipulation	No.	Peak Tangential Acceleration (G)	Time (msec)	Angular Velocity (radians/sec)	Angular Acceleration (radians/sec²)
shakes	69	9.29	106.6	60.68	1138.54
impacts	60	428.18	20.9	548.63	52,475.70

TABLE 7

Effects of neck condition and "skull" on mean peak tangential acceleration and time course of shakes and impacts

Variant	Shakes		Impacts	
	Acceleration (G)	Time (msec)	Acceleration (G)	Time (msec)
hinge neck	13.85	92.7	423.42	18.6
flexible rubber neck	5.70	93.3	427.78	21.4
stiff rubber neck	7.02	130.5	433.33	22.8
skull	9.86	107.4	436.12	20.2
no skull	8.89	103.5	427.04	21.6

TABLE 8

Effect of impact surface on mean peak tangential acceleration and time course

Surface of Impact	Acceleration (G)	Time (msec)
padded surface	380.60	24.22
metal bar	489.51	17.13

was measured from lateral neck films of several normal infants with an average age of 1 month and ranged from 3.5 to 4.5 cm; all models were therefore given neck lengths of 4.0 cm. Necks were embedded in Castolite resin* superiorly, which was also used to seal the head. The interior part of the neck was secured in dental stone.† The stuffed body was then replaced around the dental stone "thorax," with lead weights added as necessary to the thorax to reach a total body weight of 3 to 4 kg. Arms and legs were not weighted, so the slightly low total weight for age reflects an attempt to approximate trunk:head weight ratios.

Model 1 had a hinge neck made from a 360° steel hinge, 3.6 cm in width, placed in the horizontal plane to allow complete anteroposterior angulation of the head. The center of rotation was 3.3 cm below the estimated level of the skull base (approximating at the C-6 vertebral level). Model 2 had a 1.9-cm diameter hollow rubber neck with a 0.8-cm lumen. This 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. Model 3 had a 2.9-cm rubber neck with a 1.2-cm lumen. This neck was able to support the head in the vertical position but allowed full passive movement of the head. In all models, head motion was limited in the anteroposterior direction by the occiput striking the upper back and the chin striking the chest.

To test for the effect of the deformability of the model heads on impact, all models were tested with and without an external "pseudoskull" made from thermoplastic.‡ This "skull" was 1/8 in. thick and was molded to the occipital, parietal, temporal, and posterior frontal areas, with the facial area uncovered. The "skulls" weighed 170 to 200 gm.

Data were recorded from a piezoelectric accelerometer§ embedded in a small piece of thermoplastic and attached to the vertex in a coronal plane through the

* Resin manufactured by Buehler Ltd., Evanston, Illinois.

† Dental stone, Glastone Type IV, manufactured by Ransom and Randolph Co., Toledo, Ohio

‡ Polyform thermoplastic manufactured by Rolyan Medical Products, Menomonee Falls, Wisconsin.

§ Accelerometer manufactured by Endevco Corp., San Juan Capistrano, California.

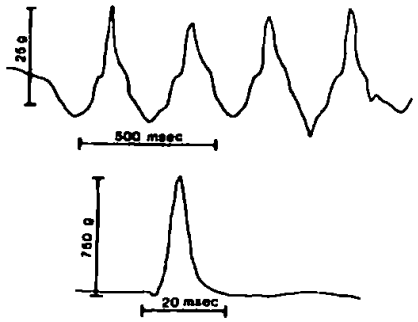

FIG. 1. Representative tangential acceleration traces for infant models undergoing shake *(upper)* and impact *(lower)* manipulations. While manipulations of the infant models were performed as described, with a series of shakes followed by an impact, the magnitude of the impact accelerations was so much greater than that associated with the shakes that different scales are used to display the respective acceleration traces.

center of the neck. Each model was subjected to repetitive violent shaking, allowing the head to travel its full excursion several times, by adult male and female experimenters. The models were held by the thorax facing the experimenter and were shaken in the anteroposterior plane, since this is the motion most commonly described in the shaken baby syndrome. At the end of each series of shakes the occiput was impacted against either a metal bar or a padded surface. Each model was tested at least 20 times. Acceleration traces were amplified and recorded.‖

Angular accelerations were calculated from the measured peak tangential accelerations by using C-6 as the center of rotation in all cases. Angular velocity was calculated as the time integral of the acceleration curve. Translational forces were assumed to be minimal.

Results

The data were collected from 69 shaking episodes ("shakes") and 60 "impacts." Typical tangential acceleration traces for shake and impact manipulations are shown in Fig. 1. The criterion for significant difference was $p < 0.01$ in all cases.

Shakes Versus Impacts. Angular acceleration and angular velocity for each shake and impact are shown in Fig. 2. Mean peak tangential acceleration for 69 shaking episodes was 9.29 G; mean peak tangential acceleration for 60 impacts was 428.18 G (Table 6). The accelerations due to impact are significantly greater than those obtained by shaking ($p < 0.0001$); on the average, impact accelerations exceed shake accelerations by a factor of nearly 50 times. Mean time interval

‖ Shock amplifier, Model 2740 A, and pulse memory unit, Model 2743, manufactured by Endevco Corp., San Juan Capistrano, California.

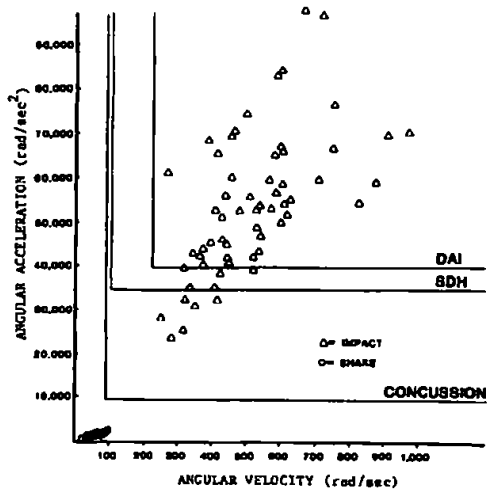

FIG. 2. Angular acceleration versus angular velocity for shakes and impacts, with injury thresholds from primate experiments scaled to 500-gm brain weight. DAI = diffuse axonal injury; SDH = subdural hematoma.

for shakes was 106.6 msec and for impacts was 20.9 msec. This difference is significant at the $p = 0.001$ level.

Effects of Neck Condition. Mean tangential accelerations and time courses for shakes and impacts for each neck condition are presented in Table 7. There is no significant difference between the hinge neck, the flexible rubber neck, and the stiff rubber neck in the mean acceleration resulting from impacts (423.4, 427.8, and 433.3 G, respectively) or in the mean time course (18.6, 21.4, and 22.8 msec, respectively). With shakes, the more flexible hinge neck is associated with higher accelerations (mean 13.85 G) than the two rubber neck models (mean 5.7 and 7.0 G) ($p < 0.001$). There is an inverse relationship between neck stiffness and time duration of a shake: the stiff rubber neck was associated with a longer time course than the more flexible rubber neck (130.5 msec and 93.3 msec, respectively) ($p < 0.001$).

Effects of "Skull." The presence of a hard thermoplastic "skull" did not change the magnitude or time course of accelerations associated with shaking of the models. The acceleration magnitude and time course were also unchanged when the models were impacted. These data are shown in Table 7.

Effects of Impact Surface. Impact against a padded surface was associated with significantly smaller acceleration (mean 380.6 G) and longer time course (mean 24.22 msec) than that against a metal bar (mean 489.5 G and 17.13 msec) ($p < 0.001$). Data are shown in Table 8.

Discussion

Clinical head injury can be classified into two major categories according to the distribution of pathological damage, whether focal or diffuse.[10] Such a distinction is important for treatment and prognosis, as well as for establishing the biomechanical conditions necessary to produce a given injury type. It has been established both experimentally and clinically that most focal injuries are associated with impact loading, resulting in contact phenomena, while diffuse injuries are associated with impulsive loading conditions resulting from acceleration-deceleration phenomena.[6] Damage to the brain occurs as a result of these biomechanical forces and from the secondary effects of ischemia due to altered autoregulation or brain swelling.

The shaken baby syndrome has been postulated to result from the effects of nonimpact acceleration-deceleration forces. It has been suggested that the back and forth movement of the head alone is sufficient to cause tearing of bridging veins, resultant subdural hematomas, and death.[8,13] The relatively large size of an infant's head, weakness of the neck musculature, softness of the skull, relatively large subarachnoid space, and high water content of the brain have been postulated to contribute to the susceptibility of shaking injuries in infants.[4,14]

While shaking alone has been considered sufficient to cause a fatal injury, the usual lack of history of the true mechanism of injury in these cases has hampered accurate clinicopathological correlations. It is of interest, however, that in a recent series of fatal cases of infantile head injuries from suspected child abuse,[5] white matter tears were found similar to those described by Lindenberg and Freytag[11] in blunt trauma in infancy. In addition, lesions in the distribution typical of diffuse axonal injury, like those found in adult head injury and in subhuman primates subjected to high acceleration-deceleration injury,[7] were described in some cases. In fact, at least one of Caffey's original cases[3] included "lacerations of the cerebral parenchyma." Shaking alone was the presumed mechanism of these injuries.

As experience has accumulated in experimental angular acceleration injury it has become clear that, besides the magnitude of the acceleration, another important biomechanical factor influencing injury type is the time interval over which the acceleration occurs. Thus, large angular accelerations occurring over shorter time periods tend to result in subdural hematoma, while longer intervals are associated with diffuse axonal injury.[6] A tolerance scale relating these two factors to resultant injury has been developed for the subhuman primate by Thibault and Gennarelli.[16] Values above certain critical limits result in a particular type of injury such as concussion, subdural hematoma, or diffuse axonal injury. When such a curve is scaled for the brain mass of an infant the size of our models, it can be seen that the angular acceleration and velocity associated with shaking occurs well below the injury range, while the values for impacts span concussion, subdural, and diffuse axonal injury ranges (Fig. 2). This was true for all neck conditions with and without skulls. A padded surface decreases the magnitude of acceleration and lengthens the time course to some extent, but these impacts also fall in the injury range.

These results are consistent with the observation that the fatal cases of the shaken baby syndrome in this series were all associated with evidence of blunt impact to the head. This preponderance of blunt trauma has also been found in at least one other series of nonaccidental head trauma in childhood in which the mechanism of injury was investigated.[9] 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." Skull fractures and scalp contusions were found at autopsy, however, most often in the occipital or parieto-occipital region. In addition, several babies had parenchymal lesions in a distribution consistent with diffuse axonal injury.[11]

While some reports on the shaken baby syndrome mention brain swelling, in most reports the subdural collections themselves have been postulated as the cause of death. In this series, all fatalities were consequent to uncontrollable brain swelling, and it is clear that drainage of the small collections present would have been useless in controlling the ICP. The problem of acute brain swelling is particularly common in the pediatric population, and its cause is poorly understood.[2] Whether high accelerations in the anteroposterior direction have some particular association to this complication remains to be investigated.

It is our conclusion that the shaken baby syndrome, at least in its most severe acute form, is 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. The most common scenario may be a child who is shaken, then thrown into or against a crib or other surface, striking the back of the head and thus undergoing a large, brief deceleration. This child then has both types of injury — impact with its resulting focal damage, and severe acceleration-deceleration effects associated with impact causing shearing forces on the vessels and parenchyma. 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.

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Appendix D

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REVIEW

Biomechanics and neuropathology of adult and paediatric head injury

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Abstract

The objective of this study was to understand the biomechanics in age-related primary traumatic brain injuries (TBI) causing initial severity and secondary progressive damage and to develop strategy reducing TBI outcome variability using biomechanical reconstruction to identify types of causal mechanisms prior to clinical trials of neuro-protective treatment. The methods included the explanation of TBI biomechanics and physiopathological mechanisms from dual perspectives of neurosurgery and biomechanical engineering. Scaling of tolerances for skull failure and brain injuries in infants, children and adults are developed. Diagnostic assumptions without biomechanical considerations are critiqued. Methods for retrospective TBI reconstruction for prevention are summarized. Mechanisms of TBI are based on the differences between the mechanical properties of the head and neck related to age. Skull fracture levels correlate with increasing cranial bone thickness and in the development of the cranial sutures in infants and in adults. Head injury tolerance levels at three age categories for cerebral concussion, skull fracture and three grades of diffuse axonal injuries (DAI) are presented. Brain mass correlates inversely for TBI caused by angular head motions and locations of injurious stresses are predictable by centripetal theory. Improved quantitative diagnosis of TBI type and severity levels depend primarily on age and biomechanical mechanisms. Reconstruction of the biomechanics is feasible and enables quantitative stratification of TBI severity. Experimental treatment has succeeded in preventing progressive damage in animal TBI models. In humans this has failed, because the animal model received biomechanically controlled TBI and humans did not. Clinical similarities of human TBI patients do not necessarily predict equivalent biomechanics because such trauma can be produced in various ways. We recommend 'reverse engineering' for in-depth reconstruction of the TBI injury mechanism for qualitative diagnoses and reduction of outcome variability.

Key words: *Adults, biomechanics, children, head injuries, mechanisms, reconstruction, youths.*

Mechanisms of traumatic brain injuries

This report addresses causation of traumatic brain injury (TBI) with special reference to critical differences in the biomechanical properties, and responses of the skulls and brains of infants, children and adults.

The incidence of TBI is highest among youths peaking at age 15–24 years. Children aged 5 and under, as well as the elderly aged 65 and over occupy similar secondary peaks suggestive of their vulnerability.[47] Unintentional injuries are the leading killer of children. Each year, more children, ages 1–14 die from unintentional injuries than from all childhood diseases combined. Under age 1, however, homicide is the leading cause of injury deaths. Head injury is the most frequent cause of death in the paediatric population, comprising between 50 and 80% of all trauma related deaths each year. Annual paediatric head injury statistics represent an enormous emotional and financial burden of 300,000–400,000 hospitalizations, 6000 to 7000 deaths, and an estimated $10 billion in annual costs for injury to our children in the USA.[74]

A recent survey by the National Center for Injury Prevention and Control (CDC) of hospitalized and fatal TBI cases in infants 0–4 years old and in youths 15–19 years old in seven states shows the TBI counts and rates per 100,000 for 1994 (Table I). Falls were the major cause of death in infants followed by motor vehicle crashes and then by intentional injuries. Youths died primarily in motor vehicle crashes, followed by falls and then by assaults.[117]

Our review of the clinical and biomechanical literature regarding the basic mechanisms of the aetiology of TBI in adults is fairly well understood.[4,23,25,26,93] The biomechanics of paediatric

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TABLE I. Causes of traumatic brain injury in infants and youths. Case counts and rates/100,000.

	Infants (0–4)				Youths (15–19)			
Causes of TBI	Case counts hospitalized & fatal	Case counts fatal only	Rate hospitalized & fatal	Rate fatal only	Case counts hospitalized & fatal	Case counts fatal only	Rate hospitalized & fatal	Rate fatal only
Falls	1124	12	42.6893	0.4558	282.5	10	11.7168	0.4148
Motor vehicle occupant	325	48	12.3434	1.823	1786	318	74.0749	13.1891
Assault	196	39	7.444	1.4812	261.5	9	10.8458	0.3735
Pedestrian	133	31	5.0513	1.774	122.5	18	5.0807	0.7466

* Selected data for 1994, reported from seven states and re-analysed by Dr D. J. Thurman. MD, NCIPC. CD3

TBI, as compared with adult TBI are not as clearly defined in biomechanical or diagnostic terms. The clarification of these diagnostic issues is important because children are very vulnerable to TBI below the ages of 3–5. They also suffer intentional, as well as unintentional injuries. Injuries remain the leading cause of childhood mortality, morbidity and disability. In order to clarify the biomechanical aetiology of primary adult and paediatric TBI, special emphasis of these important issues of the physical basis of TBI biomechanics will be given in Appendix I.

Experimental biomechanics of the skull and brain in traumatic brain injuries

Two sets of our earlier tests on experimental whiplash in adult anaesthetized Rhesus monkeys are summarized as follows: the first set was an exposure to a severe extension/flexion 'whiplash' type of head acceleration associated with a terminal minor head impact at the level of 10% probability of cerebral concussion.[80] The mechanism that was modelled in these experiments mimicked a severe rear-end collision on a torso-restrained occupant. This type of a single severe inertial load caused acute subdural haematomas (SDH), gliding contusions of the brain and spinal cord injuries in these experiments at angular velocity in the range of 500 radians/s and accelerations of 10,000 radians/s^2 for 5–6 m duration.[86] A second set of impact experiments on anaesthetized Rhesus monkeys enabled us to develop the data for impact as well as impulsive tolerance levels.[90,91] The whiplash experiments were further validated in 1968 after clinical analysis on two patients who suffered whiplash injuries in 30 mph rear end collisions to their automobiles while belted, resulting in subdural haematomas and one fatality.[86] It should be noted, however, that the magnitude of the angular acceleration and the short duration of the pulse measured in our first set of experiments are typical of head angulations occurring in impact loads and not seen in even the most violent shaking as modelled by Duhaime *et al.* until a terminal impact was given and produced the level of head acceleration as predicted for TBI in our previous experiments.[21]

In our impact and whiplash animal models similar injuries of SDH and contusions are produced, but at half the level of angular velocity by impact alone and twice that required by whiplash.[83,90,91] Scaling of injury thresholds for brains of different mass were performed in three sets of animal models and extrapolated to the brain mass of an average human.[89] Our experiments tested Holbourn's hypothesis which indicated the critical factor for brain injury as being angular acceleration and linear acceleration as being innocuous. Based on his theory and experiments on physical 2-D models, Holbourn also postulated that smaller brains would require more force than larger brains.[39] We tested the vulnerability of three primate species with increasing brain size to various levels of angular acceleration causing brain injury. Our hypothesis predicted increasing risk for brain damage in larger brains according to an inverse 2/3 power of the brain mass, ranging from 30 to 400 g in our three animal models. These experiments confirmed that larger brains were indeed more vulnerable at lower levels of angular velocity and acceleration as predicted by Holbourn. Higher levels of angular acceleration were required for causing similar injury to the smaller brains.[83,90] It is improbable that the high speed and severity of the single whiplash produced in our animal model could be achieved by a single manual shake or even a short series of manual shaking of an infant in one episode. Our discovery of a lower level of angular velocity (by 50%) in our impact experiments demonstrated the relative ease with which SDH is produced by impact as compared to whiplash alone.[88-91] Subsequently, however, our experimental results were referenced as providing the experimental basis of the 'shaken baby syndrome' (SBS) by Caffey, Gulthkelch 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.[10,33] The reality of fatal brain damage by repeated shaking of the larger adult brain and sustained over repetitive significant periods has been well documented recently in an adult Palestinian who died after repeated and sustained violent shaking in 12 sequential sessions by Israeli interrogators of the Shin Bet without evidence of head

impact. Autopsy findings discovered the findings of acute subdural haematoma, retinal haemorrhages and extensive soft tissue bruises in the shoulders and pectoral muscles.[97] Similar severity and repeated episodes of shaking in children could also result in fatality with similar neuropathologic lesions, e.g. acute SDH and retinal haemorrhage with soft tissue bruises, as well as skeletal injuries at higher levels of shaking force based on the inverse relation to the head mass. Damage to the neck and the spinal cord analogous to our experimental findings would also be expected.

In 1989, Hadley *et al.* published a report on the 'Infant Whiplash Shake Injury Syndrome'. Postmortem studies did not reveal any definitive evidence of contact phenomena to the head but the authors emphasized the frequency of injuries to the cervical spinal cord similar to that seen in our whiplash experiments.[34] Routine examination of the neck in abused children with head injuries should be examined with MRI.

In a separate series of impact experiments in rhesus monkeys, the mechanism of coup and contra-coup contusions with and without skull fracture was defined. The locations of SDH were primarily over the anterior half of the brain and fronto-temporoparietal SDH was the predominant location with no significant correlation with location of skull fracture. The rotational velocity for these experiments causing the brain lesions was measured using high-speed cinematography at 4000 frames/s. These values ranged between 150 to 250 radians, with impact linear velocities of 40–50 feet/s to occipital and frontal sites of impact.[88]

Our second set of impulsive (non-impact) experiments were also done on anaesthetized monkeys in which the head was constrained in a rigid container with the torso fully restrained, with controlled movement applied to the head neck complex. Two types of head acceleration in two groups of subjects received either *translation* of the centre of gravity (CG) of the head by one inch as compared with identical CG translation *plus* angular acceleration at increasing levels of linear acceleration. Pure translation of the head and neck could not produce hemispheric acute SDH, no diffuse petechial haemorrhages in the brain and no injuries to the cervical cord at linear accelerations in the range of up to 1000 *g*. Similar levels of linear accelerations combined with the angular motion of the head on the neck produced classical bilateral acute SDH and diffuse petechial brain haemorrhages in all subjects without impact.[83,87,91]

The distribution of the acute lesions in the latter group followed the predictions of our centripetal theory for the location of the TBI lesions which had been developed as our experimental paradigm at the onset of our programme in 1965 based on our the clinical observation of the 'one-way' disassociation of the amnesic and paralytic phenomena in TBI patients at lower levels of TBI severity and the testing of Holbourn's theory.[83,84,95] Clinical validation of the centripetal theory in adult and paediatric TBI patients was subsequently confirmed by other investigators using CT and MRI and correlations of the brain lesions with neuropsychological evaluations.[54,55,56,112,125]

A report by Duhaime, Gennarelli and Thibault *et al.* tested the effect of manually induced inertial forces of an impulsive periodic nature. Adult volunteers manually induced repetitive shaking of the head/neck of an anthropomorphic surrogate 'dummy' equivalent to a 6-month baby. The neck of the surrogate baby was replaced by a hinge to allow maximal angular head accelerations. Accelerometers on the head of the surrogate recorded linear accelerations during repeated shaking of the head of the child surrogate by volunteers. The maximum values of the head accelerations related to the angular motions of the surrogate head were found to be well below the requisite threshold for the production of the critical principal strains in the elements of the brain. In contrast to the low levels of accelerations indicative of strains in the brain during repetitive shaking, a single forced rotational motion of the surrogate by a volunteer ending in a contact of the 'dummy' head with a rigid surface at a table height, immediately resulted in higher levels of acceleration associated with the entire spectrum of TBI as found in infants.[21] Repetitive and prolonged episodic shaking by mechanical means without head impact and over a range of specific time periods has not been experimentally tested. A large animal model is required to correlate the biomechanics and physiopathology of repetitive and prolonged 'whiplash' shaking, particularly to investigate the cumulative effect of repeated shaking.

A recent experiment by Smith *et al.* has attempted to develop a model for head shaking TBI in 6-day-old rats subjected to intermittent shaking for 6 s followed by 6-s pauses repeated 60 times daily for 3 days. The authors explain that in the development of this model they were unable to produce subdural haemorrhages or neuronal cell loss by shaking alone. They required an added insult of hypoxia combined with shaking in an inverted position in order to obtain subarachnoid haemorrhages and progressive cortical degeneration.[109] The results showed that in the 6-day-old rat pup brains, only when the *shaken plus hypoxic state* was produced, a moderate to severe *subarachnoid* haemorrhage and progressive degeneration of cortical neurons were found. No subdural haematomas were produced. It is to be noted that at day 7, these rat pups have 50% of their adult brain weight and the inverted position during shaking could also produce intracranial hypertension. Unfortunately, no biomechanical

measurements such as head angular excursion, velocity or acceleration were reported for these experiments. Further experiments with appropriate biomechanical data correlated with neuropathology are required in order to develop a useful model, preferably in a larger animal model under more physiological conditions.

Principles of biomechanics

The mathematical basis of biomechanisms is given in the appendix. The causation of TBI can occur either by impact or by impulsive loading (shaking), which lead to different results. The consequences of an impact that are unique to the infant and young child can be explained on the basis of the compliant nature of the brain-case as compared to the adult, relatively non-compliant skull. In the infant, the skull is not a rigid shell structure (the cranial vault) as is found in the adult; it is better characterized as a segmented structure of loosely associated curved plates where the sutures are incapable of supporting bending loads during the early development of the child. Upon impact this structure undergoes large elastic deformation and, in some cases, even plastic deformation, both of which may result in skull fractures of various types. The shape changes of the skull associated with impact to this unique and developing skull of infants produce large strains throughout the cranium and its contents even without the onset of skull fracture and may be subjected to even greater shape changes in skull and brain when fractures do occur. It is this aspect of the mechanism of infant brain injury that needs to be further clarified.

In contrast, impulsive loading (shaking) will produce minimal if any, deformation of the skull. The biomechanical aspects of the injury mechanisms, the definition of the mechanics terminology, and direct comparison to our knowledge base in adult and pediatric head injury is intended to shed light upon the controversial and confounding interpretations of the literature in this field.[20,30] It is therefore appropriate at this point to discuss the pertinent concepts of the science of mechanics associated with TBI. These include the biomechanical behaviour of tissues undergoing dynamic loading, kinematics, head motions and, in particular, the fundamentals of dynamics of head injury and tolerance criteria.[1,23-25,32,96,126]

Biomechanical behaviour of tissues upon load application

The response of any solid structure to specified loading depends critically on its composition, geometry and, in particular, on deformation characteristics. An element may be subjected to uni-axial (one-dimensional) load or forces acting along a given axes. If several forces act along different axes, an equivalent stress or strain can be computed. Different types of stress or corresponding strain occur depending on how the load is applied. Various modes include: (a) elongation, (b) compression, (c) bending, (d) shear, and (e) torsion (twisting). This terminology is explained and illustrated in the appendix.

The brain is not a solid component, but rather a complex rheological substance, which is permeated by vascular structures and is in contact with cerebro-spinal fluid. This complex exhibits many aspects of fluid behaviour. The brain can be represented at the microscopic (axonal or neuron) level, but this would enormously complicate or possibly inhibit any analysis of regional response to loading. Due to the vast number of neural elements and current lack of knowledge of their interaction in spite of substantial local in homogeneities and anisotropies, the brain has been approximated as a uniform component whose principal properties consist of its density and viscous characteristics, subject to its extensive and pervasive vasculature. However, recent studies have identified differences in properties of various regions of the brain, e.g. gray and white matter, cortex and brain stem tissues. The stability and shape of the living brain *in vivo* is maintained by the cerebral circulation which enables the blood vessels to function as a 'soft' internal and external scaffold for the brain. The blood vessels can be represented as a series of distributed concentric tubes, each with somewhat different properties, whose overall behaviour is quasi-visco-elastic, or more simply as a single homogeneous vessel for each artery or vein. The prenatal or infant skull can be regarded as a series of nearly elastic/brittle, interlocking plates capable of resisting compression and shear, but incapable of transmitting bending across the sutures or fontanels. The adult skull, consisting of two solid bone layers sandwiching the diploë is a complex structure that is somewhat flexible; sufficiently large forced deformations of the unit frequently result in fracture at tensile strains of approximately 2%, including crushing of the diploë when radial loads are exerted. Figure 1 depicts average values of skull failure stresses in neonates, young children and adults; the resistance to fracture for an adult is 11 times greater than for the neonate.

Dynamics of head injury

The two separate types of *motion* of the head that can occur as the result of application of a force to the system are: (a) translation, where the total applied force F passes through the center of gravity of the head (exemplified by Fig. B of the Appendix) and (b) rotation, produced by the moment of a force

about the centre of gravity (or else by a 'couple', two equal, parallel and oppositely directed forces whose lines of action are distinct). An example of rotation is shown in Fig. 2. In the real world, both translation and rotation are invariably simultaneously present in varying degrees in head injuries.

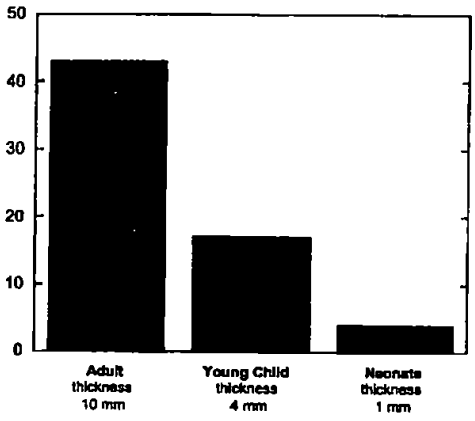

FIG. 1. Skull failure stress in the adult, young child and neonate.

Owing to the uniformity of the kinematic parameters in pure translation throughout a rigid body, which an adult skull approximates in the absence of fracture due to impact, no strain is generated in this object and, hence, no failure will occur. Uniform acceleration for a fluid, on the other hand, generates a pressure gradient in the direction of motion proportional to the acceleration that, in extreme cases, could produce trauma in the brain. Rotation produces differential displacements of adjacent spherical brain layers due to the outwardly increasing translational velocity with respect to the axis of rotation, which is most often located either at the occipital condyles or at the base of the neck. This results in shearing of the tissue, the cause of diffuse axonal injury and various forms of vascular disruptions. The relative motion of the brain with respect to the skull is also responsible for any rupture of vessels such as the bridging veins at the skull/brain interface, which is the most likely cause of subdural haematomas.[58]

Two types of force application, static and dynamic can generate damage to the skull and its contents, that produce different injuries. Static loading, although very rare, occurs slowly as exemplified by the closure of an elevator door in which a head has been imprisoned and subsequent

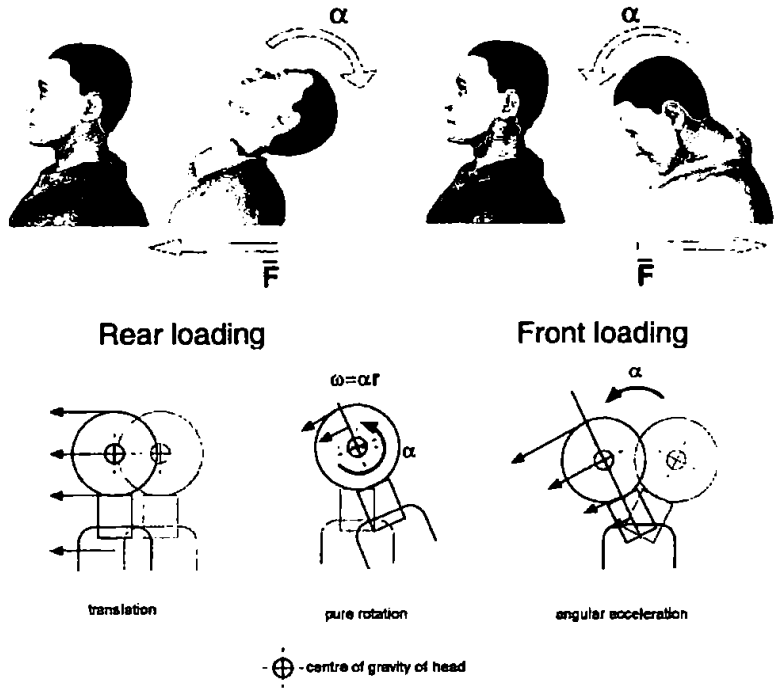

FIG. 2. Impulse loading of head/neck in rear and front loading depicting translation, pure rotation and angular acceleration.

further compression of the cranium. However, by far the two most frequent types of dynamic force application are represented by impulsive loading and by impact loading, two completely different mechanisms with totally different consequences. The first consists of movement of system elements induced by applied motion at an entirely different position of the system (or else by a fluid pressure wave, that is not relevant here). It is exemplified by a vehicular occupant whose car has sustained an impact or in tests involving high speed sleds that are suddenly arrested; the load is applied at the seat, and transmitted through the torso and neck to the head.

Continued impulsive forced motion reversal, such as would occur in the 'shaken baby syndrome' (SBS), also constitutes impulsive action where traumatic effects to the head and neck are generated by the inertial loads. The resulting differential motion of the brain and the skull is amplified by the behaviour of the neck, which is weak in infants and much stronger in adults. The resultant significant relative displacement of the brain, skull, spinal cord and neck results in failure of the para-saggital bridging veins, contusion and haemorrhages of the brain upon striking portions of the skull, and diffuse axonal injuries if significant rotation of the head is involved; most importantly, severe shaking will produce damage to the soft tissues of the neck and its contents. Although a single shake may not induce any noticeable trauma, it is possible that continued repetitive shaking over a period of time, or shaking repeated at intervals can produce significant cerebral as well as cervical spinal cord trauma in an infant.[10,34,86] Repetitive shaking may also, in some cases display additional stigmata, e.g. bruises to the extremities and body with fractures of the skeletal structures.

Impact, on the other hand, requires the rapid contact of two solid objects, such as a skull and a rigid surface (for example, a table or the floor). This is illustrated for a fall and for a blow by a blunt object on the head in Fig 3. A similar situation prevails if the head, in line with the body, were to strike a rigid surface at an angle to its sagittal plane at speeds of the order of 2–5 m/s. The duration of such contact is very short; in collisions of unbelted vehicular occupants, the contact time with the car component struck was found to be about 3/1000 s or 3 ms. Independent tests by

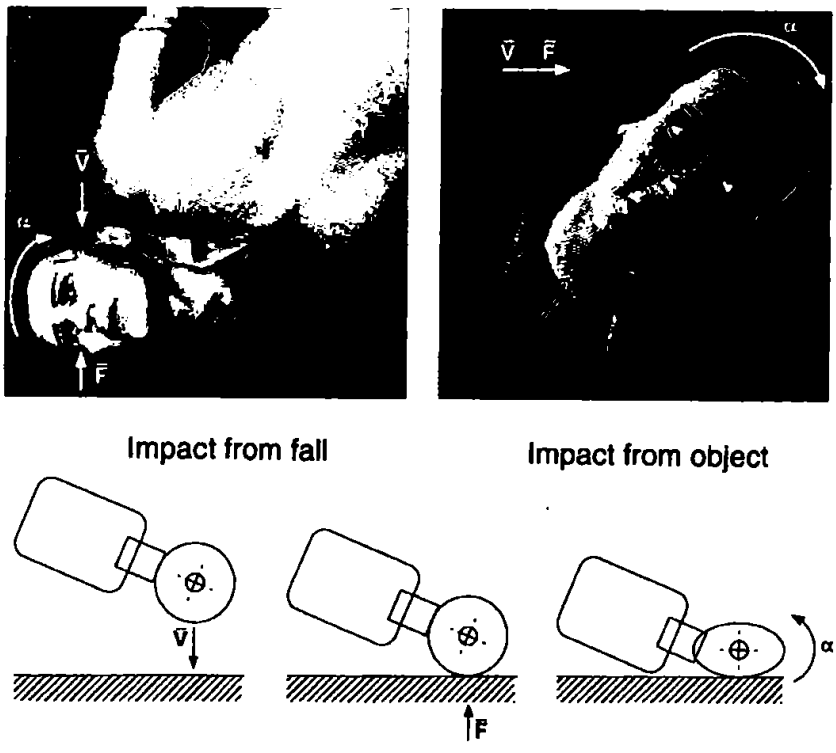

FIG. 3. Impact loading head/neck depicting impact from fall and impact from object.

Goldsmith involving the drop of an adult cadaver skull covered by a replicated scalp and filled with a brain stimulant on a wooden edge at low velocities without skull fracture indicated a contact duration ranging from 3 to 7 ms; a higher impact velocity under these conditions would reduce the contact time slightly. The degree of potential trauma is often gauged by the change in linear and angular velocity produced by the impact, although this does not relate, at least directly, to the stresses generated that are true indices of damage. Gurdjian indicated a limit of 4480 N (1000 lb) as the maximum load that can be applied to an adult head without extreme danger of serious or fatal trauma.[32] We have learned since that this is a reasonable approximation for skull fracture in adults and as an index of brain injury without additional description of the loading conditions.

The two consequences of an impact, not present in impulsive loading, are: (a) some type of contact phenomenon, such as indentation, large local deformations, a wide variety of fractures, bruising, etc., and (b) the transmission of loading effects to distal parts of the system, labelled 'wave propagation' whose manifestations often produce damaging effects at locations removed from the contact point, as exemplified by the contre-coup phenomenon. This characteristic includes the generation and release of skull deformations at positions remote from the impact location.[1,23,32] The specific structure of the interior of the skull plays a significant role in the localization of the coup and contre-coup contusion with and without skull fracture. This has been addressed experimentally in the rhesus model.[88,90]

The application and consequences of impulsive and impact loading are totally different, and they should not be linked in a single appellation, such as a 'shaken baby/impact syndrome' or 'shaken/impact syndrome' unless evidence for both types of causations are present simultaneously. The use of this dualistic term is a misinterpretation of the Duhaime, Gennarelli, Thibault study because the surrogate 'baby' was first shaken (repeatedly) and did not register loads sufficient to produce injury to the brain. Next, the head of the surrogate was impacted and exceeded the known injury criteria.[21] The levels of impact loading are from 50 to 100 times greater than the result produced by shaking alone. The impact aspect should be clearly demonstrated by a visible and/or palpable manifestation of the head injury unless the applied force is distributed by a load distributing padding, in which case impact effects may or may not be mitigated depending on the thickness and structure of the pad. Impact, including those due to falls from heights of no greater than 3–4 ft, produce forces an order of magnitude larger than can be generated by impulsive action. A possible *cumulative* effect of pure repetitive impulsive loading has not been tested in suitable animal models similar to those experiments carried out for impulsive loading analysis.[90,95]

To demonstrate this point, consider the fall of a toddler through a distance of 0.91 m (3 ft), perhaps representative of his height, by rotation about the bottom of his feet. This results in an impact velocity of 4.24 m/s (13.9 ft/s); a fall of 1.82 m (6 ft) down a set of steps produces a contact speed of 6 m/s (19.7 ft/s). These may be characterized by some as low velocity impacts, but the consequences are potentially highly traumatic: for an extremely conservative value of the contact duration of 0.01 s (10 ms), the peak force generated will be 5970 N for an adult with a head mass of 5 kg falling 1.82 m and 2280 N for a child with a head mass of 2.7 kg falling 0.9 m, respectively. This range of impact velocity can also be considered to be the reasonable levels of maximum impact produced when an infant is manually thrown on to a surface. On rigid surfaces, speeds falls from such heights produce forces that exceed skull fracture tolerance in children and adults.

By contrast, consider the case of shaken baby syndrome (SBS) where a brain mass of 800 g is involved and shaking occurs at the rate of 3 Hz with a total displacement of the infant of 0.6 m (2 ft); this is close to the maximum physically feasible conditions. This pendular motion produces a maximum acceleration of 11 g; use of where is Eq (1) eqn 1, which then provides a maximum corresponding force of 86.3 N (19.3 lb); this can be converted to an angular acceleration of about 1000 rad/s^2. All values are well below thresholds for cerebral concussion, SDH, subarachnoid haemorrhage, deep brain haemorrhages and cortical contusions.[21] Hence, the fall generates a load about 10 times greater than can be achieved by SBS and in substance can clearly produce these injuries. Thus, while it is possible to produce trauma in an infant by shaking, e.g. a SDH, particularly when shaking is prolonged and repeated at intervals, the injuries would include the cervical cord and spine, but not the brain case, nor contusions in the cerebrum or cerebellum if no impact was also imposed. It is far more likely that impacts due to falls and other causes are more probable in producing TBI by short duration impulsive loading.

Mechanisms of head injury in the adult and child

In a report by McClelland *et al*, the following statement is significant: 'Most severe TBI in children under one year of age is the result of child abuse. Some 10% of all traumatic injuries under five years of age will have a non-accidental cause. Head injury is the leading cause of death in child abuse and of

the 5% of abused children who die, the mortality of the cranial component approaches 30%.'[66] These data also emphasize the non-cranial injuries present in such cases. Understanding the fundamental biomechanics of paediatric TBI compared with adult TBI provides an insight that serves to resolve current controversies, particularly for the diagnosis of intentional versus non-international TBI. Our review of historical and current reports on paediatric TBI has also identified five assumptions in the literature concerning the mechanisms of retinal and brain injuries. A critique of these ideas will precede the review of the basic biomechanics and differential data in adults and children. These assumptions are listed below.

Intentional TBI

Child abuse is an increasing problem; between ages 0–4, 2.6 per 100 children are abused and these crimes cost an estimated 22.8 billion US dollars yearly.[74] The lack of a clear history not fitting the clinical findings is usually the starting point to analyse the available data and fit the most probable mechanism to the injuries. C. J. Hobbs has provided an important caveat stated as follows: 'The diagnosis of abuse demands absolute certainty to avoid conviction of innocent parents and unwarranted removal of children from their homes. The evidence must satisfy both the needs of clinical diagnosis and the process of law. The two most important corroborative facts in the diagnosis of abuse were the presence of multiple injuries and an inadequate explanation for the injuries.'[38] With due Hobbsian caution therefore we cite a set of assumptions in the literature concerning the mechanisms of TBI in paediatric patients that require validation.

1. That infantile head injury mechanisms, in some undefined mathematical manner, could be scaled from animal and adult injury tolerance data, based upon inertial impulsive loading, in the absence of impact.[10,33]
2. That 'low' falls in infants, e.g. less than 4 ft, are not likely to cause skull fractures, acute subdural haematoma (ASDH) and brain injury.[18–20]
3. That traumatic retinoschisis in abused infants is caused directly by repetitive shaking of the head causing brain motions, which directly produce disruption of bridging veins and ASDH. It is claimed that *similar shaking forces act on the eyes* causing the relatively dense lens to move forward and back within the ocular fluids producing retinal haemorrhages by 'abrupt tugging on the retina'.[30,118]
4. That the syndrome of 'head injured patients who talk and deteriorate or die (the TADD syndrome)' is not caused by an acute and relatively asymptomatic SDH with minimal initial sequelae and a lucid interval which can progress to late sequelae such as brain damage (the 'big black brain') or to re-bleed in the sub-acute or chronic stage following what retrospectively is found to be a trivial trauma[16–18,20,40] (personal communication from Dr Duhaime).
5. That the time interval between onset of the cause for intentional TBI injury resulting in a 'shaken baby syndrome (SBS)' is always of short duration, i.e. the time for onset of symptoms and signs of severe TBI is always brief and in the absence of a good history, timing of the injury must be extrapolated from data on accidental trauma.[20,76]

These assumptions individually and in concert are ambiguous or incorrect although they have been used as the bases for differential diagnosis of the mechanisms of paediatric head injuries usually without reference to available biomechanical analysis using reconstruction methods.

In order to discuss the mechanisms of injury to the skull, its coverings and contents there are at least two concepts that must be understood; first, we need to know the behaviour of these materials when quasi-static or dynamic forces are applied and, secondly, we must understand how external forces applied to the system translate into the stresses and strains that will challenge the functional and/or structural integrity of the components of the system. To that end the following concepts are presented:

1. To cause scalp contusion or laceration the stress must exceed the ultimate stress for the capillary network or the composite materials that constitute the matrix and vascular elements of the skin.[1,23,24,67,126]
2. To fracture the skull the ultimate strength of the bone must be exceeded.[23,31,32,71,103,114,122–126]
3. To produce subarachnoid, subdural and deep intra-cerebral haemorrhages the vascular elements at various topographic locations must be stretched beyond their ultimate strength.[1,58,116,120,126]
4. To cause an acute subdural haematoma the parasagittal bridging veins must either stretch beyond their ultimate strength in tension or fail in shear at the cortical surface or at their insertions into the venous sinuses.[58] Bridging veins can stretch to 30–35% before they tear and bleed. However, pre-loading of such veins at a lower stretch level may result in increased stiffness producing venospasm of varying degrees. This state can result in vein rupture with less force. This could explain why TADD cases with ASDH are often described as having only a 'trivial trauma'.[84,93,105,106]
5. To cause cerebral concussion the stresses must reach into the brain stem to produce loss of consciousness; lesser levels of stress will

produce amnesia without loss of consciousness but with a period of amnesic automatic behaviour.[80,83,85,87,93,94]

6. To produce moderate to severe diffuse axonal injury the axons must be stretched beyond their physiological limit or to the point of structural failure. This is most prevalent in the deep white matter such as the corpus callosum, internal capsule, rostral brain stem and basal ganglia. The basic paradigm is that external forces acting upon the head, a complex system of composite materials, will produce stresses and strains that are dictated by the nature, direction and magnitude of the applied loads and the properties of the structure. The overall distribution of stresses and strains resulting in acute lesions imaged by MRI has been documented and fits the centripetal theory discussed earlier.[26,55-57,83,87,95,112]

Two distinctly different biomechanical systems exist when one views the skull-brain structure of the adult and that of the developing child. The variants in the child, when compared with the average adult, are the constitutive properties of the materials, the geometry of the structures, the age dependent physiological response to mechanical stress and the structural properties of the head. Numerous investigations of the biomechanics of the brain injury in the adult have been conducted. Extensive bibliographical information regarding these studies has been published of which the following references are only representative. Specific injury mechanisms and tolerance criteria for scalp contusion and laceration, skull fracture, diffuse axonal injury, acute subdural haematoma, and deep intracerebral haemorrhage have been advanced.[23,25,26,32,48,58,64,65,83,84,93,94,96,103,114,116,122-124]

At this point it would be useful to discuss the role of impulsive and impact loading in their association with the various forms of head injury. An impulsive force, resulting in acceleration or deceleration of the head without head contact, is typically applied through the torso and the neck structure, thereby causing the head to be accelerated and decelerated. An example of such a situation would be a restrained occupant of a vehicle that experienced a collision. If the vehicle was stationary and struck from the rear the torso would move with the seat and frame of the vehicle, the head would lag behind and initially rotate backwards due to its inertia and would eventually follow the torso as the neck loads began to produce the necessary forces to accelerate the head. If the vehicle was in motion and impacted an object the torso would be decelerated by the restraint and the head would continue to move forward until arrested by the forces exerted by the neck. Under these conditions there would be no injury to the scalp or skull. This event is similar to the process described as shaking a baby, where the force is applied through the torso, the neck couples the torso and the head and acceleration and deceleration of the head results. The first structure to experience injury under these conditions is the neck; this is commonly known as whiplash. However, under these conditions the brain is also free to move within the cranial vault and this distortion of its shape can produce stresses within the brain that lead to injury as a function of the magnitude and direction of the induced rotational acceleration, as well as the repetitive nature of this type of whiplash that may or may not be combined with an impact.

Figure 4 compares the magnitude of the rotational acceleration or deceleration necessary to produce the level of shear stress within the brain to cause varying degrees of axonal injury. The figure also demonstrates the theoretical scaling relationship advanced by Holbourn which relates the stress to the mass of the brain and which was experimentally validated in animal models and extended to the human.[37,39,50,64,65,71,80,81,84,90,96,114] For the adult brain the rotational acceleration required to produce concussion is approximately 4500 radians/s squared and for the infant this value approaches 10,000 radians/s squared. Severe DAI in the adult brain is achieved at rotational accelerations at about 18,000 rad/s, whereas, the neonate tolerance is breached at just below 40,000 rad/s^2. These data illustrate the inverse 2/3 brain mass rule as explained earlier. Similar arguments are made for acute subdural haematoma and deep intracerebral haemorrhage, but the magnitude of the acceleration for these haemorrhagic lesions is significantly greater than that for mild diffuse axonal injury. At these levels of inertial loading, induced impulsively without contact, the neck torque in the infant would cause severe injury to the high cervical cord and spine long before the onset of cerebral

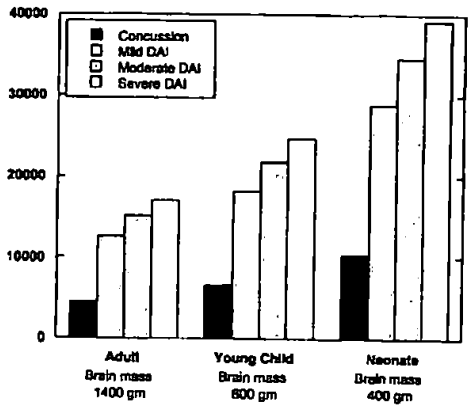

FIG. 4. Brain injury tolerance scaling in the adult, young child and neonate.

concussion.[25,51] Absence of cervical spinal cord injury would indicate a component of impact in the presence of haemorrhagic brain lesions.

In contrast to impulsive loading, impact contains the elements of inertial loading together with the contact events. Similarly, examples include the head contacting the ground as the result of a fall where the scalp compresses, the skull bends inward, stress waves propagate around the surface of the skull and through the brain material (Fig. 3 — impact from fall). These events occur with a rise time constant of the order of 100 microseconds and before the head has accelerated or decelerated to its maximum value; each event can produce failure of the materials that constitute the head structure. As the scalp is compressed by the impact, the stresses which are related to the magnitude of the force or the impact velocity, the direction of the blow, and the contact area and properties of the impacting surface all play a significant role. (Fig. 4 — impact from object) Following the contact phase the head is accelerated or decelerated giving rise to differential motion of the skull and brain. During the impact associated with a fall the skull will stop and the brain will continue to move within the cranium. This relative motion can lead to stresses which associate with the kinematical conditions. In pure translation tissue experiences compression and tension while in the angular deceleration mode the shear stresses can be very high compared with the failure criteria. The brain is a virtually incompressible material (bulk modulus = 300,000 pounds per square inch); in contrast the shear modulus is of the order of 10 pounds per square inch.[81] Therefore, velocity gradients within the brain will result in shear stresses, which can exceed failure criteria for DAI, ASDH, ASH and deep intracerebral haemorrhage.

The contact loads can produce focal injury in the absence of inertial effects. For example, skull fracture will result when the ultimate strength of the bone is exceeded. Figure 1 demonstrates the stresses necessary to fracture the skull of the adult, young child and the infant. The requisite forces are reduced as a function of the skull thickness and the moduli of the bone. Weber[122–124] provided experimental confirmation of the presence of skull fracture in each of 15 (average age 8.2 months) perfused infant cadavers. Three groups of five cadavers were dropped on stone or tile, carpeted and linoleum covered floor. The fall heights were consistently 82 cm with the cadaver in a horizontal position with head strike at the parieto-occipital zone. This produced an impact velocity of approximately 4 m/s (13 ft/s). Fractures were found in every case.

Weber also investigated whether a skull fracture could occur in unobserved falls and not be recognized by lay care takers. On the basis of his forensic pathology expertise in fatal cases and his review of clinical reports of falls from parent's arms, tables, strollers, beds and sofas, Weber reports that linear skull fracture does occur in low falls without specific symptoms. He concluded that such asymptomatic linear skull fractures can occur and not be recognized by lay caretakers, as well as by professional staff. It should be noted that prior to Weber's work, no systematic study of low level falls and fracture mechanism in infant cadavers had been carried out. Weber also emphasizes that his experimental results should not be used to exclude wrong doing by parents and guardians. His results only demonstrate the possibility that such an injury (specifically a linear fracture) from low heights are possible. Weber states, 'In all these cases one has to consider the whole situation. The so far suspected vulnerability of the infants' skull when falls occur from low heights have been confirmed as being possible by our experimental results.' In a second paper Weber also reports that only *one* out of 10 cadavers dropped on 2 cm thick rubber foam covered floor and four out of 25 drops on folded (8 cm thickness) camel hair blanket which produced skull fractures. The bone thickness in these cadavers was 0.1–0.4 mm adjacent to the fracture fissures.[123] Weber's third report was an attempt to predict the risk of skull fracture in infants (neonates to 14-month-old infants) using diaphanoscopy to locate skull transparency indicating loci of paper thin vulnerable zones in such skulls.[124]

Impact loading, as previously described, can result in stresses and associated deformations and strains that exceed adult human tolerance levels, as well as those in the child, but the weaker infant and child skulls fail at much lower levels as shown in Fig. 2 This failure is preceded by deformation of the brain with or without skull fractures. Impulsive loading on the other hand, resulting in purely inertial forces without contact, are also implicated in several important forms of brain injury in the adult. In contrast, the level of the forces necessary to produce equivalent stresses in the child are far in excess of those that can be reasonably achieved without significant injury to the cervical spinal cord and neck structures. Repetitive and prolonged episodes of shaking of the infants, as well as the adult head/neck system may produce cumulative stresses in the brain resulting in subdural haematomas and abnormally elevated intracerebral pressure. This hypothesis has not been adequately tested in animal models for TBI mechanisms as noted above. Figure 4 depicts brain injury tolerance in adults, young children and neonates.

Clinical data on paediatric TBI

The biomechanical findings described above are well validated by clinical data. One of the most extensive

studies on paediatric head trauma was reported by Berney *et al.* in 1994.[8] This was based on a consecutive, unselected series of 1812 cases of TBI in children less than 15 years old. Three categories were established by age: 0–3, 3–9 and 9–15 years. Their main conclusion was that 184 children in the 0–3-year-old category sustained low energy trauma, suffering more skull fractures, more subdural haematomas and early seizures as compared with the other two groups. The low energy level was described as falling off toboggans and equivalent low level falls, i.e. less than 3–4 ft. Children between 3–9 had rather higher energy incidents, were more often in coma and did not have subdural haematomas, but did have significant brain swelling. Children between 9 and 15 were more like young adults with more severe TBI than the previous two categories. They suffered more with extradural haematomas as compared with subdural bleeding. One of the authors' conclusions was 'the smaller the child, the lower the energy involved in the injury'.[8] These clinical data on impact injury mechanisms indicate the role of the specific biomechanical properties of baby skulls, which result in significant deformation of the skull and brain shape enabling the injuries to occur at lower levels of impact to the head and therefore the brain. All of the three age categories of children included cases of 'talk and die or deteriorate' (TADD) syndromes for each grade of energy. There were five times more TADD cases with skull fracture as compared with the non-skull fracture cases and they constituted 25% of all the ASDH cases, most of which were in the 0–3-year-old category.[8]

In 1993, Rivara *et al.* published a population based study of fall related injuries in children and adolescents resulting in hospitalization or death based on all hospital discharges for 1989 and 1990. The most common type of fall resulting in hospitalization was a relatively low fall from one level to another, e.g. from beds, tables, chairs and playground equipment (37.5% of all falls). Head injuries associated with 'skull fractures, concussion and intracerebral haemorrhages occurred to more than one-fifth of the children. This was especially common among pre-school age children in whom 42.1% had head trauma compared with only 14.4% of adolescent aged 15 through 19 years.[100]

A prospective study by Luersen *et al.* in 1988 reviewed the outcome after TBI related to age (paediatric and adult). The authors had 8814 patients of which 1906 were children younger than 14. In infants less than 2 years old, 65% had non-fatal TBI caused by falls, 20% by motor vehicle crashes and 5% by assaults. Fatal TBI at this age were caused in 20% by assault, 40% by motor vehicle crashes and 40% by falls.[59]

A retrospective study by Howard *et al.* in 1993 reviewed 100 infants who suffered TBI of which 28 also had subdural haematomas. Abuse was initially suspected in 14 of the 28 cases and investigations led to legal actions in 10 of the 14 cases. An unusual finding in this report was an apparent increased susceptibility to the development of subdural haematomas in non-Caucasian infants. Of the 28 TBI and subdural haematomas, 17 were Caucasians and 10 were non-Caucasians with one mixed-race infant. Given the overwhelmingly Caucasian population base, the number of non-Caucasian ASDH injuries was significant at 62 versus 21% for the Caucasian group ($p < 0.01$). All of the 28 cases with ASDH had well described history and evidence of head impact. Retinal haemorrhages were present in 20% of these infants. The non-Caucasian infants developed ASDH after trivial falls and with normal scalp examination as compared with Caucasian infants. The conclusion of this study was that intentional injuries were less common than unintentional TBI and shaking as the only cause of SDH was not supported.[40] The unusual vulnerability noted in non-Caucasian head injured children was also related to other published observations in Japan. Falls from sitting or standing positions were described to be the cause of ASDH and retinal haemorrhages in 26 Japanese infants as reported by Aoki & Masuzawa.[2] All were infants with acute SDH apparently due to minor head trauma without LOC and no contusions of the brain. Presentation was mostly by seizures following other minor TBI. Average age was 5.1 months and 23 were boys. All had retinal and preretinal haemorrhages. Two children died, mild retardation was found in one and epilepsy in one with the remaining 23 showing normal development. Early surgical treatment to remove the SDH clots was standard and in another case (not included in this series) the SDH was not removed because it was considered as trivial. Forty days later severe damage with multicystic encephalomalacia was confirmed.

Greenes & Schutz have recently reported a retrospective review of 101 infants less than 2 years old who were admitted for suspected TBI to a tertiary care paediatric hospital over the course of 6½ years. Nineteen of these infants, all less than 1 year old, were asymptomatic but harbouring 'occult' intracranial injuries. They had no loss of consciousness, no mental or behavioural changes, no seizures, irritability or vomiting, without retinal haemorrhages or a bulging fontanelle, and with a normal neurological examination. No history of trauma was given or witnessed for two infants. Ten suffered falls ranging from 2 to 5 feet, four fell down five to nine stair steps, one suffered from a motor vehicle crash and one from child abuse. All were alert and five were playful. Subdural haematomas were described as 'small' in seven with mild shift of midline and maximal 1-cm width in one of these. Six had epidural haematomas with

1 cm width and shift in two cases. One had both subdural and epidural haematomas. Eighteen of this group had scalp contusion or haematomas and 18 had skull fractures. No intervention other than anticonvulsants were provided in nine infants. All infants experienced no serious neurological deterioration except one subject had a brief self-limited seizure. The finding of 18 cases out of 19 harbouring 'occult' intracranial injuries also had skull fractures supports the experimental data of Weber.[123]

The authors of the report state that none of these infants required subsequent surgery or a significant degree of medical management. Specifically, they noted that 'the clinical significance of occult TBI in these patients is not entirely clear'. They also note that 'our confidence intervals are consistent with a rate of late clinical complications in these patients that may be as great as 28%'. They also realized that some TBI patients were not admitted and discharged from the ER without a diagnosis and no follow-up. A prospective study of all infants with TBI entering the ER with a uniform protocol would provide improved data to decide which infants required brain imaging and skull radiograph and a standard follow-up letter to the parents after discharge may uncover late complications such as development of acute re-bleeding in an occult subdural haematoma in a case of the TADD syndrome. What is unequivocal in this report is the conclusion that 'physicians cannot depend on the absence of clinical signs of brain injury to exclude intracranial injury in infants younger than one year of age.'[29]

An extensive review on the epidemiology and a etiology of infant TBI by De Rocco & Velardi is found in the text book titled: 'Head Injuries in the Infant and Newborn' by Raimondi, Choux & Di Rocco.[15,98] Data on two large series totalling 13,452 infants in the first 2 years of life were analysed. Most of the children who suffered head injuries were at home or in familiar surroundings. Falls were the dominant mechanism and occurred from cots, cribs, prams, high chairs and down steps. Abuse, motor vehicle injuries and falls from heights were the other mechanisms. Compared with non-intentional aetiology, intentional TBI was relatively rare in this age group.[15] In a second series by Canestri & Monzali of 5363 children up to 3 years of age, 3% were found to have suffered TBI. The majority of skull fractures (and evidence for increased intracranial pressure) occurred in infants between 9 and 17 months with the highest peak at 9 months. Falls were found to be a major cause of TBI and were grouped in two categories: falls from cots, cribs, prams, high chairs, often due to insufficient supervision, and falls against or from furniture and down steps (related to independent ambulation). In most cases the fall was from a height of about 90 cm or less (about 35 inches). Most of these falls did not result in observable injuries.[11] Infant 'Walkers' are known to be hazardous; this device caused TBI and skull fractures in 47 of 150 infants (10.6%) as described by Kavanaugh & Banco.[45]

Child abuse

Di Rocco & Velardi in their report mentioned above also reviewed intentional TBI in the context of all causes, confirming the main incidence of abuse in children under 2 years of age. They compared US data from New York and Illinois Central Registration and published data from five other US sources for a total of 705 cases along with 1719 cases observed at the Hôpital de la Timone, Marseille, France. The US data revealed a peak incidence between 9 and 12 months of age. The French data compared the relative incidence of four common aetiologies of infant and child TBI, i.e. falls, road accidents, abuse and other causes, for five categories of age. Between 0 and 2 months of age, falls were the cause in 79% of this group. Between the ages of 2 and 6 months road accidents contributed 8% of causation. From 6 to 12 months, child abuse accounted for 2% of the 1719 cohort. From 12 to 24 months, other non-specified causes were noted in 11% of the cases.[15] These data are generally in agreement with the data shown in Table I and re-analysed by Thurman from the National Center for Injury Prevention and Control (NCIPC) of the CDC.[118] The availability of CT and MRI has significantly improved the accuracy of diagnosis in TBI of all types but the biomechanical aspects of causation are not fully integrated into the nosologic skills of medical practitioners. As demonstrated by Weber, if a fall or blow to the head in infants is cushioned by a load distributing material, a range of brain lesions, including subdural and subarachnoid bleeding, intraparenchymal haemorrhages, parenchymal swelling and brain lacerations can occur without apparent external evidence of head injury. This result is due to the infant's easily deformed skull causing deformation and strains in the brain, and its vascular components as discussed in the section on biomechanics. Recent reviews and reports of intentional TBI and the outcome have provided important data on clinical and radiological information of this tragic source of brain injuries and fatalities.[5,15-19,45,74] Two aspects, however, have not been fully clarified; the mechanism of rebleeding in subdural haematomas and the biomechanics of retinal haemorrhages.

Rebleeding in the subdural haematoma (SDH)

James Barkovick in his book *Pediatric Neuro-imaging* (1995) provides a definitive statement on paediatric and adult neuroradiology. He states that the radio-

logical images (CT and MRI) of SDH in infants and children in all its states are the same as seen in adult cases with SDH, both in its acute and chronic states.[5] Three important consequences follow. First, there are no special neuro-imaging phenomena of SDH unique to the patient's age, e.g. in the density changes over time, clot retractions, onset of rebleeding in subacute and chronic SDH, mixing with CSF, and the phenomena of 'disappearing' SDH, which has been described in paediatric cases by Duhaime.[18]

A quantitative study by Mats Bergstrom *et al.* from the Karolinska Hospital on the phenomenology of rebleeding in 41 cases of SDH and nine cases of extradural haematomas (EDH) sheds useful light upon what is a somewhat obscure syndrome when autopsy data are not available.[7] Serial CT scans of the brain were analysed to plot the Hounsfield Units of each scan versus the time lapse from the time of the trauma to 4 weeks post-trauma.[84,93] Bergstrom *et al.*'s histories of these patients did not provide any evidence of 'new traumas in association with onset of symptoms. Spontaneous rebleeding may well explain the onset of symptoms as well as the attenuation values being so much higher than those of CSF and serum. It is unlikely that a sudden onset of symptoms a long time after trauma would be explained by slow continuous enlargement of a hematomas.' Finally, this report also indicates that a rapidly expanding rebleed within a relatively small sub-acute or chronic SDH caused by low level trauma with no serious neurological disturbances *ab initio* could be the most probable explanation for the sudden onset of more serious signs and symptoms requiring surgery due to a re-bleed in the SDH of a patient who was not initially a candidate for surgery at the first onset.[7]

Confirming the age of the SDH and its rebleeding potential

Apart from the standard requirement to save SDH clots for pathologic reports, recent quantitative investigations have emphasized the importance of saving the SDH during surgical removal of the clot in order to correlate the concentration of fibrinogen and fibrin monomer with the preoperative CT findings. Nomura *et al.* have classified 41 SDH cases into five categories based on the preoperative CT scans and correlated these images with the concentration of fibrinogen, which indicates rebleeding and fibrin monomer, which indicates coagulation activity using sodium dodecyl sulphate-polyacrylamide gel electrophoresis and immunoblotting. The five categories of CT images were as follows: *Layering type* SDH hyperfibrinolytically active with a highest tendency to rebleed. The *mixed density* type has also a high tendency to rebleed, but with lower hyperfibrinolytic activity. The *lower density* and *isodense* SDH had very low tendency to rebleed and low fibrinolytic activity. High density SDH are usually acute and subacute cases. In suspected child abuse and fatal cases, detailed histological studies of the clot and adjacent dura are obviously essential to determine the age of the haematoma. In SDH survivors, the clot is also an important component of the definitive diagnosis of causation when correlated with the CT findings.[77]

Hyperacute subdural haematomas

These very acute haematomas are mixed density subdural haematomas, which develop rapidly under two conditions that were first described by Greenberg *et al.* in 1985.[28] These conditions are two-fold as follows:

1. When the haemorrhage is an actively bleeding source, either from a number of bridging veins or, less commonly, from an arterial source.
2. When a coagulopathy is present. The differential diagnosis of this type of SDH by *CT findings alone* can mislead and indeed may suggest a rebleed in a subacute or chronic SDH. Surgical exploration or autopsy is necessary to prove the diagnosis of the hyper-acute nature of this SDH. A recent forensic report on a 15-month-old infant who died after intentional TBI initially presented with CT showing a mixed density SDH. This was interpreted as caused by rebleeding in an older SDH. Surgical exploration, however, discovered active bleeding and postmortem histological findings confirmed this to be a hyperacute SDH caused by child abuse. Confirmation of the nature of an SDH requires careful pathological examination of the haematoma, as well as a careful search for other stigmata, particularly soft tissue injury and fractures associated with the aetiology of intentional TBI. Soft tissue injuries have been found in 92% of 371 children who were abused.[104]

An important radiological clue for diagnosis of a rebleed occurs when a clear interface of an SDH is found separating an inner hyperdense and an outer hypodense SDH components on CT. In such a case there is good reason to assume that this appearance is the result of a rebleed in a subacute or chronic SDH. This was documented in a 1983 report in the *Journal of Neurosurgery* by Kao who reviewed 140 patients with 'chronic' SDH. Only seven patients presented with the CT findings of two components as described above. All but one of these seven cases had a definite history of head trauma more than 1 month before they developed acute disturbances and profound hemiparesis. This group was compared with seven chronic subdural haematoma patients who had only mild clinical signs and

symptoms. They were confined in bed for a period before CT and craniotomy. The CT scans in this group did not show the interface, and the layering of low and high density SDH as described in the first group. The parsimonious conclusion was that a rebleeding into a chronic SDH which was sufficiently old so as to be seen as homogeneous prior to the rebleed is real. The author suggests that a sufficient period of being in bed with the brow up before CT examination causes sedimentation in the SDH, which is seen in a small proportion of patients but may be specific for a significant amount of rebleeding to produce acute deterioration.[44]

Biomechanics of retinal haemorrhages

Although retinal haemorrhages are found in children and adults with severe CNS trauma, they are not pathognomonic for intentional TBI. The haemorrhage can be unilateral or bilateral, and may involve all the layers of the retina, as well as the choroid and vitreous. Deeper haemorrhages may be well circumscribed and have a dome shape giving rise to the appearance of retinoschisis.[27,30,31] Between 1964 and 1967 there were at least 13 publications concerning the hypotheses for the pathogenesis of retinal and optic sheath haemorrhages associated with intracranial bleeding.[75] The consensus of 10 out of the 13 concluded that the mechanism was either increased venous pressure, sudden rise of intracranial pressure (acting on orbital veins) or passage of blood through intracranial subarachnoid space with such force that intradural haemorrhages occur. The central retinal vein and its choroidal branches collapse due to the rise of pressure (Manschott). Walsh & Cogan also favoured this hypothesis.[12,121] The alternative hypothesis is *retinoschisis* caused by the whiplash effects on the head moving the brain back and forth, tearing veins and causing SDH, while the eyes similarly 'shake' within the orbits causing the dense lens to move back and forth, stressing the connections between the lens, vitreous gel and the retina, and especially the macular region resulting in retinoschisis.[30] Two recent publications are pertinent to the validity of the 'rapid rise of ICP' hypothesis. First, the human optic nerve sheath has sufficient elasticity to allow dilation in response to a rapid rise of intracranial pressure (ICP), particularly when the TBI is severe.[35] A second report on 'Head injury: cause of both brain edema and retinal hemorrhages' was presented at the 1995 meeting of the National Association of Medical Examiners. This study was a prospective, post mortem study of 169 child deaths. Brain oedema was found in 106 cases and 63 cases had no oedema. Retinal haemorrhages were found in 68 of the 106 cases with oedema and only in two cases in the 63 which had no oedema. TBI was the confounding variable, i.e. 61 of the 68 cases with retinal haemorrhages died from TBI with oedema as the cause of death. The conclusion was that TBI caused both the brain oedema and retinal haemorrhages and the increased ICP related to increased brain volume and therefore is most probably the cause of retinal haemorrhages.[22] Moreover, Dr Andrea Cibis Tongue in a critique of the Greenwald *et al.*'s retinoschisis hypothesis stated that it was 'unlikely that the basic lesion is one of retinoschisis rather than haemorrhage secondary to increased venous pressure.' This author also stated that the vascular system in infants may be more fragile and susceptible to abrupt pressure changes.[118] Green *et al.* have more recently also supported the hypothesis of intra-ocular mechanically-induced retinoschisis. Noting the direct relation of retinal haemorrhages to the presence of severe intracerebral and ocular injuries these authors conclude that increasing trauma severity 'invokes a mechanism similar to that causing subdural hematoma' by the effect of 'inertia/momentum of the vitreous body within the eye during shaking'. They further conclude that 'the mass of the vitreous (which forms a large proportion of the mass of the globe) acts upon the points at which it is attached during both acceleration, with up/down, left/right, axial and rotational movements, producing first small haemorrhages in the retina (intra-retinal and subhyaloid haemorrhage), then retinal detachment (directly, with haemorrhage having adaptive effect. Optic nerve sheath haemorrhage is also explained by this type of mechanics, i.e. 'the result of angular, rotational or axial movement about a point in the most anterior part of the optic nerve, posterior to the sclera'.[27]

The hypothesis of 'intra-ocular' retinal haemorrhages caused by orbital shaking has not been tested experimentally. The eye has a relatively very small mass compared to the brain. Biomechanical calculations based on Holbourn's inverse 2/3 rule, previously discussed, predict that, smaller masses require higher levels of force to cause damage and larger masses require less; the levels of force required for retinal bleeding by shaking to damage the eye directly is biomechanically improbable. The work of Hansen & Helmke also indicates that the role of sudden rise of ICP is more likely to cause bleeding than the 'shaken eye' hypothesis. Most probably the latter is not a testable hypothesis. By exclusion therefore, the sudden rise of ICP secondary to TBI is a valid (and testable) hypothesis.[12,35,75,118]

Principles of biomechanical reconstruction of human TBI

Application of existing biomechanical methods for in-depth reconstruction of adult and paediatric TBI are currently available and have been applied for at least two decades, primarily in the field of protection and prevention of humans in motor vehicle crashes and preventing injuries in hazardous environments,

such as playgrounds. The overwhelming bulk of this literature is published in governmental reports and journals in the fields of engineering and biomechanics. The best of these reports are developed by multidisciplinary teams including physicians, engineers and pathologists. Injuries to all parts of the body, including the head and neck are related to the causative forces utilizing the Trauma score, Abbreviated Injury Scale and the Glasgow Coma Score. For motor vehicle crashes, sources of injuries are correlated with the vehicle damage and crash circumstances. *Post hoc* reconstructions based on accident reports and medical records (even with inspection of the crash vehicles) are less informative than in-depth reconstructions closer to the time of the injury onset.

A clinical trauma nurse in the Trauma Unit and an injury reconstruction team are on standby which provide more accurate data. With the support of the National Highway Traffic Safety Administration and in collaboration with H. Champion we were able to develop for the first time an ongoing, in-depth re-construction programme for head and neck injuries. A trained nurse clinician notified the reconstruction team when the patient was admitted and the reconstruction team immediately proceeds to the crash site to gather the initial injury source data.

This strategy enables maximal data capture for correlation of the mechanics of the vehicle crash with the medical diagnoses and biomechanics of the injuries to the patient. Weekly rounds to review and merge the two data sets, biomechanical and clinical, provided the raw data base for reconstruction of the crash and the injury mechanism, as well as recording the outcome of the patient. Between 1982 and 1986, this methodology was implemented at the Shock Trauma Unit at the Washington Hospital Center. Two-hundred-and-forty-six cases were identified and 101 of these patients with head and/or neck injuries were completely studied including autopsy reports, X-ray and CT reports. Further analysis of cases in which the most complete crash and injury data sets were available, enabled physical and mathematical reconstructions, which were tested using anthropomorphic dummies seated in identical motor vehicles to reproduce the crash sequence at a sled test facility. The anatomically appropriate anthropomorphic dummies reproduced the quantitative data confirming the biomechanics of the specific injuries to the head and neck.[79] Extensive data of this type continues to be collected for injury prevention and protection purposes.[43,52,60–63,69,70,72] Biomechanical reconstruction of data in free falls have also been obtained based on the pioneering work of DeHaven and subsequently by the work of Cummins & Potter and of Snyder *et al.*[14,13,111] More recently, methods for TBI reconstruction have also became available.[3,41,60,70,101,108,119]

A research program featuring teams consisting of neurosurgeons, biomechanical engineers and adequate staff would be able to conduct in-depth reconstructions of all types of TBI injuries, including motor vehicle crashes, and falls to establish a more comprehensive data base of reconstructed TBI cases. Categories of severity will be based on a synthesis of the biochemical data, as well as the clinical, radiological and biomechanical data. In addition to CT, early stage MRI would provide more details of the acute TBI injury. This data set of biomechanically driven categorization of TBI cases, as well as the outcomes of these patients would serve as the template for predicting the vulnerability and outcome of subsequent patients of similar age, sex, mechanism and approximately equivalent levels of force under current TBI protocols. A recent report has been presented on the use of serum markers, e.g. neuron-specific enolase, creatine kinase BB, myelin basic protein and S-100 as biochemical indices for outcome predictions in TBI, as well as for stroke and subarachnoid bleeds. S-100 appears to be a promising marker in a study on 30 patients with TBI of high severity (GCS<9) and 11 patients with minor TBI (GCS > 12). Blood samples were taken 6 h after injury and after defining the outcome at discharge, which was measured by the GOS. All the minor TBI had favourable outcomes (mean S-100 + 0.35 mg/l) at discharge. The severe TBI cases fell into two groups; 31% had an unfavourable outcome with a mean S-100 level of 4.9 mg/l, while the remainder of 69% had a favourable outcome with a mean serum level of S-100 at 1.2 mg/l. All the groups differed significantly ($P < 0.05$). This work indicates a potential biochemical measure, which can be correlated with the biomechanical severity.[102]

A recent comprehensive review on TBI by Teasdale & Graham on the comparison of novel therapeutic agents for controlling the secondary progressive brain damage in animals and human TBI was a major stimulus for writing our report.[113] The authors comment on the paradoxical result of the novel experimental treatments in two categories of these subjects which were indeed puzzling. These new agents were proven to be very effective in the animal models of TBI, but failed to achieve similar benefits in TBI patients. The most probable explanation of this paradox may depend on the fact that there are at least two confounding variables, which are present in human cases of TBI. First, there is the relatively uniform population of the animal subjects compared with the relatively genetically variegated humans as suggested by Teasdale & Graham.[113] Secondly, the levels of force causing the TBI in the animal models is also uniform,

indicative of the controlling role of biomechanics causing the experimental TBI. Two uncontrolled variables exist in the human TBI research. Seeking to control secondary injuries in human TBI is significantly handicapted by biomechanically incomplete stratification whereas both of these variables are well controlled in the animal models of TBI. It follows therefore that establishing a research programme built upon a biomechanically reconstructed stratification of TBI patients receiving equivalent levels of mechanical forces, similar mechanisms, and with approximation for age, sex and other biological variables, could significantly improve the therapeutic effects of novel therapies for controlling the secondary progressive damage to the brain. The use of biological markers such as the S-100 serum factor could also be of additional value in reducing the levels of human variability.

Finally, such a programme based on biomechanics integrated with biomedical data would also serve to solve the outstanding problem of currently inadequate National and International standards for Head Injury Criteria and definition of Head Injury Tolerance. The Federal Government, and several private and industrial agencies have prescribed standards concerning certain protective devices and other modules relating to vehicular impacts, which must be satisfied in order to be accepted. One of the major dictums has involved the stipulation of peak values of *linear* head acceleration and corresponding maximum durations for which these values could be sustained. It must be emphasized that no correlation has been established between these parameters and head trauma; furthermore, such linear acceleration limits have not been related to stresses and strains that can be compared with observed cerebral damage. It should also be mentioned that the known potential damage of angular motion is not considered in these standards. These criteria use unacceptable parameters from an engineering viewpoint to correlate with trauma. It is strongly recommended that head injury criteria be established that follow the standard practice of engineering by relating conditions to levels of stress, strain, or absorbed energy.[23–25,31,65–67,79,101,107,108,111]

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Appendix: the physical basis of head injury biomechanics

Biomechanical behaviour of tissues upon load application

The deformation characteristics of all materials, including the tissues of the head, are determined experimentally under both slow (static) and dynamic loading. In this presentation, the loads and deformations will be restricted to two-dimensional considerations; this simplifies the description by avoiding the use of vector notation. The descriptions can be readily extended to three dimensions, but this will lead to some complications in concepts and mathematical expressions, particularly in the case of simultaneous rotations.

The application of force F is expressed in terms of engineering stress $\sigma = F/A_0$ where A_0 is the original cross-sectional area; the force is assumed to be distributed uniformly over the section. When compressive stresses are distributed uniformly over an entire body, this object is said to be in hydrostatic compression. Deformation is characterized by an engineering strain $\varepsilon = (L - L_0)/L_0 = \Delta L/L_0$ which represents the change of length divided by the original length for extension or compression, and by an angle in case of rotational changes. Two-dimensional deformations are depicted in Fig. A (a–e). The cases of tension (T), where an object tends to elongation, compression (C), where the element tends to contract, bending, shearing and torsion are depicted in Fig A(a) and A(b) under conditions of uniaxial loading. Bending is the result of a force applied in a direction not coincident with the central axis of the unit, resulting in the application of a moment $M = Fd$ [Fig. A(c)], where d is the perpendicular distance from the line of action of the force to the axis about which bending occurs. Simple shear [Fig. A(d)] is generated when two equal and parallel forces F generate angular deformation, while torsion is produced by a force acting perpendicular to the axis of, say, a circular bar at a distance r from the centerline; the effect of this action is the torque $\overline{T} = r\overline{F}$, which produces a twisting of the elements of the bar [Fig. A(e)]. Several of these actions can occur simultaneously.

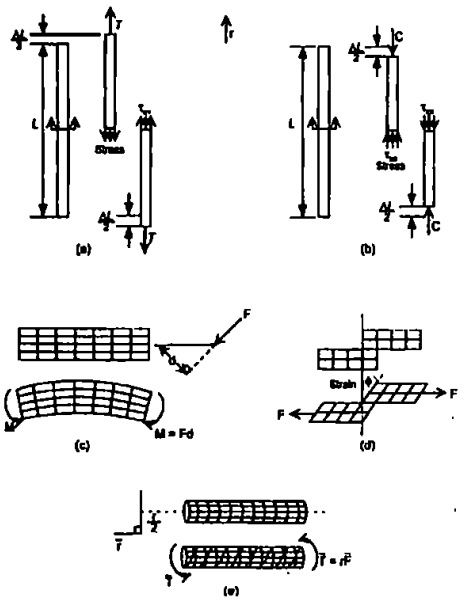

FIG. A. Uniaxial loading, stresses and deformation of a structural member. (a) Tension, (b) Compression, (c) Bending, (d) Simple shear, (e) Torsion.

The behaviour of a material is mechanically defined by the relation between stress, strain and, if important, by the rate of load application. This relation is called a constitutive equation and its characterization requires experimental measurement of the load-deformation characteristics. Many substances exhibit regions where the deformation occurs instantly upon load application and simultaneously disappears completely (or nearly so) upon force removal, representing reversible behaviour. Such a material is called *elastic* and its fabric is restored to its original condition upon unloading. Some materials stressed beyond the critical value, called the elastic limit, will exhibit permanent (or plastic) deformation resulting from irrevocable arrangement of the microstructure. However, materials permanently deformed may exhibit a measure of residual elasticity and could recover some of the deformation by a process called deformation.

When the response of the material depends significantly on the rate of load, stress or strain application, the solid is labelled as a visco-elastic substance. Furthermore, many substances, particularly biological at components, do not exhibit uniform properties either in various directions at a given location, or in moving from location to location. The first characteristic is called anisotropy, while the second is labelled inhomogeneity. In the presence of either of these complications, proper material description requires the determination of the stress-strain relation both in terms of position and direction to permit appropriate modeling of the loading and unloading response.

Failure of a solid element can occur by fracture or rupture that may take place in a brittle manner, or be preceded by permanent (plastic) changes of shape. It may also take place as the result of excessive deformation; physiological dysfunction most frequently precedes mechanical disruption. Failure limits for most engineering and many stable biological materials have been exhaustively tabulated.[1, 24,

[31, 58, 120, 126] Although some data have been obtained on the variation on mechanical properties with age, as for the skull of a neonate, child and adult,[114] there is an urgent need for additional information on this subject.

Motion characteristics of objects (Kinematics)[23]

It must be emphasized that biomechanics is an engineering discipline and, as such, employs terminology and concepts compatible with the principles of mechanics. An object may be considered as a particle, an entity of negligible dimensions compared to the path through which it travels. Its resistance to changes in motion is characterized by its mass $m = W/g$, where W is its weight and g the acceleration of gravity, 9.81 m/s² = 32.2 ft/s² on the earth. The kinematics of a particle are quantified by its displacement r, defined as the linear distance and direction from its initial position O to its final location P; by its velocity, which is the time rate of change of position, i.e, $v = dr/dt$, and by its acceleration $a = dv/dt = d^2r/dt^2$, the time rate of change of its velocity. Movement may occur in three dimensions, characterized, for example, by a rectangular coordinate system with orthogonal axes along x, y and z, and an origin O, as shown in Fig. B(a). When this motion is confined to two dimensions, say x and y, it is called planar motion [Fig. B(b)] and when it is directed along a single line, say the x-axis, it is termed rectilinear motion [Fig. B(c)]. It should be noted that, in general, the displacement r is not the same as the distance travelled; this occurs only in the case of uniaxial motion when there has been no reversal of the movement.

When the dimensions of the object are comparable or large relative to the displacements it executes, the system can no longer be regarded as a particle, but must be described as a rigid or deformable body. Rigidity can be assumed if the deformations resulting from the motion can be neglected, such as either the upper or lower arm when rotated. Even when significant deformations occur, such as brain movement during rapid acceleration of the head, the skull itself may be approximated for certain purposes as a rigid object, whereas the brain must always be regarded as deformable in the analysis of trauma. However, in view of the peculiar nature of the paediatric skull, it should frequently also be considered as non-rigid. Most biomechanical situations, particularly those involving the head/neck complex, require treatment of the anatomical components as bodies of finite dimensions.

A rigid body can execute all the movements of a particle in up to three dimensions; this is called the translational component of the object's motion. The displacements, velocities and accelerations of all particles of the object are by definition the same

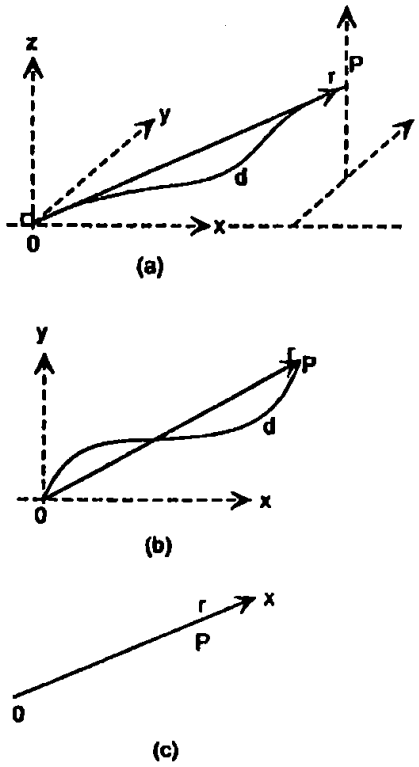

FIG. B. Displacement of an object considered as a particle. (a) In three dimensions, (b) In two dimensions [planar], (c) In one dimension [rectilinear]. In general, the distance travelled, d, differs from displacement r.

in this motion, although the reference point for their quantification is usually chosen as the centre of gravity or centre of mass G. However, in addition, such a body can either independently or simultaneously execute rotation, which consists of instantaneous circular motion of all particles of the object about a given axis, as shown in Fig. C (a–c).

The angular displacement is denoted by θ, the angular velocity by $\omega = d\theta/dt$, and the angular acceleration $\alpha = d\omega/dt = d^2\theta/dt^2$, relations that are completely analogous to the case of linear motion.

It should be emphasized that, at any instant of time, there exists a single angular velocity ω about some axis and a single angular acceleration α, either about the same axis or about a different axis; in general, these axes change with time. No motion due to rotation occur along these axes. There exists also an inertial resistance to rotation similar to the effect mass has on translation. This is characterized by the moment of inertia, I, which represents the sum of all infinitesimal mass elements, dm, of the

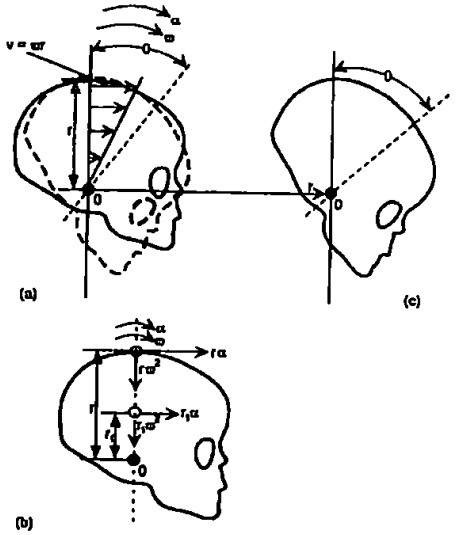

FIG. C. Rotation of the head about the occipital condyles, about an axis passing through 0.

objects, multiplied by the square of their respective distances from the axis of rotation r^2. (This is mathematically defined by the integral $I = \int_{\text{volume}} r^2\,dm$.)

In general, when the reference frame is represented by a standard rectangular (Cartesian) $Oxyz$ co-ordinate system, the rotational velocity and acceleration, which are likely to be in a direction not represented by x, y or z are usually broken up so as to represent respective components along the reference axes. For example, as indicated in Fig. C (a–c), head revolution due to the action of the neck muscles can occur in the AP direction about an axis perpendicular to the sagittal plane. Lateral bending may also take arise due to rotation about an axis perpendicular to the midplane of the sternum, as shown in Fig. D(a), whereas simple head rotation, Fig. D(b) takes place about a vertical axis.

If more than one of these rotations occur simultaneously, the magnitude of the combined speeds and accelerations, and the instantaneous axes of rotation must be obtained by the methods of vector analysis. (Note: angular displacement θ is *not* a vector because two such displacements, $\theta_1 + \theta_2 = \theta_2 + \theta_1$, the commutative law of addition, which all vectors must satisfy; however, angular velocity and angular accelerations conform to this requirement). In order to avoid this complication, head rotation will be considered in the sequel to occur only about a single, fixed axis, such as those cited above. However, it must be noted that each element of a rotating body not located on the axis of rotation experiences an instantaneous linear velocity $v = r\omega$,

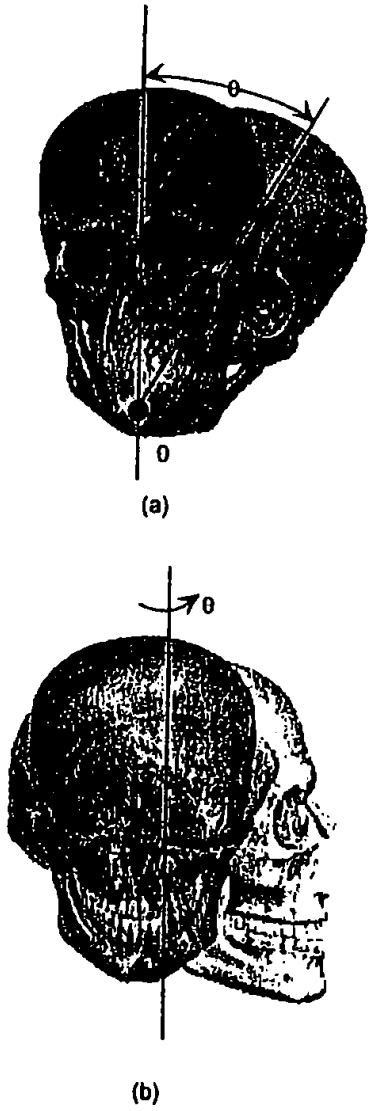

FIG. D. (a) Bending resulting from rotation about an axis perpendicular to an anterior section. (b) Head rotation about a vertical axis.

where r is again the perpendicular distance from the element to the axis (or point) of rotation. Furthermore, each such element is subjected to two simultaneous linear accelerations; one is the normal component $a_n = r\omega^2$, which is directed from the element towards the axis of rotation, and the other is the tangential component $a_t = r\alpha$ perpendicular to a_n and in the direction of the angular acceleration

α, if such acceleration exists. These components are depicted in Fig. C(a) and C(b).

Finally, a rigid body can experience simultaneous translation and rotation which, when confined to a single plane with the axis of rotation perpendicular to that plane, is called 'plane motion'. Such a situation is illustrated in Fig. C(c) for the head/neck complex executing rectilinear translation r and rotation about an axis perpendicular to the AP plane. Here, the linear velocity and acceleration components at any point of the system can be obtained by the arithmetic summation of their components in the x and y directions that are produced by the two separate types of motion. More complicated movements can be readily analysed by vectorial extensions of these methods.

Causation of head injury dynamics

This section deals with the two types of motion already described, translation and rotation, and their causation. As already indicated, pure translation can only exist when the resulting force acts on a particle or passes through the centre of gravity G of a (presumed) rigid body. In this case, the movement is governed by Newton's second law of motion:

$$F = ma = (W/g)a \tag{1}$$

where acceleration can be determined by measurement of time and distance, the mass m can be quantified from Newton's law of universal gravitation (subject too complicated to discuss here). Force F can only be defined by using eqn (1). An example of such force is the weight of an object which, when placed on the ground without any motion, is pushed upwards with the same amount of force as its weight. This action is shown in Fig. E(a); in practice, the movement of the head is restricted by the physical constraints of the neck (and its possible extensions) and by the body.

The law governing rotational motion is given by

$$M = I\alpha \tag{2}$$

where M is the applied moment about the mass centre and I is the moment of inertia about this point. Alternatively, the moment can be taken about

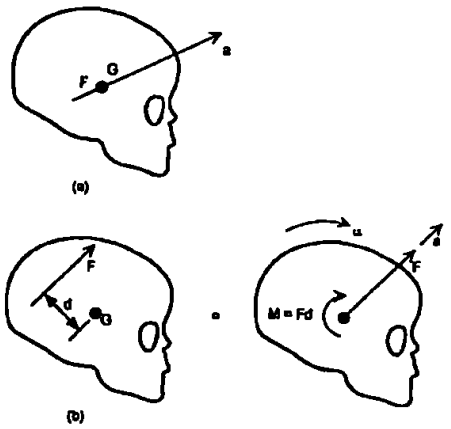

FIG. E. (a) Resulting force passes through mass centre G of a rigid body. (b) Resultant force passes at a distance d from mass centre G and equivalent force system.

a fixed point of rotation in plane motion, provided I is computed about this same position. The effect of a force displaced from the mass centre G by a perpendicular distance d from its line of action is equivalent to a combination of this force acting through the mass centre and a moment $M = Fd$ acting in the appropriate direction to provide the required rotation, as illustrated in Fig. E(b). As stated previously, such rotations produce differential displacements of adjacent spherical layers of the brain due to the outwardly increasing translational velocity of elements with respect to the axis of rotation. This generates shearing of the tissues, resulting in extreme cases in diffuse axonal injury and disruption of the bridging veins due to the difference in the motion of the skull and the brain by virtue of their differing distances from the axis of rotation and their disparate densities, that lead to differing values of the moment of inertia I.

Further descriptions of the application of the laws of motion may be found in any of a plethora of textbooks on the topic of dynamics. A condensed version of these principles is presented in Ref. 24.

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Appendix E

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Abusive Head Trauma in Infants and Children

Cindy W. Christian and Robert Block

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POLICY STATEMENT

Abusive Head Trauma in Infants and Children

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

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

ABSTRACT

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

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

Abbreviation
AHT—abusive head trauma

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HISTORY

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

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

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

DISCUSSION

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

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

THE ROLE OF THE PEDIATRICIAN

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

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with the frustration of a crying infant. They can also stress the importance of leaving a young infant or toddler in the care of adults whom the parents trust will not harm their child and can participate in comprehensive community-based prevention efforts. AHT commonly results in permanent neurologic damage and carries tremendous family and societal costs. With an aim toward prevention, the American Academy of Pediatrics recommends the following.

RECOMMENDATIONS

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

COMMITTEE ON CHILD ABUSE AND NEGLECT, 2008–2009

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Cindy W. Christian and Robert Block
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Appendix F

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Research Article

Biomechanical Evaluation of Head Kinematics During Infant Shaking Versus Pediatric Activities of Daily Living

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Abstract Abusive shaking of infants has been asserted as a primary cause of subdural bleeding, cerebral edema/brain swelling, and retinal hemorrhages. Manual shaking of biofidelic mannequins, however, has failed to generate the rotational accelerations believed necessary to cause these intracranial symptoms in the human infant. This study examines the apparent contradiction between the accepted model and reported biomechanical results. Researchers collected linear and angular motion data from an infant anthropomorphic test device during shaking and during various activities of daily life, as well as from a 7-month-old boy at play in a commercial jumping toy. Results were compared among the experimental conditions and against accepted injury thresholds. Rotational accelerations during shaking of a biofidelic mannequin were consistent with previous published studies and also statistically indistinguishable from the accelerations endured by a normal 7-month-old boy at play. The rotational accelerations during non-contact shaking appear to be tolerated by normal infants, even when repetitive.

Keywords biomechanics; traumatic brain injury; TBI; shaken baby syndrome; SBS; abusive head trauma; AHT; activities of daily living; ADL

1 Introduction

Shaken baby syndrome (SBS) has been defined as the presence of three specific findings: subdural hematoma (SDH), cerebral edema/brain swelling and retinal hemorrhage (RH) [8, 25]. This injury cluster—sometimes referred to as "the triad"—has been presumed to occur as a result of abusive shaking but not as a result of household falls, even falls down stairs [7]. The shaking hypothesis, first proposed in the early 1970s [6, 24], was seemingly accepted as settled science in 2001 in two documents: a position paper from the National Association of Medical Examiners [7] and an updated position statement from the American Academy of Pediatrics (AAP) [2]. Although the hypothesis has never been scientifically proven, practitioners working with the accepted model have accumulated years of clinical experience that convinces them that the model is correct [5].

Over the past 25 years, however, biomechanical research studies and computer modeling have raised questions about traditional thinking regarding SBS. When Duhaime et al. tried to confirm the shaking hypothesis in the 1980s using anthropomorphic test devices (ATDs), adult subjects failed to generate sufficient angular acceleration by shaking to reach the predicted thresholds for infant subdural hematoma (SDH) and diffuse axonal injury (DAI) [16]. A follow-up study published in 2003 concluded that non-contact shaking or a fall from less than 1.5 meters were less likely to cause injury than inflicted slamming against a hard surface [36].

Another team replicating Duhaime's work using alternative dummy designs, including different necks, recorded slightly higher accelerations in non-contact shaking, but peak accelerations were recorded when the surrogates' heads hit their own chests and backs [11]. Since head impact is known to cause SDH and DAI, researchers have encouraged the use of a more generic term for the symptom cluster, such as shaking-impact syndrome [15] or abusive head trauma (AHT) [9].

Computer modeling has explored refinements to the single-hinge ATD neck designs used in the Duhaime [16], Prange [36] and Cory [11] studies, predicting that more realistic neck designs, such as that used in the CRABI biofidelic mannequin, would yield lower angular accelerations [40]. As predicted, physical trials with a CRABI-6 yielded lower peak angular accelerations than what Duhaime reported (574.8 rad/s^2 [16] versus 1,138 rad/s^2 [10, 28]). Peak values across trials with the Aprica 3.4 kg anthropomorphic test device (1,436.5 rad/s^2 [28]), however, exceeded Duhaime's peak magnitude slightly, and trials with the Aprica 2.5 kg

model (13,252 rad/s^2 [28]) exceeded Duhaime's by an order of magnitude, although these figures seem to represent a different calculation strategy.

Biomechanical computer modeling has also concluded that a child's neck would break at forces lower than those required to produce the intracranial injuries associated with SBS [3,35]. Cadaveric studies have quantified the mechanical properties and strength of the human infant neck [17,31, 34], supporting the supposition that structural neck failure would result from even the accelerations reported during shaking of the Aprica 3.4 [28].

Direct experiments to determine brain injury thresholds in infants are prohibited for obvious ethical reasons. Data from field monitoring of adults and adolescents participating in contact sports have been used to set injury thresholds for mild traumatic brain injury in the more mature brain [18, 41]. Based on human data, not extrapolations from animal research, these thresholds for mild traumatic brain injury are 5–10 times greater than the rotational accelerations reported from abusive shaking of infant ATDs, particularly in studies that used a more realistic neck design. Nevertheless, the infant brain could be more vulnerable to injury from rotational accelerations than the adult and youth brain, and the repetitive accelerations presumed in shaking might not be comparable to sports contact.

Accident reconstructions of airbag-deployment injuries to infants have produced indirect data on infant injury thresholds. Klinich et al. concluded that infants in rear-facing car seats can tolerate up to 45 resultant Gs of acceleration without head injury, but may sustain fatal injury, including skull fracture and SDH, when 100 Gs or more results [14]. The auto industry's data, unfortunately, does not separate rotational from translational components. Further, the accident scenarios did not produce repetitive accelerations, and results did not reveal where the actual injury threshold might lie between 45 Gs and 100 Gs. This auto industry data is provocative, however, because it indicates that infants survive up to 45 resultant Gs, which is more than 4 times the 9.29 G and 9.9 G linear accelerations that are reported in the abusive shaking studies by Duhaime [16] and Jenny [28], respectively.

Depreitere et al. estimated the rupture threshold for adult bridging veins at 10,000 rad/s^2 [13], the same figure used by Duhaime et al. for concussion in infants [16]. Researchers have worked with even higher thresholds for infant SDH [11,16,40].

In the present study, researchers collected data from infant surrogates during abusive shaking simulations and various activities of daily living (ADLs) commonly experienced by infants. These ADLs would not be defined as abusive and would not be predicted to cause injury. Taking measurements during these activities establishes a baseline for commonly generated linear and angular head motions, allowing researchers to compare these values to injury thresholds, an approach also used by researchers trying to understand whiplash accelerations in automobile accidents [1].

	Height (in)	Weight (lb)
NCSBS demonstration doll	21.0	2.0
CRABI—12 month	29.5	22.0
KL (7-month-old baby boy)	27.0	19.2

Table 1: Anthropometry of infant surrogates.

Another data set was collected from an actual human infant at spontaneous play. He set his own level of activity and energy investment, absent any expressions of anxiety, discomfort, or neurological dysfunction. This infant's data were recorded, including rotational acceleration, which is usually considered to have the greatest potential to cause brain injury and SDH [27]. These rotational accelerations were specifically repetitive, addressing one of the criticisms of extrapolations from impact studies. Remarkably, this infant's spontaneous play resulted in rotational accelerations similar to those reported during the shaking of a 6-month CRABI biofidelic mannequin [28], and statistically undifferentiated from our measurements during shaking of a 12-month CRABI biofidelic mannequin. While our data does not establish a threshold for brain injury from repetitive rotational accelerations, it does establish a level of repetitive and cumulative rotational acceleration that is clearly well tolerated without apparent injury.

2 Materials and methods

2.1 Infant representatives

Researchers collected data from three infant representatives:

(i) a Child Restraint/Airbag-Interaction (CRABI)-12 biofidelic mannequin, weighing 22 pounds and measuring 29 inches head to toe, calibrated by and purchased from Denton ATD, Plymouth, MI, USA;
(ii) a demonstration doll, weighing 2 pounds and measuring 21 inches, sometimes allowed in court for demonstration purposes, purchased from the National Center on Shaken Baby Syndrome (NCSBS), Ogden, UT, USA;
(iii) a 7-month-old infant male, KL, weighing 19.2 pounds and measuring 27 inches, playing in his Fisher Price Deluxe Jumparoo (Mattel, Inc., El Segundo, CA, USA).

Table 1 lists the basic anthropometry of the three infant representatives, and Figures 1 and 2 show the actual infant representatives.

2.2 Subjects

Nine adult volunteers (two females and seven males, ranging in age from 20 to 77 years) subjected both the CRABI-12 and the NCSBS doll to aggressive shaking. Six of the

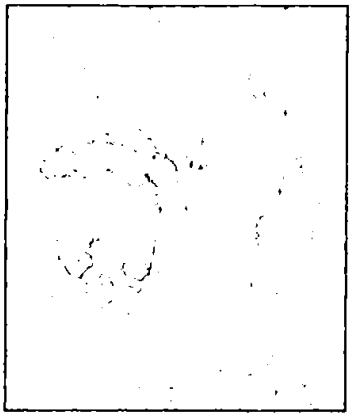

Figure 1: NCSBS demonstration doll (left) and CRABI-12 biofidelic mannequin (right).

Figure 2: CRABI-12 biofidelic mannequin (left) and 7-month-old infant, KL (right).

volunteers also handled the CRABI-12 in various ways to replicate activities of daily living (ADLs) for an infant, such as being burped or rocked.

The actual infant, KL, provided data during the normal course of his day, while playing in his Jumparoo, which is an infant's play toy manufactured by Fisher Price. At the time of the study, he met the specifications in the product's instruction manual, which cautions that it is used "only for a child who is able to hold head up unassisted and who is not able to crawl out."

Figure 3: InterSense sensors on head and torso of CRABI-12 mannequin.

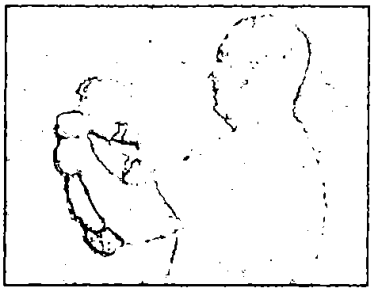

Figure 4: Male subject demonstrating aggressive shaking with NCSBS doll.

2.3 Test protocol

InterSense sensors (InterSense, Inc., Billerica, MA, USA) were secured to the head and torso of the child surrogate, as illustrated in Figure 3 for the CRABI-12 mannequin.

During the trials, the sensors transmitted raw data at 179 Hz per channel—including orientation (yaw, pitch, and roll), quaternion, angular velocity, and linear acceleration—wirelessly to a nearby computer. The sampling rate far exceeds the Niquist frequency for the shaking and pediatric ADL activities investigated.

Data was collected in three sets:

(i) from the NCSBS doll and the CRABI-12 mannequin while being shaken by an adult;
(ii) from the CRABI-12 mannequin during activities of daily living (ADLs), listed later in this section;
(iii) from the human infant at play, treated in the data analysis as an ADL.

2.3.1 Shaking

Nine adult volunteers grasped each of the two infant substitutes, the NCSBS doll and the CRABI-12 mannequin, as illustrated in Figure 4.

Figure 5: Female subject bouncing CRABI mannequin on her knee.

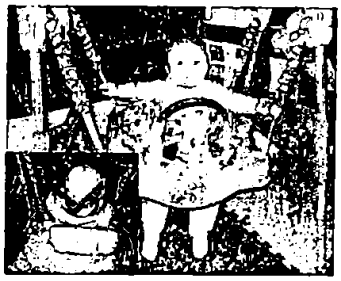

Figure 6: 7-month-old infant, KL, at play in his Fisher Price Jumparoo. Inset: InterSense sensor on back of subject KL's head.

While the sensors transmitted data, the volunteers shook the infant surrogates using three different techniques:

(i) mild shaking, to simulate resuscitative efforts;
(ii) gravity-assisted shaking, where the doll or mannequin was swung forcefully towards the ground, but without impact;
(iii) aggressive, repetitive shaking in the horizontal plane.

Each volunteer shook the infant representatives as hard as he or she could for as long as possible. Most subjects accomplished 10–20 seconds of shaking at 3–5 Hz. Each volunteer repeated the shaking twice, for a total of three trials for each ATD, per subject.

2.3.2 Pediatric activities of daily living

A subset of six adult volunteers, including two females and four males, manipulated the CRABI-12 mannequin during various pediatric ADLs, whilst head motion data was acquired using the InterSense sensors, as previously described. The investigated ADLs included:

(i) pushing the mannequin in a stroller over a smooth surface;
(ii) pushing the mannequin in a stroller over an uneven surface;
(iii) rocking the mannequin in a powered cradle;
(iv) walking on a treadmill at 2.5 mph while holding the mannequin in a baby carrier;
(v) running on a treadmill at 6.5 mph while holding the mannequin in a baby carrier;
(vi) throwing the mannequin into the air and catching it;
(vii) burping the mannequin with a back slap;
(viii) burping the mannequin with an up-and-down shake;
(ix) consoling the mannequin;
(x) bouncing the mannequin on a knee, as illustrated in Figure 5;
(xi) hitching the mannequin up onto the hip;
(xii) swinging the mannequin back and forth.

As in the shaking trials, each volunteer performed each activity three times. Only the CRABI-12 mannequin was used for these trials.

2.3.3 Infant playing

InterSense sensors were attached to the posterior aspect of the head of the 7-month-old male infant, KL, before he began one of his favorite activities, jumping in a commercially available jumping toy. Researchers collected data from KL's bouncing in 37 separate trials on two non-consecutive days, one week apart. The average minimum duration across trials was about 30 seconds.

Figure 6 illustrates the subject ready to jump.

2.4 Analysis

Using MatLab (The MathWorks, Natick, MA, USA), the investigators performed Fast Fourier Transform (FFT) analysis to isolate environmental noise data, which was removed using a phaseless 4th-order Butterworth low-pass filter, with a cut-off frequency of 50 Hz. Angular accelerations were derived, root-mean-square (RMS) values were calculated, and Head Injury Criterion (HIC-15) was computed according to (1), where HIC-15 is calculated with a period of less than 15 ms. A HIC-15 value of 390 is estimated to represent a risk of a severe head injury based on studies with the CRABI 6-month-old biofidelic mannequin [39]:

$$\mathrm{HIC} = \left[\frac{1}{t_2 - t_1}\int_{t_1}^{t_2} a\,dt\right]^{2.5}(t_2 - t_1), \qquad (1)$$

where a is a resultant head acceleration, $t_2 - t_1 < 15$ ms and t_2, t_1 were selected so as to maximize HIC.

	CRABI Resuscitative	CRABI Gravity assist	CRABI Aggressive shaking	NCSBS Resuscitative	NCSBS Gravity assist	NCSBS Aggressive shaking
AngDisp (deg)	50.8 (1.4)	121.9 (2.4)	120.8 (2.7)	71.6 (5.9)	128.6 (10.2)	167.4 (4.4)
AngVel RMS (rads-1)	12.5 (0.4)	24.3 (1.5)	25.5 (0.6)	12.5 (1.2)	35.7 (2.4)	34.6 (0.7)
AngAccel RMS (rads-2)	364.6 (20.8)	581.5 (57.2)	1068.3 (38.9)	502.9 (68.3)	995.4 (219.7)	1587.0 (79.0)
LinAccel RMS (g)	3.2 (0.1)	7.2 (0.3)	7.6 (0.2)	3.6 (0.4)	9.8 (0.1)	9.9 (0.2)
HIC-15	0.3 (0.03)	2.5 (0.2)	2.6 (0.1)	0.6 (0.2)	5.0 (0.1)	4.9 (0.3)

Table 2: Peak magnitudes, averaged across trials, recorded during different shaking techniques using the two infant surrogates (standard error of the mean in parentheses).

	Stroller (uneven)	Running (6.5 mph)	Throw in air & catch	Burping (back slap)	Bounce on knee	KL Jumparoo
AngDisp (deg)	14.2 (0.6)	59.3 (2.4)	58.6 (1.5)	12.0 (0.7)	44.2 (4.6)	77.8 (2.2)
AngVel RMS (rads-1)	2.9 (0.1)	8.3 (0.4)	7.8 (0.4)	1.3 (0.05)	6.5 (0.3)	15.6 (0.7)
AngAccel RMS (rads-2)	175.1 (0.8)	241.7 (8.8)	258.8 (19.5)	101.1 (6.0)	169.3 (7.5)	954.4 (35.0)
LinAccel RMS (g)	3.1 (0.05)	4.3 (0.2)	3.7 (0.2)	1.0 (0.1)	2.7 (0.2)	3.4 (0.1)
HIC-15	0.2 (0.02)	0.7 (0.05)	0.5 (0.05)	< 0.1 (0.02)	0.2 (0.03)	0.4 (0.02)

Table 3: Peak magnitudes, averaged across trials, during a selection ADLs using the CRABI biofidelic mannequin (standard error of the mean in parentheses).

3 Results

Table 2 reports the peak values, averaged across multiple trials and subjects, for each of the three shaking techniques performed with each of the two infant surrogates, the CRABI-12 mannequin and the NCSBS doll.

Table 3 reports the peak values, averaged across multiple trials and subjects, for a selection of pediatric ADLs, including the infant KL playing in his Jumparoo.

Figure 7 graphically illustrates the head kinematics during both infant shaking and pediatric activities of daily living. Values represent peak angular acceleration of the head, averaged across subjects and trials.

An analysis of results was conducted using SAS statistical analysis software (SAS Institute Inc., Cary, NC, USA). Findings denote that peak angular head accelerations recorded from the infant during bouncing in the Jumparoo (954.4 rad/s^2) are statistically indistinguishable at $P \leq .05$ from those recorded during aggressive shaking of the CRABI biofidelic mannequin (1068.3 rad/s^2).

Investigators also noted that values recorded during shaking of the NCSBS demonstration doll are approximately 50% higher than those recorded during shaking of the CRABI mannequin.

Most notably, even the results for aggressive shaking of the NCSBS doll are 84% below the scientifically accepted threshold for brain injury from angular acceleration.

4 Limitations of the present study

Any research using ATDs is limited by the biofidelity of the models employed.

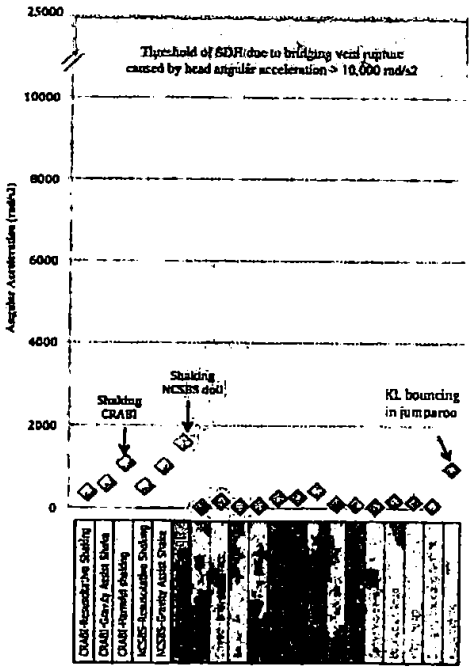

Figure 7: Head angular acceleration, in radians per second squared, during shaking (yellow) versus pediatric ADLs (blue).

Prange et al., 2003, reported that their ATD neck was specifically modified for the "worst-case scenario of no resistance provided by the neck, so that [they] could ascertain the greatest possible velocities and accelerations that can be generated by these mechanisms" [36]. The neck in their model was a single, linear metal hinge that connected the head to the torso, allowing free motion in one orientation only, neck flexion and extension in the sagittal plane. The highest-magnitude angular accelerations—2600 rad/s^2—were recorded when the model's head hit its own chest and back. Without trying to replicate the infant neck precisely, the Prange team concluded that "there are no data demonstrating that maximal angular velocities and maximal angular accelerations experienced during shaking and inflicted impact against foam cause SDH or TAI [traumatic axonal injury]".

Also in 2003, Cory and Jones reported that shaking their preliminary model produced a series of chin-to-chest and occiput-to-back impacts. Their discussion leaves open the question of how well this model reflects the neck of a human infant [11].

As illustrated in Figure 8, which was extracted from a high-speed digital video, angular displacement of the CRABI-12 neck in the sagittal plane shows a possible endpoint impact during shaking.

As in previous studies, angular accelerations of the ATD head during shaking reached maximum values at the end points of angular displacement.

When Wolfson et al. conducted calculated shaking simulations with ATDs of varying neck stiffnesses, only the models with "end-stop-type neck stiffness characteristics" produced values above predicted injury levels. More hinges added to the neck model to improve biofidelity produced lower head accelerations. The authors discounted the likelihood of impact with the back as the source of SBS symptoms with the observation "if violent impact of the head against the torso were the mechanism of intracranial injury in SBS, it is likely that findings such as bruising of the chin, chest, back and occiput would be reported" [40].

The infant KL was not photographed using a high-speed camera. The videos show no apparent contact between the child's head and body. More research is needed to investigate tissue properties and safe range of motion of the infant neck.

Figure 8: CRABI neck in the sagittal plane shows possible endpoint impact during shaking.

5 Discussion

It has been assumed for decades that aggressive manual shaking, with or without impact, produces the characteristic "triad" of SBS symptoms (subdural hemorrhages, cerebral edema/brain swelling and retinal hemorrhages) in an infant. This model is based on the hypothesis that uncontrolled motion of the infant head during shaking causes the damage directly, by tearing bridging veins to produce subdural hematoma, stretching neurons to produce diffuse axonal injury, and causing vitreous traction on the retina to produce retinal hemorrhages, schisis and folds.

Our study, however, like others before it, demonstrates that an adult's shaking of an infant surrogate does not even approach the angular accelerations generally accepted as a minimum threshold for infant SDH and DAI. These repeated experimental results undermine the fundamental thinking behind the abusive shaking hypothesis.

Type	Variations
Trauma	Accidental, falls and otherwise
	Inflicted, with impact or otherwise
Prenatal, perinatal, and pregnancy-related conditions causing intra-cranial hemorrhage	Intrauterine trauma, including abruptio placenta
	Thrombocytopenia
	Eclampsia and preeclampsia
	Chorioamnionitis
	Multiple pregnancies
	Hemolytic disease of newborn
	Prematurity
	Germinal matrix hemorrhage
Trauma at delivery	Abnormal presentation and uterine abnormalities
	Prolonged labor
	Forceps delivery
	Vacuum extraction
	Manual manipulation of the fetus
	Chemically assisted labor (pitocin drips, misoprostol (Cytotec))
Congenital malformations	Chiari malformations
	Arteriovenous malformations
	Aneurysm
	Osler-Weber syndrome
	Arachnoidal cyst
	Hydrocephalus, including extra-axial fluid collections
	Meningocele
	Syringomyelia
Venous and sinus thrombosis	Blood coagulation defects
	Leukemia
	Nephrotic syndrome
	Local infection
	Dehydration
	Hypernatremia
	Trauma induced central venous thrombosis
Genetic and metabolic conditions	Hemoglobinopathies, sickle cell disease
	Osteogenesis imperfecta
	Ehlers-Danlos Syndrome
	Von Recklinghausen's disease
	Tuberous sclerosis
	Marfan syndrome
	Menkes disease
	Polycystic kidney disease
	Glutaric aciduria
	Galactosemia
	Homocystinuria
	Alpha 1-antitrypsin deficiency
	Hemophagocytic lymphohistiocytosis, primary or secondary
	Vitamin D deficiency during pregnancy
	Others
Bleeding and/or coagulation disorders	Vitamin K deficiency
	Vitamin C deficiency
	Hemophilia A
	Hemophilia B
	Factor V deficiency
	Factor XII deficiency
	Factor XIII deficiency
	Protein S deficiency
	Protein C deficiency
	Von Willebrand disease
	Dysfibrinogenemia or hypofibrinogenemia
	Thrombocytopenic purpura
	Disseminated intravascular coagulation especially with infection or neoplasm
	Cirrhosis
	Inhibitors to clotting factors, including the following:
	– lupus erythematosus,
	– antiphospholipid antibody syndrome,
	– others
Infection	Meningitidis associated with numerous bacterial pathogens, including Streptococcus pneumoniae, Haemophilus influenzae, and Neisseria meningitidis
	Herpes encephalitis
	Cytomegalovirus encephalitis
	Infections in sinuses and/or ears
	Tonsillitis
	Toxoplasmosis
	Undetermined
Ischemic-hypoxic encephalopathy	
Vascular abnormalities	Moyamoya disease
	Kawasaki disease
	Dissecting vasculopathy
	Others
Neoplasms	Medulloblastoma and primitive neuroectodermal tumor
	Neuroblastoma
	Wilms tumor
	Leukemia
	Lymphoma
	Choroid plexus papilloma
	Xanthogranuloma
	Others
Medical interventions	Anticoagulation
	Craniotomy
	Spinal tap
	Spinal anesthesia
	Epidural anesthesia
	Subdural taps
	Intrathecal injection
	Shunts for hydrocephalus
	Placement of monitors
	Intravenous lines
	Antineoplastic therapy
	Anti-cold medications
Non-pharmaceutical toxins	Cocaine
	Lead
	Other

Table 4: Differential diagnosis for intracranial bleeding and cerebral edema.

The triad, meanwhile, is often found in children who present with seizures, which can interrupt breathing. Decreased oxygen supply can itself trigger cerebral edema. Tissue studies have concluded that brain damage in inflicted head injury results more from hypoxic-ischemic injury than from DAI [19,22,29].

Bridging vein rupture, meanwhile, is unlikely to be the source of low-volume intracranial hemorrhages in infants. Returning blood from the superior portions of the cerebral hemispheres flows through 5–8 pairs of bridging veins into the superior sagittal sinus. Given a blood flow of 50 mL per minute for every 100 grams of brain, each of these bridging veins would be expected to carry at least 5–10 mL of blood per minute. If a vein were to rupture, large volumes of subdural bleeding would be expected. However, autopsy findings in children report collections ranging from 1 to 80 mL, with 75% less than 25 mL and 50% less than 10 mL [33]. These small, thin films of subdural blood

seem clinically insignificant and inconsistent with bridging vein rupture. Researchers have suggested that in pediatric cases with minor hemorrhagic collections the blood may emanate from intra-dural vessels, rather than bridging vein rupture [32,38]. If the source of this blood is the dura, then biomechanical studies of bridging vein tolerances may not apply, yet these minor hemorrhagic collections frequently play a large role in legal proceedings.

Physicians have long recognized that the same clinical presentation can have more than one possible cause. Diagnosticians are therefore trained to apply adductive reasoning, ruling in and ruling out causes systematically, in what is known as differential diagnosis. The doctor hypothesizes the most likely cause: if a patient does not respond to treatment, or subsequent findings fail to confirm or even contradict the working diagnosis, other potential causes must be considered.

A diagnosis of SBS, unfortunately, can prematurely terminate the search for other possible causes of an infant's symptoms. A fundamental tenet of the classic SBS hypothesis is that abusive head trauma can occur in the absence of any other signs of abuse: no abrasions, no bruises, no neck or spinal cord damage, only the pattern of intracranial bleeding and swelling. This criterion—no signs of trauma—also applies to a host of other causes of intracranial bleeding and cerebral edema. Table 4, a list of current known causes, is adapted from a chapter in a reference text [37] and a medical journal article [12].

Shaken baby syndrome prosecutions, perhaps uniquely, rest primarily if not entirely on medical opinion. The same papers that established professional guidelines for identifying SBS also specified that, in cases of serious injury, the symptoms of an aggressive shaking would become apparent immediately after the assault [2,7]. With this nuance in place, the testimony of doctors is used to establish (1) that a crime was committed, (2) what actions constituted the crime and (3) when the crime occurred. Police officers who receive this information from a doctor see their jobs as to establish who was with the baby when the symptoms emerged, and then build a case against that person.

To make a diagnosis this powerful, a physician must rely on only the most solid evidence. Although the original SBS hypothesis has enjoyed decades of general acceptance, results from repeated biomechanical studies continue to undermine the reliability of the basic model, while timing of the symptoms also remains controversial [23,26] and researchers in other specialties continue to raise questions about various aspects of the classic model [4,20,21,30].

6 Conclusions

This study demonstrates that angular acceleration of the head during aggressive shaking of the CRABI biofidelic mannequin (1068.3 rad/s^2) is statistically indistinguishable ($P \leq .05$) from angular head kinematics experienced by a 7-month-old infant fervently playing in his Jumparoo (954.4 rad/s^2). Other pediatric ADLs, such as being burped or bounced on a knee, are clearly negligible. Furthermore, measured angular accelerations fall 84% below the scientifically accepted biomechanical threshold for bridging-vein rupture of 10,000 rad/s^2.

Although shaking an infant or toddler in anger is clearly ill advised and potentially unsafe, our data indicate that neither aggressive nor resuscitative shaking is likely to be a primary cause of diffuse axonal injury, primary retinal hemorrhage, schisis or folds, or subdural hematoma in a previously healthy infant.

Future research will investigate a systematic protocol for evaluating biomechanical indices associated with falls from different heights and orientations onto various surfaces.

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Appendix G

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Shaken baby syndrome: A biomechanics analysis of injury mechanisms

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Abstract

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

1. Introduction

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

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

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Table 1
Head injuries in the infant vs. the adult

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

[a] Represents different biomechanisms for the infant and adult.

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

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

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

In this study, we will present a biomechanics analysis of infant shaking and its consequences on the head-neck to determine if it is possible for the fragile infant neck to withstand SBS-defined levels of head accelerations without injury.

2. Biomechanical classification of head injury

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

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

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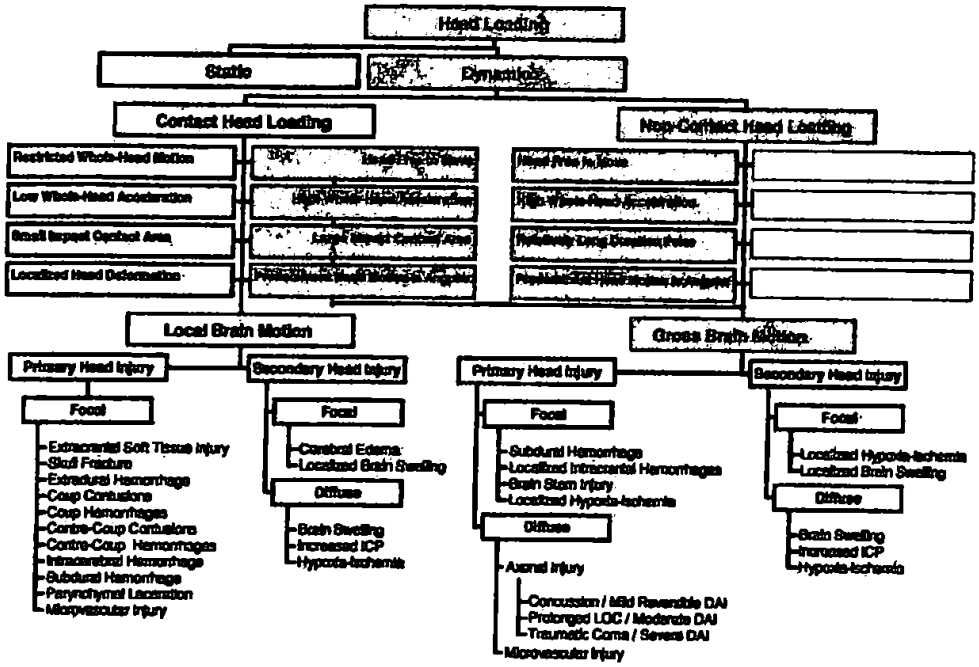

Fig. 1. Biomechanical classification of head injuries.

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

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

An infant is gripped by the chest or shoulders and forcefully shaken back and forth whipping the head in the antero-posterior direction. The nearly non-existent muscle strength of the infant neck makes the head highly susceptible to high head-whipping rotational acceleration so severe that the brain moves relative to the interior surface of the skull resulting in torn bridging veins and so acute subdural hematoma.

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

3. Biomechanical aspects of the infant anatomy

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

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

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

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

4. Injury biomechanics analysis of infantile SBS

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

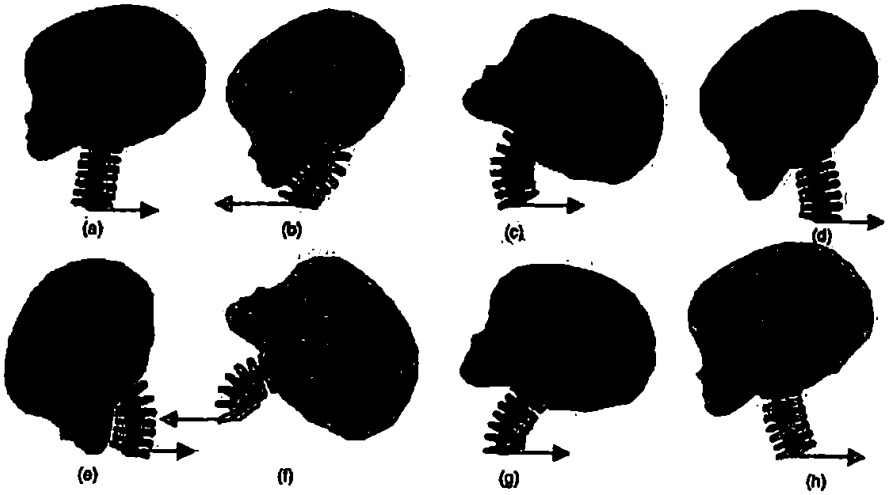

Fig. 2. Several time-history snap shots of the head-neck during chest shaking.

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

5. SBS head loading

We have necessarily described SBS in terms of head loading because the head has been central to the definition of the SBS injuries. Chest shaking forces reach the head through the neck starting at C7 and moving back and forth in wave fashion (Fig. 2) eventually transmitting forces to the cranio-vertebral junction. Along this path, the force acts on soft, flat, incompletely ossified vertebrae, and soft ligaments. The forces reach the cranio-vertebral junction and induce the infant skull to move back and forth with little resistance to pivot about the atlanto-occipital joint. The overall head motion is governed by the neck which significantly influences the head trajectory and the center of rotation. In fact, the neck must completely bear the load that keeps the head and thorax from separating. For the infant, this type of chest-shaking head motion can be thought of as a kind of "dragging" motion where the neck, acting as a tether, pulls the unsuspecting head in response to the induced motion of the chest. This has not been described as the mechanism for SBS.

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

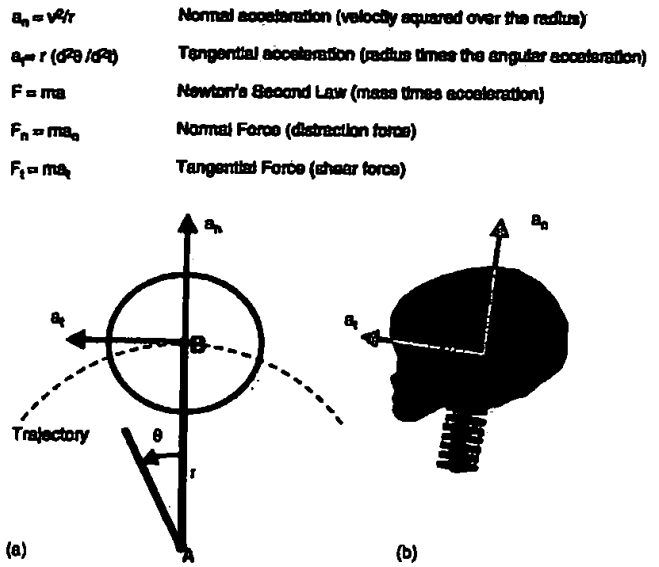

Fig. 3. Force diagram on (a) sphere and (b) head-neck schematically indicating rotational head motion about point A.

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

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

To illuminate and compare simplified head motion energies of familiar events such as car crashes, biking, walking, etc., with shaking, we calculated the free head velocity associated with each event as shown in Table 2. The table reflects conservative but realistic estimates of maximum free head velocity for each of the events. Ironically, the values show that shaking head velocity is less than the maximum free-fall velocity from height of 1 m (3(1/4) ft). This comparability of energies raises further questions concerning the forces reported for SBS and might account in part for the misconceptions and controversial views on the head injury severity of "short falls." The energy from velocity is called kinetic energy and we refer to it here as *unrealized* damage energy meaning it can be converted to potentially damaging energy through a rapid change in magnitude and/or direction. For the case of the infant head, this velocity change can come from the action of the neck on the head during chest shaking as we explained earlier or it can come from an impact action applying force directly on the head such as that from a fall. Table 2 shows the energies as multiples of units of failure energy where the failure energy was taken to be equal to the skull fracture energy in the infant. Skull fracture energy was chosen since skull fracture has been used as a conservative indicator of impact intracranial injury in infants [15,16].

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

6. Results and discussion

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

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

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

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

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

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

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

relatively less massive, head on quite a strong neck while the infant on the other hand has a relatively more massive head on a floppy, weak, neck. Consequently, the infant neck is far more susceptible to whiplash injury than that of the rhesus monkey under the similar chest-shaking head accelerations. Nearly half of the concussed animals in Ommaya's [17] whiplash experiments experienced cervical spine or brain stem injuries even with the disproportionately strong neck relative to the human infant.

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

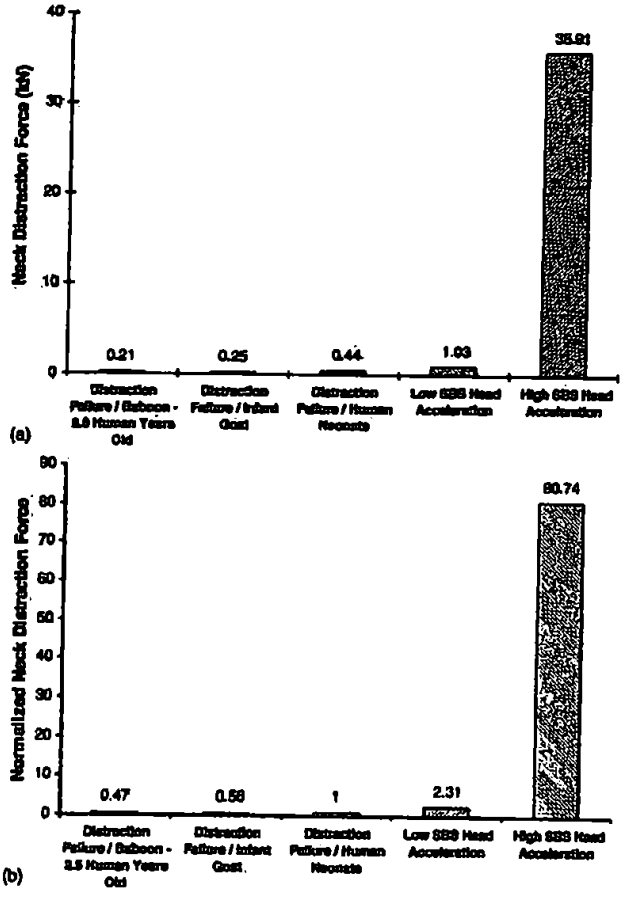

Fig. 4. SBS neck distraction force vs. (a) Structural failure of the cervical spine and (b) normalized to human neonate distraction failure data.

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

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

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

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

7. Conclusions

This study resulted in the following findings:

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

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

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

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Glossary

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

Normal: in this case "normal" means in a direction perpendicular to the surface.

G: is the designated symbol for one unit of gravitational acceleration. On earth, the value of G is 32.2 ft/s^2

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

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

E_{SI}: infant skull fracture energy.

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Response to the Letter to the Editor

I thank the writers for their comments to the editor regarding my article. At the outset, it is evident to me from the letters that the two writers seem to convey a similar construal of my paper. Therefore, I shall provide a combined response to both letters.

As their core message, the writers of the two letters suggest that my calculated SBS neck forces are too high because they are not in accord with what they calculated using the reported velocity values. Rather than concede that the velocities reported for SBS in the literature might be contradictorily low, the writer of one of the letters, instead, chose to believe that numerical errors are the reason for the dissimilarity between my numbers and theirs. In other words, it appears that these writers believe that head accelerations can, over the time duration of shaking, reach the SBS-defined levels[1] high enough to activate the customary rotational acceleration mechanism and cause subdural hematoma (SDH) but, at the same time, not necessarily cause infant neck injury.

These writers claim lower neck forces referring to dummy shaking experiments that produced far lower rotational head accelerations and they cite from an article[2] by Prange and Meyers [1] and one by Jenny et al. [2]. In effect, both writers cite acceleration values that limit the force an adult can impart on an infant and thus bound the head acceleration to far less levels than described for SBS in the literature. In fact, the writers quote a wide range of values for acceleration, which indicates that they are quite aware that human manual shaking alone may be insufficient to activate the above-mentioned rotational injury mechanism that they believe is the cause of SDH in the infant.

Clearly, various articles by M. Prange, S. Margulies, B. Meyers, C. Jenny, and others subscribe to the view that activation of the rotational acceleration mechanism of SDH demands far higher accelerations than those given by the writers in these critiques. In one of the letters, the writer refers to Prange and Meyers [1] who reported shaking accelerations of 4220 rad/s^2 and referred to Duhaime et al. [3] reporting accelerations of 1138 rad/s^2. Prange et al. [4], on the other hand, published a paper the same year that reported shaking accelerations of 2640 rad/s^2. The other writer reports on his analysis of Jenny et al. [2] data to obtain a peak acceleration of 3000 rad/s^2. Jenny et al. [2], however, reported 13252 rad/s^2 for what appears to be the same data.

Essentially, all of the rotational head acceleration values both writers quote are far below the levels described for the SBS. Their letters in effect point out that the levels of the shaking head accelerations defined for the SBS may be unrealistic and possibly unphysical. Disregarding the notion that SBS acceleration levels are unphysical, if one proceeds anyway and analyzes their application for the duration of shaking to the infant head, then a high force outcome on the neck is inevitable. Thus, the dilemma: *SBS calls for very high shaking head accelerations acting over the course of seconds to minutes to cause SDH but does not indicate what is expected to happen to the infant neck as a result of the consequent forces.*

Let me identify the difference between what I have done and what these two writers discuss. Since SBS is referred to in the literature as causing "acceleration/deceleration" brain injury, my approach was to directly analyze these accelerations applied for the time course of shaking. I chose a range that includes the highest values of accelerations reported because had I chosen lower values, the analysis would simply exclude the cornerstone mechanism of SBS and therefore, would be rendered moot. In other words, lower values would not be sufficient to activate the customary mechanism of rupturing bridging veins that SBS solely depends on for producing SDH. This of course, is the mechanism garnered for SBS from experiments where the head of a primate was potted in a metal cylinder constrained for acceleration/deceleration along a prescribed arc in a prescribed time frame. Duhaime et al. [3] evaluated this mechanism for infant shaking and impact and provided a time duration for each shaking cycle, which is much longer than that for impact. The SBS literature suggests that a shaking event consists of repeated cycles of shaking, which take place over a reported range from several seconds to more than a minute. I basically integrated the SBS accelerations over the time duration of shaking and obtained values for resulting neck forces. I agree with the writers that the neck forces I obtained are high but not because of numerical error. The forces are high because that is what results when the described SBS accelerations are applied to the infant head for the time duration of shaking.

Finally, more research is needed to advance understanding of the injury mechanisms associated with traumatic infant shaking to help detect and prevent it. It is my hope that discourse such as

DOIs of original articles: 10.1016/j.forsciint.2005.12.018, 10.1016/j.forsciint.2005.12.017.

[1] SBS is cited in the literature as having forces that are as great as those of falls from great heights, by some accounts more than 9 m (30 ft), onto hard surfaces or from high-speed motor vehicle crashes

[2] This article, cited by the writer of one of the letters as "M.T. Prange and B.S. Myers, *Evidenced-based biomechanical analysis of craniocervical inflicted neurotrauma, Inflicted Childhood Neurotrauma*, American Academy of Pediatrics, 2003, pp. 237–243", was not found. We found essentially the same citation but apparently the writer of the letter used a different title [1].

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this can help energize more interest in the provision for and conduct of innovative research in this very important area.

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Appendix H

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Anthropomorphic simulations of falls, shakes, and inflicted impacts in infants

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Object. Rotational loading conditions have been shown to produce subdural hemorrhage and diffuse axonal injury. No experimental data are available with which to compare the rotational response of the head of an infant during accidental and inflicted head injuries. The authors sought to compare rotational deceleration sustained by the head among free falls, from different heights onto different surfaces, with those sustained during shaking and inflicted impact.

Methods. An anthropomorphic surrogate of a 1.5-month-old human infant was constructed and used to simulate falls from 0.3 m (1 ft), 0.9 m (3 ft), and 1.5 m (5 ft), as well as vigorous shaking and inflicted head impact. During falls, the surrogate experienced occipital contact against a concrete surface, carpet pad, or foam mattress. For shakes, investigators repeatedly shook the surrogate in an anteroposterior plane; inflicted impact was defined as the terminal portion of a vigorous shake, in which the surrogate's occiput made contact with a rigid or padded surface. Rotational velocity was recorded directly and the maximum (peak–peak) change in angular velocity ($\Delta\dot{\theta}_{max}$) and the peak angular acceleration ($\ddot{\theta}_{max}$) were calculated.

Analysis of variance revealed significant increases in the $\Delta\dot{\theta}_{max}$ and $\ddot{\theta}_{max}$ associated with falls onto harder surfaces and from higher heights. During inflicted impacts against rigid surfaces, the $\Delta\dot{\theta}_{max}$ and $\ddot{\theta}_{max}$ were significantly greater than those measured under all other conditions.

Conclusions. Vigorous shakes of this infant model produced rotational responses similar to those resulting from minor falls, but inflicted impacts produced responses that were significantly higher than even a 1.5-m fall onto concrete. Because larger accelerations are associated with an increasing likelihood of injury, the findings indicate that inflicted impacts against hard surfaces are more likely to be associated with inertial brain injuries than falls from a height less than 1.5 m or from shaking.

KEY WORDS • brain injury • child abuse • diffuse axonal injury • subdural hematoma • children

TRAUMATIC brain injury is the most common cause of death in children.[3] Brain injuries resulting in hospitalization or death occur in at least 150,000 children per year, at a rate of more than 200 per 100,000 children. Head injury in infancy results in higher incidences of morbidity and mortality than those seen in older children, and it has become increasingly clear that the significant incidence of nonaccidental injury in the youngest patients is, in large part, responsible for this difference.[5,11,28,29]

The majority of serious traumatic brain injuries in infants and toddlers is due to child abuse, and abused children with brain injury have a worse outcome than children who sustain an accidental brain injury.[3,13] The actual mechanism of injury responsible for subdural hemorrhage, retinal hemorrhage, axonal injury, and skeletal trauma that characterize abusive head injury has been debated for decades. Caffey[4,7] first proposed the term "whiplash shaken infant syndrome" to describe the occurrence of subdural and retinal hemorrhages in response to presumed inflicted angular acceleration of the head. Others have followed with reports of subdural hemorrhage, retinal hemorrhage, and death occurring in the absence of contact injury (skull fracture, cranial bruising, or scalp swelling).[2,20] Regardless, contact head trauma remains a frequent finding in abusive head injury.[17]

Accidental falls are also a common cause of trauma found in the pediatric population, with falls accounting for 25 to 34% of hospital admissions for pediatric trauma and 6% of deaths in children due to trauma.[21,34] Nevertheless, accidental falls are a common history given by caregivers in suspected abuse cases. The suspicion of abuse often arises when the event history appears not to correspond to the injury in the child. The differentiation between causes of accidental and abusive head injury is hindered by the controversy regarding fall heights associated with serious head injures in children. Evidence exists to support the hypothesis that short falls do not cause serious injury and the critical height for a fall to cause death is substantial (> 10 ft).[4,10,34,36,49,55] Simultaneously, however, others contend that relatively short falls can occasionally cause injuries associated with high mortality rates, such as SDHs, epidural hematomas, and skull fractures.[8,18,19,21,40,43,44,46,54] Because the mechanical responses experienced by the head and the in-

Abbreviations used in this paper: ANOVA = analysis of variance; DAI = diffuse axonal injury; SDH = subdural hematoma; TAI = traumatic axonal injury; Δt = duration of the maximum change in angular velocity; $\Delta\dot{\theta}_{max}$ = maximum (peak–peak) change in angular velocity; $\ddot{\theta}_{max}$ = peak angular acceleration.

TABLE 1

Comparison between body measurements in infant and surrogate

Body Measurement	Infant	Surrogate
head weight (kg)	0.77–0.87*	1.13
head circumference (transverse plane [cm])	39.5†	40.5
head height (inferior to superior [cm])	13.5†	12.6
head breadth (right to left [cm])	10.5†	11.4
head length (anterior to posterior [cm])	14.1†	12.6
distance from shoulder to top of head (cm)	14.7†	15.1
distance from axis of rotation (C5–6) to top of head (cm)	15.4‡	14.7
distance from axis of rotation (C5–6) to center of gravity of the head (cm)	9.5‡	9.2
distance from axis of rotation (C5–6) to base of skull (cm)	3.3*	4.5
distance from axis of rotation (C5–6) to transducers (cm)	not applicable	18.0
total body weight (kg)	3–4* 4.8§	4.83
breadth of shoulders (cm)	17.6†	17.8
head/body weight ratio	0.23* 0.24‖	0.23

* Weight of 1-month-old infant according to Duhaime, et al., 1987.

† Measurements obtained in an infant 0 to 3 months of age according to Schneider, et al.

‡ Distance according to Swishchuk.

§ Weight of average male or female 6-week-old infant according to Kuczmarski, et al.

‖ Ratio in a 6-week-old infant according to Jensen.

jury tolerances associated with shaking, shaking with impact, and falls have not yet been established, the differentiation between accidental and inflicted head injury is problematic. Objective information regarding these parameters is needed to determine if some circumstances are more hazardous than others during accidental and inflicted traumatic situations.

Previously, dolls that were anthropomorphically matched to human infants were shaken with and without impact;[12] investigators reported that during an inflicted impact the angular deceleration of the head is 45 times that observed during a vigorous shake, exceeding scaled thresholds for concussion, subdural hemorrhage, and DAI. In that study, however, the angular (rotational) motions were calculated on the assumption that there was a fixed center of rotation, and were not measured directly. Moreover, falls were not simulated, only inflicted shakes and impacts. In this study, we have created an improved anthropomorphic surrogate of a 1.5-month-old infant, and directly measured the rotational velocities experienced by the head of the surrogate during shakes, inflicted impacts, and short-distance falls. This research also extends that of previously published studies by examining the effect of different contact surfaces on the rotational response of the infant's head during both abusive and accidental injury scenarios. By comparing the responses, our findings are an important step toward distinguishing the potential for head injuries caused by rotational motion during contact and purely inertial events, and during accidental and inflicted injury scenarios.

Materials and Methods

Construction of the Anthropomorphic Dummy of an Infant

Head Design. A dummy was designed, constructed, modified, and improved to create a more biofidelic and durable anthropomorphic surrogate of a 1.5-month-old infant than that used previously. The head of a toy doll (La Baby; JC Toys Group, Inc., Miami, FL) was used to represent the head of the anatomically correct 1.5-month-old anthropomorphic surrogate. Because the center of gravity and the majority of an infant's body mass are located in the head and torso, the distribution of the weight of the arms and legs of the infant were incorporated into the weight of the torso. The surrogate's total body weight, 4.8 kg (10.6 lb), was matched to that of a 1.5-month-old infant whose body weight lies within the 50th percentile.[25] Using previously reported measurements, the distributed masses of the head and body were adjusted to mimic those of 1.5-month-old infant by creating a head/total body weight ratio of 0.235[12,25] (1.13-kg head mass). The breadth, length, and width of the head were measured and are in good agreement with those obtained in a 0- to 3-month-old infant in the 50th percentile (Table 1).

Neck Design. One-month-old infants have very compliant necks with little muscle tone and control of head movement.[9] The weak flexor and extensor muscles of the neck allow a significant lag between the head and torso when raising the infant to a sitting position and lowering him or her back to a lying position.[9] The normal movement of the neck has been described in the context of qualitative neurological examinations and developmental assessments in children; however, no quantitative information is available on the biomechanics of the human infant neck. In light of the absence of detailed quantitative information about the kinematics of infant necks in the literature, we fashioned a hinged neck with negligible resistance for the dummy, as previously published.[12] In this way, measurements would reflect a worst-case scenario of no resistance provided by the neck, so that we could ascertain the greatest possible velocities and accelerations that can be generated by these mechanisms. One end of a heavy-duty stainless-steel strap hinge was rigidly attached to the skull material on the surrogate's head; the other end was rigidly attached to the torso. The hinge was used as the neck joint in the surrogate to allow resistance-free motion in extension and flexion with no movement in other directions. The center of rotation of the hinge was located 9.2 cm inferior to the center of the mass of the head, and corresponded to the junction between C-5 and C-6 measured by magnetic resonance imaging.[31] Although the fixed center of rotation in the dummy would result in an overestimation of rotational acceleration because the actual centers of rotation are likely to be higher in the cervical spine, the relatively short cervical spine, compared with the head size of a typical young infant, minimizes the influence of this idealization. Regardless, this simplification once again errs on the side of a worst-case scenario.

Skull and Scalp Material. Another design consideration was the representation of the stiff skull with a flexible scalp. Experiments were performed to determine appropriate "skull" and "scalp" materials to use in the construction of the dummy. The mechanical properties of the orthopedic-grade copolymer polypropylene (2.25 mm thick; American Plastics, Fort Worth, TX) were tested and determined to lie between those of infant skull and suture.[31,42] The plastic was heated and molded to the head of the surrogate and allowed to cool to room temperature. The instrumentation mounting bracket was attached securely to the skull, creating a rigid connection among the instrumentation, surrogate skull material, and head. A latex rubber material (Mold Builder [1.25 mm thick]; ETI, Fields Landing, CA) with properties similar to scalp[11,21,42] was used to cover the occipital portion of the polypropylene skull; this material remained adherent throughout the tests.

Head Instrumentation

An angular rate sensor (model ARS-01; ATA Sensors, Albuquerque, NM) was securely attached to the top of the dummy's head via a lightweight bracket, and was adjusted to measure rotations with an axis of rotation oriented perpendicular to the sagittal plane. This transducer location was selected to be remote from the impact site, so as not to damage the transducer in the impact and fall experiments. Any similar location on the head and transducer orientation would have yielded the same rotational velocity data. The velocity channel was sampled at 10,000 scans per second and filtered using a digital Butterworth low-pass filter (DADiSP/2000; DSP Develop-

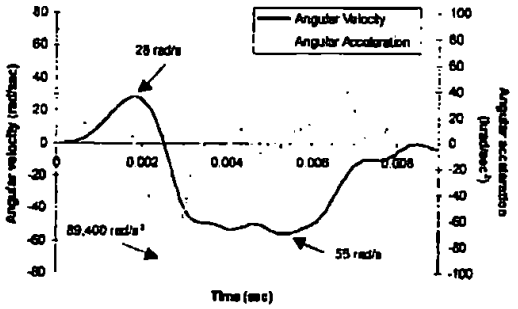

FIG. 1. Graph showing a representative angular velocity and acceleration trace for the impact produced from a 0.9-m (3-ft) fall onto concrete.

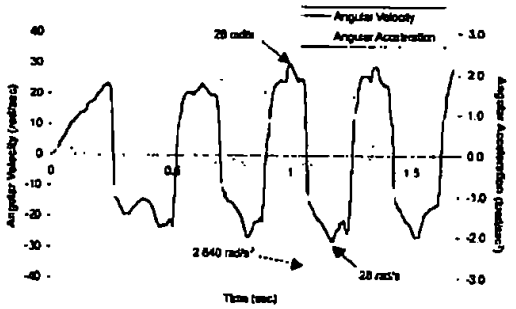

FIG. 2. Graph showing a representative angular velocity and acceleration trace for a shaking event.

ment Corp., Newton, MA) as specified by the Society of Automotive Engineers.[50] Simulations of falls and inflicted impacts that involved contacting hard materials (carpet pad, concrete, or lab bench) were processed through a low-pass filter with a cutoff frequency of 1000 Hz, according to the standard SAE J211-1 channel frequency Class 1000 used for measuring head accelerations during automotive crash tests.[50] Because shakes and impacts against the foam mattress had pulse durations much longer than impacts against hard surfaces, these signals were processed through a low-pass filter with a cutoff frequency of 250 Hz to eliminate the noise from the velocity signal.

Abuse and Accident Reconstruction

A custom-designed drop-test apparatus was constructed to allow the dummy to be dropped consistently from 0.3-m (1-ft), 0.9-m (3-ft), and 1.5-m (5-ft) heights. The surrogate was suspended supine from three points (two on the body, one on the head) with the height of the dummy's head placed slightly lower (< 3 cm) than the body to ensure that the surrogate's occiput was the first point to contact the surface. To reproduce the shaking of an infant, the surrogate was grasped firmly by its torso and held at chest level. The dummy was then shaken back and forth, making sure that during each shake the head went through a complete range of motion from full extension to full flexion. Each episode included at least five shakes, with the final shake concluding with an inflicted impact of the surrogate's occiput against one of three materials located at approximately the volunteer's waist level (0.9 m from floor). The sequence was divided into two segments, the "shaking event" and the "inflicted impact event," for analysis. Volunteers were instructed to use maximum effort during vigorous shaking and impact, and not to release or throw the dummy during impact.

Three materials were used in the fall and inflicted impact simulations: a piece of 10.2-cm (4-in)–thick foam from a crib mattress, a section of 6.35-mm (0.25-in)–thick carpet pad, and a hard surface (concrete floor for falls, stone bench top for inflicted impacts). These materials were tested and the average linear elastic moduli of the foam and carpet pad were measured and found to be 24.8 and 621 kPa, respectively,[42] whereas the elastic modulus of concrete in compression has been documented to range from approximately 20 to 60 GPa.[41] These data demonstrate the wide range of contact surface characteristics that were used in the household fall situations.

Statistical Analysis

After filtering the measured angular velocity for each event, angular acceleration was calculated by taking the derivative of the angular velocity–time history trace. Only the rotation caused by the initial impact was analyzed in traces for falls and inflicted impacts. For each shaking event, only the shake with the greatest angular acceleration was analyzed out of the series of shakes in the episode. Each event was analyzed to find the $\Delta\dot{\theta}_{max}$, the Δt, and the $\ddot{\theta}_{max}$ during this interval.

A total of 134 fall events were reconstructed from various heights onto different surfaces with at least 14 falls for each height–surface combination. A representative trace of the measured angular velocity and calculated angular acceleration during a fall is shown in Fig. 1.

Sixty-one shaking and impact sequences were performed by six different adult volunteers (four male and two female volunteers ranging in weight from 50–100 kg). Nearly all shaking episodes (60 of 61) consisted of a shake and an inflicted impact segment in which the surrogate's occiput made contact with one of three surfaces (foam in 18 episodes, carpet pad in 20 episodes, and a composite bench top in 22 episodes). Typical angular velocity and angular acceleration measurements from a series of shakes and inflicted impacts are shown in Figs. 2 and 3, respectively.

To determine overall differences between falls from different heights and onto different surfaces, three separate two-way ANOVAs were used to analyze the $\Delta\dot{\theta}_{max}$, $\ddot{\theta}_{max}$, and Δt individually. A Tukey test for multiple comparisons was also performed to determine the differences between each individual type of fall. The measurements from shakes were compared with inflicted impacts against different surfaces by using one-way ANOVA to determine the significance of the test mode and the Tukey test to determine differences between each pair of test modes. A Dunnet test for multiple comparisons to a control was used to compare the $\Delta\dot{\theta}_{max}$, $\ddot{\theta}_{max}$, and Δt from all falls, comparing the effects of the various surface materials by using shakes as the control or standard in the Dunnet test. The same analysis was repeated in a sequence of Dunnet tests by using inflicted impacts against foam, carpet pad, or bench top as the control group in each evaluation. Statistical significance was defined at a probability value of 0.05 or less for each analysis.

Results

Falls Onto Padded and Unpadded Surfaces

A two-way ANOVA revealed a significant overall increase in $\Delta\dot{\theta}_{max}$ and $\ddot{\theta}_{max}$ ($p < 0.001$; Fig. 4 *upper* and *center*) and a decrease in Δt with falls onto harder surfaces and from greater heights ($p < 0.001$; Fig. 4 *lower*). A Tukey test revealed no significant effect of the height of the fall in the measured $\Delta\dot{\theta}_{max}$ or $\ddot{\theta}_{max}$ during falls onto foam and showed that both kinematic measurements at a given height were significantly less for falls onto foam than for those onto carpet pad or concrete. Falls from a given height onto the carpet pad and concrete were indistinguishable, except for the $\ddot{\theta}_{max}$ during 1.5-m falls. For a particular surface, no significant difference between 0.9- and 1.5-m falls was found in the measurements of $\Delta\dot{\theta}_{max}$ or $\ddot{\theta}_{max}$; however, measurements of Δt were significantly different when comparing fall heights onto foam, with the Δt decreasing as the height of the fall increased.

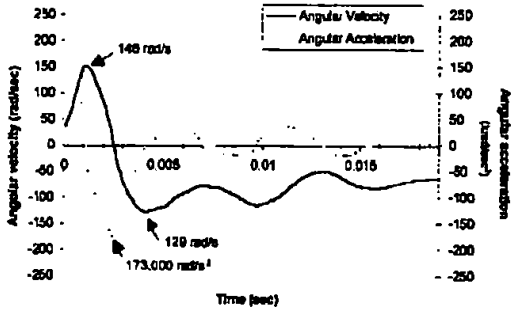

FIG. 3. Graph demonstrating representative angular velocity and acceleration trace for an inflicted impact against a bench top.

Shakes and Inflicted Impacts

Averaged across volunteers, the values of $\Delta\dot{\theta}_{max}$ and $\ddot{\theta}_{max}$ during inflicted impacts against foam were greater than, although not significantly different from, those measured during shaking events (Fig. 5 *upper* and *center*). Inflicted impacts against foam, however, had a significantly shorter average Δt than shaking events (Fig. 5 *lower*). Inflicted impacts against the carpet pad and rigid bench surface were indistinguishable. Taken together, inflicted impacts against these two hard surfaces resulted in an approximately 39 times greater $\ddot{\theta}_{max}$, a three times greater $\Delta\dot{\theta}_{max}$, and a 53 times shorter Δt than the response measured during shaking.

Shakes had a statistically similar $\Delta\dot{\theta}_{max}$ to those of 0.3-m falls onto concrete and carpet pad, and a similar $\ddot{\theta}_{max}$ to that of falls onto a foam mattress. Shaking resulted in a significantly longer Δt than any fall events. Inflicted impacts against foam had a similar $\ddot{\theta}_{max}$ to that of falls onto a foam mattress and a similar $\Delta\dot{\theta}_{max}$ to that of falls onto concrete. Inflicted impacts against a hard surface resulted in a similar Δt to those of falls onto carpet pad and concrete, and had a significantly greater $\Delta\dot{\theta}_{max}$ and $\ddot{\theta}_{max}$ than all fall scenarios.

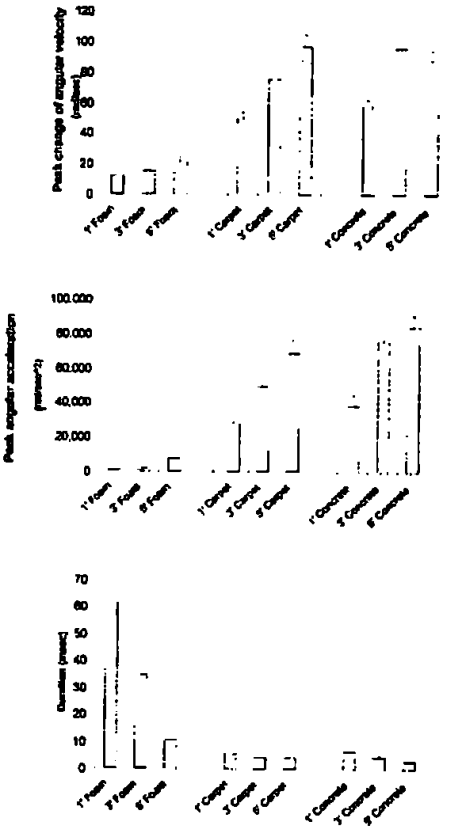

FIG. 4. Bar graphs showing the $\Delta\dot{\theta}_{max}$ *(upper)*, $\ddot{\theta}_{max}$ *(center)*, and Δt *(lower)* for falls from different heights onto different surfaces. The bars depict mean values and the error bars indicate standard errors.

Discussion

The focus of this study was to determine the rotational response of the head of an infant that is experienced during low-height falls and inflicted head injuries. Rotational motions have been shown to cause a diffuse pattern of strains and injury throughout the brain, whereas translational motion causes more focal damage.[37] For this reason, with the exception of epidural hematoma, falls have often been considered benign, because they are assumed to be essentially translational events. Nevertheless, although a fall may have predominantly translational components, its terminus (contact with an often immobile object) may produce significant rotational events and the brain may also experience rapid changes in rotational velocity and deceleration. It was the purpose of this study to measure these rotational events and to compare them with those created by shaking, impacts after free falls, and inflicted impacts.

In the impacts against carpet pad and concrete simulated in this study, the occiput made contact before the torso and then rotated, producing a significant angular motion of the head relative to the thorax. We found that the rotational accelerations and changes in rotational velocity experienced by the head during impact increased with the height of the fall. These larger rotational responses can be attributed to the higher linear velocities reached as drop height (and thus potential energy) increases. Harder surfaces absorb less energy than deformable materials during contact, causing the head to rebound more; the head thus experienced significantly larger rotational accelerations during contact with the concrete and the carpet pad, compared with contact with foam, during falls and inflicted impacts. Conversely, when the head contacted the foam it was pocketed in the foam, such that both torso and head moved together, producing only a very small rotational response. It should be emphasized that the foam material used in these tests was unencased, and the addition of a plastic or other covering might alter the deceleration pattern. For this reason, one cannot extrapolate tests performed using unencased foam to impacts against a covered mattress or padded furniture.

Anthropomorphic simulations

Head rotations during shakes occurred over significantly longer time periods than any other event. Thus, although shakes had a similar $\Delta\dot{\theta}_{xx}$ to that of a 0.3-m (1-ft) fall onto concrete and carpet pad, this change in velocity occurred over a much longer period, producing a significantly lower $\ddot{\theta}_{xx}$. Furthermore, shakes had a similar $\ddot{\theta}_{xx}$ to that of falls onto the mattress foam, but because the Δt is longer, shakes had significantly higher $\Delta\dot{\theta}_{xx}$ values.

There has been much debate on whether shaking alone is sufficient to cause the typical primary brain injuries seen in inflicted neurotrauma in infancy, specifically, SDH and/or TAI, or whether impact is necessary. Recent evidence suggests that injury to the cervicomedullary junction may be found in some cases of fatal inflicted head injury, and the role of this finding in the pathophysiology of apnea, hypoxia, and secondary cellular events is, at present, incompletely understood. Regardless, the focus of this study was to investigate the biomechanical causes of primary brain injuries by using rotational forces as a benchmark for the incidence of acute intracranial events such as failure of parasagittal bridging veins or axonal tears. To mimic inflicted impacts, the shaking scenarios described earlier were concluded with an impact of the surrogate's occiput against one of three surfaces. Our results demonstrate that measurements made during inflicted impacts against a carpet pad were not significantly different from those made during impacts against a rigid surface (lab bench top). Although the pad was less stiff than the bench top, the carpet pad used was only 6.35 mm (0.25 in) thick and the force of the inflicted impact completely compressed the pad during impact. During impact the carpet pad therefore exhibited the same properties as the underlying bench top during the later stages of contact. Inflicted impacts against carpet pad and bench top produced a significantly greater $\Delta\dot{\theta}_{xx}$ and $\ddot{\theta}_{xx}$ than those experienced under all other conditions simulated in this study. In contrast, the inflicted impact against the thicker (10-cm) foam mattress did not fully compress the foam, resulting in a $\Delta\dot{\theta}_{xx}$ and $\ddot{\theta}_{xx}$ that were lower than those associated with inflicted impacts against harder surfaces and similar to those associated with shaking. To summarize, inflicted impacts against the carpet pad and bench top had a three times greater $\Delta\dot{\theta}_{xx}$ and a 39 times greater $\ddot{\theta}_{xx}$ than shaking. These results suggest a higher likelihood of injury from inflicted impacts against hard surfaces than from vigorous shaking, or from falls of 1.5 m or less.

Sixteen years ago, we published results found using a less sophisticated anthropomorphic dummy of a 1-month-old infant,[12] and found similar $\Delta\dot{\theta}_{xx}$ and $\ddot{\theta}_{xx}$ ratios between shakes and impacts to those in the current study. In the current study, we constructed a more biofidelic 1.5-month-old dummy by using skull and scalp material with properties similar to those in infants. We measured rotational velocity directly and expanded the study to include falls and a wide variety of contact materials. Despite these improvements, the current study has several limitations. The dummy designed for this study included a simplified representation of the infant skull and neck that potentially influenced loads during falls and impact events. First, the dummy's skull was made from a solid homogeneous 2.25-mm-thick sheet of copolymer polypropylene. The actual braincase of an infant consists of bone plates connected by compliant sutures that allow substantial deformation of the skull. The compliant skull deforms during the birthing process and permits normal expansion of the brain and soft tissues during infancy and childhood; it also allows large changes in shape during impact loading.[31] These skull deformations would slow the period of contact during an impact, and decrease the resulting angular accelerations. The solid plastic "skull" of the dummy is therefore not an exact representation of the separate bone and suture material of an infant, and it could cause an overestimate of the response measured during impact events. Studies including more accurate skull and suture representations, which can also be used to measure skull contact forces, are now underway; the important data obtained in these studies will be crucial for computational simulations of skull and brain injuries caused by these focal loads experienced during impacts.

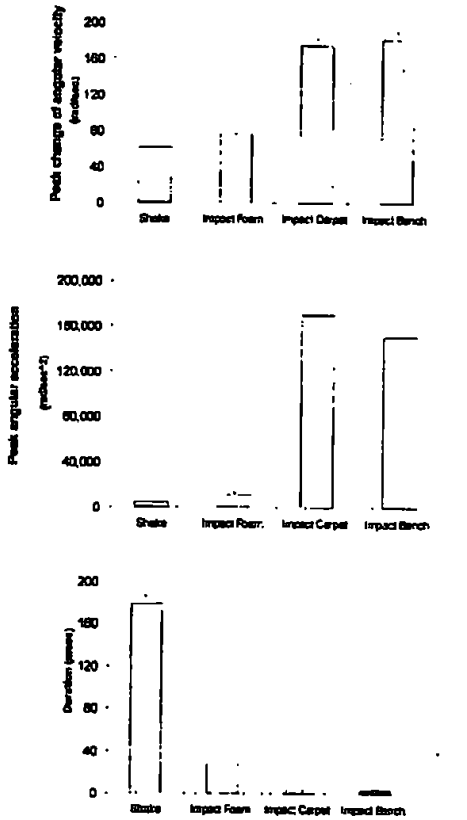

FIG. 5. Bar graphs showing the inflicted $\Delta\dot{\theta}_{xx}$ *(upper)*, $\ddot{\theta}_{xx}$ *(center)*, and Δt *(lower)* for shakes and impacts against different surfaces.

A second limitation of the current study is the inability of the surrogate's neck to mimic the properties of a real infant's neck. At present, no detailed quantitative information is available to validate the biomechanical properties of the human infant neck. Qualitatively, as a child matures, the neck stiffness increases, offering more resistance to a rotational motion of the head relative to the torso. Nevertheless,

young infants (< 2 months old) have little muscle tone in their necks and cannot support the weight of their heads.[9] Thus, the low-resistance hinge representation may be appropriate for a newborn but not an older child. Furthermore, the cervical spine consists of a series of vertebrae, allowing for rotation of the neck in different locations and directions; the hinge used to model the neck motion in the dummy has a fixed point of rotation, only allowing anteroposterior flexion and extension. The hinge does not provide resistance to motion or dampen responses between the torso and neck. More accurate kinematics of the infant neck would allow for translation as well as rotation of the head, and a moving center of rotation. These differences in the kinematics of the neck result in an overestimation of the rotational motions experienced during falls and inflicted events. Although the biofidelity of the surrogate has not been established, these experiments do provide an upper boundary for the measured head response to shaking, inflicted impact, and falls. When data are available on properties of the neck, a more biofidelic model should be created to ensure accurate measurements during inflicted and accidental injury scenarios.

A third limitation of the study is that the model represents a child in the 50th percentile for body and head mass. A heavier child with the same neck development would experience higher impact energy in a fall or inflicted impact due to the larger mass, and likely would experience a concomitant larger rotational velocity and acceleration than a smaller child. It is difficult to speculate, however, whether the volunteers could have generated the same peak acceleration with a heavier child. Considering that each sequence was a maximum effort, it is likely that the shakes would have resulted in a lower acceleration and velocity in a heavier dummy. Thus we would anticipate a greater disparity among the data obtained during shakes, impacts, and falls with increasing body and head mass.

Subdural hematoma and TAI are among the most common findings in serious head injuries in infancy and in those associated with nonaccidental causes. Both these injury types have been produced by, and correlated to, the angular velocity or angular acceleration of the head.[1,15,16,32] Rotational motions have been shown to cause a diffuse pattern of strains and injury throughout the brain, whereas translational (linear) motion causes more focal damage.[24,37] Although the anthropomorphic dummy test data are useful to evaluate the rotational response of the head caused by falls and inflicted injury events, the results of the dummy tests cannot be used to predict whether such rotations are sufficient to cause injury. Regional tissue thresholds specific to the infant would be required to predict injury on the basis of local intracranial stresses or strains produced by the rapid rotations. Such thresholds are currently unavailable for the pediatric population. In lieu of this information, we used a more qualitative approach to determine injuries likely to occur during simulated events. Specifically, we correlated measured accelerations and changes in velocity with injuries documented from controlled cadaver, animal, and human experiments in which the response is often measured directly and the exact details of the injury event are carefully recorded. Using dimensional analysis, angular velocities and accelerations from the different animal, human, and cadaver experiments were scaled to the infant as a function of brain mass (420 g).[38] These results were compared with the different rotational responses measured in the minor fall and inflicted impact events simulated in the dummy experiments performed in this study.

Impacts from falls from 0.3, 0.9, and 1.5 m onto mattress foam, and from 0.3 m (1 ft) onto carpet pad produced the lowest rotational accelerations and changes in velocity of all falls examined in this study. Weber[34] found only a 10% chance of skull fracture when infant cadavers were dropped from a similar height onto a similar surface. No data have been collected from animal and human experiments that were conducted at the low levels of $\ddot{\theta}_{res}$ and $\Delta\dot{\theta}_{res}$ measured during falls onto foam. Values of $\ddot{\theta}_{res}$ and $\Delta\dot{\theta}_{res}$ similar to those measured during 0.3-m falls onto carpet pad were recorded from head rotations of instrumented models of boxers with no occurrence of concussion, skull fracture, SDH, or TAI of the brain.[39] All cases of SDH and DAI in human cadaver studies and primate (rhesus monkey and baboon) rotational inertial experiments[1,15,32] had considerably greater angular accelerations and velocities when scaled to a human infant than an average $\ddot{\theta}_{res}$ and $\Delta\dot{\theta}_{res}$ produced by falls onto foam. Correlating these experimental data, it is highly unlikely that serious or fatal injuries occur during falls onto an unencased foam mattress from a height of 1.5 m or less, or from 0.3 m falls onto a carpet pad.

At the rotational responses calculated during impacts after 0.3-m (1-ft) falls onto concrete and 0.9-m (3-ft) falls onto carpet pad, it is not clear if serious injuries occur. The experimental evidence shows both the absence and occurrence of serious head trauma. Specifically, although human infant cadaver drop test studies have demonstrated an 80 to 100% occurrence of skull fractures,[44,53] subhuman primate inertial studies[1,15,32] and adult cadaver studies[35] have shown both the presence and absence of intracranial hemorrhage and acute SDH.

No experimental data could be found with a $\ddot{\theta}_{res}$ and $\Delta\dot{\theta}_{res}$ similar to those of the most severe falls, that is, 1.5-m (5-ft) and 0.9-m (3-ft) falls onto concrete and 1.5 m (5 ft) falls onto carpet pad. At lower values of $\ddot{\theta}_{res}$, however, impacts to the cadaver head caused intracranial bleeding, and nonhuman primates experienced SDH and DAI. The absence of data at this highest tier underscores our uncertainty regarding the occurrence of serious injury at these levels. These falls represent an extreme limit, producing the maximum rotation because of the hinge neck, the occipital contact site, and the purely sagittal rotation. In reality a fall is likely to include mixed rotational directions that would decrease the actual acceleration in the sagittal plane and the potential for SDH. For these idealized situations of falls onto hard surfaces, experimental data thus support at least the possibility of intracranial injuries caused by these most severe falls measured in this study.

There were no experimental data at values of $\ddot{\theta}_{res}$ and $\Delta\dot{\theta}_{res}$ similar to those measured during shaking or inflicted impacts against foam. For example, shakes and inflicted impacts against foam each had a $\Delta\dot{\theta}_{res}$ similar to that associated with noninjurious head rotation in boxers,[39] but these human tests produced a significantly greater $\ddot{\theta}_{res}$. The most severe inflicted impact against foam approached, but was still less than, the values of $\Delta\dot{\theta}_{res}$ and $\ddot{\theta}_{res}$ that produced SDH in adult rhesus monkeys and cadavers.[1,15,32] To summarize, there are no data demonstrating that the $\Delta\dot{\theta}_{res}$ and $\ddot{\theta}_{res}$ experienced during shaking and inflicted impact against foam cause SDH or TAI in infants.

Finally, although the $\ddot{\theta}_{max}$ and $\Delta\dot{\theta}_{max}$ measured during inflicted impacts against carpet pad and rigid bench top were significantly greater than those associated with all other scenarios tested in this study, no animal, human, or cadaver experiments at these levels of $\ddot{\theta}_{max}$ have been published. The majority of inflicted impacts against these hard surfaces produced a $\Delta\dot{\theta}_{max}$ and $\ddot{\theta}_{max}$ greater than the scaled rotational responses that produced fatal acute SDH in adult primates and intracranial bleeding in adult cadavers.[1,15,35] Approximately half of these inflicted impacts also exceeded the scaled $\Delta\dot{\theta}_{max}$ and $\ddot{\theta}_{max}$ that produced axonal injury in adult baboons.[32] Given this experimental data, angular velocities and accelerations measured during inflicted impacts against hard surfaces would likely produce SDH and, possibly, TAI in an infant.

These injury projections should be interpreted with caution, because differences in species, age, material properties, geometry, and direction make scaling experimental angular acceleration and velocity measurements to infants problematic when based on differences in brain mass alone. To avoid the limitations of using scaled loads from animal and cadaver experiments to investigate real life events, case studies of minor falls in infants were also used to examine injuries that occur as a result of falling from different heights. Unfortunately, these falls are rarely witnessed, load measurements of the event are lacking, contact surface information is rarely given, and the population studied generally includes a broad age range, rather than just newborns. To increase the specificity of our comparisons, we included only case studies of children reported to be younger than 3 years old.

Skull fracture has been reported as a result of minor falls in children younger than 3 years.[11,18,19,23,27,47,52,53] Reports of falls 0.6 m or less note an absence of skull fracture.[44,48,53,55] Studies have documented cases of skull fracture from minor falls from heights estimated to be 0.9 m (3 ft)[18,19,47,52] and 1.2 to 1.5 m (4–5 ft)[55] with no fatalities. Importantly, several studies have also shown an increase in the risk of skull fracture with the increased hardness of surfaces contacted after a free fall.[27,54] To summarize, these case study data provide evidence that minor falls (< 1.5 m) can cause skull fracture, especially if the impact occurs on a hard surface.

Case studies of infants younger than 3 years old have shown that SDH, TAI, and death are rarely caused by impact from falls from 1.5 m (5 ft) or less. In a 3.5-year retrospective study, Chadwick and colleagues[8] found no fatalities resulting from 1.5- to 2.75-m (5–9-ft) falls. In that study the authors did find three to seven deaths in children younger than 3 years of age with a history of falling from approximately 0.6 to 1.2 m (2–4 ft), but the majority of these cases had associated injuries attributed to abuse. Cases of playground falls reported by Plunkett[40] revealed only six fatal falls ranging from 0.6 to 1.8 m during an 11.5-year period. The causes of death in all six children were determined to be SDH or cerebral edema. Studies of falls occurring in hospitals from an estimated height of 0.61 to 1.2 m (2–4 ft) do not show any case of SDH or death.[22,30,36] Other case studies have shown no incidence of SDH or death caused by falls from 0.9 m (3 ft).[48,55] To our knowledge, no case study contains a report of serious brain injuries or death caused by falls from 0.3 m (1 ft) or less. Similar to the results of the animal and cadaver experimental data, these data show that, although the possibility exits, SDH and death from minor falls are unlikely.

Conclusions

This paper presents the rotational response of the head of an infant, measured using an anthropomorphic dummy, during minor falls, shakes, and inflicted impacts. In general, the $\ddot{\theta}_{max}$ and $\Delta\dot{\theta}_{max}$ increased with increasing fall height and surface hardness. The measured angular velocity and acceleration during minor falls were similar to those associated with shaking; however, inflicted impacts against hard surfaces produced a significantly greater $\ddot{\theta}_{max}$ and $\Delta\dot{\theta}_{max}$ than a 1.5-m fall onto concrete. Because larger rotational acceleration and velocities are associated with a higher likelihood of injury, these findings suggest that inflicted impacts against hard surfaces may be more frequently associated with clinically significant inertial brain injuries than vigorous shaking or falls from less than 1.5 m. In addition, there are no data showing that the $\Delta\dot{\theta}_{max}$ and $\ddot{\theta}_{max}$ of the head experienced during shaking and inflicted impact against unencased foam is sufficient to cause SDHs or primary TAIs in an infant.

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Neurosurgical forum
Letters to the editor

Aneurysm Size

TO THE EDITOR: I read with interest the article on the association of aneurysm size and the extent of subarachnoid hemorrhage (SAH) (Russell SM, Lin K, Hahn SA, et al: Smaller cerebral aneurysms producing more extensive subarachnoid hemorrhage following rupture: a radiological investigation and discussion of theoretical determinants. *J Neurosurg 99:* 248–253, August, 2003).

Abstract

Object. The goal of this study was to determine the relationship between aneurysm size and the volume of subarachnoid hemorrhage (SAH).

Methods. One hundred consecutive patients who presented with acute SAH, which was diagnosed on the basis of a computerized tomography (CT) scan within 24 hours postictus and, subsequently, confirmed to be aneurysmal in origin by catheter angiography, were included in this study. The data were collected prospectively in 32 patients and retrospectively in 68. The volume of SAH on the admission CT scan was scored in a semiquantitative manner from 0 to 30, according to a previously published method.

The mean aneurysm size was 8.3 mm (range 1–25 mm). The mean SAH volume score was 15 (range 0–30). Regression analysis revealed that a smaller aneurysm size correlated with a more extensive SAH ($r^2 = 0.23$, $p < 0.0001$). Other variables including patient sex and age, intraparenchymal or intraventricular hemorrhage, multiple aneurysms, history of hypertension, and aneurysm location were not statistically associated with a larger volume of SAH.

Conclusions. Smaller cerebral aneurysm size is associated with a larger volume of SAH. The pathophysiological basis for this correlation remains speculative.

Data on mean SAH volume scores for aneurysms grouped by size (as presented in Table 5 and Fig. 3 in the article) seem to be inconsistent. As shown in Table 5, an aneurysm size of 6 to 10 mm was associated with a mean SAH volume score of 10.7, whereas a mean score of about 13 is depicted in Fig. 3. Similarly, data on larger aneurysms (11–15 mm, mean SAH volume score 5.1 compared with ~10) and greater than 15 mm (mean SAH volume score 1.6 compared with ~8) are not corresponding. Given the high variability of the SAH volume scores, I wonder if the presentation of the data is erroneous.

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RESPONSE: The data presented in Table 5 and Fig. 3 were reviewed using our statistical analysis program. The means graphically depicted in Fig. 3 were correct, but the means reported in Table 5 were incorrect. The probability value remained highly significant, however. We have provided a revised version of Table 5. We apologize for the inaccuracy, and thank Dr. Hasselblatt for highlighting this error to the readership.

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TABLE 5

*Mean SAH volume scores for aneurysms grouped by size**

Aneurysm Size (mm)	No. of Lesions	Mean SAH Vol Score†	SEM
1–5	35	20.2	1.3
6–10	39	14.0	1.3
11–15	18	9.9	1.9
>15	8	8.8	2.8

* SEM = standard error of the mean.

† The mean volume of SAH decreases with increasing aneurysm size ($p < 0.0001$).

Rotational Injury

TO THE EDITOR: This article (Prange MT, Coats B, Duhaime AC, et al: Anthropomorphic simulations of falls, shakes, and inflicted impacts in infants. *J Neurosurg 99:* 143–150, July, 2003) extends the work of two of the authors (Duhaime and Margulies) published by this journal 16 years ago.[1] The methodology used in the most recent paper, although more complex, seems to have produced results similar to the original work in one important aspect.

Abstract

Object. Rotational loading conditions have been shown to produce subdural hemorrhage and diffuse axonal injury. No experimental data are available with which to compare the rotational response of the head of an infant during accidental and inflicted head injuries. The authors sought to compare rotational deceleration sustained by the head among free falls, from different heights onto different surfaces, with those sustained during shaking and inflicted impact.

Methods. An anthropomorphic surrogate of a 1.5-month-old human infant was constructed and used to simulate falls from 0.3 m (1 ft), 0.9 m (3 ft), and 1.5 m (5 ft), as well as vigorous shaking and inflicted head impact. During falls, the surrogate experienced occipital contact against a concrete surface, carpet pad, or foam mattress. For shakes, investigators repeatedly shook the surrogate in an anteroposterior plane; inflicted impact was defined as the terminal portion of a vigorous shake, in which the surrogate's occiput made contact with a rigid or padded surface. Rotational velocity was recorded directly and the maximum (peak–peak) change in angular velocity ($\Delta\dot{\theta}_{xx}$) and the peak angular acceleration ($\ddot{\theta}_{xx}$) were calculated.

Analysis of variance revealed significant increases in the $\Delta\dot{\theta}_{xx}$ and $\ddot{\theta}_{xx}$ associated with falls onto harder surfaces and from higher heights. During inflicted impacts against rigid surfaces, the $\Delta\dot{\theta}_{xx}$ and $\ddot{\theta}_{xx}$ were significantly greater than those measured under all other conditions.

Conclusions. Vigorous shakes of this infant model produced rotational responses similar to those resulting from minor falls, but inflicted impacts produced responses that were significantly higher than even a 1.5-m fall onto concrete. Because larger accelerations are associated with an increasing likelihood of injury, the findings indicate that inflicted impacts against hard surfaces are more likely to be associated with inertial brain injuries than falls from a height less than 1.5 m or from shaking.

One of the conclusions of the original paper is ". . . the shaken baby syndrome, at least in its most severe acute form, is 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." In the current work, the authors state: "In addition, there are no data showing that the $\Delta\Theta_{max}$ and the Θ_{max} of the head experienced during shaking and inflicted impact against unencased foam is sufficient to cause SDHs [subdural hematomas] or primary transient ischemic attacks in an infant."

Given the above conclusion by the same authors who conducted the same experiments with differing methodologies, published in the same journal 16 years apart, it would seem appropriate at this point to pose the question, "Why, then, need shaking be invoked as a causative mechanism in intracranial injury in infants at all, since according to both series of experiments, it does not seem to be a factor in causing such injury?"

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Reference

1. Duhaime AC, Gennarelli TA, Thibault LE, et al: The shaken baby syndrome. A clinical, pathological, and biomechanical study. J Neurosurg 66:409–415, 1987

RESPONSE: Whereas our recent paper has a considerably broader focus that emphasizes falls and the influence of contact surface, Uscinski, Thibault, and Ommaya are correct in pointing out consistency with results reported in our earlier paper.[1] In both papers we measure relatively small rotational velocities and/or accelerations during vigorous shaking, and low height falls are below the thresholds for severe primary brain injuries. At these levels, however, there is a paucity of data in humans, animals, and cadavers, and therefore we cannot postulate whether brain injury would be associated with these events. To summarize, new research is needed to determine if injuries can occur in the brain, cervicomedullary junction, or cervical spinal cord as a result of a single or series of head rotations at these low magnitudes, and if these injuries are primary or secondary in nature. Therefore, we cannot yet answer if shaking can cause intracranial injury in infants, and use of terminology that includes this mechanism should be avoided.

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Reference

1. Duhaime AC, Gennarelli TA, Thibault LE, et al: The shaken baby syndrome. A clinical, pathological, and biomechanical study. J Neurosurg 66:409–415, 1987

Cerebral Metabolism

TO THE EDITOR: I read with interest the article by Zauner, et al. (Zauner A, Clausen T, Alves OL, et al: Cerebral metabolism after fluid-percussion injury and hypoxia in a feline model. *J Neurosurg* 97:643–649, September, 2002).

Abstract

Object. Currently, there are no good clinical tools to identify the onset of secondary brain injury and/or hypoxia after traumatic brain injury (TBI). The aim of this study was to evaluate simultaneously early changes of cerebral metabolism, acid–base homeostasis, and oxygenation, as well as their interrelationship after TBI and arterial hypoxia.

Methods. Cerebral biochemistry and O_2 supply were measured simultaneously in a feline model of fluid-percussion injury (FPI) and secondary hypoxic injury. After FPI, brain tissue PO_2 decreased from 33 ± 5 mm Hg to 10 ± 4 mm Hg and brain tissue PCO_2 increased from 55 ± 2 mm Hg to 81 ± 9 mm Hg, whereas cerebral pH fell from 7.1 ± 0.06 to 6.84 ± 0.14 ($p < 0.05$ for all three measures). After 40 minutes of hypoxia, brain tissue PO_2 and pH decreased further to 0 mm Hg and 6.48 ± 0.28, respectively ($p < 0.05$), whereas brain tissue PCO_2 remained high at 83 ± 13 mm Hg. Secondary hypoxic injury caused a drastic increase in cerebral lactate from 513 ± 69 μM/L to 3219 ± 490 μM/L ($p < 0.05$). The lactate/glucose ratio increased from 0.7 ± 0.1 to 9.1 ± 2 after hypoxia was introduced. The O_2 consumption decreased significantly from 18.5 ± 1.1 μl/mg/hr to 13.2 ± 2.1 μl/mg/hr after hypoxia was induced.

Conclusions. Cerebral metabolism, O_2 supply, and acid–base balance were severely compromised ultra-early after TBI, and they declined further if arterial hypoxia was present. The complexity of pathophysiological changes and their interactions after TBI might explain why specific therapeutic attempts that are aimed at the normalization of only one component have failed to improve outcome in severely head injured patients.

In this study of experimental brain trauma the authors monitor the cerebral energy metabolism and O_2 consumption rate and arrive at conclusions with direct clinical implications. The experimental approach of using a technique for cerebral microdialysis that is available also during neurointensive care is of particular interest. Unfortunately, the study is burdened with severe methodological problems, which render the conclusions questionable.

Evaluation of extracellular or whole tissue lactate/pyruvate ratio is a generally accepted decades-long way of describing cerebral cytoplasmic redox states.[1] The lactate/pyruvate ratio reflects the equilibrium of the lactate dehydrogenase (LDH) reaction and, irrespective of the species studied, the cerebral lactate/pyruvate ratio is close to 20 under physiological conditions. During ischemia and hypoxia there is a rapid shift in the LDH equilibrium and the ratio increases to very high levels due either to a simultaneous increase in lactate and decrease in pyruvate (ischemia, hypoxia with relative ischemia) or to a pronounced increase in lactate and a moderate increase in pyruvate (pure hypoxia). In studies using microdialysis the relative recoveries of lactate and pyruvate are similar and the lactate/pyruvate ratio is accordingly not affected by technical variations such as length of dialysis membrane, perfusion rate, and so on. In the study by Zauner, et al., the lactate/pyruvate ratio was 306 ± 176 in control animals. This value is an obvious technical artifact. It is also difficult to understand how the measured mean levels of lactate (513 ± 69 μM/L) and pyruvate (8 ± 2.8 μM/L) can result in this mean lactate/pyruvate ratio. Since the given lactate/pyruvate baseline value does not represent a control level, it is not possible to interpret the (nonexisting) effects of FPI or subsequent hypoxia. In consideration of the experimental conditions (general anesthesia, 10 mm dialysis membrane, perfusion rate 2 μl/

Appendix

I

Cases: *See, e.g., Del Prete v. Thompson*, 10 C 5070, 2014 WL 296094 (N.D. Ill. Jan. 27, 2014) (discussing lucid intervals, biomechanics, rebleeds); *Ex parte Henderson*, 384 S.W.3d 833 (Tex. Crim. App. 2012) (discussing accidental causes); *Aleman v. Vill. of Hanover Park*, 662 F.3d 897 (7th Cir. 2011) (discussing the agreement that a lucid interval was likely); *State v. Edmunds*, 308 Wis. 2d 374, 2008 WI App 33, 746 N.W.2d 590, 593 (Ct. App. 2008) (discussing alternative causes, lucid intervals, biomechanics, and the shift in medical opinion).

Medical literature: *See, e.g.*, Mark Donohoe, *Evidence-Based Medicine and Shaken Baby Syndrome Part I: Literature Review, 1966-1998*, 24 Am. J. Forensic Med. Pathology 239, 241 (2003) (concluding 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."); 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); Ronald H. Uscinski, *Shaken Baby Syndrome: An Odyssey*, 46 Neurol. Med. Chir. (Tokyo) 57 (2006) ("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."); Waney Squier & Julie Mack, *The Neuropathology of Infant Subdural Haemorrhage*, 187 Forensic Sci. Int. 6 (2009) (concluding that the very specific anatomy and physiology of the infant dura should cause us to consider alternative mechanisms to bridging vein rupture as the source of SDH in this age group); 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).

Secondary Sources: *See, e.g.*, Deborah Tuerkheimer, *The Next Innocence Project: Shaken Baby Syndrome and the Criminal Courts*, 87 Wash. Univ. L. Rev. 1, 5, 18 (2011); 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) (setting forth the support for SBS/AHT and containing multiple references to the medical literature); Keith A. Findley, Patrick D. Barnes, David A. Moran, Waney Squier, *Shaken Baby Syndrome, Abusive Head Trauma, and Actual Innocence: Getting It Right*, 12 Hous. J. Health L. & Pol'y 209, 312 (2012) (responding to the Narang article and containing multiple references to the medical literature).

Appendix J

FILED

APR 16 2010

SONYA KRASKI
COUNTY CLERK
SNOHOMISH CO. WASH.

SUPERIOR COURT OF WASHINGTON
FOR SNOHOMISH COUNTY

THE STATE OF WASHINGTON, Plaintiff, v. MORRIS, MICHAEL J. Defendant.	No. 09-1-02223-7 MOTION FOR A PRELIMINARY DETERMINATION OF WHETHER OR NOT A FRYE HEARING IS NECESSARY

A preliminary hearing for April 30, 2010 was noted by a court order that entered on March 12, 2010 to determine whether or not a Frye hearing is necessary in this case. The defense desires a Frye hearing to decide if the previous general acceptance of Shaken Baby Syndrome in the mainstream medical community can now be challenged. The issue on April 30th is whether or not the defense can make an adequate offer of proof to support having a Frye hearing. If a Frye hearing is ordered, we will then need this case pre-assigned to a judge for a multiday Frye hearing.

Respectfully submitted this 15 day of April, 2010.

HALLEY R. NUPP, #23331
Deputy Prosecuting Attorney

AFFIDAVIT OF CERTIFICATION:

I am a duly appointed Deputy Prosecuting Attorney for Snohomish County and make this affidavit in that capacity. This affidavit is based upon information and belief that:

The Defendant, Michael J. Morris, is currently charged with Assault of a Child in the Second Degree (DV) that occurred on May 29, 2009 and involved his 6 week old daughter, A.M.

The Defendant was interviewed out-of-custody by the Brier police department. He voluntarily came to the police station for the interview. Once there, even though he was out of custody, he was read his Miranda rights, which he waived, and during his interview with police he did admit to shaking his daughter twice during the time period she sustained her injuries.

The Defendant told police that he was the sole person in charge of his daughter during the time of the incident, that he did shake his daughter twice, and that afterwards he ran with his daughter in his arms to the neighbors house. He stated that his daughter's head injury may have occurred while he was running because he did not have her head properly supported. He also remembered that he was holding his daughter one time when she got a hold of a knife, and he "kind of dropped" her on his lap when he went for the knife.

The Defendant later e-mailed his wife indicating that he did shake A.M.

A.M. has suffered traumatic brain injury that will impact her for the rest of her life. When she left the hospital she was still on a feeding tube and was having difficulty moving her arm on the right side of her body. There is a strong likelihood that she will be blind.

The evidence is that before A.M. was in her father's care that night she was healthy and having no issues or distress. There is no other evidence of a third party having contact with the child during this time.

Medical tests were conducted on A.M. She was found to have a bruise under her left chin. She had bilateral retinal hemorrhages. There was no indication of fractures, broken

bones, or scalp swelling. She was found to have significant brain injury, which included prominent intracranial bleeding.

Dr. Feldman with Children's Hospital was involved with her case. He indicated that A.M.'s injury was attributed by him to severe whiplash acceleration forces. Intracranial hemorrhages and severe retinal hemorrhages accompanied by schesis cavities such as A.M. sustained, result from this type of head injury mechanics. He further indicated that A.M. lacked either physical or radiological findings to indicate that she had impact injury to the head. He did indicate that impact against a soft surface would increase the experienced whiplash deceleration forces and likely would not leave evidence of surface or skull impact trauma, and as such he cannot exclude an associated impact.

Dr. Feldman at trial will testify regarding the physical findings and his expert opinion regarding the abusive head trauma in this case.

I certify (or declare) under penalty of perjury under the laws of the State of Washington that the foregoing is true and correct.

HALLEY R. HUPP, #23331
Deputy Prosecuting Attorney

DATED: 4/15, 2010, at the Snohomish County Prosecutor's Office.

ARGUMENT

A Frye hearing is needed when a party seeks to introduce novel scientific evidence. The purpose of a hearing is to test the reliability of new science. Shaken Baby Syndrome has been used in the medical literature since 1962. It is merely a term which is used to describe a

constellation of medical symptoms. It is merely a descriptive term. There is nothing new or novel about it.

To be admissible, expert testimony concerning novel scientific evidence must both satisfy both Frye and ER 702. State v. Copeland, 130 Wash.2d 244, 256, 922 P.2d 1304 (1996). **Frye analysis need not be undertaken with respect to evidence that does not involve new methods of proof or new scientific principles from which conclusions are drawn.** (emphasis added). Ruff v. Department of Labor and Industries of State of Washington, 107 Wn.App. 289, 300, 28 P.3d 1 (2001)(citing State v. Russell, 125 Wn.2d 24, 69, 882 P.2d 747 (1994).

Courts across this country have from the past to the present found Shaken Baby Syndrome to be generally accepted in the medical community. The Court in State v. Leibhart, 266 Neb. 133, 662 NW2d 618 (2003) wrote

> In this respect, we note that expert testimony regarding shaken baby syndrome has been previously admitted by courts in this state." See, State v. Reynolds, 240 Neb. 623, 483 N.W.2d 155 (1992); State v. Wolcik, 238 Neb. 883, 472 N.W.2d 732 (1991). See, also, Farm Bureau Ins. Co. v. Witte, 256 Neb. 919, 594 N.W.2d 574 (1999). We also note that for some time, courts in other states have found shaken baby syndrome to be a generally accepted diagnosis in the medical community. State v. Lopez, 306 S.C. 362, 412 S.E.2d 390 (1991); State v. McClary, 207 Conn. 233, 541 A.2d 96 (1988); Matter of Lou R., 131 Misc.2d 138, 499 N.Y.S.2d 846 (1986).

In State v. McClary, 207 Conn 233, 541 A.2d 96 (1988) the court wrote:

> "[T]his court now finds that 'shaken baby syndrome' (sometimes denoted as 'shaken infant syndrome' or 'shaken child syndrome' ...) is a generally recognized medical condition, and 'the state of the pertinent art or scientific knowledge [is] sufficiently developed to permit a reasonable opinion to be asserted' (People v. Torres, [128 Misc.2d 129, 134, 488 N.Y.S.2d 358 (1985)])." Matter of Lou R., 131 Misc.2d 138, 142, 499 N.Y.S.2d 846 (1986). "[T]he theory of cause of death here, violent shaking, resulting in subdural hematoma, has been accepted as legally sufficient in other cases." State v. Evans, 74 N.C.App. 31, 36, 327 S.E.2d 638 (1985); see also Dabbs v. State, 518 So.2d 825 (Ala.Cr.App.1987); People v. Kelley, 662 P.2d 168, 170 (Colo.1983); State v. Ostlund, 416 N.W.2d 755, 760-61 (Minn.App.1987); State v. Munici. Slip. Op. 1987 WL 20206, Ohio App.

Cuyahoga County, No. 52579 (Nov. 19, 1987); State v. Schneider, Slip. Op. 1984 WL 3719, Ohio App. Lucas County CA No. 6-84-214 (Dec. 21, 1984).

In Washington State evidence of "Shaken Baby Syndrome" was admitted in State v. Daniels, 124 Wn.App 830, 103 P.3d 249 (2004) and State v. Fero, 125 Wn.App. 84, 104 P.3d 49 (2005), as well as, numerous unpublished cases. The list of court cases is endless, but the fact is that the defense is at this point challenging something that has been a generally accepted medical condition for a very long time.

Further, in this case the Defendant admitted to shaking the victim. Dr. Feldman will simply testify that shaking can cause the injuries presented. He will also testify regarding the degree of force necessary to cause the injuries. He will not be testifying to novel scientific evidence.

In order for a Frye hearing to be warranted in this case the defense must show that there has been a change in the mainstream medical community and that the community no longer finds the constellation of injuries that have been labeled Shaken Baby Syndrome ("SBS") to be generally accepted as indications of abusive head trauma.. They cannot make that offer of proof because there has been no such change. The American Academy of Pediatrics published an article in "Pediatrics" in May 2009 entitled "Abusive Head Trauma in Infants and Children" that recognized that neurologic injury can occur from both shacking a child and/or blunt impact.

A preliminary hearing is warranted for making this determination because otherwise either party could demand a Frye hearing in every criminal or civil case that contains scientific testimony, even if the scientific evidence is not novel, which would substantially delay the court's

time as well as generate huge costs for the citizens of Washington State and Snohomish County.

HALLEY R. HUPP, #23331
Deputy Prosecuting Attorney

DATED: 4/15, 2010, at the Snohomish County Prosecutor's Office.

Appendix K

RECEIVED
APR 0 - 2010
PROSECUTING ATTORNEY
FOR SNOHOMISH COUNTY
BY
FOR

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SONYA KRASKI
COUNTY CLERK
SNOHOMISH CO. WASH.

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

The State of Washington,

Plaintiff,

vs.

MICHAEL J. MORRIS,

Defendant.

09-1-01071-9

DEFENSE REPLY BRIEF

I. FACTS AND PROCEDURAL POSTURE

On May 29, 2009, Michael Morris was caring for his infant daughter, Alexis, while his wife took their other child to a doctor's appointment. As Mr. Morris fed Alexis, she began to cough and choke. Mr. Morris attempted to help clear her airway, but she soon began to turn blue. At that point, Mr. Morris attempted to revive Alexis but was not successful. He ran across the street to a neighbor's home to get a ride to the hospital.

Mr. Morris contacted his wife on the way to the emergency room and she met him there, taking Alexis inside. While at Stevens, a number of scans and tests were run and Alexis was intubated. She was shortly transferred to Harborview Medical Center, then to Children's Hospital after a few days.

The first scans taken of Alexis's brain at Stevens and Harborview showed no mass effect and that the white and grey matter of the brain were "normal in density."

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The intial scans showed a "tiny" 1 millimeter subdural hemorrhage. Further, the scans showed no edema or hydrocephalus. These initial scans also showed fluid and/or other blockage in Alexis' mastoid air cells and middle ear.

Dr. Feldman reviewed these scans as well as scans taken later at Children's Hospital, noting in his report dated May 31, 2009 (but edited and submitted on June 1, 2009), "patchy alteration of gray-white distinction." Dr. Feldman indicated a number of tests would be "helpful re timing of her injuries" but did not later expound on whether these tests were done and what the results were.

At a pretrial hearing on March 12, 2010, the defense indicated to the State that they would be submitting briefing and requesting a Frye hearing on the issue of whether the term "Shaken Baby Syndrome" was currently scientifically accepted and could be used at trial. The State indicated that the defense was required to have a hearing to request a Frye hearing but the defense was not in agreement. The State then filed this motion.

II. A PRELIMINARY HEARING IS NOT WARRANTED WHEN A PARTY REQUESTS A FRYE HEARING

The State indicates, without cited authority, that "in order for a Frye hearing to be warranted in this case, the defense must show that there has been in a change in the mainstream medical community and that the community no longer finds the constellation of injuries that have been labeled Shaken Baby Syndrome (SBS) to be generally accepted as indications of abusive head trauma." This is an incorrect statement of the law and an inaccurate recitation of how Frye challenges are handled in Washington.

For some pretrial issues, a hearing is required to determine if a criminal defendant will be able to challenge pieces of evidence. This is the case in for a Franks hearing, in which a defendant seeks to challenge the truthfulness of factual statements

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in an affidavit for a search warrant. Franks v. Delaware, 438 U.S. 154, 98 S.Ct. 2674, 57 L.Ed.2d 667 (1978). In the case of a Franks hearing, a preliminary offer of proof is required and the defense must make "substantial preliminary showing that a false statement knowingly and intentionally, or with reckless disregard for the truth, was included by the affiant in the warrant affidavit." Id. at 155 – 156. This is because the court begins with a presumption that the affidavit supporting the search warrant is valid, which then shifts the burden to the defense to show that it is not. Id.

No such presumption or shifting of burden exists in the case of a Frye hearing. The burden remains on the party who is offering the evidence to show that it is generally accepted in the scientific community. At no time does the burden shift to the party challenging the evidence. Only *full* acceptance of a process in the relevant scientific community obviates the need for a Frye hearing. State v. Russell, 125 Wash.2d 24, 41, 882 P.2d 747 (1994), cert. denied, 514 U.S. 1129, 115 S.Ct. 2004, 131 L.Ed.2d 1005 (1995) (emphasis added).

The State cites to Ruff v. Department of Labor and Industries, 107 Wn.App. 289, 300, 28 P.3d 1 (2001) for the proposition that a Frye hearing is not required here. However, the holding in Ruff makes it clear that this case presents exactly the situation in which a Frye hearing *is* required. In Ruff, Division One delineated the differences in fact-specific situations applying scientific principles and those in which the principles themselves are being challenged. Ruff, 107 Wn.App. at 301. Here, the defense is not arguing that the term "Shaken Baby Syndrome" should not or does not apply to these facts, but instead that the diagnosis itself is no longer generally accepted and the terminology itself should be excluded from trial. The facts in Ruff are very similar to the case at hand – there medical testimony was ruled inadmissible because the expert's opinion as to causation and diagnosis did not meet the generally accepted thinking of

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the medical community. Ruff, 107 Wn.App. at 305. That is the exact argument that defense is making in this case.

In requesting a hearing to determine if a hearing is necessary, the State is essentially asking the Court to make a ruling without legal basis. There is no case law or statutory authority to determine what standard the court would use at this hearing. Must the defense make a showing by a preponderance to get a Frye hearing? By clear and convincing evidence? The State does not indicate what burden or methods of proof would be used here, nor whose burden it is. The State's request for a ruling on whether a hearing is necessary has no basis in law.

III. THE FRYE STANDARD

In Washington State, the standard for determining if evidence based on scientific procedures is admissible is that set forth in Frye v. United States, 293 F. 1013, 1014, 34 A.L.R. 145 (D.C.Cir.1923) (rejecting precursor to the modern polygraph test). The rule is well established:

> [E]vidence deriving from a scientific theory or principle is admissible only if that theory or principle has achieved general acceptance in the relevant scientific community.
>
> In Re the Personal Restraint of Young, 122 Wn.2d 1, 56 (1993).

Here however, in response to the State's motion, the question is not whether Shaken Baby Syndrome (SBS) meets the Frye standard, but rather whether a Frye hearing is merited at all.

IV. A FRYE HEARING IS NECESSARY

In recent years Shaken Baby Syndrome has come under intense scrutiny in both scientific literature and the criminal justice system. This alone merits a Frye hearing, where a determination as to whether the science of SBS does or does not meet the above cited Frye standard of general acceptance will be made.

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1. Past Admissibility Does Not Preclude a Frye Challenge

The State cites two cases where evidence of SBS was admitted (State v. Daniels, 124 Wn.App. 830, 103 P.3d 249 (2004) & State v. Fero, 125 Wn.App. 84, 104 P.3d 49 (2005)). However, simply because a general scientific practice is commonly recognized and has been found to be admissible in the past does not mean that such practice is immune from a Frye challenge. State v. Bander, 150 Wn.App 690, 712, 208 P.3d 1242 (2009). The cases cited by the State date from 2004 and 2005 and, in neither instance, did the trial nor appellate courts consider a Frye challenge to the scientific evidence regarding SBS.

2. Challenges to SBS in Scientific Literature

When courts attempt to integrate new scientific knowledge into law, it is often scientific literature that leads the way. Scientific literature should be considered here, to determine whether a Frye hearing is merited. In reviewing Frye challenges, appellate courts consider scientific literature above and beyond the trial record. State v. Sipin, 130 Wn.App. 403, 415, 123 P.3d. 862 (2005). It is likewise appropriate for trial courts to consider scientific literature, because "it is impractical in many instances for a true cross-section of scientists to testify at a hearing." Id., at 751 (citing State v. Copeland, 130 Wn.2d 244, 261 (1996). Here, where no experts will testify, it is clearly appropriate for the court to consider the same.

Recent scientific literature casts doubt on the validity of SBS, and therefore its admissibility under the Frye general acceptance standard. The most current citation in the State's motion is a May 2009 article in "Pediatrics" entitled "Abusive Head Trauma in Infants and Children." This article itself notes the changes in SBS science and acceptance:

> Although the term is well known and has been used for a number of decades, advances in the understanding of the mechanisms and clinical spectrum of injury associated with

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> abusive head trauma compel us to modify our terminology to keep pace with our understanding of pathologic mechanisms. Although shaking an infant has the potential to cause neurologic injury, blunt impact or a combination of shaking and blunt impact cause injury as well. Spinal cord injury and secondary hypoxic ischemic injury can contribute to poor outcomes of the victims. The use of broad medical terminology that is inclusive of all mechanisms of injury, including shaking, is required. (http://pediatrics.aappublications.org/cgi/content/full/123/5/1409?maxtoshow=&hits=10&RESULTFORMAT=1&title=abusive+head+trauma+in+infants+and+children&andorexacttitle=and&andorexacttitleabs=and&andorexactfulltext=and&searchid=1&FIRSTINDEX=0&sortspec=relevance&resourcetype=HWCIT)

Other scientific literature make the same argument, and stronger. This literature argues that "the scientific underpinnings of SBS have crumbled over the past decade as the medical establishment has deliberately discarded a diagnosis defined by shaking." *The Next Innocence Project: Shaken Baby Syndrome and the Criminal Courts*, Deborah Tuerkheimer, 87 Wash. U. L. Rev. 1, 11 (2009). Further still, some of the recent literature argues that reasons other than shaking are the cause of the harm in these types of cases:

> In spite of the many scientific studies that uncritically accept the existence of SBS, an adequate link between shaking a baby and the two SBS markers has never been scientifically verified and the latest evidence points strongly to organic reasons for the babies' distress, as opposed to shaking. *Shaken Baby Syndrome: A Questionable Scientific Syndrome and a Dangerous Legal Concept*, 2003 Utah L. Rev. 1109, Summary (2003).

Clearly, the most recent scientific literature, including the article cited by the State, indicates a change in the diagnostic criteria for the constellation of symptoms previously designated as "Shaken Baby Syndrome." At the very least, a change in terminology exists and should be explored via a Frye hearing.

3. Challenges to SBS in the Criminal Justice System

The criminal justice system has begun to recognize the flawed science of SBS:

"On January 31, 2008, Audrey Edmunds was granted a new trial on the basis of an evolution in scientific thinking. For the first time, a court examining the foundation of shaken baby syndrome (SBS) concluded that it had become sufficiently eroded that a new jury probably would have a reasonable doubt as to the defendant's guilt." *Tuerkheimer*, at 7 (citing *Edmunds*, 746 N.W.2d at 599).

The criminal justice system has begun to take note that new science has cast doubt on the reliability of SBS as a means of prosecution. Again, at the very least, this recognition merits a Frye hearing where this shift can be explored.

V. CONCLUSION

The procedural posture of this case, relevant case law and scientific and legal scholarship indicate the necessity of a Frye hearing. Further, the Court should not preclude the Defendant's right to challenge scientific evidence proposed to be presented against him without hearing from experts on both sides. A preliminary determination should not be made without this testimony and the defense requests a testimonial Frye hearing be scheduled before the June 11, 2010 trial date.

DATED this 27 day of April, 2010.

Respectfully submitted,

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Appendix L

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FILED
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SONYA KRASKI
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SNOHOMISH CO. WASH.

SUPERIOR COURT OF WASHINGTON
FOR SNOHOMISH COUNTY

THE STATE OF WASHINGTON, Plaintiff, v. MORRIS, MICHAEL J. Defendant.	No. 09-1-01071-9 RESPONSE TO DEFENSE REPLY BRIEF

I. Motion in Limine to not use the term "Shaken Baby Syndrome"

It appears from the defense brief that what they are really requesting is that neither the State nor the expert(s) involved in this case use the label "Shaken Baby Syndrome". This is a label that has been useful to make people aware of this issue, and to help doctors become aware of what to look for when treating a child. It is not a label that has been used by the experts in this case, nor is it a label the State plans on using at trial. The exception might be the articles expert(s) reference, but I would think the State and the defense can come to an agreement that allows the expert(s) to refer to the articles in this arena without using the actual label "Shaken Baby Syndrome."

II. The need for a Frye hearing has simply not be establish.

The defense in this case is confusing the label "Shaken Baby Syndrome" with the basic medical science being used in this case. The science used in this case is no different than a doctor testifying regarding whiplash in an auto accident case, a medical examiner talking about cause of death in a homicide case, a doctor testifying in a stabbing case, or someone at the crime lab testifying about chemicals found in the individual's blood. The expert in this case is not using some new novel scientific technique, gadget, theory, or method. In fact he is using the same medical science that would be applied if any of us went to the doctor after suffering whiplash. It is truly basic medical information that is generally accepted in the scientific community and it does not warrant a Frye hearing. Further, even if the label "Shaken Baby Syndrome" is falling out of favor, there is no need for a Frye hearing because the State is agreeing not use that label.

Dr. Feldman in his report dated 8/7/2009 writes that the injuries were attributed to severe whiplash acceleration forces. He indicated that the victim lacked either physical or radiological findings to indicate that she had impact injury to the head. He did indicate, however, that impact against a soft surface would increase the experienced whiplash deceleration forces and likely would not leave evidence surface or skull impact trauma. He will also testify that it is not plausible that a respiratory event caused her head trauma, though a respiratory event could have resulted from the head trauma.

The Defense has provided neither affidavits from experts nor any other evidence calling into question Dr. Feldman's findings in this case or calling into question the general acceptance of his theories and methods. Further, Dr. Feldman neither used the label "Shaken Baby

Syndrome", nor did he indicate that the victim's injuries were only consistent with injuries caused by shaking the victim.

If a defense expert is hired and does call into question Dr. Feldman's diagnosis, that would be appropriate testimony for trial, not for a Frye hearing. A Frye hearing is only required to establish that there is new novel science. Dr. Feldman's diagnosis is consistent with Abusive Head trauma, and as the defense indicates in their brief, Abusive Head Trauma was again recognized by the general scientific community as recently as May 2009.

III. The Next Innocence Project: Shaken Baby Syndrome and the Criminal Courts

The Defense response is really encapsulated by the controversial and very much criticized law review article of Deborah Tuerkheimer, The Next Innocence Project: Shaken Baby Syndrome and the Criminal Courts. The argument is that the last person with a deceased child should not be convicted solely by an expert saying the victim died of "Shaken Baby Syndrome". Inherent in that argument are facts like those in State v. Edmunds, 308 Wis.2d 374, 746 N.W.2d 590 (2008) where there is no other evidence, other than the expert(s), regarding who injured the victim and how the victim was injured.

This case is fundamentally different than Edmunds and the cases that are the focus of the law review article. The Defendant in this case admitted to holding the victim straight out from his chest and then shaking her for at least five seconds. He then admitted to shaking her again for another five seconds. Given the Defendant's admissions, there is no question at trial regarding whether or not he shook the victim. The question for the expert(s) is simply whether or not such shaking could cause the injuries in this case.

Deborah Tuerkheimer on page 10 of her law review article makes it clear that her article challenges convictions made solely on the triad of medical findings labeled "Shaken Baby Syndrome." However in footnote 58 on pages 10 and 11 she writes, "By this, I mean those whose convictions rest exclusively on the presence of retinal hemorrhage and/or subdural hematomas. In contrast, a sizeable number of SBS prosecutions rely on corroborative evidence beyond the triad; convictions which result in these cases are therefore less dramatically undermined by recent scientific developments." Stated in the context of this case, because the Defendant admitted he shook his child, the mechanism for the injury is really not at issue, and the medical evidence is offered to simply support that the behavior admitted to could in fact cause the injuries to the victim.

The defense argument and the law review article also both depend heavily on the Edmunds case. It is important to note that Edmunds case was not decided on a Frye standard, and the issue in that case was not whether or not "Shaken Baby Syndrome" was generally accepted in the scientific community. In that case the defense expert agreed with the State that the injuries were consistent with the child being shaken. The defense was that someone else could have shaken the child, and the child could have died later from the shaking. The appellate court in Edmunds overturned the case on the basis of new expert evidence that was discovered and that the defense did not use at trial. The ruling of the Edmunds court was not that "Shaken Baby Syndrome" could not be used at trial, but rather the defense should have the option to bring in expert(s) of their own to contradict that testimony. As such the court in Edmunds overturned the case and sent it back for a new trial.

IV. Conclusion

The defense in their reply brief argues that the "Court should not preclude the Defendant's right to challenge scientific evidence proposed to be presented against him without hearing from experts on both sides."

Pursuant to ER 702 it is appropriate for the defense to have an expert(s) testify at trial and present his/her opinion and expertise regarding the nature of the injuries in this case. The State has no objection to that. But, the right to challenge scientific evidence at trial, is different than requesting a Frye hearing. For there to be a Frye hearing the State's expert must be relying on scientific theory and/or method that is not generally accepted in the scientific community. Dr. Feldman will testify regarding the abusive head trauma he observed in this case and will give his expert opinion about the possible causes for it. The theories and methods he will rely on are basic medicine generally accepted in the scientific community, and have been accepted by courts throughout this country.

The State respectfully requests that Court denies the defense request for a Frye hearing.

HALLEY R. HUPP, #28331
Deputy Prosecuting Attorney

DATED: 4/28/2010, 2010, at the Snohomish County Prosecutor's Office.

Appendix M

Last, First MI:		DOB:	HGT:	WGT:	Hair:	Eye:
MORRIS, MICHAEL, J.		8/27/88	5'8"	163	B	B
Address:	**City:**			**State**	**Zip code:**	
2508 Nike manor Rd.	Brier			WA.	98036	
Home #:	**Work #:**		**Cell #:**			
(573) 424-1015			573-424-1015			
Date of Statement:	**Time:**		**Name of Officer:**			
JUNE 02, 2009	1205 pm		MURPHY			

Miranda Warnings Applicable ☑ Yes ☐ No

Initials

- You have the right to remain silent. MM
- Anything you say can be used against you in a court of law. MM
- You have the right at this time to talk to an attorney and have him present with you while you are being questioned. MM
- If you cannot afford to hire an attorney, one will be appointed to represent you before questioning, if you wish. MM
- You can decide at any time to exercise these rights and not answer any questions or make any statements. MM

Narrative

ON MAY 29 2009 AT 1930 hours MY wife took our 18 month old daughter to a doctors apointment I was left home alone with was for less than ten min. After feeding my youngest which I was doing before my wife left Alexis started to spit up her milk I acted as any caring parent would do I made sure see was alive and check vitals. After she had spit up, she quit breathing. I was holding her in my hands and I shook her to get her to respond to me I did it once the the second time for at lowest 5 sec. this time harder After I layed her down to check vitals she wasn't breathing. I ~~the~~ took her in my arm and ran, I ran as fast as I could go. I ran to Dicrass the street to a friend's house to go to the hospital, when we were in the car she was starting to take small gasp of air. we then arived to the hospital. Alexis also hit her neck when let go at her bk my 15 mos had a nike I don't think she hit hard. Also as I ran to the other friends house I did not have her head supported to me as I was running which would make her head bounce around.

I certify (or declare) under penalty of perjury under the laws of the State of Washington the foregoing is true and correct. Furthermore, I made this statement voluntarily.

Signature of person giving statement

Signature of witness

Appendix N

Patrick Lee

[redacted]

From: pat.k.lee@gmail.com
Sent: Friday, June 12, 2009 1:45 PM
To: Patrick Lee
Subject: Fw: I did shake her after she stoped...

------ SMS Text ------
From: 15734291202
Sent: Jun 12, 2009 12:53
Subject: I did shake her after she stoped...

I did shake her after she stoped breathing brit. She would not respond to me am i a bad parent im so sorry baby i forgot this is all my falt Sent via BlackBerry by AT&T

80

Appendix O

40

BIOMECHANICS OF HEAD TRAUMA IN INFANTS AND YOUNG CHILDREN

Susan Margulies, PhD, and Brittany Coats, PhD

INTRODUCTION

Traumatic brain injury (TBI) is a leading cause of death and disability among children and young adults in the United States, and it results in over 2500 childhood deaths, 37,000 hospitalizations, and 435,000 emergency department visits *each year*.[1] Head injury in infancy results in higher morbidity and mortality than that seen in older children, and it has become increasingly clear that the significant incidence of inflicted injury in the youngest patients is in large part responsible for this difference.[2,3] Skull fracture, intracranial bleeding, and traumatic axonal injury are the most common findings in serious head injuries in infancy and those associated with non-accidental causes. Determining the biomechanical conditions that cause these injuries and others in infants and older children will yield more effective prevention, diagnosis, and treatment strategies, as well as provide valuable information to help discern between accidental and inflicted injury etiologies.

MECHANICS OF TRAUMATIC BRAIN INJURY

Head injuries often occur with rapid head accelerations or decelerations that produce injurious brain deformations, or head impact that can deform or fracture the skull and produce underlying local brain deformations. Most frequently, both inertial (acceleration-deceleration) and contact mechanisms occur together such as in motor vehicle crashes, falls, and accidental and abusive impacts. Although rapid accelerations can be linear or rotational, high angular acceleration and velocity of the head are often correlated with intracranial hemorrhages and traumatic axonal injury in the brain.[6-12]

The underlying pathology of traumatic diffuse axonal injury (DAI) is the widespread damage to axons in the white matter of the brain, and the level of immediate neurological impairment is correlated with the extent and severity of axonal damage.[13] Biomechanical analyses of primate and porcine inertial models of DAI indicate a link between brain material deformation (tissue strain) and white matter injury.[14,15] Galbraith[16,17] confirmed this finding, showing that uniaxial tensile strain, not stress, caused short- and long-term neural dysfunction in unmyelinated axons. Subdural hematomas (SDHs) are caused by the rupture of the parasagittal bridging veins. In a primate inertial model for SDH,[18] biomechanical analysis revealed strong association of SDH with the elongation of the bridging veins beyond their ultimate or rupture strain as the brain moved relative to the skull during sudden acceleration or deceleration.[11] Taken together, these studies demonstrate that tissue deformation is closely associated with the primary axonal and vascular damage found in DAI and SDH.

Previous studies have attempted to define head injury tolerances for adults and children by determining the maximum load experienced by the head without injury. The most common head injury load tolerance, used extensively in automobile occupant safety research, is the head injury criterion (HIC), which is calculated from the acceleration of a head motion over a certain time interval.[19] Unfortunately, the HIC was derived from impact experiments on adult cadavers, does not differentiate between rotational and translational loads, and is not sensitive to the direction of head movement, making it an ineffective parameter for pediatric head injury.

Traditionally, a biomechanical analysis of head injury in the infant and young child assumes that they respond as miniature adults, with identical tissue properties and injury thresholds.[19-23] Using an engineering approach called *dimensional analysis*, critical inertial loading conditions associated with SDH and DAI can be scaled from the adult to the infant as a function of brain mass.[24,25] These analyses conclude that the lower brain mass of the young child allows the pediatric brain to sustain higher rotational accelerations than its adult counterpart before the onset of injury. However, differences between adults and children in tissue composition, mechanical properties, and brain vulnerability can influence the predictions of injury from scaling. As we describe later in this chapter, studies have shown significant age-related differences in brain and skull properties, indicating that the pediatric head cannot be modeled as a miniature adult head. Unfortunately, the biomechanical data used in most scaling approaches involve adult tissue composition, mechanical properties, and injury thresholds, and so must be interpreted with caution. Pediatric biomechanical models incorporating age-specific data are necessary to accurately mimic the pediatric head response to impact or rotation.

MATERIAL RESPONSE OF PEDIATRIC BRAIN AND SKULL TO LOADS

To date, human,[26] porcine,[26,27] and rodent[28] brain tissue testing studies reveal that when undergoing large deformations, the immature brain is approximately twice as stiff as adult brain. This finding is due, in part, to the presence of unmyelinated axons in the pediatric brain. Biomechanical models have reported that axons, rather than the surrounding matrix of astrocytes and oligodendrocytes, contribute more effectively to the stiffness of brain tissue.[29] Lipids have a low shear modulus,[30] suggesting that the increasing volume of myelin in the brain will decrease the effective shear modulus (stiffness) of brain tissue. Axons in the pediatric brain are undergoing rapid myelination during the first year of life and do not reach adult levels of myelination until approximately 18 months old. Although the pediatric brain is stiffer than adult brain, two studies[31,32] found human pediatric dural tissue to be approximately half as stiff as adult dura,[33] assuming similar thicknesses across age. Because the meninges provide support and protection to the brain during trauma, the pediatric brain might be less protected against loads than the adult brain. Material tests on pia and arachnoid in children and adults, however, is needed before this assertion can be made.

At birth, the skull begins as a single cortical bone layer approximately 1 mm thick, but as the bone continues to develop, the single layer of cortical bone differentiates into three layers: an outer table of cortical bone, middle spongy diploe layer, and an inner table of cortical bone, with a composite thickness of 5 to 6 mm. In a series of studies on fetal cranial bone, McPherson and Kriewall[34,35] report a significant increase in cranial bone stiffness (elastic modulus) with increased fetal development (20-40 weeks' gestation). More recently, Coats and Margulies[36] measured the elastic modulus in infants (36 weeks' gestation to 1 year) and found an increase in stiffness with age, such that the adult modulus is more than 30 times higher than the 1-month-old infant,[37] the 1-year-old is 18 times higher than the infant,[36] and the adult is 1.5 times higher than the 6-year-old.[38] Human infant cranial suture was 35 times lower than pediatric cranial bone, and tolerated significantly larger deformations prior to fracture or rupture, indicating that the infant braincase can withstand large shape distortions prior to fracture, unlike the adult skull.[36]

ANIMAL MODELS OF PEDIATRIC TRAUMATIC BRAIN INJURY

In adult head injury, limits of acceleration associated with concussion have been suggested based on measurements made from instrumented football players[39] and boxers,[40] and those specific for SDH and DAI have been suggested based on scaled rotational accelerations from inertial head injury studies in adult primates.[19,41] In pediatric head injury, there are no human data on load tolerances causing concussion, SDH, or DAI, but pediatric animal models are used to investigate mechanisms of brain injury under controlled impact or inertial conditions. Most pediatric animal models for head injury have evaluated the effect of contusional brain injury on the immature rat brain.[42-44] These models have been particularly useful in characterizing the focal injuries that often occur with head impact, primarily to the gray matter.[45-48]

Adelson and colleagues[47] have developed a model of diffuse brain injury in 17-day-old rats. However, unlike humans, rodents have lissencephalic brains and little white matter, as well as a very different maturational sequence from humans. With little white matter, DAI is difficult to characterize in the rat.[6,49] Developmentally, the rodent brain has its growth spurt entirely in the postnatal period, whereas humans peak in brain growth at the time of birth,[50] and the small brain size of rodents poses challenging biomechanical constraints when designing a device to create an animal model for inertial injury (widespread axonal injury, SDH). Because biomechanical analysis of mechanisms of pediatric brain injury depends on animal models that include salient differences between human infant and adult brains, such as differential development of the gray and white matter, regional cerebral metabolism and blood flow, changes in receptor density and distribution at different ages, and changes in biomechanical properties, rodent models are limited in their ability to provide data relevant to understanding head injury in human infants and young children.[51]

In contrast, piglets provide many advantages in modeling the immature human brain; the overall shape, gyral pattern, and distribution of gray and white matter is similar in pigs and humans. The growth pattern of the postnatal brain is similar to that of human infants; brain weight nearly doubles between the neonatal and juvenile periods and triples to reach the adult weight.[50,52,53] The response of the piglet to hypoxia and ischemia appears to parallel that in human infants. Cerebral blood flow and metabolism are similar, and the piglet brain matures in a similar manner with respect to myelination and brain electrical activity.[50,54-58] Visual-evoked response remains immature until 2 weeks after birth.[59] Whereas rodent models of TBI have limited their focus to only contusion injury, piglet TBI models have investigated fluid percussion[45] and inertial rotation injuries,[12] in addition to contusion injury,[60-63] that produce distributed white matter and SDH in infant and juvenile pigs.

Some fluid percussion studies report the pediatric brain to be more vulnerable to injury,[44,45] whereas other weight-drop (contusional) studies report no significant differences between the adult and pediatric animals.[64] The applied load in these studies, however, is not precisely scaled across age for infants and adults. In a study with the applied load scaled to age, Duhaime et al[61] produced cortical lesions across three age groups of piglets (5 day "infant," 1 month "toddler," and 4 months "adolescent") and reported that 1 week after injury the infant piglets had the smallest cortical lesion, and the adolescent group had the largest at 1 week after injury, indicating that the younger infant brain was either less vulnerable to acute injury and/or was able to have a quicker recovery than the older pigs. In inertial injury studies in piglets and adult pigs experiencing the same angular velocity and acceleration, Raghupathi and Margulies[12] reported at only 6 hours after injury that the piglets had 3.4 times more injured axons per area than the adults, indicating that the pediatric brain is more vulnerable to acute rotational injury than the adult. A later study by Duhaime and colleagues[65] confirmed age-dependent temporal pattern, such that lesion

volume 1 day after cortical impact injury in infant piglets was larger than adolescents, but that lesion volume for the infant pigs was smaller at 7 days and 30 days after injury. Finally, in a subsequent study from the Margulies lab,[66] repeated rapid horizontal head rotation 15 minutes after the initial rotation produced significantly more regions of injured axons than animals experiencing a single rotation, suggesting a cumulative effect of injury.

Interestingly, recent data indicate that there is a directional dependence on pediatric head injury,[67] with sagittal rotations resulting in the worst clinical findings with the greatest loss of consciousness, 100% apnea, and lowest cerebral blood flow, and the best outcomes from coronal rotations. Inertial head injury studies in the adult primate also report significant effects of rotational direction on brain trauma,[7,68] but differences between head and neck orientation in the biped compared with the quadruped make direct comparison difficult. In summary, animal models demonstrate that injury severity depends on developmental age, rotational direction and/or impact location, time after the injury, the number of insults to the brain, and the time between those insults.

Although animal models are useful for determination of injury response and time course, as well as development of new diagnosis and treatment strategies, they cannot be used to determine local tissue deformations that are primarily responsible for acute TBI, or determining minimum accelerations or impact forces necessary to produce concussion, SDH, or DAI. The inability to measure intracranial deformations hinders the extension of these animal model results to other types of loads (faster rates, different directions, distributed rather than focal loads) and to humans.

MECHANISMS OF ABUSIVE HEAD TRAUMA

In infants and toddlers, the majority of serious TBI is due to child abuse,[3] and abused children with brain injury have a worse outcome than those with accidental brain injury.[69] Although contact head trauma is a frequent finding in abusive head trauma,[70] many researchers believe that shaking alone can cause SDH, retinal hemorrhage, and death.[71,72] These authors argue that the lack of evidence of contact injury (skull fracture, cranial bruising, or scalp swelling) is evidence of shaking alone. If the infant's head strikes a padded surface such as a mattress, however, the force of the impact is widely dissipated and might not be associated with evidence of impact, even though the brain decelerates rapidly. The mechanism of injury responsible for SDH, retinal hemorrhage, and skeletal trauma that characterize abusive head trauma has been debated during the past decade. Duhaime et al[21] suggested that impact was necessary for SDH by demonstrating that the magnitude of angular deceleration is 45 times as great when an infant anthropomorphic surrogate model's head is struck as when it is shaken alone, and that the thresholds for concussion, SDH and DAI are exceeded at the moment of impact. Prange and others[73] later confirmed these findings and demonstrated that vigorous shaking produces accelerations that are significantly smaller than the rebound the head experiences from a 1-foot fall onto concrete. Unfortunately, surrogates only yield insight into the accelerations and contact loads experienced during a fall or simulated abusive scenario and cannot predict injuries. Furthermore, the utility of surrogates is compromised when their biofidelity or humanness (e.g., resistance of the neck to flexion/extension, compliance of the head and thorax, mass of the head/torso/limbs) is not considered in the construction of the surrogate. As more data regarding the kinematics and mechanical properties of children become available, the response of surrogates should be validated with human child data to ensure that they mimic children's responses.

COMPUTATIONAL MODELS OF PEDIATRIC HEAD INJURY

To relate the deformation occurring within the brain to a specific applied contact force or acceleration-deceleration of the head, finite element computational models are often used. These computational models are useful in extending the experimental results beyond scenarios tested in the experiments and to develop prevention and protection strategies. Many computational models of brain injury have been developed for the adult, but only a few have been developed for the child.[74-79] Many of these models have idealizations that can limit their validity, such as the use of simplified material properties and geometries. If assigned realistic parameters, computational models are a powerful tool to predict the stress and strain distributions throughout the brain, but are not capable of predicting the incidence of tissue injury. Tissue injury thresholds, or specific mechanical limits of the tissue related to functional or structural failure, must be developed to determine whether a focal tissue distortion predicted by the model would produce a specific injury, such as edema or axonal injury.

ROLE OF BIOMECHANICS IN ABUSIVE HEAD TRAUMA

Biomechanics provides empirical data regarding the global and focal mechanisms of head injury. Each biomechanical tool (animal experiments, human and animal tissue testing, anthropomorphic surrogate studies, and computational models) provides a crucial piece of the puzzle, but the best approach to understanding mechanisms of head injuries in child abuse is to combine the information from each of these tools and compare with data from clinical studies to answer the question: What mechanisms cause what injuries in children of what age? Before a comprehensive answer can be given, however, more research is needed to define tissue injury thresholds and to create biofidelic anthropomorphic surrogates and computational models that mimic the child's responses. Continued research in these areas will open pathways for enhanced traumatic head injury prevention, detection, and treatment strategies specific to infants and toddlers.

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Appendix P

41

THE CASE FOR SHAKING

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INTRODUCTION

A causal link between abusive trauma and subdural hemorrhages (SDHs) during infancy was first proposed by Guthkelch[1] in 1972. Shortly thereafter Caffey[2,3] suggested the term *whiplash shaken infant syndrome* to describe infants with abusive subdural and retinal hemorrhages but without external evidence of physical trauma. The term *shaken baby syndrome* (SBS) has been used subsequently to describe the combination of intracranial injury (brain injury and/or intracranial hemorrhage) and retinal hemorrhages from an abusive mechanism involving a rotational acceleration of the head caused by violent infant shaking. Intracranial injuries include acute and/or chronic SDH, axonal injury (AI), gliding contusions, cortical tears, and intracerebral edema. Retinal hemorrhages are reported in 30% to 100% of cases; averaging several series yields an approximate frequency of about 80%.[4]

In 1987 Duhaime and colleagues[5] in a landmark study reported evidence of impact injury in 63% of cases, and 100% of fatal cases of SBS. Subsequent publications have confirmed evidence of impact (e.g., scalp ecchymosis, soft tissue swelling, and skull fracture) in many infants with abusive head injuries. Duhaime's study also evaluated the accelerational forces generated by both shaking and impact using doll models, comparing these forces with established injury thresholds and suggesting that the forces acting on an infant's head during shaking alone were insufficient to cause SDH or AI. In this study only impact could generate forces sufficient to reach these injury thresholds, leading the authors to suggest that the term *shaken impact syndrome* was a better description of the causal mechanisms involved. The American Academy of Pediatrics has recently recommended that the term *abusive head trauma* (AHT) be used to avoid drawing any conclusions about the underlying mechanistic cause of the injuries.[6]

Over the past several years, the relationship between shaking and intracranial injury has come under intense scrutiny and criticism, with several authors questioning the assertion that shaking alone can achieve established injury thresholds for SDH or brain injury, and therefore, infant shaking, no matter how violent, cannot cause or contribute to the pathophysiology of AHT.[7-10] The logical conclusion is that abusive head trauma cannot be alleged in the absence of physical signs of external injury from impact, leading to widely disparate expert medical opinion in individual court cases about whether or not abuse has occurred. The arguments against shaking reached such a fevered pitch in the United Kingdom that all such cases were re-reviewed and in three cases sentences were changed based, in part, upon a "new understanding" of the biomechanics of infant shaking.

Unfortunately, nobody has yet marshaled a coherent and comprehensive argument in support of shaking as a causal mechanism for abusive head injury. This chapter reviews the available literature to make such an argument, emphasizing the following key concepts:

1. The biomechanical data regarding various injury thresholds are based upon experimental observations performed in *adult primates*. There is no evidence to support that injury thresholds derived from these studies are applicable to infant brains, and recent evidence suggests that injury thresholds in infant brains are considerably less than in adults.
2. The experimental primate studies used single-cycle rotational injuries with approximately 30-msec cycle times; there is no evidence that the injury thresholds derived from these experiments are applicable to the biomechanical parameters (repetitive 250- to 350-msec cycle times) during violent infant shaking.
3. Recent studies also suggest that secondary metabolic responses of infants to head injuries are both quantitatively and qualitatively different than in older children or adults; moreover, those injuries identified as abusive in nature by clinical criteria seem to engender an even greater secondary metabolic response.
4. These metabolic responses might reflect developmental *critical periods* during which the infant is uniquely susceptible to the effects of head injury and particularly to abusive head injuries. The injury thresholds for initiating these cascades have not been determined.
5. The clinical spectrum (both neurological presentation as well as neuroimaging and pathological findings) that follows *accidental* head injuries in infants and children has been well described in the literature in numerous publications. The extent, character, profile, and pattern of these accidental injuries are overwhelmingly consistent, and are wholly different from both the types and pattern of inflicted injuries.
6. A number of studies have addressed the clinical and pathological features of admitted (or confessed) cases of abusive head injury. Although evidence of blunt force trauma (impact) is reported in 20% to 63% of these cases (even among those cases in which only shaking was admitted), violent infant shaking remains a strikingly

common element in these confessions, with shaking admitted in up to 71%. In addition, a well-documented case of "shaken adult syndrome" having retinal and SDHs and AI has been described,[11] suggesting that shaking can be injurious even beyond infancy.

7. Although it is obvious that some of these confessions are erroneous, *in order to accept the thesis that shaking alone* cannot *achieve injury thresholds for brain injury or intracranial hemorrhage,* every *case of admitted shaking would have to involve numerous systematic and consistent lies,* something that defies logic.

BIOMECHANICS

A Brief Primer

A number of elegant biomechanical analyses of infant shaking have already been published. (See references 12 and 13 for examples of contrasting views.) In this text, Margulies provides a more detailed analysis of the biomechanics of AHT. (See Chapter 40, "Biomechanics of Head Trauma in Infants and Young Children.") A brief review, however, will help to better understand the arguments put forth by various authors. A *force* (measured in Newtons), when applied to an object (such as the brain) will result in a deformation, the magnitude of which will depend upon the magnitude and direction of the force, the rate at which the force is applied, the surface area over which the force is applied, and the *intrinsic material properties of the object to which the force is applied. Stress* is defined as the force divided by the surface area over which the force is applied (measured in Newtons/m^2, or Pascals). The application of force to an object results in a *deformation* of the object. The amount of deformation is called *strain.* The *Elastic (or Young's) Modulus* is the ratio of stress to strain and is a constant for any uniform material. The smaller the elastic modulus, the more readily deformation occurs.

An *elastic* deformation is one in which the deformation is proportional to the force applied and is entirely reversible. The brain and skull, however, like many biological substances, are *viscoelastic,* meaning that the elastic properties will vary depending upon the rate of application of a force. For events of short duration in which the force is rapidly applied, the brain is relatively resistant to deformation (i.e., it is relatively "stiff"). In contrast, for events of longer duration during which forces are applied more slowly, deformation is greater (i.e., the brain is "less stiff"). For short duration events, the deformation, or strain, is proportional to force multiplied by the time. Since force is directly proportional to mass times the acceleration ($F = ma$) and velocity is proportional to acceleration times the time ($v = at$), then the strain for events of short duration is proportional to mass and velocity; strain is dependent more upon the velocity of an applied load for short duration loads. For events of longer duration, the acceleration of the applied load is the more critical factor.

Studies of the biomechanics of traumatic brain injuries began with Holbourn's studies in 1943,[14] which suggested that brain injury results from shear strains in which tissues deform as they translate past one another in a gliding fashion, rather than by compressive or tensile strains in which the tissues are either compressed or pulled apart. He postulated that *impact loading* (a focal blow) to a stationary head results in localized deformation of the skull and underlying brain, inducing maximal shearing in the superficial tissues and producing *superficial contusions.* In contrast, *impulse loading,* or accelerational brain injuries, produce higher degrees of diffuse shear strain that is distributed deeper in the brain substance. He suggested that *linear* acceleration produces primarily compressive strains and therefore little deformation (since brain is relatively incompressible), whereas *rotational* acceleration produces higher degrees of shear strain that would be more injurious. He also recognized the importance of viscoelasticity in the generation of brain injuries and proposed that the transition between short-duration (velocity-dependent) events and long-duration (acceleration-dependent) events occurred at 2 to 200 msec. Finally, he concluded that sagittal rotations produced the greatest midline shear and therefore were most likely to injure the bridging parasagittal veins and produce SDH. These strains were reproduced in gelatin brain models that, although lacking some important features (such as the falx and tentorium), nonetheless supported these basic conclusions.

Applicability of Biomechanical Studies to Human Infant Shaking

Experimental verification of Holbourne's theories came during the 1960s with studies by Gennarelli, Thibault, Ommaya, and others at the University of Pennsylvania.[15-19] In these studies, *adult non-human primates* were subjected to rapid single accelerations of increasing magnitude while seated on a sled, with the head either immobilized (producing linear head acceleration) or not (producing angular acceleration). Critical thresholds capable of producing loss of consciousness (concussion), SDH, and AI were established in three different primate species having different brain masses. These results suggested (1) The thresholds for rotational velocity and acceleration were on average 50% higher for whiplash events not associated with impact compared with those events associated with impact, (2) the critical thresholds were inversely proportional to the brain mass (specifically [brain mass]$^{2/3}$) in the three species examined (Figure 41-1), and (3) SDH was more frequent following short-duration events lasting 5 msec or less, whereas AI more commonly followed longer-duration events lasting >6 msec. It is important to again point out that these injury thresholds were established for *adult non-human primates undergoing single cycle accelerational events.*

In 1987 Duhaime and colleagues[3] attempting to biomechanically model the forces that could be generated by shaking and impact, studied a "biofidelic" doll model having a head filled with wet cotton to approximate the weight distribution of a human infant head. Peak velocities and accelerations generated by either shaking the doll or impacting the doll against either a hard or soft surface were compared against the established injury thresholds derived from the previous experimental primate studies. Duhaime concluded that shaking alone (with cycle times of approximately 100-250 msec) achieved maximum velocities and accelerations well below the identified thresholds for SDH or AI (Figure 41-2). On the other hand, impact injuries against hard or soft surfaces (with cycle times of approximately 30 msec) consistently exceeded these injury thresholds. The authors concluded that shaking *alone* was unlikely to generate

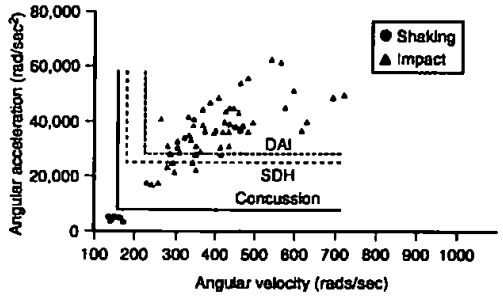

FIGURE 41-1 Illustration of injury thresholds for producing diffuse axonal injury *(DAI)*, subdural hemorrhage and concussion as derived from adult primate data. *(Adapted from Duhaime AC, Gennarelli TA, Thibault LE, et al: The shaken baby syndrome. A clinical, pathological, and biomechanical study. J Neurosurg 1987;66:409-415.)*

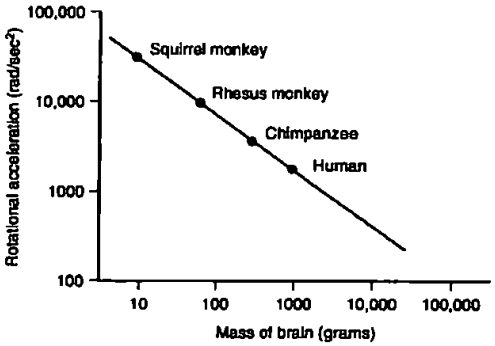

FIGURE 41-2 Relationship between injury thresholds in various species based upon mass scaling. Thresholds were experimentally derived from adult specimens of each of the three primate species studied; theoretical injury thresholds for adult humans are extrapolated from the experimental data on primates assuming the injury threshold is proportional to $1/m^{2/3}$, where m = brain mass. *(Adapted from Ommaya AK, Fisch FJ, Mahone RM, et al. Comparative tolerances for cerebral concussion by head impact and whiplash injury in primates. Warrendale, PA, 1970, Society of Automotive Engineers.)*

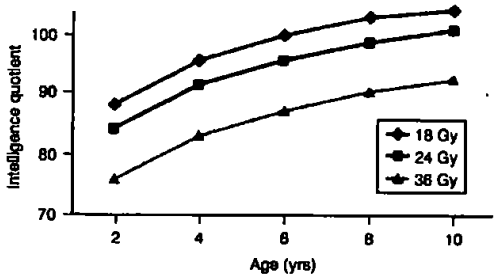

FIGURE 41-3 Effect of age on the effect of external beam whole brain radiation therapy on the intelligence quotient (IQ). For each radiation dose, the IQ declines as the therapy is delivered at progressively younger ages. *(Adapted from Silber JH, Radcliffe J, Peckham V, et al. Whole-brain irradiation and decline in intelligence: The influence of dose and age on IQ score. J Clin Oncol 1992;10:1390-1396.)*

enough force/load to produce SDH and AI described in the SBS and suggested that a *combination of shaking plus impact* was more common, and perhaps even necessary, to achieve the requisite injury thresholds.

The thrust of much of the medical research over the ensuing two decades has focused on proving (or disproving) that shaking alone can (or cannot) achieve the injury thresholds derived from the original experimental primate studies. There are several concerns with both the methodology and underlying assumptions of this line of research. First, the biofidelity of the original Duhaime model, particularly as it relates to the kinetics of neck motion, is in question, and significant differences in peak velocity and acceleration have been achieved simply by varying the parameters of the neck model in various studies.[20-22]

More critical, however, are the difficulties with the underlying assumptions: (1) The differences in thresholds for brain injury (SDH and AI in particular) can be accounted for *solely* by size scaling (smaller brains are less susceptible to injury than are larger brains), (2) brain injury thresholds (after size scaling) are identical for both mature and immature brains, and (3) the injury thresholds for single impulse whiplash injuries as determined in the primate studies are comparable to cyclic impulse loading (shaking). Simply put, *can injury thresholds derived from adult non-human primates undergoing a single acceleration on a sled be reliably extrapolated, using only mass scaling, to a human infant undergoing repetitive shaking?*

An Infant Is not Simply a Small Adult

Although it is intuitively obvious, an infant is not simply a scaled-down adult (and even less, a small adult monkey). A number of physical features of the infant brain are significantly different, including (1) The relative size of the head to the body, (2) the strength of the neck muscles, (3) the water content of the brain, (4) the degree of myelination, and (5) the relative absence of nodes of Ranvier (which in adults form the locus for traumatic AI).

Two well-known examples illustrate the fallibility of using the response to external forces in an adult (or even an older child) to predict the response in an infant. In the first example, the effect of radiation injury to the developing human brain as a function of age, the results are quantitative and predictable—cranial radiation to treat cancer results in a decline in IQ that is dependent upon both the radiation dose and child's age (Figure 41-3). In the second example, the effects of eye patching on vision, the response of the infant is qualitatively different and unexpected compared with the response of the older child or adult. A child >6 years of age undergoing a period of monocular eye patching will sustain no significant lasting visual effects, whereas an infant subjected to the same period of monocular eye patching will suffer a significant and lasting visual loss that is not predicted by the response of the older child.[23]

Experimental evidence from animal models also suggests that the responses of the developing brain to traumatic brain injury (TBI) are significantly different from those of the adult brain. The developing piglet brain is a reasonable model for

studying the effects of trauma on developing brain tissue. Experimental evidence has suggested that (1) The developing brain sustains greater injury at a lower threshold than adult brain, and (2) the effects of repetitive impulse loading are cumulative. The elastic shear modulus of the 2- to 3-day piglet brain (corresponding to a 1-month-old human) is significantly lower (that is, the tissue is more readily deformed) than that of the adult porcine brain, suggesting that injury thresholds are significantly lower for infant brains than predicted by simple mass scaling,[24] and no minimum threshold for injury has yet been established for piglet brains. Moreover, piglets subjected to two impulse loads within 15 minutes sustain greater injury at lower peak rotational velocities than do piglets undergoing single impulse loads[25]; the effect of *repetitive cyclic events over seconds* that would be analogous to shaking has not been systematically studied.

Neither has the effect of natural (or harmonic) frequency on brain injury thresholds been examined. The natural frequency is an inherent property of a material that results in a summation of forces that are applied cyclically at the natural frequency. The natural frequency has been estimated at between 5 and 10 Hz (corresponding to cycle times of 100-200 msec) for non-human primate brains, and between 4 and 5 Hz (corresponding to cycle times of 200-250 msec) for human brains.[18,26] The natural frequency for human brain is well within the frequency range for shaking (4-10 Hz) as derived from Duhaime's study.[3] A study by Thibault and Margulies[27] suggests that both adult and infant brains are increasingly susceptible to repetitive (or *oscillatory*) loading at lower frequencies down to 20 Hz, the lowest frequency studied; the effects of lower frequency events have not yet been evaluated.

THE BIOCHEMICAL RESPONSE OF THE DEVELOPING BRAIN TO ABUSIVE HEAD INJURY

Mounting evidence suggests that the biochemical and metabolic responses to head injury are significantly and fundamentally different in the young infant than that of the older child or adult. These biochemical responses are likely developmentally regulated and occur during "critical developmental periods" during which the developing brain is uniquely susceptible to outside forces that at other times would be innocuous. These developmentally regulated critical periods are well known throughout developmental biology and are responsible for the visual loss to monocular eye patching described previously.

The metabolic responses to abusive head injury have been thoroughly reviewed by Kochanek and colleagues,[28-30] and the interested reader is referred to these and other reviews for more complete information. The purpose here is to synthesize and condense the evolving information about this complex topic to explain how the infant response in general, and the response to abusive head injury in particular, differ in important ways from the responses seen in older children and adults. TBI sets in motion a number of metabolic cascades which contribute to *secondary* neuronal injury and death hours or days following injury. These secondary insults can be divided mechanistically into five broad categories: (1) excitotoxicity, (2) ischemia, (3) inflammation, (4) oxidative stress and free radical damage, and (5) apoptosis or programmed cell death. For each, the immature brain has been shown to be more vulnerable than the developed brain and, in many cases, uniquely susceptible to secondary metabolic insults.

Glutamate, an excitatory amino acid, has been implicated as one of the most clinically significant mediators of excitotoxicity both in animal models and in humans. Glutamate exerts its effects through the activation of both *N*-methyl-D-aspartate (NMDA) receptors and non-NMDA receptors. Increased levels of glutamate sufficient to cause neuronal cell death have been described in the cerebrospinal fluid (CSF) of both adults and children after TBI,[31,32] and its effects have been studied extensively. These include the initiation of a calcium-mediated metabolic cascade that leads to activation of intracellular proteases, lipases, and endonucleases; the release of superoxide radicals and nitric oxide; and ultimately, cell death. Ruppel and colleagues[32] demonstrated significantly greater rises in CSF glutamate after TBI both in young children (<4 years of age) and those with abusive injuries. Peak CSF glutamate levels were *sevenfold greater* after abusive injuries compared with accidentally injured controls, and the elevations were much more sustained compared with the response to accidental injuries. These studies suggest that TBI in infants can produce a quantitatively different glutamate response, and that abusive injuries appear to be particularly injurious in this regard. Other studies suggest that the metabolic *response* to glutamate is also age dependent. The glutamate NMDA receptor is developmentally regulated with its glutamate-modulated activation producing a greater calcium influx in the immature brain. Intracerebral injection of NMDA produces a much greater degree of damage in immature rat brains than in adults.[33] Finally, developing neurons are more susceptible to this excitotoxic cascade than are mature neurons.

Cerebral ischemia is common in the first 24 hours following TBI and is more common in children than adults.[34] Secondary ischemic injuries play an important role in abusive head injury, particularly in the genesis of the severe global hypodensities or "big black brain" on CT scans commonly seen in abusive head injuries.[35] Diffusion-weighted MRI confirms multifocal and global ischemic abnormalities in these infants without any demonstrated macrovascular (arterial or venous) occlusion on magnetic resonance angiography or venography.[36,37] *In vitro* studies in adult pigs suggest that the brainstem is inherently vulnerable to shear injury compared with the cerebrum,[38] and pathological studies in fatal cases of AHT have found *traumatic* AI at the cervicomedullary junction and upper cervical spinal cord,[12,39-42] with secondary *ischemic* AI at more rostral levels of the neuraxis.[41,42] This has led to the hypothesis that *focal* AI at the lower brainstem, cervicomedullary junction, and/or upper cervical spinal cord can produce transient apnea and/or circulatory changes that lead to global ischemic encephalopathy.[40,41]

Molecular responses to infant TBI might also contribute significantly to ischemic brain injury. Endothelin-1, a peptide with potent vasoconstrictor properties, is significantly increased in the CSF of infants and children with TBI,[15] and in a piglet model of TBI.[44] Endothelin-1 influences cerebral autoregulation in juvenile and particularly newborn piglets,

and the response is mitigated by treatment with endothelin-1 antagonists.[44]

Another pair of molecular species, adenosine and vascular endothelial growth factor (VEGF), protects against ischemic injury. Adenosine stimulates the formation of VEGF following TBI. VEGF is vasoactive, angiogenic, and neuroprotective; it increases vascular permeability, stimulates angiogenesis, protects against ischemia, and reduces glutamate-induced excitotoxicity. Although both adenosine and VGEF are increased in CSF following TBI in infants and children, the increase is less following AHT compared with accidental injuries.[45]

Another secondary metabolic injury pathway that is set in motion following TBI results in oxidative cellular damage. Free radicals such as superoxide, hydroxyl radicals, hydrogen peroxide, and oxidative nitrogen species such as peroxynitrite interact with a number of intracellular molecules, causing damage to cell membranes, proteins, and nucleic acids. F2-isoprostane, a marker of lipid peroxidation, is increased in the CSF of children following TBI.[46] In an experimental model of daily shaking in 6-day-old rats, hydroxyl radicals were increased following the third shaking episode; both this oxidative response and intracerebral hemorrhage were diminished by pretreatment with tirilazad, an antioxidant.[47]

A number of inflammatory mediators have been implicated in secondary cellular damage following TBI. Tumor necrosis factor (TNF), interleukin (IL)-1, IL-6, and IL-8 are increased in the CSF of both adults and children following TBI and produce vasomotor paralysis, vasodilation, hyperemia, and tissue edema.[48-50] A number of inflammatory reactants are particularly increased in infants and in victims of abusive head injury including IL-4, IL-6, IL-8, IL-10, IL-12, the soluble intracellular adhesion molecule-1, and quinolinic acid.[51-53] In particular, quinolinic acid rises significantly *earlier* following AHT compared with accidental TBI, which might reflect either prior episodes of head injury or a delay in seeking medical care following AHT.[54,55]

Apoptosis is a developmentally regulated energy-dependent process that results in cell death and is characterized histologically by DNA fragmentation and pyknosis; the resulting DNA fragments are readily identified in immunohistochemical preparations. In contrast, *necrosis* is a cell death resulting from a metabolic failure with swelling and cell lysis. Apoptosis occurs throughout embryogenesis and early childhood and is responsible for a variety of developmentally regulated processes such as remodeling of the digits by elimination of interdigital tissue, and the development of nephrons to form the kidney. Apoptosis in the brain is important for the elimination of neurons and remodeling of intercellular connections during brain and spinal cord development.

Apoptosis is principally regulated by mitochondrial membrane cytochrome *c*, which is increased in the CSF of children and infants with TBI, particularly in victims of abusive head injury and following fatal accidental head injuries.[56,57] In addition, Bcl-2, which regulates cytochrome *c* and is protective against apoptosis, is *higher* in the CSF of children with TBI, but remarkably *lower* (and not significantly different than controls) in the subset of infants with abusive head injury.[57]

Table 41-1 summarizes those CSF markers that are increased to a greater degree in abusive head trauma compared with accidental trauma in young infants. The net result of these five metabolic cascades (excitotoxicity, ischemia, inflammation, oxidative stress, and apoptosis) is that infants, particularly those suffering AHT, are at particularly greater risk for secondary cellular damage and death. Markers of both glial injury, such as myelin basic protein, and neuronal injury and death such as S-100 protein and neuron-specific enolase, are higher, rise earlier, and are more sustained in younger infants following AHT compared with older children and those with accidental injuries.[58,59] In particular, the sustained rise in neuron-specific enolase suggests that neuronal death is ongoing, likely the result of secondary metabolic destruction. To what degree the activation of these destructive metabolic pathways is dependent on young age as opposed to mechanism of injury is unclear, since most severe *infant* head injuries are also abusive and a multivariate analysis is not yet possible with the small sample sizes studied. *Nonetheless, it is clear that the metabolic response of the immature brain to TBI is both quantitatively and qualitatively different than that of the developed brain, and the thresholds necessary to initiate these metabolic cascades in the developing brain has never been examined properly.*

Table 41-1 Relative Changes in Cerebrospinal Fluid Markers Following AHT Compared with Accidental Neurotrauma

Marker	Physiological Role	Change (Compared with Accidental Trauma)
Glutamate	Neurotoxin	↑↑↑
Quinolinic acid	Neurotoxin	↑↑, more rapid peak in AHT
ICAM-1	Inflammation	↑
Cytokines	Inflammation	↑
Bcl-2	Neuroprotectant	↓
Adenosine	Neuroprotectant	↓
Procalcitonin	Neuroprotectant	↓
S-100 Protein	Neuronal marker	↑
MBP	Myelin marker	↑
NSE	Neuronal marker	↑

ACCIDENTAL BRAIN INJURIES—SPECTRUM AND PATTERN OF INJURIES

Research by Prange and colleagues[20] using biofidelic dummies suggested that shaking results in changes in both angular velocity and angular acceleration comparable to a fall from a 1.5-meter height onto concrete. In fact, "short fall defenses" have been used by various expert witnesses to explain the injuries in many court proceedings involving

alleged abusive injuries. Plunkett[60] has published "evidence" suggesting that short falls can be fatal. It therefore seems reasonable to contrast the severity and pattern of injuries seen in cases of alleged AHT with those that are commonly seen following short falls as an example of impact injury. A wealth of studies have described the injuries after short falls in young children, including falls at home from sofas, beds, high chairs, or changing tables; falls from cribs while in hospitals; and falls down stairs (with or without walkers). Both the published literature and the author's 17-years experience as a full-time pediatric neurosurgeon are consistent—the spectrum of injuries includes skull fractures and small contact epidural, subdural, or subarachnoid hemorrhages (typically underlying the impact site) or contusions. Among 1815 pediatric short falls (<10 feet) reviewed by Alexander et al,[61] 44 (2.4%) had skull fractures and only 8 (0.04%) had any intracranial injuries. Among 1037 pediatric stairway falls (including 524 in walkers), 34 (3.2%) had skull fractures, 13 (1.2%) had intracranial injuries, and 1 (0.09%) died. A more recent epidemiologic analysis of short falls (<1.5 m) among children in California by Chadwick et al[62] suggested that the odds of death resulting from short falls was less than 0.48 per million children per year. Even the evidence provided by Plunkett,[60] consisting of a review of over 75,000 reports of childhood injuries from playground equipment reported to the U.S. Consumer Product Safety Commission, identified only 18 deaths, an incidence of less than 0.02%; moreover, half of the deaths were caused primarily by either large space-occupying extra-axial (subdural or epidural) hemorrhages or large arterial distribution cerebral infarctions (one with identified post-mortem vertebral artery dissection) that are rarely seen in AHT.

Many comparative studies have confirmed that the nature and pattern of intracranial injuries following accidental falls is distinctly different from that following AHT.[63-70] Summarizing the findings in these studies, AHT differs significantly from accidental injury in the following ways: (1) More frequently causing prolonged coma and lower initial Glasgow Coma Scores, (2) having both more frequent, repeated, and more difficult to control seizures, (3) having more frequent SDHs *and* more extensive or diffuse SDHs yet *less* frequent epidural hemorrhages, (4) having much more frequent retinal hemorrhages (approximately 75%-80% on average) *as well as* more extensive, multilayered retinal hemorrhages, and retinoschisis; and (5) having a significantly higher mortality and worse long-term neurological outcomes. In addition, these injuries—both the pattern and extent—are observed both in cases that lack *any* history of trauma, and among those having other recognized abusive injuries such as bite marks, patterned soft tissue injuries, rib fractures, classic metaphyseal skeletal injuries, and others.

The wealth of evidence suggests that, although there are rare exceptions, inertial (impact) injuries from falls generate a consistent pattern of injury from an impact such as rare skull fractures and even rarer focal intracranial injuries. These injuries are well tolerated, resulting in serious intracranial injury or death in only rare isolated cases. In contrast, infants with AHT consistently present with injuries that are clinically, radiographically, and pathologically distinct from accidental injuries. Perhaps most importantly, clinical studies confirm that although evidence of impact is certainly present in a proportion of cases of AHT, impact is significantly less common in AHT than in accidental injuries.[64] How, then, are we to explain the mechanics of these injuries?

CLINICAL EVIDENCE OF SHAKING—PERPETRATOR CONFESSIONS

Confessed perpetrators in various studies have admitted to shaking alone, shaking with impact, or impact alone. In one study, 46% admitted to shaking only, 29% to impact only, and 25% to a combination of shaking and impact. Combining the first and third groups, 71% of the total admitted to shaking with or without additional impact.[70] There is no disputing the physical evidence of impact in a proportion (35%-63% of cases) of confessed abusive head injury, even among those who confessed to shaking only. Nor is there any argument that impact increases the applied force and, from a strictly biomechanical standpoint, makes the injuries potentially worse. However, if the biomechanical data are accepted at face value, then shaking without impact should *never* achieve sufficient force to produce prolonged traumatic coma, AI, or SDHs. The evidence from perpetrator confessions suggests otherwise.

For example, Starling[71] reviewed 171 cases of AHT, comparing 81 having perpetrator confessions with 90 having none. Of the 81 cases having perpetrator confessions, 69 had additional clinical evidence sufficient to make a reasonable determination regarding the mechanism of abuse (shaking alone, shaking with impact, or impact alone). Among the 32 cases with admitted shaking alone, 4 (13%) had evidence of impact injury, whereas evidence of impact injury was found in 5 of 17 cases (29%) of admitted shaking plus impact, and 12 of 20 (60%) of cases of admitted impact only. Neither the presenting clinical features nor the ultimate outcomes were dependent upon the mechanism described by the perpetrator. The frequency of SDHs was similar in all three groups, and retinal hemorrhages were *more* common in the cases of admitted shaking only. The authors concluded that shaking alone is sufficient to cause both subdural and retinal hemorrhages. A more recent review of perpetrator confessions (excluding the study by Starling above) found legitimate evidence of intracranial injury by shaking alone in 11 of 54 (20%) cases, providing further support for the importance of shaking as a mechanism of injury despite the author's conclusion that there is no "... support for many of the commonly stated aspects of the so-called shaken baby syndrome."[72]

One underlying weakness in all studies of confessed perpetrator admissions is the possibility (perhaps even the likelihood) that the perpetrator is either lying outright about the mechanism of injury, or omitting critical portions of the story. Unfortunately, this inherent weakness will likely not be overcome without the development of an unequivocal lie detector test. It is striking, however, that perpetrators consistently volunteer the same mechanism of injury—shaking the infant—when they are asked to describe what happened. *This common and consistent admission by the perpetrator to shaking the infant (whether or not cranial impact injury is described or discovered by physical examination, radiographic studies, or autopsy) overwhelmingly suggests that shaking is an important component of infant abusive TBI and is, in fact, sufficient to cause the intracranial injuries found in AHT. To suggest otherwise (as required by the biomechanical*

evidence) would require that every confessed perpetrator has to have been consistently and universally *lying about the same phenomenon, something that defies logic and common sense.*

TOWARD A BETTER UNDERSTANDING OF THE MECHANISMS OF SHAKING-INDUCED TRAUMATIC BRAIN INJURY

It is becoming increasingly clear from both neuroimaging studies[36,37,73,74] and post-mortem analyses of fatal cases[40,41] that the widespread cerebral and axonal damage in cases of AHT are, in fact, ischemic rather than directly traumatic in nature. There is also mounting evidence that the primary damage involves a previously unemphasized upper cervical spinal cord or brain stem injury, especially in fatal cases.[39-42] The presence of uncontrolled early seizures, so prevalent during the initial 24 to 72 hours after both AHT[68] and hypoxic-ischemic encephalopathy (HIE),[75,76] but rare following accidental trauma,[68] also strongly supports an ischemic pathophysiological basis for AHT. Whether the cerebral ischemia is primary and due to the apnea so commonly reported in these cases,[71,77] or due to secondary metabolic cascades as discussed above, is uncertain. Acute elevations of glutamate in particular could be a common final pathway for seizures in both AHT and HIE.[76] What is abundantly clear, however, from multiple clinical observations is that shaking, with or without impact, plays an important role in the pathogenesis of infant abusive brain injuries. Unfortunately, we have spent decades trying to determine whether infant shaking can achieve "magical" thresholds for brain injury—thresholds derived from experimental studies on adult primates and applied to human infants based solely upon mass scaling with no recognition of the many unique anatomical, biochemical, and developmental determinants of the developing infant brain. A simple biomechanical approach to injury modeling for AHT is therefore unlikely to get us any closer to the truth since we will never be able to know the specific injury thresholds for initiating these complex metabolic injury cascades in infants.

There are many similarities between the current controversies surrounding infant shaking and those that led to the discovery of the nature of insect flight. For centuries scientists were unable to fully explain insect flight, which seemed to violate known Newtonian principles. The prevailing experimental approach prior to the 1980s was to apply known, steady-state aerodynamic principles (those that govern fixed wing airplane flight) to insect flight, which, unfortunately, failed to fully explain how insects could fly. Insects placed in experimental wind tunnels could generate less than half of the lift required to maintain flight. Eventually, studies elucidated the mechanism: a dynamic leading edge vortex, generated by changes in the wing's position during flight, which spirals air along the wing's leading edge and creates the additional lift required for flight. Nobody would have suggested prior to this discovery that insect flight was impossible because it "lacked a sufficient factual base"—the observations spoke for themselves! However, it took a quantum change in thinking to discover the underlying mechanism.[78]

Similarly, understanding the pathophysiological role of shaking in AHT will likely require a quantum change in our thinking from the classical biomechanical paradigms of the 1970s to a model that takes into account new observations regarding metabolic pathways, the role of brainstem and/or upper cervical spinal cord injury, or other factors yet undiscovered. To those who argue that the contribution of shaking to the pathophysiology of AHT is a hypothesis lacking a sufficient evidentiary base, 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. The thrust of current research should be to better understand why this occurs, rather than dismiss these important observations out of hand.

A quote from Richard Feynman[79] seems an appropriate way to end this chapter: "In general we look for a new law by the following process. First we guess it. Then we compare the consequences of the guess to see what would be implied if this law that we guessed is right. Then we compare the result of the computation to *nature*, with experiment or *experience*, compare it directly with *observation*, to see if it works. If it disagrees with experiment it is wrong. In that simple statement is the key to science. It does not make any difference how beautiful your guess is. It does not make any difference how smart you are, who made the guess, or what his name is. If it disagrees with experiment (or experience), ... it is wrong."[79]

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Appendix Q

July 15, 2014

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RE: *State of Washington v. Michael Morris*

Dear Ms. Torres,

I am in receipt of your correspondence and materials regarding the above named criminal action. The purpose of this report is to present an evidence-based evaluation of the validity of the State's evidence that the diagnoses of subdural hematoma, retinal hemorrhage, and retinoschisis were a reliable proxy for abusive head trauma. This report outlines the methods and results used to evaluate the State's evidence, as well as my conclusions.

My summary conclusions in this matter are as follows: In the absence of additional diagnostic findings indicative of physical trauma, the diagnoses of subdural hematoma combined with retinal hemorrhage and retinoschisis are not a reliable proxy for abuse. More often than not (approximately 4 out of 5 times), a determination of abuse based solely on the presence of these findings, and in the absence of additional findings of injury (as was the case in State v. Morris), will be erroneous.

The bases for this opinion begin on page 3 of this report.

My methods in this case pertain to the fields of forensic medicine and epidemiology. Forensic medicine refers to the intersection of medicine and law, and in particular medicolegal investigation of causation. Epidemiology is defined as the scientific study of disease and injury in populations, including prevalence, risk, and incidence in specific populations. Inferential conclusions regarding the prevalence, incidence, risk, and causation of disease must be based on the proper interpretation of epidemiologic study and the proper application of epidemiologic

methods. Epidemiology is the scientific basis for the evaluation of general causation. The scientific field that dictates how probabilities may be inferred from epidemiologic data and methods and how the inferences can be applied to individuals or groups of individuals in a legal setting is forensic epidemiology. Forensic epidemiology provides the scientific basis for the evaluation of specific causation, to the extent that probability or likelihood of causation may be evaluated. The methods applied in this report are consistent with those outlined in the Reference Manual on Scientific Evidence, published by the Federal Judicial Center as a publication of the National Academies of Science (3rd Edition, 2011).

My qualifications to perform the analysis and render the opinions described herein are as follows:

I am a doctor of medicine and an epidemiologist, and my fields of expertise are forensic medicine and epidemiology. My relevant education is as follows: I hold a doctor of medicine degree from Umeå University in Umeå, Sweden, a PhD in public health with a major focus in epidemiology from Oregon State University, and an MPH in epidemiology and biostatistics from Oregon State University. My post-doctoral fellowship is in forensic pathology. I serve as a full Affiliate Professor of Epidemiology and Psychiatry at Oregon Health and Science University School of Medicine, in the Departments of Public Health and Preventive Medicine and Psychiatry, and an Adjunct Professor of Forensic Medicine and Epidemiology at the Institute of Forensic Medicine, Faculty of Health Sciences, Aarhus University, Aarhus, Denmark.

I have more than 30 scientific publications pertaining to injury biomechanics, including a book for the Society of Automotive Engineering, and have taught injury biomechanics, including the biomechanics of abusive head trauma, in a faculty peer-reviewed course at OHSU for the past 15 years. I have served as a consultant on head and spine injury biomechanics to state and federal government, as well as the U.S. Congress.

I serve as an Associate Editor of The Spine Journal, Physical Medicine & Rehabilitation, and the Journal of Forensic Biomechanics, and was co-editor in chief of the Journal of Whiplash and Related Disorders. I have published more than 150 scientific papers, abstracts, book chapters and books on topics that include traumatic injury to the spine and head. *Additionally, I have several publications on the validity of various diagnostic findings as an indication of child abuse, including abusive head trauma.* I have provided testimony in more than 300 civil and criminal trials in state and Federal courts throughout the United States, Canada, and Australia. Please see my CV for further details.

Analysis and Conclusions

Relevant facts:
My understanding of the facts in this case, in brief, are as follows:
In 2009 Michael Morris was charged with first degree assault of a child for the alleged assault of his six-week old daughter, Alexis Morris (AM). The case went to trial in June 2011 and Mr. Morris was convicted. At trial, the State presented the testimony of Dr. Kenneth Feldman, a pediatrician and child abuse expert and Dr. Erin Herlihy, an ophthalmologist, *inter alia*. The State's theory was that on May 29, 2009, Mr. Morris assaulted AM during the 10-15 minutes he was left alone with her after his wife left him alone with the baby to go take the older child to the doctor. Mr. Morris's then-wife testified that when she left, Mr. Morris was feeding AM, and that she was gone for about 10-15 minutes before she received a call from the neighbor that there was an emergency. Mr. Morris testified that he was feeding AM her bottle when she vomited through her nose and went limp, and then would not respond. He ran across to the neighbor's house to get help and the neighbor drove them to the hospital. At the hospital, the ER doctor saw a small bruise on her neck and a CT scan revealed a small subdural bleed. She was airlifted to Harborview, where the CT scan findings were mostly the same and retinal hemorrhage was also diagnosed. Dr. Kenneth Feldman, a pediatrician, was asked to consult by Harborview due to suspicion of child abuse. Two days later, AM was transferred to Children's Hospital, where further imaging revealed brain damage and where bilateral profuse retinal hemorrhaging, *possibly* with schisis cavities, was noted.

Mr. Morris was questioned by police and admitted to shaking the child twice in the process of trying to revive her, and after she had gone limp. The State theorized that Mr. Morris shook AM before she collapsed (not after as he explained) and that he shook her hard (not lightly as he described).

The defense presented the testimony of two experts, a clinician and a radiologist. The clinician opined that AM may have had viral meningitis, given all her symptoms (including and leading up to May 29). The radiologist gave a differential diagnosis that included a lack of oxygen or blood flow to the brain, a bleeding or clotting problem, infection, or trauma (accidental or non-accidental). None of those things could be ruled out. There was testimony about an extended intubation she underwent at Children's Hospital, where she experienced very low levels of oxygen for extended periods.

Assumptions of undisputed facts:

- Alexis Morris (AM) was six-weeks old on 5/29/09
- AM lost consciousness and was unresponsive on 5/29

- AM had difficulty breathing on 5/29
- AM was seen at the ER at 8:20 p.m. on 5/20 and was taken there immediately after her collapse by her father and a neighbor
- AM had a full fontanelle
- AM had a yellowing, older bruise below her chin (older than 5/29). Etiology of bruise is undetermined.
- AM had a small amount of scleral bleeding (blood in the white of her eye) (date unknown)
- AM had fluid in the middle ear (mastoid sinus) (detected by CT scan)
- AM had subdural bleeding ("SDH") (the exact date of the SDH is unknown but it's acute, meaning less than 7-10 days old)
- AM had retinal bleeding ("RH") (the exact date of the RH is unknown but it can be assumed that it's fresh, within one week)
- AM had retinoschisis (splitting of the retina) (date unknown)
- AM had no other bruising or pattern of bruises
- AM had no skull fractures
- AM had no detected scalp swelling or bruising
- AM had no external signs of trauma
- AM had no fractures; hospital skeletal surveys were normal
- AM was intubated on 5/29 and then again on 5/31
- AM had relatively minor brain damage, detected via MRI after 6/1
- AM had a lower than normal body temperature on 5/29 and 5/30
- AM had a high temperature (fever) on 5/31 and for days afterwards
- Michael Morris was alone with AM for approximately 15 minutes on 5/29
- Prior to 5/29, AM was cared for by multiple people, primarily her mother
- AM had been taken to the doctor on 5/26 for a cough, congestion, loss of appetite, and foul-smelling diaper.
- Otherwise, AM appeared normal or mostly normal to her caretakers before and on 5/29 until the acute event that resulted in taking her to the hospital

Facts in dispute:

- Michael Morris confessed to shaking his daughter. There is agreement that he admitted to shaking, but disagreement as to whether it's a "confession" and as to how hard he shook her.
    - The State argued that his admission to shaking her is suspicious because he didn't tell anyone initially; the State argued he shook her before she collapsed and that he minimized what he actually did.

  - The defense argued that he admitted to resuscitative shaking, which is what he did after AM did not respond to ordinary stimulus
- There was impact to the head. There is agreement that there is no external sign of impact, but the State argued that since there was no autopsy, bruising on the underside of the scalp could not be detected, and so the possibility of scalp bruising cannot be ruled out.
- AM had an infection (viral meningitis). The State's expert argued she had non-specific signs of illness (also consistent with a head injury) and that a test for viral meningitis was useless. The defense expert argued she may have had viral meningitis and that it can cause the findings at issue.
- AM had a botched intubation attempt. The defense expert argued that the intubation attempt on 5/31 went awry and resulted in extended periods of low oxygen, which could explain the MRI findings seen on 6/1 noting damage to the brain itself. The State experts argued that the intubation was nothing out of the ordinary and that brain damage is evolving and was a result of the inflicted injury on 5/29.

Analysis:

In this case the pivotal issue is whether or not the diagnostic findings of subdural hematoma (SDH) and retinal hemorrhage (RH) serve as an accurate proxy for the expert medical inference that the cause of the conditions are intentional abuse. The utility of any test is highly dependent on its accuracy, and accuracy is determined by various measures of how often a test result is correct.

For any test there are at least four possible results: 1) a true positive, in which the test correctly identifies tested subjects with the condition of interest; 2) a true negative, in which the test correctly identifies test subjects who do not have the condition of interest; 3) a false positive, in which the test is positive even though condition is not present, and; 4) a false negative (FN) in which the test is negative even though the condition is present.

Two important measures that allow for the assessment of the accuracy of a test are the sensitivity and specificity of the test. *Sensitivity* refers to the percentage of tests that are positive when a condition of interest is present. The sensitivity of a test is also referred to as the true positive rate of the test. *Specificity* refers to the percentage of tests that are positive when the condition of interest is absent. Specificity is also known as the true negative rate. The equation (1 – specificity) gives the *false positive* rate, another important measure of test accuracy. In other words, if the specificity of a test is 80% (*i.e.* 80% of the time the test accurately identifies individuals without the condition

of interest), then the false positive rate is 20% (20% of the time individuals without the condition are identified as having it).

Another important factor is how often the condition of interest is expected to be present before the test is given. This measure is called *prevalence*. One cannot understand what a positive test means without knowing something about these 3 measures (true and false positive rate and prevalence).

The following 2 examples illustrate this principle using tests with identical true and false positive rates (the values are just for the examples):

1. Consider a test for norovirus (a virus that is commonly associated with food poisoning) that has a true positive rate of 90% and a false positive rate of 10%. If norovirus causes 80% of all food poisoning cases (*i.e.* the prevalence of the tested condition is 80%), then the test would yield true positive results in 90% of the 80 cases caused by norovirus, for 72 true positives. The test would also yield false positive results in 10% of the 20 remaining cases not caused by norovirus, for 2 false positives. Therefore, there is a 72:2, or 36:1 probability that a positive test for norovirus is a **true positive**. This would be a pretty reliable test for the presence of norovirus.

2. In contrast, consider a test for HIV antibodies with the same true and false positive rates as the norovirus test, but given to the population of white males applying for life insurance. The prevalence of HIV in this population would be quite small, far less than 1%. If we gave the test to 100 men, we would have no more than 1 true positive (assuming 1 in 100 has the disease), but we would have 10 false positives among the remaining 99 healthy men. Thus, there would be a 10:1 probability that a positive test was a *false positive*. Obviously, such a test would be a highly unreliable test for the presence of HIV antibodies.

In the present case, the State's expert witness Dr. Feldman has asserted that the finding of SDH and RH are highly reliable indicators of abuse. In doing so, he has used the presence of SDH and RH as *tests* for a history of abuse as a cause of the injuries. Dr. Feldman further testified during the trial in this matter on the accuracy of these findings as a test for abuse, as follows:

> *"There's another evidence-based study looking at retinal hemorrhages in that retinal hemorrhages picked up abusive head injury...75 percent of the time and it was specific for head injury about 95 percent of the time...and when you get to splitting of the retina such as*

> *this child was eventually found to have, they didn't see it in anything but abusive head trauma." 6/3/11 RP 12-13.*

In this section of his testimony Dr. Feldman testified that retinal hemorrhage alone has a true positive rate of 75% for abusive head trauma, and a true negative rate of 95%, and thus a false positive rate of 5%. He also indicated that retinal splitting (retinoschisis) was only found when there had been abuse, and this has a 100% true positive rate for abuse. Dr. Feldman did not indicate which publication he was referring to for these figures, or how reliable it was.

Most importantly, Dr. Feldman gave no explanation regarding how often the determination of abuse would be correct when SDH and RH are the *only* possible findings of abuse present; *i.e.* when there is no additional diagnostic or circumstantial evidence that abuse has taken place. The last point is important, because there appears to be no distinction on the part of the State's expert between the true positive rate for abuse when SDH and RH are present among other findings of abuse, and when they are the sole findings thought to be representative of abuse, as was the case in *State v Morris*.

What is needed to assess the validity of the State's expert's assertions regarding the relationship between SDH and RH and abuse is a valid and large data set against which the accuracy of SDH and RH can be tested as a determinant of abuse. For this reason an analysis of national hospital inpatient data was performed for this case. The database used for this analysis was the Kids' Inpatient Database (KID). The KID is made available by the Healthcare Cost and Utilization Project (HCUP) of the Agency for Healthcare Research and Quality of the US Department of Health and Human Services. The KID provides a survey sample of discharge records for approximately 3 million unweighted pediatric hospital stays in the United States one out of every three years, and is ***the most reliable source of information available for what is occurring with hospitalized children in US hospitals***. The KID is nationally representative, and estimates about 7 million weighted pediatric hospital stays per survey year. The KID provides extensive information regarding each hospital inpatient stay including patient age, sex, discharge diagnoses, and procedures performed, *inter alia*. More information about the KID can be accessed at http://www.hcup-us.ahrq.gov/kidoverview.jsp.

For the present case the analysis performed was as follows:
We accessed the 2000-2009 KID (4 years total) and selected every non-newborn child aged 3 years or less who had a diagnosis of retinal or subdural hemorrhage, or retinoschisis (ICD-9 codes 362.81, 852.2x, 432.1, or 361.1). The outcome of interest was the rate of diagnosed child abuse (ICD-9 codes 995.54 and 995.55) among those with and without additional diagnoses of trauma

and/or injury (ICD-9 codes 800.xx-999.xx). Frequencies and odds ratios (with associated 95% confidence intervals) were calculated using widely used statistical software (SAS 9.4).

The results of the analyses were as follows:
There were a total of 13,409 children with a diagnosis of either retinal or subdural hemorrhage. Of these, 4,062 (30.3%) had a diagnosis of child abuse. There were 7,814 children with only a diagnosis of subdural hemorrhage (no retinal hemorrhage). Of these 5,653 (72.3%) had an additional diagnosis indicating trauma/injury, and 2,161 (27.6%) did not. Of those who had an additional trauma/injury code, 1,050 (18.6%) had a diagnosis of child abuse, versus 30 (1.4%) of those who did not. The resulting odds ratio for abuse given additional trauma/injury was 16.4 [95% CI 9.6, 27.8]), meaning that a child with SDH and additional diagnosis of injury is 16.4 times more likely to have a diagnosis of abuse as well. Conversely, this finding also meant that if there is no additional injury diagnosis the child is 1/16th as likely to have been abused. ***With only 1.4% of children with SDH and no additional diagnosis of trauma also diagnosed as abused, SDH alone is a very poor proxy for abuse.***

There were 2,623 children with only a diagnosis of retinal hemorrhage (no subdural hemorrhage). Of these 2,014 (76.8%) had an additional diagnosis indicating trauma/injury, and 610 (23.2%) did not. Of those children with an additional trauma/injury code, 1,142 (56.7%) had a diagnosis of child abuse, versus 53 (8.7%) of those who did not. The resulting odds ratio for abuse given additional trauma/injury was 13.9 (95% CI [8.5, 22.7]). ***Again, retinal hemorrhage alone, with no additional injury diagnosis, is a very poor proxy for abuse.***

Finally, we examined those children with injuries most like AM: those with both retinal and subdural hemorrhage but no other findings of physical injury. There were 2,972 children with both retinal and subdural hemorrhage diagnoses. Of these children, 2800 (94.2%) had an additional diagnosis indicating trauma/injury, and 173 (5.8%) did not. Of those who had an additional trauma/injury code, 1,759 (62.8%) had a diagnosis of child abuse (children *not* like AM), versus 30 (17.3% of the total) of those who did not (children like AM). The resulting odds ratio for abuse given additional trauma/injury was 8.2 (95% CI [4.7, 14.2]). Thus, when the two conditions are both present AND there are additional findings of traumatic injury there is a more than 50% probability of abuse. **But, when no additional findings are present there is a less than 20% probability of abuse.**

Adding the relatively rare diagnosis of retinoschisis did not increase the reliability of the RH and SDH as proxies for abuse. There were very few cases of all 3 diagnoses (only 33 total, with 31 with additional diagnoses of traumatic injury, and only 2 cases without additional diagnoses). Of the 31 cases with additional diagnoses, there were 22 (71%) that were associated with abuse and 9%

(29%) that were not. Of the 2 cases with no additional diagnosis of injury, none (0%) were also diagnosed as abused.

As a result of the preceding it can be concluded that Dr. Feldman's assertion that there is a 5% false positive rate for abuse when retinal hemorrhage is present is incorrect. **In fact, when there are no additional findings of trauma, RH alone as an indication of abuse has a 91% false positive rate.** Further, the claim that all cases of retinoschisis are associated with abuse is also incorrect, and this relatively rare diagnosis cannot be used as a proxy for abuse.

Thus, whatever the basis for Dr. Feldman's testimony regarding the reliability of RH and retinoschisis as indications for abuse, it was erroneous.

To the extent that Mr. Morris' conviction was based on the evidence that the diagnostic findings seen in AM were a reliable indication of abuse, it was in error.

The preceding opinions have been given as reasonable medical and scientific probabilities.

Very truly yours,

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Updated 6-14

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EDITORIAL ACTIVITIES

Associate Editor:
OA Epidemiology, 2014-present
J of Forensic Biomechanics, 2010-present
The Spine Journal 2007-present
PM&R, official scientific journal of the American Academy of Physical Medicine and Rehabilitation 2008-present
Scandinavian Journal of Forensic Medicine, 2012-present
Editorial Board Member:
The Spine Journal 2004-present
International Research Journal of Medicine and Medical Sciences
Egyptian Journal of Forensic Sciences 2010-present
Journal of Case Reports Practice 2014-present
Austin Journal of Public Health & Epidemiology 2014-present
Co-Editor in Chief, *Journal of Whiplash-Related Disorders* 1999-2006
Editorial Committee Member, *Spineline* 2004-2009
Peer reviewer:
Orthopedics
Spine
The Spine Journal,
Lancet
Annals of Biomechanical Engineering
Journal of the American Board of Family Medicine
Journal of Forensic and Legal Medicine
Acta Neurologica Scandanavica

Medical Science Monitor
Pain Research & Management
Journal of Back and Musculoskeletal Rehabilitation
American Society for Testing and Materials (ASTM)
Biosecurity & Bioterrorism
Annals of Medical and Health Sciences Research
Neurorehabilitation and Neural Repair
International Research Journal of Medicine and Medical Sciences,
Jurimetrics
Law, Probability, and Risk

COURSES TAUGHT

PHPM 574 Forensic & Trauma Epidemiology
Department of Public Health and Preventive Medicine
Oregon Health & Science University School of Medicine
Portland, Oregon 2006-2013

Principles of Forensic Epidemiology
Forensic Psychiatry Fellowship
Department of Psychiatry
Oregon Health & Science University School of Medicine
Portland, Oregon – 2011 to present

PHPM 503 Thesis Advising
Department of Public Health and Preventive Medicine
Oregon Health & Science University School of Medicine
Portland, Oregon 2005-present

PHPM 507 Injury and Trauma Epidemiology
Department of Public Health and Preventive Medicine
Oregon Health & Science University School of Medicine
Portland, Oregon 1999 – 2005

Forensic Epidemiology and Bioterrorism
Charles County Department of Public Health
College of Southern Maryland, Waldorf, Maryland 2014

ACTIVITIES and HONORS

Member, Scientific Advisory Board, International Conference on Forensic Inference and Statistics. August 2014, Leiden, The Netherlands

Reviewer, National Aeronautical Space Administration (NASA) 2011

Past president, International Cellular Medicine Society, 2009 to 2012

Founding member, International Cellular Medicine Society, 2009

Member, Research Planning Committee, North American Spine Society 2007-2009

Member, Complementary Medicine Committee, North American Spine Society 2007-2009

Special Deputy Sheriff (Forensics), Vehicular Homicide Investigator, Clackamas County, Oregon, 2007-present

Member, Crash Reconstruction and Forensic Technology (CRAFT) multidisciplinary law enforcement fatal crash investigation team, Clackamas County, Oregon, 2002-present
Consultant Forensic Trauma Epidemiologist to the Medical Examiner Division of the Oregon Department of State Police – Occupant Kinematics, 1999-2006
Deputy Medical Examiner, Marion County, Oregon. 2000-2005
Moderator, Engineering sciences section, American Academy of Forensic Sciences 62nd Annual Meeting, Seattle, WA 2010
Co-Chair, International Whiplash Trauma Congress V, Lund, Sweden. 2011
Co-Chair, International Whiplash Trauma Congress IV, Miami, FL. October 2007.
Co-Chair, International Whiplash Trauma Congress III, Portland, OR. June 2006.
Co-Chair, International Whiplash Trauma Congress II, Breckenridge, CO. February 2005.
Co-Chair, International Whiplash Trauma Congress I, Denver, CO. October, 2003
Co-Chair, Forensic Section, International Traffic Medicine Association. Budapest, Hungary. September, 2003
Member, Blue Ribbon Panel Congressional Task Force on roller coaster-induced brain injury. Funded by a grant from the National Institute of Child Health and Human Development 2002-2003
President, Spinal Injury Foundation. Denver, CO 2002-2009
Member, Marion-Polk County C.R.A.S.H. Team - Occupant Kinematics Consultant 1999-2004
Scientific Chair, North American Whiplash Trauma Congress. Victoria, British Columbia 1999

BOARD CERTIFICATION AND ORGANIZATIONS

ACTAR Accredited Crash Reconstructionist, Accreditation Commission for Traffic Accident Reconstruction, #1581
American Academy of Forensic Science, Pathology/ Biology section
Fellow, International College of Chiropractic
Diplomate, American Academy of Pain Management
Certification in basic and advanced crash reconstruction - Northwestern University
Member, American College of Epidemiology
Member, Association for the Advancement of Automotive Medicine
Member, Sigma Xi Scientific Honor Society
Member, Society of Automotive Engineers
Past member, International Traffic Medicine Association
Inactive member, North American Spine Society
Past member, Forensic Accident Reconstructionists of Oregon

GRANTS

2011-2015 World Health Organization – research grant for Rwandan study of relationship between genocide and suicide and homicide victimization and offending. $200,000 over 4 years.

2010-2011 Centers for Disease Control (Administered by National University of Rwanda and OHSU) SPH/CDC $200,000 over 4 years.

2002-2003 National Institute of Child Health and Human Development – Blue Ribbon Task Force on Roller Coaster Associated Brain Injury. $75,000.

DISSERTATION SUPERVISION/MENTORING

Frank Franklin Ph.D., J.D. candidate, Earle Mack School of Law, Drexel University (2013)
Bonnie Colville-Ebeling MD – PhD candidate (University of Copenhagen, Faculty of Health Sciences, Department of Forensic Medicine) – (2012-present)
Dimitrios Papadakis BSc, MRes, Dr.rer.nat. (2012-present) mentoring
Wendy Leith MS – MPH candidate (2012-present)
Konrad Dobbertin – MPH 2011
Apostolo Alexandridis – MPH 2011
Wilson Rubanzana MD – PhD candidate (National University of Rwanda, School of Public Health) (2010-present)
Catherine Maddux-Gonzalez – MPH 2009
Laura Criddle MS, RN – PhD 2008
Peter Harmer PhD – MPH 2006

PUBLICATIONS

Peer-reviewed journal articles

1. Centeno CJ, Pitts J, Al-Sayegh H, **Freeman MD.** Efficacy of autologous bone marrow concentrate for knee osteoarthritis with and without adipose graft (in review)

2. Centeno CJ, Pitts J, Al-Sayegh H, **Freeman MD.** Anterior cruciate ligament tears treated with percutaneous injection of autologous bone marrow nucleated cells; a pilot study. *PM R* (in review)

3. **Freeman MD**, Cahn PJ, Franklin FA. Applied forensic epidemiology. Part 1: medical negligence. *OA Epidemiology* 2014 Jan 18;2(1):2.

4. Koehler S, **Freeman MD**. Forensic epidemiology; a methodology for investigating and quantifying specific causation. *Forens Sci Med Path* 11/2013; DOI:10.1007/s12024-013-9513-8

5. Rubanzana W, Hedt-Gauthier B, Ntanganira J, **Freeman MD**. Exposure to genocide as a risk factor for suicide in Rwanda. *Int Epidemiology* (in review)

6. Centeno CJ, **Freeman MD**. Percutaneous injection of autologous, culture-expanded mesenchymal stem cells into carpo-metacarpal hand joints: A case series with an untreated comparison group. *Wien Med Wochenschr* 2013:DOI 10.1007/s10354-013-0222-4

7. **Freeman MD**, Eriksson A, Leith W. Head and neck injury patterns in fatal falls: epidemiologic and biomechanical considerations. *J Forensic Legal Med* 2013:http://dx.doi.org/10.1016/j.jflm.2013.08.005

8. Colville-Ebeling B, **Freeman MD**, Banner J, Lynnerup N. Autopsy practice in forensic pathology – evidence-based or experience-based? A review of autopsies performed in a case of multiple, simultaneous deaths. *J Forensic Legal Med* (in press)

9. **Freeman MD**, Eriksson A, Leith W. Injury pattern as an indication of seat belt failure in ejected vehicle occupants *J Forensic Sci*, 2014 doi: 10.1111/1556-4029.12482

10. Dobbertin KM, **Freeman MD**, Lambert WE, Lasarev MR, Kohles SS. The relationship between vehicle roof crush and head, neck and spine injury in rollover crashes. Accid Anal Prev. 2013 Apr 25;58C:46-52. doi: 10.1016/j.aap.2013.04.020. [Epub ahead of print]

11. Centeno CJ, Schultz JR, Cheever M, **Freeman M**, Faulkner S, Robinson S. A Case Series of Percutaneous Treatment of Non-Union Fractures with Autologous, Culture Expanded, Bone Marrow Derived, Mesenchymal Stem Cells and Platelet Lysate. *J Bioengineer & Biomedical Sci S2:007* doi:10.4172/2155-9538.S2-007

12. Woodham M, Woodham A, Skeate JG, **Freeman MD**. Long-Term Lumbar Multifidus Muscle Atrophy Changes Documented With Magnetic Resonance Imaging; A Case Series. *Radiology Case* 2014;8(5):27-34

13. Wendlova J, **Freeman MD**. The Slovak Regression Model of Fall-Related Femoral Neck Fracture Risk. *Journal of Forensic Biomechanics* Vol. 4 (2013), Article ID 235595, 5 pages doi:10.4303/jfb/235595

14. **Freeman MD**, Dobbertin K, Kohles SS, Uhrenholt L, Eriksson A. Serious head and neck injury as a predictor of occupant position in fatal rollover crashes. *Forensic Sci Int* 2012;222:228–33.

15. **Freeman MD**, Kohles SS. An examination of the threshold criteria for the evaluation of specific causation of mesothelioma following a history of significant exposure to chrysotile asbestos-containing brake dust, *Int J Occ Env Hlth* 2012;18(4):329-36.

16. **Freeman MD,** Fuerst M. Does the FDA have regulatory authority over adult autologous stem cell therapies? FDCA 21 CFR 1271 and the Emperor's New Clothes. *J Transl Med.* 2012 Mar 26;10(1):60.

17. **Freeman MD,** Everson T, Kohles SS. Forensic epidemiologic and biomechanical analysis of a pelvic cavity blowout injury associated with ejection from a personal watercraft (jet-ski). *J Forens Sci* 2012 doi: 10.1111/j.1556-4029.2012.02250.x

18. **Freeman MD,** Kohles SS. Plasma levels of polychlorinated biphenyls, non-Hodgkin lymphoma, and causation. *J Environ Public Health* 2012;2012:258981. doi: 10.1155/2012/258981. Review.

19. Centeno CJ, Fuerst M, Faulkner SJ, **Freeman MD.** Is cosmetic platelet-rich plasma a drug to be regulated by the Food and Drug Administration? *J Cosm Derm* 2011;10:171–3.

20. Centeno CJ, Schultz JR, Cheever M, **Freeman M,** Faulkner S, Robinson S, Hanson R. Safety and Complications Reporting Update on the Re-Implantation of Culture-Expanded Mesenchymal Stem Cells Using Autologous Platelet Lysate Technique. *Cur Stem Cell Res & Ther* 2011;6(4):XX

21. **Freeman MD,** Kohles SS. Application of the Hill Criteria to the Causal Association of Post-Traumatic Headache and Assault. *Egypt J Forensic Sci* 2011;1:35-40.

22. **Freeman MD,** Kohles SS. Application of the Bradford-Hill Criteria for Assessing Specific Causation in Post-Traumatic Headache. *Brain Inj Prof* 2011;8(1):26-8.

23. **Freeman MD,** Kohles SS. An Evaluation of Applied Biomechanics as an adjunct to systematic specific causation in forensic medicine. *Wien Med Wochenschr* 2011;161:1-11.

24. Uhrenholt L, **Freeman MD,** Jurik AG, Jensen LJ, Gregersen M, Boel LW, Kohles SS, Thomsen AH. Esophageal injury in fatal rear-impact collisions. *Forensic Sci Int* 2010 Oct 5. [Epub ahead of print] PubMed PMID: 20932695.

25. **Freeman MD.** A Bayesian assessment of unexplained fracture as a forensic test of child abuse; quantification of uncertainty using the Error Odds approach. *Acta Medicinae Legalis et Socialis* 2010:179-84.

26. Nystrom NA, Champagne LP, **Freeman MD,** Blix E. Surgical fasciectomy of the trapezius muscle combined with neurolysis of the spinal accessory nerve; results and long-term follow-up in 30 consecutive cases of refractory chronic whiplash syndrome. *J Brachial Plexus and Peripheral Nerve Injury* 2010:5;7.

27. Centeno CJ, Schultz J, Cheever M, Robinson B, **Freeman MD**, Marasco W. Safety of autologous MSC transplantation: an in vivo MRI study of transplanted MSCs culture-expanded using a novel, platelet-lysate technique. *Cur Stem Cell Res & Ther* 2010;5:81-93.

28. Dagenais S, Gay RE, Tricco A, Mayer, JM, **Freeman MD**. North American Spine Society Contemporary Concepts in Spine Care: Spinal Manipulation Therapy for Acute Low Back Pain *Spine J* 2010 Oct;10(10):918-40.

29. Uhrenholt L, Schumacher B, **Freeman MD**. Road traffic fatalities in Aarhus Police District in 2000-2004 - medical investigations and legal consequences. *Ugeskr Laeger.* 2010 Sep 27;172(39):2683-2687. Danish

30. **Freeman MD**, Woodham M, Woodham A. The role of the lumbar multifidus in chronic low back pain; a review. *PM R* 2010 Feb;2(2):142-6.

31. **Freeman MD**, Centeno CJ, Kohles SS. A systematic approach to clinical determinations of causation in symptomatic spinal disc injury following motor vehicle crash trauma. *PM R* 2009;1(10):951-6.

32. **Freeman MD**, Rosa S, Harshfield D, Smith F, Bennett R, Centeno CJ, Kornel E, Nystrom A, Heffez D, Kohles SS. A case-control study of cerebellar tonsillar ectopia and head/neck (whiplash) trauma. *Brain Injury* 2010;24(7-8):988-94.

33. **Freeman MD**, Kohles SS. Applications and limitation of forensic biomechanics; a Bayesian perspective. *J Forensic Legal Med* 2010;17:67-77.

34. **Freeman MD**, Nystrom A, Centeno C, Hand M. Chronic whiplash and central sensitization; do a trigger points play an important role in pain modulation? *J Brachial Plex Peripher Nerve Inj* 2009 Apr 23;4:2.

35. **Freeman MD**, Hand ML, Rossignol AM. Applied Forensic Epidemiology: A Bayesian evaluation of forensic evidence in a vehicular homicide investigation. *J Forensic Legal Med* 2009 Feb;16(2):83-92. Epub 2008 Oct 21

36. Centeno CJ, Busse D, Kisiday J, Keohan C, **Freeman M**, Karli D Regeneration of meniscus cartilage in a knee treated with percutaneously implanted autologous mesenchymal stem cells. *Med Hypothese.* 2008 Dec;71(6):900-8.

37. Centeno CJ, Schultz J, **Freeman M**. Sclerotherapy of Baker's cyst with imaging confirmation of resolution. *Pain Physician* 2008 Mar-Apr;11(2):257-61.

38. Centeno CJ, Busse D, Kisiday J, Keohan C, **Freeman M**, Karli D. Increased knee cartilage volume in degenerative joint disease using percutaneously

implanted, autologous mesenchymal stem cells. *Pain Physician* 2008 May;11(3):343-53.

39. **Freeman MD**, Centeno CJ. A fatal case of secondary gain; a cautionary tale. *Amer J Case Reports* 2008;9:97-103.

40. Centeno CJ, Elkins W, **Freeman M**, Elliott J, Sterling M, Katz E. Total Cervical Translation as a Function of Impact Vector as Measured by Flexion-Extension Radiography *Pain Physician* 2007 Sep;10(5):667-71.

41. **Freeman MD**, Rossignol AC, Hand M. Forensic Epidemiology: A systematic approach to probabilistic determinations in disputed matters. *J Forensic Legal Med* 2008;15(5):281-90.

42. Centeno CJ, Kisiday J, **Freeman MD**, Shultz JR. Partial regeneration of the human hip via autologous bone marrow nucleated cell transfer: a case study. *Pain Physician* 2006;9:135-7.

43. Croft AC, **Freeman MD**. Correlating crash severity with injury risk, injury severity, and long-term symptoms in low velocity motor vehicle collisions. *Med Sci Monit* 2005 Oct;11(10):RA316-21. Epub 2005 Sep 26.

44. **Freeman MD**, Croft AC, Nicodemus CN, Centeno CJ, Welkins WL. Significant spinal injury resulting from low-level accelerations: A case series of roller coaster injuries. *Arch Phys Med Rehab* November 2005;86:2126-30.

45. **Freeman MD**, Croft AC, Rossignol AC, Elkins W. Chronic neck pain and whiplash: a case/control study of the relationship between acute whiplash injuries and chronic neck pain. *Pain Res Manag* 2006;11(2):79-83.

46. Centeno CJ, **Freeman MD**, Welkins WL. A review of the literature refuting the concept of minor impact soft tissue injury. *Pain Res Manag* 2005;10(2):71-4.

47. Centeno C, Elliot J, Elkins W, **Freeman M**. A prospective case series of fluoroscopically guided cervical prolotherapy for instability with blinded pre and post radiographic reading. *Pain Physician* 2005;8(1):

48. Centeno CJ, Elkins WL, **Freeman M**. Waddell's signs revisited? *Spine* 2004 Jul 1;29(13):1392

49. **Freeman MD**, Nelson C. Injury Pattern Analysis as a means of driver identification *Laboratory Medicine* 2004;35(8):502-5.

50. **Freeman MD**, Olson D. Hemifacial tic following a low-speed motor vehicle crash. *J Whiplash Rel Dis* 2004;3(1).

51. **Freeman MD**, Nelson C. Injury pattern analysis as a means of driver identification in a vehicular homicide; a case study. *Forensic Examiner* Spring 2004;13(1):24-8.

52. Croft AC, Haneline MT, **Freeman MD**. Low speed frontal crashes and low speed rear crashes: is there a differential risk for injury? *Annu Proc Assoc Adv Automot Med. 2002;46:79-91.*

53. Croft AC, Herring P, **Freeman MD**, Haneline MT: The neck injury criterion (NIC): future considerations. *Accid Anal Prev* 2002;34(2):247-55.

54. **Freeman MD**, Croft AC, Rossignol AM. A critical evaluation of the methodology of a low back pain clinical trial *J Manipulative Physio Ther* 2000; 23(5):363-4.

55. **Freeman MD**, Croft AC, Rossignol AM, Weaver DS, Reiser M. A review and methodologic critique of the literature refuting whiplash syndrome. *Spine* 1999;24(1):86-98.

56. **Freeman MD**, Croft AC, Rossignol AM. Whiplash Associated Disorders (WAD) - Redefining Whiplash and its Management" by the Quebec Task Force: A Critical Evaluation. *Spine* 1998;23(9):1043-9.

57. **Freeman MD**, Fox DD, Richards TR. The superior intracapsular ligament of the sacroiliac joint: confirmation of Illi's ligament. *J Manipulative Physiol Ther* 1990;13(7):374-90.

Non-peer-reviewed publications

1. **Freeman MD**. The problem with probability. *Trial* March 2006, 58-61.

2. Croft AC, **Freeman MD**. Auto insurers and their new role in whiplash prevention—new rules, new risks, new tests. *Forum* 35(3):9-13, 2005.

3. O'Shanick G, Varney N, **Freeman MD**, et al. Blue Ribbon Panel Review of the Correlation Between Brain Injury and Roller Coaster Rides (Report to US Congress, Funded by the National Institute of Child Health and Human Development). February 25, 2003.

4. Croft AC, Herring P, **Freeman MD**, Centeno C, Haneline MT, Baric JJ: Late (chronic) whiplash injury. Public health perspectives amidst a controversial literature. JACA 40(8):26-32, 2003.

5. **Freeman MD**. Don't fall for defense fallacies. *Trial* 2000.

6. Seroussi R, **Freeman MD**. A review of original research by Brault et al. "Clinical response of human subjects to rear-end automobile collisions" *Injury Forum* 2000;2(5):49-50.

7. **Freeman MD**. The epidemiology of acute and chronic whiplash injury in the U.S. Proceedings of HWS-Distorsion (Schleudetrauma) & Leichte Traumatische, Hirnverletzung. Invaliditat und Berufliche Reintegration. Basel, Switzerland. June 29-30, 2000.

8. **Freeman MD**. Meta-analysis of whiplash prognosis studies. Proceedings of Whiplash 2000, Bath, England. May 16-18, 2000. pp 102-24.

9. **Freeman MD**, Croft AC, Reiser M. [reprint of A review and methodologic critique of the literature refuting whiplash syndrome] *Trial* March 1999

10. Croft AC, **Freeman MD**. From railway spine to whiplash. *Topics in Clinical Chiropractic* (Trauma) 1998;5(3):54-61.

11. **Freeman MD**, Croft AC, Reiser M. Die epidemiologie des Schleudertraumas - wo liegt die Schelle Zur Verletzung? (The epidemiology of whiplash - is there a reliable threshold for whiplash injury?) *HWS-Distortion (Schleudetrauma) & Leichte Traumatische, Hirnverletzung*. Edited by Ettlin TM and Mürner J. June 25-6, 1998:99-118.

12. **Freeman MD**. The first CIREN conference: motor vehicle crash-related trauma and biomechanical engineering. *JACA* 1998;35(4):54-61.

Thesis

1. **Freeman MD**. The Role of Forensic Epidemiology in Evidence-Based Forensic Medical Practice. Thesis for completion of Doctor of Medicine degree, Umeå University. Responsible publisher under Swedish law: the Dean of the Medical Faculty of Umeå University, Printed by: Print och Media, Umeå, Sweden 2013. ISBN: 978-91-7459-729-5 ISSN: 0346-6612

2. **Freeman MD**. A study of chronic neck pain and whiplash injuries. Thesis for completion of Doctor of Philosophy degree, Oregon State University. *UMI Dissertation services*, Ann Arbor, MI. 1998:9820108.

3. **Freeman MD**. Analysis of lumbar spinal strength using the doubly multivariate repeated measures design. Thesis for completion of Master of Public Health degree, Oregon State University. 1995 (unpublished)

Books

1. **Freeman MD**, Budowle B, Zeegers M, Eds. Forensic Epidemiology; Principles and Practice. Elsevier, Amsterdam, NL. 2014 (projected)

2. Nordhoff L, **Freeman MD**, Siegmund GP. Human Subject Crash Testing: Innovations and Advances. Society of Automotive Engineers, Detroit MI 2007

3. Berardinelli D, **Freeman MD**, DeShaw A. From Good Hands to Boxing Gloves; How Allstate Changed Casualty Insurance in America. Trial Guides 2008

4. **Freeman MD**. Litigating Major Auto Injury and Death Cases; Forensic Science (volume 2 of Litigating Major Auto Injury and Death Cases, Koehler K, Freeman MD). ThomsonWest:2006

5. **Freeman MD**. Litigating Minor Impact Soft Tissue Cases; Forensic Medicine (volume 2 of Litigating Minor Impact Soft Tissue Cases, Koehler K and Freeman MD). ThomsonWest:2001

Book Chapters

1. **Freeman MD**, Haneline M. Unintentional and Intentional Injuries. Chapter 7, in *An Introduction to Public Health in Chiropractic*, Jones & Bartlett.

2. **Freeman MD**, Centeno CJ, Kornel E. Acute cervical spine trauma. British Medical Journal, *BMJ in Practice*. Available online at https://online.epocrates.com/noFrame/showPage.do?method=diseases&MonographId=944

3. Hunter OK, **Freeman MD**. Cervical Sprain and Strain. eMedicine from WebMD. Updated July 15, 2009. Available at: http://emedicine.medscape.com/article/306176-overview.

4. **Freeman MD**. Forensic considerations in the mild traumatic brain injury case. In: Mild Traumatic Brain Injury: Onset, Consequences, & Outcomes Eds. O'Shanick G, Varney N. Springer (in press)

5. **Freeman MD**. The epidemiologist as forensic scientist pp 178-9. In: Principles and Practice of Epidemiology: An Engaged Approach, Rossignol AM. McGraw-Hill, NY, NY 2007

6. **Freeman MD**, Nordoff LS. Crash Injury Thresholds, (Ch. 14) in Nordhoff LS. Motor vehicle collision injuries: Biomechanics, diagnosis & management. Jones and Bartlett Publishing, Boston MA 2005.

7. Murphy D, **Freeman MD**. Management of neck pain and related disorders. In Principles of Chiropractic Text, Haldeman S, Editor in Chief. Third Edition, 2004: 969-998.

8. **Freeman MD**, Croft AC. The Controversy over Late Whiplash: Are Chronic Symptoms after Whiplash Real? in: Whiplash Injuries Edited by M. Szpalski and R. Gunzburg. Lippencott-Raven. September 1997

Scientific and Peer-reviewed Conference Proceedings and Abstracts

1. Williams K, **Freeman MD**, et al. The investigation of a cluster of fentanyl overdose deaths: how the use of epidemiologic surveillance and outbreak methods resulted in the rapid identification of the source of a public health crisis. *Proceedings of 2014 National Association of Medical Examiners annual meeting.* September 19-23, 2014, Portland, OR (pending)

2. **Freeman MD**. Forensic Applications of Epidemiology in Civil and Criminal Litigation. *9th International Conference on Forensic Inference and Statistics* August 19-22, 2014, Leiden, NL (pending)

3. Rubanzana W, **Freeman MD**, Hedt-Gauthier B. Exposure to genocide and risk of suicide in Rwanda: a population-based case-control study. *20th IEA World Congress of Epidemiology.* August 2014, Anchorage, AK (in review).

4. **Freeman MD**, Uhrenholt L. Investigation of a disputed mechanism of diffuse axonal injury following a low speed frontal crash. *Proceedings of 65th Annual Meeting of the American Academy of Forensic Sciences* 2014 Feb 17-22: Seattle

(WA). 1984:367-8.

5. **Freeman MD**, Cahn P. Cause of death following opiate overdose; a forensic epidemiologic investigation of comparative risk. *Proceedings of 65th Annual Meeting of the American Academy of Forensic Sciences* 2014 Feb 17-22: Seattle (WA). 1984:396.

6. Rubanzana W, **Freeman MD**, Hedt-Gauthier B. Risk factors for homicide victimization in Rwanda, post Genocide: A forensic epidemiological investigation. *Proceedings of 65th Annual Meeting of the American Academy of Forensic Sciences* 2014 Feb 17-22: Seattle (WA). 1984:564-5.

7. Colville-Ebeling B, **Freeman MD.** An Evaluation of the Discriminatory Power of Clinical Diagnostic Findings of Abusive Head Trauma in U.S. Hospitals. *Proceedings of 65th Annual Meeting of the American Academy of Forensic Sciences* 2014 Feb 17-22: Seattle (WA). 1984:346-7.

8. Hatten B, **Freeman MD,** Horowitz Z. Coral Snake Envenomations 2001-2011: Antivenin Use and Outcomes. Proceedings of the Society for Academic Emergency Medicine. 2013, Annual Meeting in Atlanta, Georgia, May 14-18, 2013 (in press).

9. Rubanzana W, **Freeman MD.** Exposure to effects of genocide as a risk factor for intentional death in Rwanda: a forensic epidemiological investigation. *Scand J Forens Med* 2012;18(1):117.

10. **Freeman MD**, Dobbertin K, Kohles SS, Uhrenholt L, Eriksson A. Serious head and neck injury as a predictor of occupant position in fatal rollover crashes. *Scand J Forens Med* 2012;18(1):34-5.

11. **Freeman MD**, Uhrenholt L. Self-defense or attempted murder? A combined ballistic and traffic crash reconstruction of a Texas shooting. *Scand J Forens Med* (2012;18(1):51.

12. **Freeman MD.** Applied forensic epidemiology: the evaluation of individual causation in wrongful death cases using relative risk. *Scand J Forens Med* (2012;18(1):25.

13. Uhrenholt L, Webb A, Pedersen M, Christensen HW, **Freeman MD.** Does whiplash trauma result in somatic injury. *Scand J Forens Med* (2012;18(1):121

14. **Freeman M**, Uhrenholt L. Rollover collisions; the effect of restraint use on skull vault fractures. 2011. Poster session presented at Årsmøde i Dansk Selskab for Retsmedicin og Dansk Selskab for Ulykkes- og Skadeforebyggelse [The Danish Traffic Medicine Society of the Danish Society for Forensic Medicine] November 3-5, 2011] Grenå, Denmark.

15. Uhrenholt L, **Freeman M**, Jurik AG, Jensen LL, Gregersen MEG, Boel LWT et al. Evidence of somatic injury in rear-impact collisions - esophagus injuries. 2011. Poster session presented at Årsmøde i Dansk Selskab for Retsmedicin og Dansk Selskab for Ulykkes- og Skadeforebyggelse [The Danish Traffic Medicine Society of the Danish Society for Forensic Medicine] November 3-5, 2011] Grenå, Denmark.

16. Uhrenholt L, **Freeman M**. How microscopy can explain traffic crash-related cervical spine injury. 2011. Poster session presented at Årsmøde i Dansk Selskab for Retsmedicin og Dansk Selskab for Ulykkes- og Skadeforebyggelse [The Danish Traffic Medicine Society of the Danish Society for Forensic Medicine] November 3-5, 2011] Grenå, Denmark.

17. Nystrom NA, **Freeman MD.** "Trigger Point" Surgery for Soft Tissue Pain in Chronic Whiplash Syndrome. *J Rehab Medicine* 2011;Suppl 50:27.

18. **Freeman MD**, Kohles SS. Scientific and Legal Criteria for Evaluating Injury Causation Following Whiplash Trauma. *J Rehab Medicine* 2011;Suppl 50:20.

19. Rosa S, **Freeman MD**, Harshfield D. Restoration of Normal Cerebrospinal Fluid Flow in 2 Cases of Confirmed Cerebellar Tonsillar Ectopia with Long-Term Headaches, Following Use of The Atlas Orthogonal Instrumented Manipulation Technique. *J Rehab Medicine* 2011;Suppl 50:14.

20. **Freeman MD**, Centeno CJ. "Whiplash-Associated Disorders [WAD]" – the persisting lexicon of a failed venture. *J Rehab Medicine* 2011;Suppl 50:6-7

21. Kohles SS, **Freeman MD.** Mathematical Models Characterizing the Probability of Trigger Event, Ambient-Risk, and Coincidental Influences on Inductive and Abductive Conclusions of Specific Causation. *Annals of Epidemiology* 2010;20(9):713-4.

22. Centeno MD, **Freeman MD**, Schultz J, Cheever M, Faulkner S, Hanson R, Kohles S. Clinical Percutaneous Implantation of Autologous, Culture-Expanded MSCs into Peripheral Joints. *Orthopedic Research Society*, 2011 Annual Meeting in Long Beach, California, January 13-16.

23. Nystrom A, **Freeman MD**. Central sensitization is a reversible response to focal soft-tissue neck pain in chronic whiplash. *2010 American Academy of Orthopedic Surgeons Annual Meeting* March 9-13, 2010, New Orleans, LA.

24. **Freeman MD.** The Error Odds method of objectively assessing bioengineering based claims of causation; a Bayesian approach to test validity quantification (Special joint session of Jurisprudence and Engineering Sciences) *Proceedings of 62nd Annual Meeting of the American Academy of Forensic Sciences* Feb 2010, Seattle, Washington.

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Freeman MD. Analysis of the whiplash literature with emphasis on research out of Quebec and Saskatchewan. *Saskatchewan Medical Group and Coalition Against No-Fault.* Saskatoon, Saskatchewan. September 2000.

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Appendix R

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Review

Forensic Epidemiology: A systematic approach to probabilistic determinations in disputed matters

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Abstract

Forensic medicine testimony often relies upon terms of probability to enhance the strength of the testimony. Such terms must have a demonstrably reliable and accurate basis; otherwise their use is speculative, unjustified, and potentially harmful. Forensic Epidemiology is introduced as a framework from which probabilistic testimony can be assessed in settings in which it is either proffered or encountered. In this paper, common forensic uses of probability are reviewed, appropriate methods for presenting such testimony are proposed, and inappropriate uses of probability and epidemiologic concepts and data, as well as a logical fallacies commonly observed in forensic settings are presented. A previously unpublished logical fallacy, the "Prior Odds" Fallacy, is also introduced.
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Keywords: Probability; Epidemiology; Forensic; Prior Odds Fallacy; Sensitivity; Specificity; Positive Predictive Value

1. Introduction

In 1999, British solicitor Sally Clark was convicted in a United Kingdom court of murdering two of her children. Both of the infants died within weeks of birth under circumstances originally diagnosed by some experts as sudden infant death syndrome (SIDS) or cot death.[1] An important witness for the prosecution was the prominent pediatrician Sir Roy Meadow, who testified that the probability of two cot deaths in one family was exceedingly remote; about 1 in 73,000,000. The miniscule probability that the deaths were due to natural causes was used by the prosecution as evidence that the deaths were homicidal. Meadow was a well-known and often-used prosecution witness in similar proceedings, having been the first to promulgate the concept of Munchausen Syndrome by Proxy (MSbP) in which a parent injures or sickens a child as a means of procuring medical attention.[2] Meadow's Law, a heuristic attributed to Meadow that pertains to multiple cot deaths in families, states that unless otherwise proven, one death is tragic, two is suspicious, and three is murder.[3]

In the Clark case, the estimate of 1 in 73,000,000 was derived from squaring the observed risk of a single cot death in an affluent non-smoking family; estimated at 1 in 8500. Meadow's testimony created a furor among statisticians, with the president of the Royal Statistical Society

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writing an open letter of complaint to the Lord Chancellor,[4] and the British Medical Journal publishing an editorial concerning the error of allowing non-qualified experts to testify regarding unsound statistically based arguments.[5] The primary complaint with Meadow's testimony was that from a statistical perspective, he treated the specific risk to the Clark family for cot death like they were a randomly selected family with no predilection for the event in question. In other words, Meadow considered the one family in 8500 with a cot death to simply be unlucky, with no biological or environmental diathesis for the condition, rather like the probability that one will roll a six with a die. Thus, according to Meadow, the second death was no more likely than the first, just as a second six is no more likely than the first, and the probability of each can be multiplied to arrive at the probability of both.

The most obvious problem with this thinking was that it presumed that all possible biologic and environmental risk variables for cot death have been investigated and are known, and that none were present in the Clark family; an over simplification to the point of deception. The approach could be compared to the superficially convincing claim that an injury by lightning strike is a random event, and thus a second such injury would be no more likely than the first. In reality, an individual who works on a golf course in a geographic area with frequent thunderstorms is much more prone to a lightning strike injury than is an office worker who resides in an area where such storms are rare.

The guilty verdict was appealed, based in part on the problems with Meadow's statistical claims. The conviction eventually was overturned when it was found that a witness had failed to reveal evidence that one of the children's deaths may have been associated with a *Staphylococcus aureus* infection, among other problems.[6] The public attention brought to the Clark case also focused attention on the lack of validated criteria necessary for a diagnosis of MSbP, with the result that several murder convictions that had resulted from Meadow's testimony were reviewed.

The tragedy of the Clark case raises the question of how such a chain of events could have occurred; how could testimony that is fundamentally flawed be used to imprison an innocent person? An issue of even greater concern is how often similar superficially convincing testimony is used effectively in criminal and civil proceedings, resulting in an unjust verdict. What is apparent from the many different experts from a variety of disciplines who gave opinions regarding the faulty testimony in the Clark case, is that not only is there uncertainty regarding what constitutes valid testimony on some issues, but that there is also a degree of uncertainty as to who should be setting the standards for such testimony. As an example, while it is not difficult to determine that the relevant expert to testify in a medical malpractice claim against an oncologist whose patient died from hypovolemic shock while receiving autologous stem cell therapy is another oncologist, who is the appropriate expert to address a claim that the patient would have died within 5 years, on a more likely than not basis?

Any expert opinion that addresses the probability, risk, incidence, or prevalence of an event occurring or not occurring in an individual or a population is an opinion that must have a foundation in valid epidemiologic concepts and data. Epidemiology is most simply defined as the scientific study or analysis of populations having similar disease or injury characteristics. The proper application of epidemiologic concepts and data to forensic issues is the practice of Forensic Epidemiology. The term was first introduced by Loue in 1999[7] and later adopted by the US Centers for Disease Control and Prevention (CDC) in 2003 as a narrowly focused Public Health Law Program module designed to aid with the investigation of acts of bioterrorism.[8] The subject matter covered by the term Forensic Epidemiology has since been expanded to cover the multitude of areas in which epidemiologic terms, concepts, and data may be applied in a forensic venue.[9]

2. Forensic Epidemiology

The practical application of Forensic Epidemiology (FE) concerns both the recognition of the improper use of epidemiologic concepts and data as well as the use of such testimony to help prove the assertion of one side or another. For this reason, this paper is organized in three sections; the first will help the reader identify the most common scenarios in which experts (both epidemiologic and non-epidemiologic) use terms and concepts of epidemiology in forensic venues. The next section describes appropriate uses of epidemiologic concepts and data in forensic venues. The final part of the paper is devoted to common fallacies associated with epidemiologic and probabilistic testimony in forensic venues. Because population-based inferences are often-used to support variety of expert opinions, the tenets of FE, as presented in this paper, are directed at all experts who give opinions regarding medicolegal issues in forensic venues.

2.1. Common testimony types involving FE concepts

Probability serves as the basis for many decisions people make on a daily basis. When one purchases a new DVD player and opts not to also purchase an extended warranty, one is evaluating, consciously or subconsciously, a series of probabilities. Many factors – how much the unit costs, one's prior experience with DVD player failure, and how long one typically keeps electronics before replacing them with an updated model – affect the decision that it is less than likely that the expenditure on the warranty is justified. If one is given some additional knowledge, for example, that one out of every three DVD players will require a costly repair within a year after the manufacturer's warranty expires, then one can assess the risk one is taking in not purchasing the warranty.

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In a similar fashion, data and terms of probability may be used to sway judge and jury fact finders in a forensic setting, because assigning a weight to an opinion is a common method of strengthening testimony. These opinions affect how a fact finder perceives issues such as causation, negligence, and injury severity and prognosis that dictate trial outcomes.

2.1.1. A "reasonable probability"

The use of probabilistic language is inescapable in the forensic setting, since the standard for admitting expert testimony is that it is rendered as being "more probable or likely than not" or as a "reasonable probability" or "reasonable medical probability"; all relatively interchangeable terms.[10] In some jurisdictions, this standard for expert opinion is assigned a value that must be exceeded before the testimony is admissible; the expert must be "more than 50% certain" that the opinion is correct. Using probabilistic language for such testimony is somewhat of a mischaracterization of an internal process of the expert, who has opined that he is more certain than not that his opinion is accurate or true, regardless of the methods used to arrive at the opinion. There is no way objectively to weigh all of the processes that make up such a standard, because experts potentially are influenced by many different factors that may cause them to favor a particular opinion. One example of an exception, in which the decision processes of the expert can be externally scrutinized, is when a data set is described and an opinion is rendered that a particular outcome lies inside or outside of an error range or confidence interval bracketing the average of the data set. In such an instance, an opinion that it is a reasonable probability that an outcome would *not* take place, for instance, the failure of a medical device, could be reached based upon the application of valid and relevant epidemiologic/statistical tenets to the relevant data.

Probabilistic opinions may be expressed in terms of the:

- *Incidence of an occurrence or condition.* This is expressed as a rate, with a number of affected persons per some denominator. For example, the rate of traffic crash fatalities in the United States in 2004 was 1.45 per 100,000,000 vehicle miles traveled, 14.59 per 100,000 persons, 18 per 100,000 registered vehicles, and 21.54 per 100,000 licensed drivers.[11] Although the numerator and denominator are different for each estimate they all represent the same total number of deaths for 2004.
- *Prevalence of an occurrence or condition.* Prevalence is essentially a cross-section of a population at a given point in time. It is expressed as a proportion or percentage, such as the prevalence of cancer in the United Kingdom is approximately 2%, meaning that one out of every 50 live persons has a diagnosis of cancer in the UK.[12] Prevalence can be estimated from the incidence and survival rate of a condition.
- *Risk of an occurrence or condition.* Typically given as a proportion or percentage, this is the most commonly used and abused epidemiologic concept in forensic testimony. Risk may be expressed in absolute terms, *e.g.* the risk of dying in a motor vehicle crash in a given year is approximately 1 in 6500,[13] or as a relative risk (typically presented as a ratio, but also as a difference), such as the lifetime risk of dying in a car crash is more than 23,000 times greater than dying from a snake bite.[3] Misunderstanding is rife in such claims, however. For example, while it is reasonable to conclude that one is significantly less likely to die from a snake bite than in a traffic collision, this does not mean that handling a venomous snake is safer than driving a car. The average person's exposure to a venomous snake, in terms of duration, may be more than 23,000 times less than their exposure to a motor vehicle; thus the incidence of snake bite death may be significantly higher per unit of time of exposure than that of motor vehicle death per exposure for the same unit of time.

Opinions involving risk often rely upon probabilistic language, and this in turn may lead to a lack of specificity. For example, it is reasonable to opine that not wearing a seatbelt increases the risk of ejection in the event of a rollover crash, an important determination in some forensic venues, as it may indicate contributory negligence of the occupant to his or her own injuries for failure to wear a seatbelt. On the other hand, if the occupant was not wearing a seatbelt, and was *not* ejected but still fatally injured, the presence or absence of a seatbelt is a significantly smaller factor for injury frequency and severity, particularly if there is a great deal of vehicle roof crush that may have resulted in severe head and neck injury to a properly positioned and restrained occupant.[14] Quantification of the difference in risk of injury between the two scenarios would be important in helping a fact finder (judge or jury) determine whether the lack of a seatbelt was a significant factor in the case in question.

The following are a sample of common forensic opinions that state or imply probability (*NB* – the validity of the opinions is not addressed):

- *It is more likely that the patient will need future surgery as a result of the injury*
  - This is a prediction of an event that has not yet occurred. It implies that the future *incidence* of surgery for those who have the injury is higher than for those who do not. Such claims do not necessarily have to be based upon published epidemiologic data, because they can be a statement of clinical experience, based on one or both of two observations: that patients with the injury in question go on to have the surgery more often than patients who do not, or that an disproportionately large percentage of patients who have the surgery have a history of the injury. Similar claims, of what has been observed

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and thus is *possible* or *plausible*, are the converse of statements of what is *impossible*, with regard to the level of substantiating data needed for a valid conclusion. As an example, if one wanted to determine if any red headed subjects were included in a group of 100, a sample size of one could establish the fact, if a read head was included in the sample. Conversely, if one wanted to establish that there were no red heads in the group one would have to examine the hair color of every group member. The level of proof required to validate a claim of possibility or plausibility is significantly less than what is required to establish impossibility or implausibility.

- *If the occupant had worn his seatbelt the injury would not have occurred*
  - The statement implies that the *risk* of injury for similar crashes with similar occupants who are restrained is 0. Unlike the previous opinion, such a statement implies a basis in data gathered from large samples of crashes and occupants that are similar to the case in question, as it implies impossibility of an outcome not just within the realm of the expert's experience but for all restrained occupants exposed to the same type of collision.

- *The disc herniation was not caused by the fall because the patient did not have immediate acute pain, something that would have been expected with a traumatic disc herniation*
  - This is a statement of prevalence for a certain condition at a certain point in time; it refers to the status of all patients with a traumatic disc herniation shortly after the trauma has occurred. The claim forces an inference that 100% of such patients will have pain immediately following injury. It would be unusual, if not unheard of, to find a population sample that would allow for such a definitive and broadly sweeping inference. In some cases, however, there may be a physiologic reason for such a statement, as for example, when the presence or absence of evidence of hemorrhage is used to determine whether injury may have occurred pre or post-mortem.

- *Retinal hemorrhage in an infant is reliable indicator of shaken baby syndrome*
  - This claim sounds like an estimation of point prevalence, but in fact it is a statement of prevalence ratio (also known as odds) as it implies a comparison of the finding of retinal hemorrhage in violent assault versus some other trauma. Because many shaken baby syndrome homicide prosecutions are defended with the assertion that the injuries resulted from a fall or some other precipitating unintentional trauma, the claim implies that the incidence of retinal hemorrhage in infants that have sustained an unintentionally self-inflicted injury is very small – at or near zero, and that the incidence of retinal hemorrhage in infant victims of assault is significantly higher.[15] As a hypothetical example, it might be said that only 1% of infant fatalities that result from an unintentional injury result in retinal hemorrhage, whereas 75% of confirmed cases of shaken baby syndrome have the same finding. Thus, a finding of retinal hemorrhage is at least suggestive of homicide, absent any other evidence. What is less clear is how such evidence would be presented when there is a smaller difference between the two prevalence estimates, *e.g.* 30% versus 50%, and at what point the difference becomes insignificant from a evidentiary perspective. This is further discussed in Section 2.2.3.

2.2. Principles of applied Forensic Epidemiology

This section of the paper focuses on how FE is used to formulate or substantiate an opinion, and the principles governing such an application. FE is of little use in describing something that has already occurred and been observed; this is the job of the clinician. However, when there are questions regarding causation of injury, or multiple potential causes, or unknown outcomes, the probability that one cause played a greater role than another must be weighed, and this often requires the interpretation of data derived from epidemiologic study. An example would be a crash-related head injury associated with a multiple impact collision scenario, including both frontal and near side impacts, with the forensic question of which impact caused the injury. An FE approach would consist of evaluating the probability of a head injury for a near side impact in which the occupant's head is highly likely to sustain a high acceleration contact with an unyielding structure such as the B-pillar, in comparison with a frontal crash scenario in which peak head acceleration is typically lower. The basis for the opinion would have to come from analysis of real world data, as illustrated in the example in Fig. 1. The analysis may already exist in the literature or it may need to be conducted *de novo* for the purposes of the forensic investigation. The probabilistic data can not

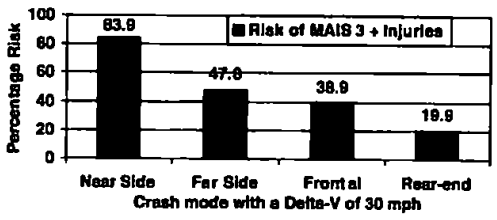

Fig. 1. Adapted from [Augenstein J, Perdeck E, Bowen J, Stratton J, Singer M, Horton T, Rao A. Injuries in near-side collisions. In: Proceedings of the 43rd annual conference of the association for advancement of automotive medicine; 1999. p. 139–58]. (MAIS 3+ refers to injuries rated as serious or greater on the Abbreviated Injury Scale.)

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conflict with the real evidence; for example, if the medical evidence clearly showed an injury to the occupant's face then this would be given more weight that the probabilistic evidence. The FE conclusion should support, or be supported by, the evidence in the case.

Another application of FE is in the "what if" scenario in which an undisputed outcome is compared to a theoretical outcome if a predicate action or event had been different. For example, in a case of alleged medical negligence, in which an encapsulated ovarian granuloma tumor has been incompletely removed, the effect might be that the tumor is advanced from a FIGO Stage I (contained within the ovary) to a Stage II (spread to the pelvis). With adequate data, and based on the alternate "what if" scenario of the surgeon performing the procedure completely, the 5-year survival probability for the actual stage of the tumor (II) can be compared to that of the stage of the tumor had the alleged negligence not taken place (I).[16]

FE also has demonstrated utility in the criminal prosecution of alcohol-associated vehicular homicides in which some or all occupants were ejected, and there was dispute as to who was driving; the decedent or an ejected, as well as intoxicated, survivor.[17] This application of FE, called injury pattern analysis, evaluates injury patterns associated with various occupant positions, and relies upon probabilistic weighting of observed injury distribution and nature versus expected anatomic distribution and pattern of injury based upon observational epidemiologic study of similar types of collisions.[18]

2.2.1. Causation

Standards for epidemiologic determinations of cause and effect were first laid out in a systematic fashion by Hill in 1969.[19] Hill outlined nine criteria by which determinations of causation could be made when there is substantial epidemiologic evidence linking a disease or injury with an exposure, *e.g.* smoking and lung cancer. The criteria have since been modified and distilled by others[20–23] but they all comprise three basic elements:

1. There must be a biologically plausible link between the exposure and the outcome. Traumatic loading and bony fracture would be a straightforward example of a plausible link. An example of an implausible link would be trauma and leukemia. Plausibility is a very low threshold that can be overcome with relatively weak evidence, such as from small observational studies (case studies or case series with small numbers of subjects), or from the results of well-designed experiments with many subjects. Analogy also is a valid method of establishing plausibility; if forceful loading from one type of trauma can cause an injury, than forceful loading from another type of trauma may be a plausible cause of the same kind of injury.
2. There must be a temporal relationship between the exposure and the outcome. Quite obviously, the outcome cannot pre-exist the exposure. Less obviously, the outcome cannot postdate the exposure by a time period that is clinically considered to be too long or too short to relate the two. This determination is highly dependent upon the specifics of any case. For example, benzene exposure to the skin will cause symptoms of irritation that are apparent within 1 day, however, changes to the blood system may not be apparent for months. A crash-related injury to the spine may not be apparent for a day or two or even a week, but will not be completely latent for 2 months prior to causing symptoms. On the other hand, an injury that causes acute symptoms to the spine may mask or overlap with other symptoms resulting from, for example, a concomitant shoulder injury. Such an injury may not be apparent for some time following the original traumatic episode. The determination of etiology is typically made based on clinical judgment on a case-by-case basis, rather than from clearly delineated guidelines or principles.
3. There must not be any *likely* alternative explanations for the symptoms. The term "likely" is of critical importance, as, for example, it is not sufficient to simply point out that a patient with back pain following trauma is obese, that obesity is related to back pain, and thus it is more likely that the obesity rather than the trauma caused the back pain. For an alternative etiologic explanation to be considered more likely than an alleged exposure it must be both biologically plausible and have a stronger temporal relationship to symptom onset than the alleged exposure. If plausibility is present and temporality is relatively comparable, then two exposures can be compared by examining the dose–response of each exposure. This term typically refers to the magnitude and intensity of each exposure, but for the purposes of FE may also refer to outcome risk. An example of a comparison of dose–response is seen in the above example comparing the probability of head injury for a near side impact collision versus a frontal impact. Both are biologically plausible mechanisms of head injury and both occurred at the same time; however, the near side impact scenario has an established higher head injury risk.

It is common practice for clinicians, rather than epidemiologists, to make determinations of causation in individual patients. A clinician's causal determination incorporates the patient's history and the results of examinations and tests with the clinician's experience and training to arrive at a conclusion regarding causality. Such determinations, however, may not violate any of the three basic elements of causation.

2.2.2. Strength of evidence

In 1993, the United States Supreme Court issued an opinion in a case called Daubert v. Merrell Dow Pharmaceuticals Inc. This case set new standards for evidentiary hearings in the United States, in which the judge acts as a gatekeeper for proposed scientific testimony.[24] The case

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concerned the alleged teratogenic effects of the drug Bendectin, used primarily for pregnancy-associated morning sickness. The plaintiff in the case had brought forth evidence from a variety of experts who cited *in vitro*, animal, and chemical studies as a basis for their collective opinion that Bendectin caused the birth defects that were the subject of the lawsuit. In response, the defense produced an epidemiologist expert who presented an analysis of epidemiologic (observational) studies of women who had used the drug, and opined that there was no relationship between the use of Bendectin and birth defects. A lower court had ruled that the experimental evidence presented by the plaintiff was insufficient to establish causation in light of the epidemiologic evidence of the defendant. When the case reached the Supreme Court the Justices ruled in favor of the defendant, affirming the ruling of the lower courts and establishing a new set of criteria for the admissibility of expert scientific testimony. The Daubert decision helped to highlight the use and misuse of forensic scientific evidence to establish or question causation. In this decision, a causal relationship suggested or refuted by an animal or cadaveric study is insufficient proof for establishing the etiology of an injury or disease when there is contradictory observational evidence. The latter includes clinical determinations of causation that do not violate the three basic elements of causation noted above. Fig. 2 illustrates the hierarchy of evidence strength in terms of its utility in establishing causality. The Daubert decision solely addresses evidence that is intended to support or refute the first element of causation, biologic plausibility. Temporality and likely alternative explanations are primarily clinical determinations and thus largely unaffected by Daubert.

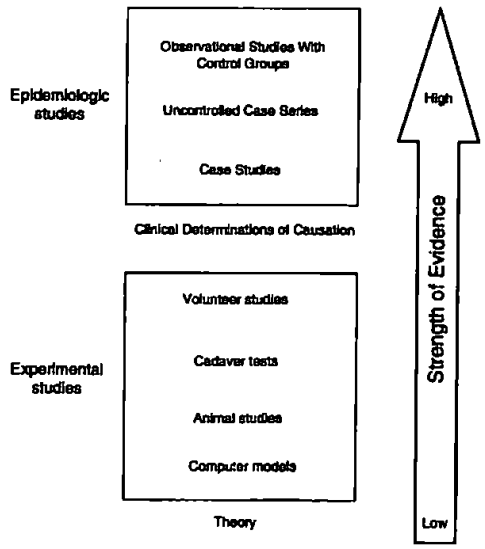

Fig. 2. Hierarchical depiction of evidence strength for causal determinations modeled from the Daubert decision.

2.2.3. Sensitivity, Specificity, and Positive Predictive Value

These are fundamental epidemiologic concepts that are critical to appropriate weighting of testing results. These concepts are equally important for understanding the precision or reliability of a particular opinion based upon the interpretation of a fact in evidence using an established set of criteria. For any test or criterion there are at least four possible results: true *positive* (TP), in which the test correctly identifies the guilty (or liable) party, true *negative* (TN), in which the test correctly identifies the innocent party, and false *positive* (FP) and false *negative* (FN), in which the test incorrectly identifies the innocent as guilty or the guilty as innocent, respectively. In a criminal forensic setting, the sensitivity of a test indicates the percentage of guilty defendants the test correctly identifies as guilty, and is calculated by dividing the true positives (the correctly identified guilty defendants) by all of the guilty defendants, included those incorrectly identified as innocent (TP + FN). The specificity of the test indicates the percentage of innocent defendants that the test will correctly identify, and is calculated by dividing all of the correctly identified innocent defendants (the true negatives) by all of the innocent defendants (TN + FP). The most important parameter of a test or criterion that may be used in a forensic venue is its Positive Predictive Value (also called Predictive Value Positive), as this value indicates how often the test is correct when it indicates guilt. As such, PPV measures the potential for harm when a particular test is used as an isolated index of guilt, as it also demonstrates the proportion of innocent defendants incorrectly identified as guilty. Table 1 is matrix that illustrates these measures.

For purposes of illustration, an example of the utility of Positive Predictive Value can be made with the claim made earlier in this paper, that retinal hemorrhage (RH) is a reliable indicator of shaken baby syndrome (SBS). For the following example it is assumed that the claim that RH is "reliable" equates to a sensitivity of 90% and a specificity of 75%; that is, RH is present in 90 out of 100 cases of SBS, and 75% cases of non-SBS death will not have RH. If one were to present these statistics as support for the opinion that a pediatric death resulted from SBS because RH was present it would likely be given a great deal of weight in a forensic setting. If the contrasting defense theory is that the fatal injury and RH resulted from a fall instead of a violent assault, the Positive Predictive Value (PPV) of RH as an indicator of SBS can help a jury determine the weight they should give to the evidence. In order to calculate PPV, however, it is necessary to know more about the data underlying the sensitivity and specificity calculations. If, for example, there are 200 cases of SBS deaths annually, and this results in 180 (90%) with findings of RH, and there are 1000 cases of non-SBS head injury annually, with only 250 (25%) with findings of RH, then the PPV is only 42% (see Table 2), and a determination of SBS based

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Table 1
2 × 2 matrix illustrating the relationship between the Sensitivity, Specificity, and Positive Predictive Value of a test for guilt

		Criterion			
		+	–		
Guilt	Yes	True Guilty (TP)	False Innocent (FN)	All Guilty (TP + FP)	Positive Predictive Value TP/(TP + FP)
	No	False Guilty (FN)	True Innocent (TN)	All Innocent (FN + TN)	Negative Predictive Value TN/ (FN + TN)
		All + tests (TP + FN) Sensitivity TP/ (TP + FN)	All – tests (FP + TN) Specificity TN/ (FP + TN)	All cases (TP + FP + FN + TN)	

TP and FP are True Positive and False Positive and TN and FN are True Negative and False Negative.

upon the finding of RH alone would be improper (*NB*: the figures used in the above example are solely for illustration).

2.3. Common forensic fallacies involving epidemiologic concepts

2.3.1. Prior Odds Fallacy

The Prior Odds Fallacy, described for the first time in this paper, is related to the Prosecutor's Fallacy, in which the pre-event or predictive odds associated with a piece of evidence are presented to the jury as a gauge of guilt.[25] An example of the Prosecutor's Fallacy is as follows: a rare blood type, present in only 1% of the population and matching that of a suspect is found at a crime scene. The prosecutor uses this evidence to suggest that there is a 99% probability that the suspect is guilty. The reason the inference is a fallacy is that while the matching blood type is *suggestive* of guilt, the 99% figure is unrelated to the *certainty* of guilt. For example, in a city of 1,000,000 there would be 10,000 people with the same blood type as the suspect. Or, the crime may have taken place in an ethnically homogenous community where 90% of the local denizens have the rare blood type.

In contrast with the Prosecutor's Fallacy, the Prior Odds Fallacy, typically offered in injury litigation settings as evidence against or for causality, is not suggestive of either. The Prior Odds Fallacy is seen when the low probability of an occurrence, *e.g.* possessing a winning lottery ticket, is used to cast doubt on the accuracy of the observation of the occurrence.

The following example illustrates the fallacy: a woman is involved in a rear impact collision that results in minimal damage to her vehicle, and is subsequently diagnosed with a permanent spine injury by her doctor. The insurer defending the case hires a doctor who examines the patient and opines that the majority of crash injuries recover spontaneously within a matter of months following a crash with minimal damage, and therefore it is highly improbable that the signs and symptoms of permanent injury are related to the collision in question. The Prior Odds Fallacy was committed in the example when the pre-crash or "prior odds" of contracting a permanent injury (say 1 in 20 or 0.05) was used suggest a correspondingly high probability (19 out of 20, or 0.95) that the original doctor's determination of causation was in error. The fallacy occurs due to the fact that there is no relationship between the probability of injury in the general population exposed to minimal damage crashes (0.05) and the frequency of clinician error in determining causality in patients that have been exposed to minimal damage crashes (unknown, but unlikely to be 0.95). The pre-event probability of an occurrence is not a valid measure of whether the occurrence took place; either it did or it did not (0.0 or 1.0). As a simple example, deaths resulting from plane crashes are exceedingly rare, however, a pathologist's clinical observations of a decedent following a plane crash would not be considered to be in error because the death was unlikely to have occurred.

The fallacy can be further illustrated with the example of the roll of a six-sided die. The probability that a six will be rolled is 1 in 6 (17%), and the probability that something other than a six will be rolled is 5 in 6 (83%). In this example,

Table 2
2 × 2 table illustrating the Positive Predictive Value of retinal hemorrhage presence as a gauge of guilt

		Retinal hemorrhage			
		Present	Absent		
SBS	Yes	True Positive (TP) 180	False Negative (FP) 20	All SBS + (TP + FP) 200	Positive Predictive Value TP/ (TP + FN) 42%
	No	False Positive (FN) 250	True Negative (TN) 750	All SBS – (FN + TN) 1000	Negative Predictive Value TN/ (FP + TN) 25%
		All cases with RH (TP + FN) 430	All cases without RH (FP + TN) 770	All deaths (TP + FP + FN + TN) 1200	
		Sensitivity TP/(TP + FP) 90%	Specificity TN/(FN + TN) 75%	Prevalence of SBS in all deaths (TP + FP)/(TP + FP + FN + TN) 17%	

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the result of the roll is recorded by a hypothetical machine that has been found to have an error rate of one in 50 observations, so that the roll is misidentified in 2% of cases. The Prior Odds Fallacy would occur if a 6 was rolled and subsequently identified as such by the machine (with a 2% error rate), but it was asserted that there was an 83% probability that the result was something other than a 6 (83% probability the machine is wrong).

The error rate of clinical determinations of causality in minimal damage crash injuries is not known, but it is not likely to be very large. Such determinations would depend upon the observation of the three criteria of causation described earlier in this paper. Conversely, the error rate resulting from the introduction of evidence that invokes the Prior Odds Fallacy, if a judge or jury determination is based upon such evidence, would be calculated as follows:

$$\text{Prior Odds Fallacy error rate} = 1 - E_c,$$

where E_c is the actual error rate in clinician observations of causation. Although there are no published data on such errors, for the purposes of this paper the error rate is assigned a value of 0.05, theoretically taking into account cases in which false patient or physician attribution of causation has occurred. Thus, using the values stated above, the rate error in fact finder determinations when the Prior Odds Fallacy is accepted as evidence is $1 - 0.05 = 0.95$ or 95%.

In criminal cases the only fact finder determination is the guilt or innocence of the accused. This scenario differs from civil litigation, in which the fact finder must determine (a) whether the defendant acted negligently, and if so then (b) whether the act of negligence is causally related to the alleged injuries, and if so, then (c) the amount of damages to be awarded. The Prior Odds Fallacy is primarily directed at the causation determination in civil litigation. Further, of the three causation elements identified earlier in this paper (biologic plausibility, temporality, and the lack of a likely alternative explanation), the Prior Odds Fallacy is directed mainly at biologic plausibility, relying upon the implication that a low prior odds (*e.g.*, only one in 20 will be injured) is an indicator of *implausibility*. In fact, a very low level of scientific or clinical evidence is required to assert a *plausible* biologic association between a noxious exposure and an injury outcome, as plausibility is either present or not, regardless of degree. Thus, assertions of low frequency of association between an exposure and an outcome are irrelevant to biologic plausibility.

The Prior Odds Fallacy also occurs in plaintiff expert testimony that intended to support causality in civil litigation. An example is sometimes seen in crash injury cases, in which photographs of extensive vehicle damage are used to elicit testimony that the degree and extent of injury observed in a patient are consistent with the crash, implying a low likelihood of error in the observation of causal association between the subject crash and the diagnosed injuries. Regardless of whether the frequency of injury is 5%, 10%, or 40% for similar crashes, diagnostic error is independent of pre-event probability of injury.

2.3.2. Fallacies contributing to lower prior odds estimates

All of the following fallacies are used to establish an erroneous pre-event probability that would then be applied to causation via the Prior Odds Fallacy:

(a) *Non-representative sample fallacy.* Results observed in one side of a population distribution curve cannot be used to argue thatthe other side does not exist or is rare. An example is seen in the human subject crash testing literature in which some authors have concluded that there is a crash speed injury threshold below which injury is unlikely or impossible in the general population[26,27] with the intent that the thresholds be applied in medicolegal settings as a litmus test for causation.[28] The results of testing of the hardiest members of the population who are not injured until they are exposed to high crash forces (arrow "A" in Fig. 3) cannot be used to exclude the existence of, or in any other way define the distribution of the members of the population who are injured when exposed to low crash forces (arrow "B" in Fig. 3). The use of volunteer crash tests to establish an injury threshold in the general population has been criticized as unscientific, as the studies underlying the proffered forensic opinion suffer from (1) inadequate study numbers of subjects, vehicles, and crash conditions (contributing to random variation), (2) non-representative study samples (crash test volunteers cannot be said to represent the full spectrum of the motoring public with regard to injury susceptibility), and (3) they are conducted under non-representative conditions (crash tests are designed to minimize participant injury risk).[29] The fallacy also occurs when *in vitro*, *ex vivo*, animal model, computer model, and other surrogates are used as a basis for establishing or questioning causation.

(b) *Appeal to statistical authority.* Juries are more likely to be convinced of the validity of testimony when it is supported with a reference to statistics or statistical

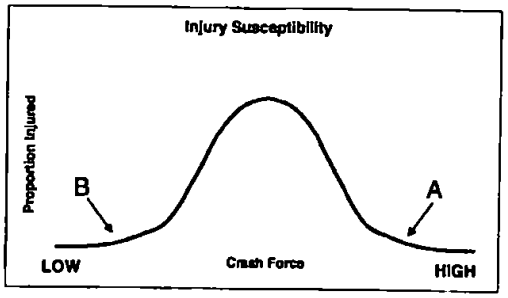

Fig. 3. Theoretical normal distribution of the general population by degree of crash severity required to cause injury.

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language, regardless of the appropriateness of the statistical reference. An extreme example was seen in the Sally Clark case in which probability of innocence was given in very precise terms (1 in 73 million), however, non-specific probabilistic language tends to accomplish the same goal. The claim that an opinion is "highly likely" or "highly unlikely" rather than merely "likely" or "unlikely" is an example weighting an opinion without precision; unless the terms are defined and data are presented to substantiate the claims, such opinion weighting is speculative and potentially harmful. The same is true for the use of "reasonable certainty" versus "reasonable probability"; with no substantiating data the former is no more than an attempt to bolster the persuasiveness of an opinion with misleading language. Caution must be used when assessing for this fallacy; it does not occur when a clinician makes a claim based on clinical experience. For example, the claim that. "it is highly unlikely the surgery will result in a substantial impairment" is a reasonable conclusion for a surgeon to draw regarding one of his own patients, when the claim is based upon his experience performing the surgery and observation the results of the surgery. In contrast, it would not be appropriate for a clinician to claim that her clinical experience allows her to draw the conclusion that certain injuries are highly unlikely to require treatment, because many patients may leave this doctor's care and seek care elsewhere, unbeknownst to her. In the two examples above, the former claim is based upon a reasonable sample of the population to which the claim is to be extrapolated (patients who have surgery). The latter claim is less valid because the patients seen by any one clinician may be non-representative of the general injured patient population (*i.e.* specialist practices), and thus limit the extrapolability of the clinician's experiences.

(c) *Impossibility fallacy.* This occurs when an expert opines that a causal relationship is impossible; the claim implies both 100% clinical observation error rate (as a result of the Prior Odds Fallacy) and a nearly census of population-based data from which to infer the claim of 0 incidence. The fallacy also occurs when an expert claims that a causal relationship is always present, implying zero uncertainty in the opinion. The easiest way to understand this fallacy is to picture a box full of 100 rubber balls that can be either red or black. A statement that there is a red ball in the box (red ball possible) could be verified with a sample of only one ball, if the ball was red. In contrast, a statement that there are no red balls in the box (red ball impossible) would require an examination of every ball in the box. The claim may incorporate other fallacies as well, such as Non-representative sample fallacy, *e.g.* because no disc herniation has ever been observed in volunteer crash testing it is impossible to herniate a disc under similar circumstances.

(d) *Straw man fallacy.* This fallacy stems from the use of unvalidated constructs or inappropriate proxies for causal mechanisms. A good example is seen in the literature regarding whiplash injury; some authors have compared peak accelerations recorded at the head during activities such as sneezing[30] or skipping rope[31] to the accelerations observed in volunteer crash testing. The fallacy occurs when peak head acceleration is used as a proxy for injury risk, so that the improper conclusion is drawn that skipping rope and crash-related trauma have the same injury potential. Because whiplash injury occurrence is associated with numerous variables aside from peak acceleration, such as gender, occupant bracing, vehicle and seat variations, *inter alia*,[28] selecting a variable that is only loosely correlated with injury occurrence as an index of the probability of injury presence lies at the heart of this fallacy.

2.3.3. Base rate fallacy

This fallacy has been described by others as applying to a variety of scenarios but is frequently overlooked in forensic medicine testimony.[32] This fallacy occurs when the base rate of a finding or occurrence in a relevant comparison population is erroneously overlooked while the prevalence of the same finding in the target population is used as evidence in favor of one side or another. An example was presented previously in this paper, in the shaken baby syndrome (SBS) example. While it is important for the fact finder to be made aware that retinal hemorrhage is present in 90% of SBS cases at post-mortem examination, it is equally important to know the prevalence of the same condition in all of the relevant non-violent assault injury mechanisms as well, as discussed earlier in this paper in Section 2.2.3.

3. Conclusions

The 19th century essayist and novelist Charles Dudley Warner (1829–1900) is credited with the quote "Everyone complains about the weather but no one does anything about it". In some ways, the quote is apropos for the widespread but unsystematic use of probability in forensic medicine, in that everyone uses it but not everyone understands it. The purpose of this paper, in which the concept and some of the applications of Forensic Epidemiology have been introduced, is to fill a void that presently exists in forensic medicine with the addition of a general heading under which the proper and improper forensic use of probability is systematically described. As demonstrated by the tragedy of the Sally Clark case, there is little doubt that the use of probability in forensic medicine is in need of

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standardization; there is a high potential for continued harm and injustice if nothing is done in this regard.

Better and more explicit heuristics are needed to describe and implement the concepts introduced in this paper for the wide variety of circumstances encountered in forensic medicine. A few recommendations are as follows:

- Be alert for the language of probability or epidemiology in forensic opinions.
- When epidemiologic data are referenced as a basis for an opinion, evaluate the propriety of their use. Are the sample population and circumstances sufficiently similar to allow for extrapolation to the facts in the present case?
- When in doubt regarding causal determinations, return to the three essential elements of causation: biologic plausibility, temporality, and lack of likely alternative explanation.
- When a clinical outcome is known, be aware of the potential for Prior Odds and other fallacies.
- If a test or criterion is set as an evidentiary standard, determine if the Specificity, Sensitivity, and Positive Predictive Value is known or can be determined for the test or criterion. Use these tools to help determine the real utility of the test or criterion in a forensic setting.

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Appendix S

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IN THE SUPERIOR COURT OF THE STATE OF WASHINGTON

IN AND FOR SNOHOMISH COUNTY

STATE OF WASHINGTON Plaintiff, vs. MICHAEL MORRIS, Defendant.	)	NO. 09-1-01071-9 DEFENSE MEMORANDUM IN SUPPORT OF A NEW TRIAL

NEWLY DISCOVERED EVIDENCE SUPPORTS THAT CONTENTION THAT A NEW TRIAL SHOULD BE GRANTED.

Granting a new trial because of newly discovered evidence is appropriate when:

(1) will probably change the result of the trial;
(2) was discovered since the trial;
(3) could not have been discovered before the trial by the exercise of due diligence;
(4) is material;
(5) and, is not merely cumulative or impeaching.

State v. Savaria, 82 Wn.App. 832 (1996) (overruled on other grounds by State v. C.G., 150 Wn.2d 604 (2003)).

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(425) 339-6300

In the trial of State v. Michael Morris the primary allegation was that Mr. Morris had assaulted his daughter, A.M., via shaking or shaking combined with impact. There was significant medical testimony as to her injuries (specifically subdural hemorrhage and retinal hemorrhage). Most relevant to this motion, there was also significant testimony about the possibility of a "lucid interval." This was relevant to the trial, and the defense, in that Mr. Morris had only been alone with A.M. for approximately 10 minutes at the time she began exhibiting symptoms. The State's expert, Dr. Feldman, testified that lucid intervals are "unlikely" but that they do happen.

The newly discovered evidence in this case is a new, incredibly similar injury to A.M. . Information from A.M.'s current doctor in St. Louis indicates (via attached affidavit) that the current injury is likely to be non-accidental and that non-accidental shaking has been occurring for quite sometime. Mr. Morris has been in custody for nearly seven months. This evidence would lead to a strong inference that someone other than Mr. Morris inflicted the 2009 injury on A.M.

The new evidence could not have been discovered before trial even by due diligence, as the new injury occurred after the trial's completion (although fewer than 30 days after sentencing).

Taking all this information into consideration, the Court should grant a new trial based on newly discovered evidence: (1) The new medical evidence would probably change the result of the trial. (2) The new evidence was discovered since the trial. (3)

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The evidence could not have been discovered before trial by the exercise of due diligence. (4) The evidence is material. (5) The evidence is not merely cumulative or impeaching.

In conclusion, the defendant respectfully requests this Court to exercise its discretion to grant a new trial based on newly discovered evidence.

DATED this _25___ day of July, 2011.

Respectfully submitted,

CASSIE C TRUEBLOOD - WSBA #37829
Attorney for Defendant

SNOHOMISH COUNTY PUBLIC DEFENDERS
1721 HEWITT AVENUE - SUITE 200
EVERETT, WASHINGTON 98201
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Appendix

T

Tonya Davis

From: Goodkin, Jarett [Jarett.Goodkin@co.snohomish.wa.us]
Sent: Tuesday, August 02, 2011 6:34 PM
To: Cassie Trueblood; Tonya Davis
Cc: Scott, Kala; 'Ross Adams'; Paul, Lisa
Subject: State v. Michael Morris 09-1-01071-9

Cassie and Tonya:

Today, I spoke with Dr. Kutz, who is the Child Abuse Specialist at Cardinal Glennon Hospital. Brittany Morris gave him permission to speak with me. There is no mention of old and new findings in the Ophthalmology records at Cardinal Glennon. The hemorrhaging referenced relates to the most recent incident. He does not see anything in the records that is consistent with Dr. Andreone's statement that "shaking has been occurring for quite some time." He has been trying to catch up with her to discuss the situation. He believes that there may have been a word missing that changed the meaning of the sentence. He stated that A.M. had bruising on her forehead and buttocks, and she also has devastating brain injury. The bruising on both the buttocks and forehead are consistent with having occurred during the same time period and either at the same time or within a relatively short period of time prior to the brain injury. Based on his understanding of the sequence of events, everything points to these injuries occurring at the babysitter's home. In addition, he would be hard pressed to find any phenomenon that would lead him to believe that the prior injuries themselves (SDH and RH or even resulting seizure disorder) had any role in explaining the child's current condition. Given the nature of these injuries, he also does not believe "lucid interval" comes into play. In his opinion, the findings (bruising and brain injury) are the result of abusive trauma. The bruising on the forehead suggests impact. Due to the severity of the injury, he would have doubts as to whether the 11 year old could have caused these injuries. The babysitter's mother was supposedly out with her boyfriend when the incident happened. It does not currently appear that Brittany Morris is implicated given that she dropped the child off more than a day and a half before the incident. A.M. is off of life support and currently breathing on her own. However, the only thing she is doing is occasionally opening her eyes. The prognosis remains poor. Apparently, Ms. Morris once again believes that the most recent incident is the result of an accident.

I also spoke with Detective Colvin. He had also just spoken with Dr. Kutz. His investigation is on-going. He hopes to have more information to share with us in the next couple of weeks.

You can reach Dr. Kutz at (314) 268-4146. If you call him, he will talk to the mother about whether she is willing to give her permission to speak with you. If you send him a release from your client, he will check with their legal counsel about whether there is any issue with it given your client's conviction. However, he does not believe at this point that the conviction presents any problems with the release.

You can also contact Detective Colvin at (573) 718-2448. However, he told me that he will give you the same information that he gave me...he is investigating the matter and hopes to have more information in the next few weeks.

If you have any questions, please let me know.

Jarett A. Goodkin
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(425) 388-6360 (fax)
jgoodkin@co.snohomish.wa.us

Tonya Davis

From: Cassie Trueblood
Sent: Monday, August 29, 2011 12:26 PM
To: Tonya Davis
Subject: FW: Michael Morris 09-01071-9

Here's the latest.....

From: Goodkin, Jarett [mailto:Jarett.Goodkin@co.snohomish.wa.us]
Sent: Monday, August 29, 2011 12:27 PM
To: Cassie Trueblood
Subject: Michael Morris 09-01071-9

Cassie:

I think that is the quickest response from a doctor that I have ever received. I just spoke with Dr. Kutz. A.M. was transferred to a rehabilitation facility. He does not know her current condition. He did speak with Dr. Andreone. She had misinterpreted the Ophthalmology findings. The records indicate multiple retinal hemorrhages involving different layers. She originally thought it meant there were old hemorrhages and new hemorrhages. However, the hemorrhages were all acute. There were no old hemorrhages, and there is nothing in the records to suggest the existence of the old hemorrhages, which had previously resolved as shown by the records from St. Louis Children's Hospital that we had in discovery. Based on what he knows, he is still of the opinion that something happened while A.M. was with the babysitter and her mother.

I will let you know when I hear back from Detective Colvin. Unless he has something significantly different, I will be asking for your client to be moved from the jail to DOC. Please let me know if I need to get a date from Ben for a hearing.

Thanks,

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Appendix U

Pediatric Resident Admission Note

Admit Date: 7/17/2011 2:49 AM

Chief Complaint
Subdural Hematoma - transfer from PICU

History of Present Illness
Alexis Morris is a 2 y.o. female with past medical history significant for shaken baby syndrome at 6 weeks of age resulting in a seizure disorder is being transferred from the PICU to General Pediatrics for continued medical management of a likely non-accidental trauma resulting in a subdural hematoma. She initially presented to an OSH following a reported fall from bed while under a babysitters supervision. She was actively seizing, given versed and rapid series intubation. She had a fixed and dilated pupil and was given mannitol. CT showed right subdural hematoma and midline shift and was transferred to Cardinal Glennon.

She had severe cerebral edema, increased ICP, and severe right hemispheric injury. Neurosurgery inserted ICP monitors. She had a decompressive right hemicraniectomy, with evacuation of the subdural hemorrhage and a right subgaleal drain placed.

Alexis was intubated from admission, and was extubated on 7/31. Since extubation, she has been found to have stridor as well as significant retractions with respiration. Scope by ENT found post-cricoid areas and arytenoids erythematous and red. Likely due to irritation of NG tube and reflux. She was on 6 L HFNC, but has been weaned to 2 Liters HFNC at 30% FiO2.

She was found to be positive for MRSA on day 2 of admission. Respiratory cultures of 7/28 and 8/2 were found to be growing enterobacter cloacae. This is thought to be due to aspiration. She is on 5 days of tobramycin nebulized and cefapime.

Ophthalmology was consulted and found multiple retinal and vitreous hemorrhages. Initial skeletal survey did not show fractures. Social services are involved in the care of this patient.

Past Medical History
No birth history on file.
Past Medical History

Diagnosis	Date
• MRSA (methicillin resistant staph aureus) culture positive *trach aspirate; nasal*	7/18/11;7/19/11

No past surgical history on file.

Allergies
No Known Allergies

Family History
No family history on file.

Social History
History

History and Physical (continued)

Social History Narrative

- No narrative on file

Father is in jail out of state for child abuse of this patient

Review of Systems

Review of Systems
Constitutional: Positive for activity change.
Respiratory: Positive for stridor.
Neurological: Positive for seizures.

Exam

Vitals
BP: 93/66 mmHg (08/05/11 1900)
Temp: 99.6 °F (08/05/11 1600)
Pulse: 162 (08/05/11 1900)
Resp: 43 (08/05/11 1900)
SpO2: 100 % (08/05/11 1900)
Weight: 11.9 kg (26 lb 3.8 oz) (08/05/11 0400)
General: Audible stridor, visible retractions, eyes open and deviated to the right
Head: Swollen on right with hemicraniectomy site covered with dressings
Eyes: Pupils reactive, eyes deviated to the right
Cardiovascular: regular rate and rhythm, normal S1 and S2, no murmurs
Chest: Stridor, prominent upper airway noise
Abdomen: soft, non-tender, non-distended and no hepatosplenomegaly or masses
Neuro: Left arm hypertonic, +Babinski on left, questionable Babinski on right

Labs/Objective

Head CT results below.

Active Hospital Problems

Diagnoses	Date Noted
• Retinal hemorrhage, bilateral	08/05/2011

Seen on exam on 7/17 during NAT workup. Optho recommending follow up "at first available appointment".

Follow up Dr. Davitt scheduled for November 2, 2011 at 12:45

- Feeding problem — 08/05/2011

Has not nippled feeds since before TBI 7/17/11. OT working with her on oral stimulation.

Continue OT involvement
Current feeding: Pediasure NG 110mL Q3hrs for goal of 880kcal/day
Nexium 10mg Qday for reflux
Will follow up on OT PO challenge to determine risk for aspiration. Will likely need GI consult for Gtube feedings.

•

History and Physical (continued)

Muscular hypertonicity 08/05/2011
Resulting from TBI.

Transfer to RJ in process
Continue baclofen 2mg BID per neurology

- Infection caused by Enterobacter cloacae 08/05/2011
  Trach aspirate from 7/26, 7/28, 7/31 growing Enterobacter cloacae. S/p 7 days clindamycin

  Continue Cefepime and tobi nebs for 5 more days (to end 8/10)

- Tachypnea 08/01/2011
  Increased work of breathing and stridor since extubation on 7/31. S/p dexamethasone and racemic epinephrine. Stable on HFNC. ENT did bedside scope above the chords and found post-cricoid area and arytenoids and very edematous and red, likely resultant from NG tube and reflux.

  Continue HFNC as needed, wean as tolerated.
  Change to Nexium 10mg per NG daily
  Will have OT work on oral stim/feeding technique to determine earliest opportunity to pull NG tube and do all PO

- Closed head injury with loss of consciousness of unknown duration 07/17/2011
  2WF c h/o shaken baby syndrome with epilepsy secondary to this who fell about 3 feet from a bed today hitting her head. She had a seizure and was taken to OSH where she was intubated and given mannitol 2/2 a fixed and dilated R pupil. Transferred to CGH with CT showing SDH and midline shift.

  CT OSH: unclear image with tilt
  CT CGH (3h later): R convexity acute SDH ~3mm maximal width; partial effacement of R lateral ventricle, 6mm R to L midline shift, R frontoparietal edema, cerebellar tentorium and posterior cerebral falx SDH; basal cisterns not effaced
  Other injuries: bruising on forehead

  Now s/p decompressive craniectomy, hyperventilation, ventricular CSF drainage, Hyperosmolar therapy. Had R subgaleal drain placed 7/18 by neurosurgery.

  Will need prolonged PT/OT/ST at RJ- transfer process currently ongoing
  Protective helmet being arranged
  Per Dr. Kutz' initial consult, consider need for rpt skeletal survey 4 weeks from initial injury (8/17)

- Hematoma, subdural, with loss of consciousness, traumatic 07/17/2011
- Seizures 07/17/2011
  Had h/o seizures from first "shaken baby" injury. Off antiepileptics since Jan. Now with another TBI. Fourteen hours of bedside video EEG monitoring show severe slowing and disorganization of left hemisphere background patterns (apparently with some endogenous state differentiation, and intermittent rhythms that resemble deeper sleep states), along with profound suppression of background activity diffusely over the right hemisphere. No epileptiform features were seen,

however, and multiple typical clinical events were not associated with epileptiform change in the EEG.

Per Neurology, continue keppra

Elisa R Pincus, MD

CC: No primary provider on file.

Phone: None
Fax: None

H&P signed by David A Wathen, DO at 08/06/11 1015

Author:	David A Wathen, DO	Specialty:	Pediatrics	Author Type:	Physician
Filed:	08/06/11 1015	Note Time:	08/06/11 0934		

Pediatric Attending Admission Note

Admit Date: 7/17/2011 2:49 AM

I have interviewed the patient/family and examined this patient. I have reviewed the findings of the resident. My findings (key elements and supplemental information) are as follows:

Chief Complaint
fell from bed

History of Present Illness
Alexis Morris is a 2 y.o. female who was a victim of shaken baby syndrome at 6 wks of age. She again presented on 7/17/11 with reports that she fell from her bed. Trauma surgery and neurosurgery evaluated her and noted that she had severe cerebral edema, increased ICP, and severe right hemispheric injury. Neurosurgery inserted ICP monitors. She had a decompressive right hemicraniectomy, with evacuation of the subdural hemorrhage and a right subgaleal drain placed. She was intubated at admission and admitted to the PICU in critical condition. She was extubated on 7/31 and since has had some significant stridor that was evaluated by ENT. They found significant erythema and edema of the vocal cords thought to be secondary to irritation from ETT, NG tube and possibly reflux. SHe has not been noted to have any seizures since admission but has been maintained on AED's.

Other Pertinent History
Past Medical History

Diagnosis	Date
• MRSA (methicillin resistant staph aureus) culture positive	7/16/11;7/19/11

History and Physical (continued)

trach aspirate; nasal
Shaken baby syndrome with resultant seizure disorder and developmental delay

Exam
BP 89/58 | Pulse 159 | Temp 99 °F | Resp 33 | Wt 11.8 kg (25 lb 9.2 oz)
Gen: Awake and in NAD
Eyes: pupils deviated to right throughout exam with occasional movement to midline, does not focus on objects
Head: dressings on right parietal and frontal head, clean and dry
Neck:supple, no LAD, no masses
Heart: normal S1/S2, no murmur
Lungs: Coarse rhonchi B/L, likely transmitted upper airway sounds, equal air entry, no stridor this am
Abd: Soft, Non-tender, non-distended, Normal bowel sounds, no organomegaly
Ext: Pt not spontaneously moving extremities, PROM of extremities this am with some hypertonicity noted especially at end of ROM

Lab/Other Information
8/5 - BMP normal; CBC reveals decreased WBC from previous day and normocytic anemia

Active Hospital Problems

Diagnoses	Date Noted
• Retinal hemorrhage, bilateral	08/05/2011

Seen on exam on 7/17 during NAT workup. Ophtho recommending follow up at first available appointment.

Plan:
Follow up Dr. Davitt scheduled for November 2, 2011 at 12:45

• Feeding problem 08/05/2011
Has not nippled feeds since before TBI 7/17/11. OT working with her on oral stimulation. Pt currently receiving NG tube feedings of pediasure 110mL Q3hrs with goal of 880kcal/day.

Plan:
Continue OT involvement
Nexium 10mg Qday for reflux
Will follow up on OT PO challenge to determine risk for aspiration. Will likely need GI consult for Gtube feedings.

• Muscular hypertonicity 08/05/2011
Likely resulting from closed head trauma and brain injury. Pt will require intensive therapies including PT and OT to prevent contractures.

Plan:
Pt would likely benefit from inpatient stay at R-J specialty hospital
Continue baclofen 2mg BID per neurology

• Infection caused by Enterobacter cloacae 08/05/2011
Trach aspirate from 7/26, 7/28, 7/31 growing Enterobacter cloacae. S/p 7 days clindamycin and currently receiving cefepime and tobi nebs

Plan:
Continue Cefepime and tobi nebs, to end 8/10

- **Stridor** 08/01/2011

  Increased work of breathing and stridor since extubation on 7/31. S/p dexamethasone and racemic epinephrine. Stable on HFNC. ENT did bedside scope above the chords and found post-cricoid area and arytenoids and very edematous and red, possibly resultant from NG tube and reflux.

  Continue HFNC as needed, wean as tolerated.
  Change to Nexium 10mg per NG daily
  Will have OT work on oral stim/feeding technique to determine earliest opportunity to pull NG tube and do all PO

- **Closed head injury with loss of consciousness of unknown duration** 07/17/2011

  2 yo female with h/o shaken baby syndrome with seizures secondary to this presented with reported fall from bed and resultant subdural hematoma. She had a seizure and was taken to OSH where she was intubated and given mannitol 2/2 a fixed and dilated R pupil. Transferred to CGH with CT showing SDH and midline shift.

  CT OSH: unclear image with tilt
  CT CGH (3h later): R convexity acute SDH ~3mm maximal width; partial effacement of R lateral ventricle, 6mm R to L midline shift, R frontoparietal edema, cerebellar tentorium and posterior cerebral falx SDH; basal cisterns not effaced
  Other injuries: bruising on forehead

  Now s/p decompressive craniectomy, hyperventilation, ventricular CSF drainage, Hyperosmolar therapy. Had R subgaleal drain placed 7/18 by neurosurgery.

  Will need prolonged PT/OT/ST at RJ- transfer process currently ongoing
  Protective helmet being arranged
  Per Dr. Kutz' initial consult, consider need for rpt skeletal survey 4 weeks from initial injury (8/17)

- **Hematoma, subdural, with loss of consciousness, traumatic** 07/17/2011
- **Seizures** 07/17/2011

  Had h/o seizures from first "shaken baby" injury. Off antiepileptics since Jan. Now with another TBI. Fourteen hours of bedside video EEG monitoring show severe slowing and disorganization of left hemisphere background patterns (apparently with some endogenous state differentiation, and intermittent rhythms that resemble deeper sleep states), along with profound suppression of background activity diffusely over the right hemisphere. No epileptiform features were seen, however, and multiple typical clinical events were not associated with epileptiform change in the EEG.

  Plan:
  Continue Keppra at present dosing
  Neurology has evaluated pt

See resident's H&P further details.

David A Wathen, DO
314-294-5623

CC: No primary provider on file.

Phone: None
Fax: None

H&P signed by David A Wathen, DO at 08/07/11 0718

Author:	David A Wathen, DO	Specialty:	Pediatrics	Author Type:	Physician
Filed:	08/07/11 0718	Note Time:	08/06/11 1246		
Related Notes:	Original Note by: Stuart F Cooner filed at 08/06/11 1322				

Pediatric Medical Student Admission Note

Admit Date: 7/17/2011 2:49 AM

Chief Complaint
Traumatic Brain Injury/Subdural Hematoma. PICU Transfer

History of Present Illness
Alexis Morris is a 2 y.o. female who presented to the ED with probable non-accidental trauma resulting in a subdural hematoma. She has a PMHx significant for shaken baby syndrome at 6 weeks of age which resulted in a seizure disorder and her father being incarcerated for child abuse. Her initial presentation to CG was from an OSH with a hx of 'falling off a bed'. When she arrived at CG she was non-responsive, seizing, and had a fixed dilated right pupil. Opthalmology noted bilateral retinal and vitreal hemorrhages. She also had a large bruise on her head. Seizure was controlled with versed and intubation. She was diagnosed with a right subdural hematoma with massive midline shift which completely compressed the right lateral ventricle. Over the course of her stay at CG, Alexis had a ICP monitor placed, a Burr hole drilled, and eventually a hemicraniectomy to relieve the increased ICP. Alexis has been in the PICU since admission from the ED. On 7/31, Alexis was extubated. After this, she was found to have severe inspiratory stridor with some retractions/use of accessory muscles. ENT was consulted and found subcricoid and arytenoid edema/inflammation likely from intubation. She was on 6L 30% FiO2 via HFNC. She has since been gradually weaned to 2L 30%FIO2 although stridor remains prominent. She has also been found to have MRSA and an enterobacter infection, both of which are being treated with antibiotics. ~~Neurologic deficits are obvious.~~

Past Medical History
No birth history on file.
Past Medical History

Diagnosis	Date

History and Physical (continued)

- MRSA (methicillin resistant staph aureus) culture positive 7/18/11;7/18/11
  trach aspirate; nasal

No past surgical history on file.

Immunizations
Immunization status: Unknown.

Allergies
No Known Allergies

Family History
No family history on file.

Social History
History

Social History Narrative
- No narrative on file

Review of Systems
Constitutional: Decreased spontaneous activity
Eyes: Positive for deviation
Respiratory: Positive for retractions and stridor
Cardiovascular: None
Gastrointestinal: Negative for nausea, vomiting, hemetemesis, jaundice
Genitourinary: female
Musculoskeletal: Positive for spasticity, paralysis
Neurological: Positive for seizures, weakness bilateral hand(s), bilateral arm(s), bilateral leg(s), bilateral ankle(s), bilateral foot, bilateral feet,

Exam
Vitals
BP: 108/61 mmHg (08/06/11 1152)
Temp: 98.8 °F (08/06/11 1152)
Pulse: 143 (08/06/11 1152)
Resp: 43 (08/06/11 1152)
SpO2: 100 % (08/06/11 1152)
Weight: 11.6 kg (25 lb 9.2 oz) (08/06/11 0400)
General: ill appearing, lethargic, uncommunicative, in moderate distress
Head: Large right cranial mass at hemicraniectomy site
Eyes: Pupils: are equal, round, and reactive to light
Extraocular Movements: sustained bilateral right gaze deviation
Cardiovascular: regular rate and rhythm, normal S1 and S2, no murmurs
Chest: Inspiratory noises prominent. Stridor and retractions evident at rest. Some wheezes noted
Neuro: Right limbs are weak with decreased spontaneous movement. Left arm and leg are hypertonic, spastic. Positive babinski on left, questionable on right.

Appendix V

Copies To
Juvenile

Poplar Bluff Police Department

330 N Second Street, Poplar Bluff, MO 63901

Supplemental Narrative Report

Report Date	Type of Incident	Complaint No.
07/16/2011 2200	ABUSE OF CHILD (F)	2011-32[redacted]

COPY

Date / Time	Officer ID / Name	Secondary Officer ID / Name	Description
02/23/2012 1440	48 Det. J. Kelsay #48	48 Det. J. Kelsay #48	CASE STATUS

During the course of this investigation, foul play has been suspected however a guilty party could not be found responsible for the injuries to Alexis Morris. I made contact with investigator Nelvia Johnson and was advised she was unable to substantiate this report. Mrs. Johnson stated the family worked with a caseworker, and Alexis is back at home.

This report will be considered closed until further leads arise.

[redacted]

Officer		Approving Officer	
48 Det. J. Kelsay #48	/ /	#39	/ /

Page 1 of 1 Printed 02/23/2012 1536

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Appendix W

REVIEW

Evidence-Based Medicine and Shaken Baby Syndrome

Part I: Literature Review, 1966–1998

Mark Donohoe, MD

(*Am J Forensic Med Pathol* 2003;24: 239–242)

In recent years, there has been a clear move toward basing medical practice and opinions on the best available medical and scientific evidence. This process has been termed *evidence-based medicine* (EBM) and involves a review of the quality of evidence that is available in various diseases and fields of inquiry within medicine.

This is the first of 2 articles that attempts to formally rank the available medical scientific evidence by internationally accepted methods, to determine the degree of confidence that can be held on various claims about the condition termed *shaken baby syndrome* (SBS). Areas with good scientific evidence are identified, and shortcomings in the research and publications on the subject are addressed.

Approximately half of all indexed medical publications on the subjects of SBS and shaken-impact syndrome were published before 1999 and half since that time. Given that 1998/1999 is regarded as the turning point in acceptance of the tenets and practice of EBM, it seemed reasonable to assess the quality of evidence before 1999 and compare it with the quality of evidence on the same subject matter since that time. At the conclusion of Part II, the 2 periods are compared to determine the extent to which EBM has affected the field of SBS in terms of quality of available evidence.

The aim of this review is to be neutral on the subject of SBS. Neutrality is difficult to define in this field, in part because of the polarization of opinions on the highly emotional subject of infant injury and death and in part because of clear data deficiencies arising from difficulties in performing experiments. It is clearly unethical to intentionally shake infants to induce trauma, and there is an obvious problem with studies and reports that rely on either indirect or disputed evidence of the occurrence, severity, or type of trauma.

Many studies lacking these critical data make the obvious logical error of selecting cases by the presence of the very clinical findings and test results they seek to validate as diagnostic. Not surprisingly, such studies tend to find their own case selection criteria pathognomonic of SBS.

Neutrality in this review simply means that there is no selective quotation of the available literature, and literature is not chosen to support any particular view. The assessment is of the methods and quality of the actual research, and until this assessment is complete, the content, findings, and recommendations are irrelevant. At the end of the ranking, those studies that achieve the highest QER scores are reviewed for their content, findings, and recommendations. Their outcomes are collated, and the entire published data set is then reviewed as a whole to determine the summarized recommendations, noting areas of agreement, conflict, or controversy. From this, the problems with the published evidence are noted, and data gaps are identified. Recommendations can then be made according to the summarized data.

In assessment of the quality of the available scientific evidence, the author has taken an approach recently defined worldwide as an appropriate scale for review of quality of evidence. This approach has been described recently in context of setting Australian clinical guidelines.

Genuine hypothesis testing requires use of appropriate research methodologies, including collection of relevant control data, and suitable statistical analysis. The interpretation of individual study findings may be constrained by factors such as whether the cohort examined was adequately representative of the patient population in general. Replication across studies and in independent research centers is a key factor in the reliability of evidence.

Compelling evidence comes from consistent findings in 2 or more well-constructed, controlled trials or population-based epidemiologic studies (i.e., level I or level II evidence). By contrast, clinical practice guidelines with level IV evidence represent consensus statements of the expert panel according to clinical experience and limited scientific data. Although these statements may influence current practice,

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they are likely to be modified in response to further research findings. Data from a single case series without control subjects provide little more than a stimulus for subsequent hypothesis testing.

Quality of Evidence Ratings

I: Consistent evidence obtained from more than 2 independent, randomized, and controlled studies or from 2 independent, population-based epidemiologic studies. Studies included here are characterized by sufficient statistical power, rigorous methodologies, and inclusion of representative patient samples. Meta-analysis of smaller, well-characterized studies may support key findings.

II: Consistent evidence from 2 randomized controlled studies from independent centers, a single multicenter randomized controlled study, or a population-based epidemiologic study. Data included here have sufficient statistical power, rigorous methodologies, and the inclusion of representative patient samples.

III-1: Consistent evidence obtained from 2 or more well-designed and controlled studies performed by a single research group.

III-2: Consistent evidence obtained from more than 1 study but in which such studies have methodologic constraints, such as limited statistical power, or the inclusion of patient samples that may be nonrepresentative.

III-3: Evidence obtained from a single case study or a selected cohort study.

III-4: Conflicting evidence obtained from 2 or more well-designed and controlled studies.

IV: Consensus opinions of authorities according to clinical experience or descriptive reports.

SBS: Literature Review (1966–1998)

Overview and Methods

The entire Biomednet Medline database (http://www.biomednet.com/db/medline) was searched by using the search term *shaken baby syndrome* and Internet Explorer in late November 1998. Other articles identified that had not yet been indexed on MEDLINE but had been published were also included.

The entire set of retrieved articles was reviewed, and those in which SBS was only peripherally mentioned or in which SBS was unrelated to the original article were omitted. Letters and brief correspondence were also discarded, unless they added new information or data on SBS. Articles in non-English journals that lacked an English abstract were also generally excluded from assessment.

These exclusions reduced the initial list of 71 articles to 54, which were reviewed, categorized, and ranked according to the QER above. To these was added the important study by Jayawant et al. from *BMJ* of December 5, 1998. The editorial was omitted because it added nothing to the original article.

It was impossible to review the full original article in many cases, although all of the major articles were reviewed in full. The remainder was assessed for categorization by using the authors' abstracts.

Each article was assigned to 1 of 4 categories: (1) randomized controlled trial; (2) case series with or without controls (with date, series size recorded); (3) single case reports; and (4) other, including review articles, opinion pieces, and articles on social implications.

Results of Quality of Evidence Ratings

Fifty-four articles or abstracts were reviewed. One was a randomized controlled trial.[34] This trial was not relevant to the general topic of SBS because it assessed a diagnostic technique (electroretinograph) that proved unsuccessful in diagnosis. Twenty-six were case series.[1,2,7,9,11,13–15,18,19,26,28–30,33,36,39,42,45–50,52,53] Twenty-five were retrospective studies, and 1 was prospective.

In total, 307 SBS cases were claimed to have been assessed among the 23 articles in which numbers of SBS patients were provided, with a mean study size of 13 cases and a median of 7 cases per series.

Selection criteria for SBS cases were unstated in 12 articles, based on presumption or suspicion in 10, and confirmed in 4 by confession or conviction. Two studies had appropriate control groups, 3 had inappropriate control groups, and 21 were case series without control groups. Twelve studies were case reports:[3–5,8,10,17,22,24,31,32,51,55]

Retinal pathology in suspected SBS, 5 cases

Blunt head injury at autopsy, 1

Subdural hemorrhage (SDH) and retinal hemorrhage (RH) as a result of fall and chest compression, 1 case

Shaking causing traumatic aneurism, 1 case

Arteriovenous malformation, not SBS, as cause, 1 case

Intentional asphyxia and shaking of 15-week-old baby, 1 case

Magnetic resonance imaging value in diagnosis, 1 case

Raised intracranial pressure as cause of RH, 1 case

Fifteen were "other" articles[6,12,16,20,21,23,25,27,37,38,40,41,43,44,54]

Historical reviews of SBS,10 articles

Opinion papers without original material, 3 articles

Social issues and SBS, 1 article

Review of imaging in SBS (computed tomography versus magnetic resonance imaging), 1 article

RESULTS

The randomized controlled trial was unrelated in any aspect of interest in this review and addressed a method of assessment of the retinae that proved to be unsuccessful.

Of the case series, the flaws noted above are relevant. All but 1 was retrospective, and all but 5 had no control population to compare cases with. Three of those that did

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include controls chose insufficient or inappropriate controls (head impact trauma, without healthy controls or other illness unrelated to head injury). This shortcoming would normally have excluded these studies from the literature review (because they do not fulfill criteria for inclusion). Given the difficulties inherent in assessment of SBS and of identifying appropriate control groups, however, these articles have been included as QER III-2.

In studies with confirmed (i.e., admitted or observed) trauma and SBS, there were few common findings, apart from the presence of SDH accompanied by RH in 80% of examined cases. Some articles attempt to measure other risk factor, and the study by Jayawant et al.[42] is noteworthy in this respect.

Finally, the "other" articles do little but summarize opinions and summarize past data. Such articles do not add to the quality of understanding of the condition, nor are they necessarily accurate in what has become a rather emotionally charged area of research and polarized opinion.

In this article, the quality of evidence, rather than the predominance of findings, is being assessed. The issue of the evidence for SBS appears 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 evidence.

Data Gaps Identified

There exist major data gaps in the medical literature about SBS. There is a very obvious lack of clear definition of cases. For valid studies, some method of determining cases of actual proven shaking must be found, and appropriate control groups (trauma without shaking, other illness, healthy controls) must be defined and assessed blindly. This gold standard has yet to be achieved in even a single study in the field of SBS. There is a lack of useful and specific laboratory or other markers proven to identify SBS. There is poor definition and quantification of the social and family risk factors to provide guidance on likelihood of abuse for a given set of circumstances. Last, there is a strong need for a checklist or other diagnostic or management tool to assess cases and to quantify index of suspicion of shaking.

CONCLUSIONS

There was no evidence on the subject of SBS that exceeded QER III-2 by the end of 1998, which means that there was inadequate scientific evidence to come to a firm conclusion on most aspects of causation, diagnosis, treatment, or any other matters pertaining to SBS.

The majority of evidence achieved only a level of QER IV, opinions that shed no new light upon SBS and did not add to knowledge about SBS. Many of the authors repeated the logical flaw that if RH and SDH are nearly always seen in SBS, the presence of RH and SDH "prove" that a baby was shaken intentionally. Many other studies assumed that the presence of RH and SDH was sufficient to make the diagnosis of SBS in terms of case selection.

The remainder of articles are QER III-3, and as noted above, the inclusion of case series without controls would normally not occur. Thus, the data available in the medical literature by the end of 1998 were inadequate to support *any* standard case definitions, or *any* standards for diagnostic assessment.

Before 1999, there existed serious data gaps, flaws of logic, inconsistency of case definition, and a serious lack of tests capable of discriminating NAI cases from natural injuries. By 1999, there was an urgent need for properly controlled, prospective trials into SBS, using a variety of controls. Without published and replicated studies of that type, the commonly held opinion that the finding of SDH and RH in an infant was strong evidence of SBS was unsustainable, at least from the medical literature.

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Appendix X

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ORIGINAL PAPER

Confessed abuse versus witnessed accidents in infants: comparison of clinical, radiological, and ophthalmological data in corroborated cases

Matthieu Vinchon · Sabine de Foort-Dhellemmes · Marie Desurmont · Isabelle Delestret

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Abstract
Background The diagnosis of inflicted head injury (IHI) or accidental trauma (AT) in infants is based on clinical, radiological, and/or ophthalmological findings such as subdural hematoma (SDH), encephalopathy, retinal hemorrhage (RH), and signs of impact. As a consequence, the diagnostic value of these criteria is difficult to assess because of a circularity bias.
Purpose In order to assess the predictive value of these diagnostic criteria avoiding circularity, we studied prospectively corroborated cases of head injuries in infants, comparing IHI with AT.
Material and methods We selected 45 cases of confessed IHI and 39 cases of AT having occurred in public places. Patients were systematically evaluated clinically, with computed tomography scanner and ophthalmological evaluation by a trained ophthalmologist. RH was rated as absent, mild, and severe, according to the depth and extent of the RH.
Results Brain ischemia was found in 26.7% of IHI; the most prominent elements in favor of IHI were SDH, severe RH, and absence of signs of impact; the predictive diagnostic values of these three features were 0.685, 0.961, and 0.830, respectively; however, only severe RH in the absence of ocular impact was specific of IHI. When all three features were combined, the specificity was 100%, but the sensitivity was only 24.4%.
Conclusion Our study confirms the high diagnostic value of RH, SDH, and signs of impact for the differential diagnosis between AT and IHI. The evaluation of head injuries in infants requires a high level of awareness and thorough and systematic examination by a trained multidisciplinary team.

Keywords Subdural hematoma · Infant · Inflicted head injury · Shaken baby syndrome · Accidental trauma

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Introduction

The differential diagnosis between inflicted head injury (IHI) and accidental trauma (AT) in infants is of major legal importance and should be based on medical evidence. However, this evidence is difficult to gather: In IHI, the principal witness is generally the perpetrator and the history of trauma is generally absent or insincere, and AT mostly occurs at home in the absence of independent witness. Several series have dealt with corroborated AT [25, 35, 40] or IHI [1, 30], but the clinical and radiological data were not precisely described, and comparison between AT and IHI is lacking in the medical literature.

The hallmarks of shaken baby syndrome (SBS) are subdural hematomas (SDH), encephalopathy, and retinal hemorrhage (RH) [10, 20]. Since in most series the diagnosis of IHI is based on the presence of these features, evaluation of the diagnostic value of these criteria is flawed

by a bias of circularity [3, 8, 20, 29, 33, 39]. Other biases relate to the age range of the patients: In series mixing infants with older children, IHI and SDH were more common in infants while AT and impact-related lesions were prevalent in juvenile [7, 27]. In previous studies limited to head injuries in infants, we found that the pathophysiology of the SDH was age specific rather than cause specific [35, 38]. As a result, the real diagnostic value of SDH for the diagnosis of SBS is debated. Evaluation of the diagnostic value of RH is also problematic; RH have been reported in rare cases of AT; however, fundoscopy is performed mainly in cases of suspected abuse [2, 8, 15]. In addition, many studies give scanty details regarding RH; however, the latter one is of paramount importance for the diagnosis of IHI [20].

In order to provide evidence-based characterization of clinical, ophthalmological, and radiological lesions found in IHI and AT, we studied cases with undisputed AT or IHI from our prospective database of head injuries in infants. Feldman et al. earlier proposed to classify infantile head traumas along a spectrum, from certain abuse (confessed) to certain accident (witnessed), through highly probable abuse, probable abuse, undetermined, probable accident, and highly probable accident [8]. We selected both extremes of this spectrum in order to compare the clinical, ophthalmological, and radiological features of IHI and AT and their outcome. The purposes of our study were to provide reliable elements for the differential diagnosis between AT and IHI and to test the diagnostic value of these features.

Materials and methods

We started in 2001 a prospective register collecting medical information on all traumatic head injuries in infants. All children under the age of 24 months referred alive to the emergency room, the pediatric intensive care unit (ICU), or our neurosurgical department were included. Our institution is the sole facility for the management of pediatric neurosurgical cases for a four million population area. The methodology and the first 150 cases of this series were published earlier [38]. We selected cases of corroborated IHI and AT; cases of obstetrical trauma were excluded. Corroborated AT was defined as an accident having occurred in a public space in front of independent witnesses. Corroborated IHI was defined as abuse confessed by the perpetrator; information on the confession was obtained by a forensic pediatrician (MD) from judicial sources during expertise or after the judicial hearings were made public.

Information was gathered regarding the mechanism of trauma, and the presence of risk factors such as perinatal illness (diseases affecting the prenatal period, traumatic or premature birth, neonatal diseases requiring hospitalization), presence of a step parent, and familial dysfunction (history of violence, substance abuse, neglect, and/or psychiatric disorder). The delay from trauma to admission, first computed tomography (CT) scan, and occurrence of intracranial hypertension was calculated based on the time of trauma. The timing of AT is generally precise and unequivocal. Regarding IHI, precise timing is considered less reliable; because several reports have established that symptoms of IHI generally occur immediately after assault [1, 30, 31], we used the time of clinical onset of symptoms reported on admission as an approximation for the time of abuse.

Clinical findings, including neurological symptoms, signs of impact, peripheral lesions, and life-threatening events were collected. All patients had at least one head CT scan. Radiological data included the delay of the CT relative to clinical onset, the presence of skull fracture, extradural, meningeal bleeding, the presence of hyperdense and/or hypodense lesions, ischemic lesions, and the maximal thickness of pericerebral collections on axial view. For statistical analysis, scalp swelling, skull fracture, extradural hematoma, and focal brain contusion were pooled together and labeled as "radiological signs of impact".

Fundoscopy was performed systematically by an ophthalmologist for all cases of trauma, and in case of RH, repeated controls and seriated pictures were made by a trained neuro-ophthalmologist. The RH were rated as reported earlier by Vinchon [37] and illustrated in Fig. 1. RH were either absent, mild (preretinal: superficial, splinter, or flame-shaped; or intraretinal, dot, or blot-shaped), moderate (preretinal, pearl-shaped with a diameter less than twice the papilla), or severe (large preretinal, associated with other types, diffuse with or without retinoschisis).

The evaluation of cases suspect of IHI also included a complete clinical examination by a pediatrician (ID), extensive biological evaluation to rule out clotting deficiency, and a skeletal survey using X-rays and/or isotopic bone scan. Whenever the possibility of child abuse or neglect arose, the multidisciplinary team (neurosurgeon, pediatrician, forensic doctor, psychologist, and social worker) met and discussed whether the case should be referred to justice, to social network follow-up, or not be regarded as suspicious. Following the French legislation, cases reported to the judiciary were then investigated by the city attorney with hearing of all adults involved in the child's care at the time of trauma.

The clinical outcome was evaluated using the Glasgow Outcome Scale (GOS), with 1—meaning normal life, 2—mild sequels, 3—severe sequels, 4—vegetative state, and 5—dead. Whenever a child with SDH presented with

Fig. 1 Grading of RH in infants. RH were either absent (not illustrated), mild (preretinal: superficial, splinter, or flame-shaped; or intraretinal, dot or blot-shaped; a) moderate (preretinal, pearl-shaped with a diameter less than twice the papilla; b), or severe (large preretinal, associated with other types, diffuse with or without retinoschisis; c)

intracranial hypertension not relieved by one or more subdural puncture, a subduroperitoneal shunt was inserted following a protocol described previously [34]. We calculated the delay to onset of intracranial hypertension as the time between trauma and either the first subdural puncture or subdural drainage.

Statistics

Continuous variables were tested using Student's *t* test; binary variables were tested using the chi-square test. For the variables relevant to the differential diagnosis between AT and IHI, the positive and negative predictive values were calculated and tested using the chi-square test for significance.

Results

From May 2001 to February 2009, we collected 412 cases of head injuries caused by AT or IHI in infants; 124 (30.4%) were considered IHI, and 288 (69.8%) were considered AT. Among these 412, 39 AT (13.5) and 45 IHI (36.3%) were corroborated. These 84 patients constitute the clinical material of the present study.

The mean age was significantly younger in the IHI group. A male predominance was found in both groups: The male/female ratio was 1.81 in the IHI group, compared with 1.44 in the AT group; the difference was not significant. Perinatal illness was more common in IHI, although not significantly; family dysfunction was significantly more prevalent in IHI. The mechanism of trauma in the IHI group was SBS in 30 cases and beaten baby syndrome in 15. The AT group breaks down as follows: passenger in a car (19), defenestration (five), baby in a carriage hit by a vehicle (three), fall from arms (three), from a supermarket trolley (two), on a sidewalk (two), from a hospital bed (two), from a seat (one), and with a walker in stairs (one). The delay from trauma to admission was shorter in the AT group but without reaching statistical significance. The delay between trauma and initial CT scanner, however, was significantly shorter in the AT group. Patients with IHI presented significantly more often with seizures, coma, raised intracranial pressure, and neurological deficit. Scalp swelling was found in a minority of children with IHI; a notable exception was fatal IHI, seven of the ten children who died of IHI having scalp swelling, and four of these having skull fracture; the fifth case of IHI with skull fracture survived with major sequelae. AT was associated in most cases with swelling of the scalp. Systemic failure considered life threatening was also more common in AT, without reaching statistical significance, while peripheral fractures were significantly more common in IHI. The constitution of the series, medical past history, clinical presentation, and delay to presentation and to first CT scanner are summarized in Table 1.

SDH was the main diagnosis in the majority of children with IHI but was also found in a large minority of AT. By contrast, impact-related lesions (linear fractures, depressed fractures, extradural hematomas, and focal brain contusion) were found almost exclusively in AT; the difference was highly statistically significant. All cases of trivial fall (from arms or from hospital beds) resulted in minor lesions (skull fractures in two, mild subarachnoid hemorrhage in three), with spontaneous and quick recovery. The main diagnoses in AT and IHI are compared in Fig. 2. SDH was present on CT scanner in a majority of patients but significantly more often in IHI. The density of the lesions was mixed (hypo- and hyperdense) in the majority of patients in both groups, significantly more often in IHI. Brain edema/ischemic brain damage was almost specific of IHI. Fundoscopic data were available in 44 cases of IHI (97.8%) and 35 cases of AT (89.8%), respectively. While severe RH were found in the

Table 1 Clinical presentation in the IHI and AT groups

	Inflicted	Accident	*p*
N	45	39	
M/F	29/16 (1.81)	23/16 (1.44)	0.24
Perinatal illness	22 (48.9%)	11 (28.2%)	0.055
Socio-psy	20 (44.4%)	2 (5.1%)	<0.001
Stepparent	5	1	NS
Age (months)			
Mean	3.8	8.1	<0.001
Median	3.2	5.5	
Extremes	0.8–18.3	0–23.9	
Delay to referral (hours)			
Mean	57.0	21.2	0.09
Median	12.0	3.5	
Extremes	0–646	0–396	
Delay to CT (days)			
Mean	2.3	0.5	0.026
Median	0.8	0.1	
Extremes	0–27	0–3.7	
Clinical features			
Seizures	31 (68.9%)	5 (12.8%)	<0.001
Somnolence	18 (60.0%)	12 (30.8%)	0.38
Coma	20 (44.4%)	9 (23.1%)	0.04
Deficit	20 (44.4%)	8 (20.5%)	0.02
Raised intracranial pressure	30 (66.7%)	9 (23.1%)	<0.001
Swelling	1 (2.2%)	30 (76.9%)	<0.001
Impact on head	17 (37.8%)	34 (87.2%)	<0.001
Other impact	11 (24.4%)	6 (15.4%)	0.30
Peripheral fracture	12 (26.7%)	2 (5.1%)	0.008
Transfusion	10 (22.2%)	10 (25.6%)	0.71
Life threat	3 (6.7%)	8 (20.5%)	0.060

majority of cases of IHI (56.8%), RH were found in only six cases of AT: mild, flame-shaped in five, and severe in only one patient, who had facial trauma suggesting impact on the globe. Overall, severe RH in the absence of direct impact appeared specific of IHI. The proportions of the different grades of RH in AT and in IHI are compared in Fig. 3. Radiological and ophthalmological features are summarized in Table 2.

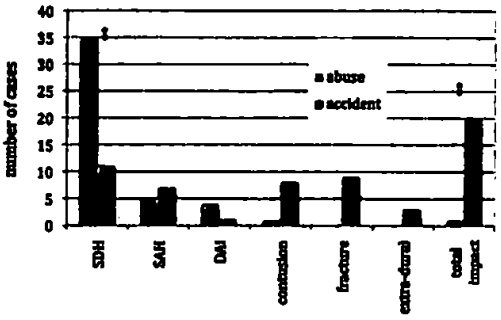

Fig. 2 Main diagnosis in the AT and IHI groups. The "total impact" subgroup represents the sum of cases with contusion, fracture, or extradural hematomas. $^{**}p<0.01$

Thirty-two patients developed a SDH requiring drainage, significantly more often in IHI than in AT; the delay between trauma and intracranial hypertension was longer in AT, but the difference was not significant. The mean duration of assisted ventilation and overall stay in ICU was longer in IHI; the overall hospital stay was also significantly longer in IHI, which was caused at least in part by the legal procedure. Twelve children died as a consequence of the trauma, significantly more in IHI (ten cases, 22.2%) than in AT (two cases, 5.1%). Eleven of the 12 children who eventually died were comatose on presentation. Both fatal AT were high-velocity traffic accidents with systemic failure. At last control, the number of patients with normal overall status (GOS 1) was significantly higher in AT (Fig. 4). Regarding neurological sequels, only epilepsy was significantly more common in

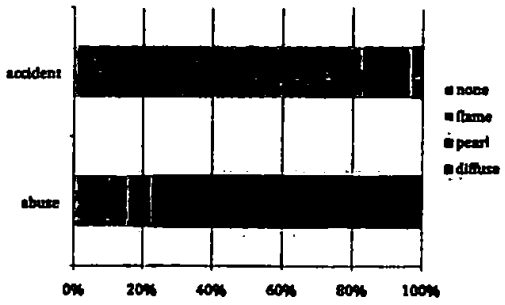

Fig. 3 Retinal hemorrhage in the AT and IHI groups. Although most cases of abuse were associated with severe hemorrhage, seven had no hemorrhage, and three had only mild hemorrhages. Conversely, mild RH were found in five cases of AT, and one had severe hemorrhage caused by direct facial impact. We conclude that severe retinal hemorrhage is pathognomonic of IHI but admits a number of false negatives

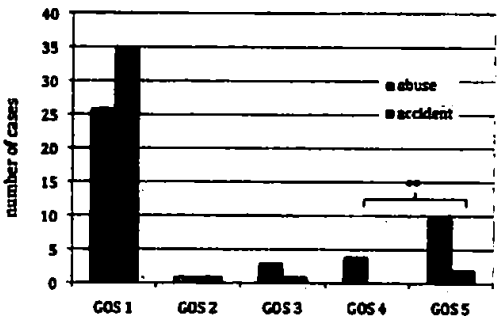

Fig. 4 Clinical outcome in the AT and IHI groups, according to the Glasgow Outcome Score (*1* normal life, *5* dead). The *number* of cases with severe morbidity (*GOS 4*) or mortality was very significantly higher in the IHI group (**p=0.002)

IHI. However, the mean age at last control was less than 2 years in 55 patients, and we assume that with a longer follow-up, the differences between the two groups will become more pronounced. Data on immediate and subsequent outcome, duration of hospital stay, occurrence of raised ICP, and requirement for subduroperitoneal drainage are summarized in Table 3.

The sensitivity, specificity, positive predictive value, and negative predictive value of brain ischemia, SDH, severe retinal hemorrhage, and signs of impact, relative to the diagnosis of IHI, are summarized in Table 4. Comparison of the corroborated series with uncorroborated cases shows that fatal IHI was probably overrepresented in the corroborated group (ten of 45 versus three of 77 in the uncorroborated group, NS). The majority of cases of corroborated AT were traffic accidents, generally more violent than trivial household accidents; however, three cases of uncorroborated trauma were fatal (two falls with walker, one fall in the stairs without walker).

Table 2 Radiological and ophthalmological findings in the IHI and AT groups

	Inflicted	Accident	p
CT findings			
Subdural collection	37 (82.2%)	17 (43.6%)	<0.001
Mixed density image	39 (90.7%)	17 (53.1%)	<0.001
Brain ischemia	12 (26.7%)	1 (3.2%)	0.0023
Extradural	1 (2.4%)	5 (13.9%)	NS
Contusion	7 (15.6%)	10 (25.6%)	0.25
Fracture	5 (11.4%)	26 (66.7%)	<0.001
Total impact	10 (22.2%)	26 (66.7%)	<0.001
Retinal hemorrhage			
None	7 (15.9%)	29 (82.9%)	<0.001
Mild	3 (6.8%)	5 (14.3%)[a]	NS
Moderate	9 (20.5%)	0	NS
Severe	25 (56.8%)	1 (impact)	<0.001

[a] Percents represent the ratio of the number of features on the total number of available data; data may be missing, producing different percents for the same absolute value

Table 3 Early and later evolution in the IHI and AT groups

	Inflicted	Accident	p
Delay to raised ICP (days)			
Mean	4.9	7.0	0.441
Median	1.6	4.5	
Subdural drainage	25	7	<0.001
Mean duration (days)			
Ventilated	3.3	1.4	0.056
In ICU	4.3	1.9	0.042
In hospital	16.1	9.0	0.0002
Last control			
Follow-up (months)	17.3	19.0	0.76
Age (months)	20.9	27.1	0.23
Developmental delay	8 (23.5%)	2 (5.4%)	NS
Motor delay	9 (26.5%)	4 (10.8%)	0.218
Deficit	7 (20.6%)	2 (5.4%)	NS
Epilepsy	10 (29.4%)	1 (2.7%)	0.008
Behavior	5 (14.7%)	1 (2.7%)	NS
Vision	9 (26.5%)	3 (8.1%)	0.108
GOS 1	26 (57.8%)	35 (89.7%)	0.001
GOS 2	1	1	NS
GOS 3	3	1	NS
GOS 4	4	0	NS
GOS 5	10 (22.2%)	2 (5.1%)	0.026

Table 4 Diagnostic value of brain ischemia, subdural hematoma, retinal hemorrhage, and absence of signs of impact, for the diagnosis of child abuse

	Sensitivity	Specificity	Positive predictive value	Negative predictive value	Chi-square (p)
Brain ischemia	0.267	0.971	0.921	0.505	<0.01
SDH	0.822	0.552	0.685	0.724	<0.001
Severe RH	0.556	0.974	0.961	0.655	<0.001
Absence of scalp swelling	0.978	0.769	0.830	0.968	<0.001
SDH and diffuse RH and no scalp swelling	0.244	1	1	0.534	<0.001

Note that the absence of signs of impact has roughly the same diagnostic value for IHI as has severe retinal hemorrhage. These figures show that the sensitivity, specificity, and positive and negative predictive values of these three features are uneven but that the specificity and positive predictive value of the three combined was 100% (at the cost of a loss of sensitivity)

Discussion

The diagnosis of IHI is based on clinical, radiological, and ophthalmological criteria. The differential diagnosis between AT and IHI is a frequent dilemma in medical and legal practice: "the dire consequences of either false-positive or false-negative diagnosis intensify the need to establish accurate diagnostic criteria" [27]. The aim of our study was to evaluate the diagnostic value of these criteria. It was therefore necessary to dispose of another diagnostic criterion as a gold standard in order to avoid circularity; we chose to select cases of witnessed AT and confessed IHI.

Reliability of data

Our study was intended to select data as reliable as possible. We considered that accidents occurring in public spaces or confession of abuse by the perpetrator give the best possible assurances for the diagnosis of AT and IHI, respectively.

Details of abuse as reported by abusers may be unreliable, however, because they seek to underestimate the violence of abuse and the repetition of trauma and often lack precision in timing [19, 29]. Regarding the notion of abuse itself, we think the information on confessions obtained from the judiciary can be trusted. In IHI, the absence of immediate, spontaneous confession, the longer delay to referral, and the context of guilt confirm that shaking was abusive, not playful. We have limited data regarding details of the confession; however, the dating generally appeared consistent with the time of clinical onset as initially reported when the child was admitted. We have few data on the perpetrator and the mechanism of abuse, as detailed in other studies [1, 30, 31]; instead, our focus was on the clinical, radiological, and ophthalmological data in IHI versus AT.

Accidental traumas occurring in private spaces cannot escape a measure of doubt over possible concealed abuse [40]. In a nonverbal child, everything depends on the value of the witnesses, who should be independent and reliable: In the cases reported by Gardner and by Piatt, the witnesses (the 5-year-old brother of the victim and the parents of the victim, respectively) cannot be regarded as such [9, 23]. On the contrary, traumas occurring in public spaces are generally seen by independent witnesses; traffic accidents are investigated by the police and insurance and considered to be definitely accidental [27]. Exceptional trivial AT occurring in hospital (fall from a bed) also belong to the corroborated category [22], as do exceptional cases caught on video [25].

Analyzing data in categories defined by these same data represents a bias of circularity. In many series studied by radiologists, ophthalmologists, pathologists, and examiners, the criterion for IHI was that the cases had been declared abusive by a team of clinicians [10, 12, 20, 33, 39]. These authors ignored the fact that the IHI team had based their diagnosis on the same data, which are the subject of their study. In studies conducted by clinicians, the criterion for IHI is generally that the lesions are considered not explained by the mechanism of the alleged trauma [3, 5, 8, 16, 38]. The basis on which these lesions are considered incompatible with the trauma may be subjective, and these studies are thus not immune of circularity either [29]. In some studies, a laudable effort was made to avoid the circularity bias, by artificially setting aside RH from the criteria of IHI [8, 24]. This method appears unrealistic in retrospective studies and could be dangerous in prospective studies by underestimating IHI. How can the circularity ring be broken? We think that our study, with prospective inclusion of all cases of head injuries, systematic ophthalmological evaluation, and selection of cases based on nonmedical data, allows the evaluation of the medical data in IHI and AT with reasonable certainty, even though the selection of corroborated cases introduces other biases.

Selection bias derives from the pattern of accrual of basically any study. In autopsy series, the trauma is a priori more violent than in survivors, and more severe lesions should be expected. The high prevalence of impact lesions

in SBS (the shaken impact theory) [4] or of brain ischemia [10] could thus be an overestimation due to a selection bias in autopsy series. Our study does not escape a selection bias, as attests the higher mortality rate in corroborated compared to noncorroborated IHI. Also, fatal AT in corroborated and noncorroborated cases resulted from different mechanisms, with different lesion patterns. The higher mortality and morbidity in IHI, compared to AT, can likewise be artificially raised by a selection bias. In the literature, the mortality rate in IHI varied between 27% and 61% according to series [18], which suggests that the threshold for the diagnosis of IHI was highly variable. What raises concern, however, is that if a trauma is all the more suspect of abuse that its consequences are dire, conversely, mild cases of IHI are likely underdiagnosed. Cases of apparently isolated SDH have been eventually diagnosed as IHI after a thorough investigation [21]. Undiagnosed IHI will possibly recur, and several studies have shown that recurrent IHI bears a poor prognosis [13, 14, 34]. This emphasizes the need for a high level of awareness for IHI in all cases of head injuries in infant and for an adequate and thorough evaluation.

The age of included patients introduces another bias because the lesion pattern depends on the age of the patients as well as on the mechanism. In series including children of all ages, Ewing–Cobbs noted that the age was different in the AT and IHI groups [6], which muddles the evaluation of the different traumatic patterns in AT and IHI. Although the present study included only infants (less than 24 months), the mean age was still significantly lower in IHI, which may account partly for a higher proportion of SDH in this group. However, the median age was only 5.5 months in the AT group, which corresponds to the peak prevalence of SDH [34]. Our opinion is that the lesser prevalence of SDH in the AT group relates to the higher proportion of patients with purely extradural, impact-related lesions, rather than to the age bias.

Differential diagnosis between AT and IHI

The diagnosis of SBS is classically based on the triad—SDH, RH, and encephalopathy [11, 22, 29]—while other authors refer to another triad: SDH, RH, and absence of signs of impact [26]. Overall, our study confirms the diagnostic value of all of these latter features for the diagnosis of IHI.

In our study, the clinical manifestations of encephalopathy were often minimal, and brain ischemia was detected on CT scanner in only 26.7% of patients with IHI. Magnetic resonance imaging has a much higher yield than CT for brain lesions but has some disadvantages (impractical in young children, low sensitivity for blood in early studies), and we do not use it routinely for mild cases [36]. CT scanner is the most commonly used diagnostic tool for head injuries in infants [33]. We think that encephalopathy is more a marker of severity than a mainstay of the diagnosis of IHI.

Impact lesions are prominent in AT, in particular traffic accidents, characterized by extensive lesions of the skull and its covering, which may be life threatening on account of blood loss and coagulopathy [32]. In case of SDH, the presence of signs of impact are considered important diagnostic features in favor of AT [28, 35]. Since a negative finding is of no value unless the absent feature is systematically and prospectively recorded, we looked systematically for clinical and/or radiological signs of impact in all cases of infantile head trauma. In a previous study, we found signs of impact in a minority of cases of IHI [38]. It appears from the present study that this negative finding was a very significant element of the differential diagnosis between AT and IHI, with the same diagnostic value as severe RH; when combined with SDH and severe RH, the predictive value regarding IHI was 100%, confirming the value of the "Ontario triad" [26]. These finding are in apparent contradiction with the "shaken impact" theory, which states that shaking alone does not provide the required energy to produce diffuse brain damage [4]. As discussed above, we think that a selection bias may explain the higher incidence of impact lesions in fatal IHI [4, 10]. Reciprocally, IHI has been under diagnosed in the past, and many minor cases (without impact) may have been overlooked [14]. The prevalence of signs of impact in IHI thus varies between 100% and 33% according to the series [16, 18, 26], reflecting the investigators' personal opinion, the intensity of the pursuit of evidence of impact, and the zeal for declaring cases to the judiciary. In our series, we found that seven of ten cases of fatal IHI had signs of impact, and four had a skull fracture. We conclude that shaken impact baby syndrome represents a subgroup of IHI with a worse prognosis but that SBS without impact can also be fatal, as shown earlier [1, 18]. Whatsoever, we do not think that the absence of signs of impact lessens in any measure the perpetrator's responsibility in the child's illness.

SDH is a cardinal feature in IHI, occurring in the majority of cases [10, 16, 21]. However, subdural collections are also found in infants after AT [35], meningitis, craniotomy, and overdrainage by shunts. Their pathogenesis should be viewed as a common final pathway of many different processes affecting the meninges at an age of special hydrodynamic fragility. The lower prevalence of SDH in AT likely reflects the higher rate of impact-related, purely extradural lesions in this group. SDH alone thus cannot be considered indicative of IHI, but we found that its association with severe RH and absence of signs of impact is virtual certainty of abuse. Classically, SDH in

SBS are mixed-density collections, which has long been considered a sign of rebleeding indicating repeated assault [6]; however, we found that mixed density SDH are also found in AT [35], mixed density indicating sedimentation rather than rebleeding [33]. Our present study confirms these earlier findings.

The high value of RH for the diagnosis of IHI is well established [20, 24]; in corroborated series, RH were noted in 75–100% of cases [18]. In our study, 97.8% of cases of IHI and 89.9% of cases of AT underwent fundoscopy, and RH were noted in 84.1% and 17.1% of these, respectively. The main problems with RH are thus false positives (RH in AT) and false negatives (absence of RH in IHI). In a series of corroborated IHI, Starling et al. found no RH in 18% of SBS [31]. As said above, the value of negative findings depends on how intensely they are looked for. Serial imaging of the retina shows that severe RH can resolve over a period of a week (unpublished data); it may also be reminded that RH is not a feature of the beaten baby syndrome.

RH have also been reported in corroborated AT, like traffic accidents, defenestration, or staircase accident [2, 5, 15, 35]. For these reasons, some authors have stated that the specificity of RH for IHI is "not supported by scientific evidence" [17]. Plunkett also reported RH in four of the six fatal AT who underwent fundoscopy, suggesting that the severity of trauma plays a role [25]; however, it is generally the absence of RH in major AT like traffic accidents that struck the authors.

In previous studies, we found that RH associated with AT were mild, while RH caused by IHI were severe [35, 38]. Other authors have shown that in IHI, the severity of RH was also correlated with the severity of brain damage [20]. In order to enhance the diagnostic value of RH and minimize false positives, it appeared important to establish a grading. We have proposed since 2004 a four-tier grading based on easily indentified features [37, 38]; however, it was difficult to validate the diagnostic value of this grading because of the circularity bias. The present study, basing the diagnosis of IHI on confession rather than medical features, eliminates this bias and confirms the very high predictive value of severe RH. Severe RH were found in the majority of cases of IHI (56.8%); only one case of AT had severe RH, was likely caused by craniofacial trauma, as published before [17]. We conclude that severe RH in the absence of facial trauma are specific of IHI.

Conclusions

The paucity of data gained from animal experiments is still a major handicap to our understanding of the natural history of traumatic intracranial lesions in infants. Advances rely heavily on clinical data as a major source of information. However interpreting these data is delicate, especially in traumas which are either concealed or occur in private spaces. In order to be exploitable, studies must be calibrated, selective, and prospective. We think data gathered from corroborated cases of IHI and AT avoid the circularity bias and allow comparisons. Our study confirms the diagnostic value of the SDH, severe RH, and absence of signs of impact for the differential diagnosis between IHI and AT. The importance of the differential diagnosis between IHI and AT requires systematic evaluation by a trained multidisciplinary team.

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Appendix

Y

A Systematic Review of the Diagnostic Accuracy of Ocular Signs in Pediatric Abusive Head Trauma

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Topic: To review systematically the diagnostic accuracy of various ocular signs for pediatric abusive head trauma (AHT).

Clinical Relevance: Intraocular hemorrhages (IOH), perimacular retinal folds, traumatic retinoschisis and optic nerve sheath hemorrhages have been reported as cardinal signs of AHT. The evidence base supporting the accuracy of this interpretation, however, has not been systematically reviewed.

Methods: A systematic keyword search of MEDLINE, EMBASE, and Evidence-Based Medicine Reviews was conducted for original studies reporting ocular findings in AHT. Articles were graded using a checklist for systematic reviews of diagnostic accuracy.

Results: The initial search yielded 971 articles, of which 55 relevant studies were graded, and 20 studies met inclusion criteria and were included in the review. The overall sensitivity of IOH for AHT was 75% and their specificity was 94%. Intraretinal hemorrhage at the posterior pole was the most common finding, although extensive, bilateral, and multilayered IOH were the most specific for AHT. Optic nerve sheath hemorrhages had a sensitivity and specificity for AHT of 72% and 71%, respectively. Traumatic retinoschisis and perimacular retinal folds were reported in 8% and 14% of AHT, respectively, but were not reported in other conditions.

Conclusions: Prospective, consecutive studies confirm that IOH in infants—particularly bilateral, extensive, and multilayered—are highly specific for AHT. Optic nerve sheath hemorrhages are significantly more common in AHT than in other conditions, in autopsy studies. Traumatic retinoschisis and perimacular folds are present in a minority of AHT, but rarely seen in other conditions.

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Pediatric abusive head trauma (AHT) is a significant cause of morbidity and mortality, with an estimated incidence of 29.7 (95% confidence interval, 22.9–36.7) per 100 000, in infants <1 year old.[1] The spectrum of injury and clinical presentation is broad, and less severe cases are often difficult to diagnose due to unreliability of the history, nonspecific symptoms, and lack of external signs of injury.

The mechanism of injury in AHT is thought to be severe rotational acceleration secondary to violent shaking, direct impact, or a combination of the two. Since the first observation of retinal trauma in an abused infant 45 years ago,[2] intraocular hemorrhages (IOH) have been considered a hallmark of AHT. It has also been thought that perimacular retinal folds, traumatic retinoschisis, and multilayered IOH extending to the retinal periphery were highly correlated with repeated acceleration–deceleration injury.[3–7]

The validity of the diagnostic criteria for AHT and the specificity of any retinal findings for abuse have been called into question. An evidence-based appraisal of the literature up to 1998 reported inadequate scientific evidence to validate most aspects of AHT.[8] Some criticisms of the evidence base were the frequency of circular logic, lack of control groups, inconsistency in definition of cases, and a lack of prospective studies. Although these criticisms may have been valid for the period studied, this article was limited in its scope and did not specifically address retinal findings. Immense interest in this area has subsequently led to a number of detailed prospective studies being conducted.

A recent review[9] reported evidence from prospective studies, supporting the diagnosis of abuse in infants with head injury and IOH, and a correlation between severity of intraocular and intracranial pathology. This review, however, did not mention how articles were considered as being relevant and did not employ a systematic approach to filtering studies or assessing the risk of bias.

The primary objective of this article is to review, systematically, the accuracy of IOH, perimacular retinal folds, traumatic retinoschisis and optic nerve sheath hemorrhages, in diagnosing AHT in infants who do not have a credible history of accident or a medical condition.

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A summary of the findings from relevant studies will be presented, in an attempt to make evidence-based clinical recommendations pertaining to the sensitivity and specificity of these clinical signs for AHT.

Sources and Methods of Literature Search

Inclusion Criteria

Types of Studies. Studies were included that allowed conclusions to be made regarding sensitivity or specificity of ocular findings for AHT. Sensitivity can be calculated from the frequency of ocular findings in a consecutive series of patients with AHT. Specificity can be calculated from the frequency of ocular findings in consecutively recruited control groups. Thus, the types of studies eligible for inclusion were case-control studies, cohort studies, or consecutive case series.

Population. Eligible studies included infants or children with AHT. Although there is no universally accepted reference standard for the diagnosis of AHT, cases that are accompanied by confessions, witnessed by reliable third parties, or confirmed in legal proceedings are commonly accepted as being valid cases. In addition, we accepted as valid, cases that were confirmed by multidisciplinary assessment using standard criteria.

Details. All articles had to include, at a minimum, the frequency of ocular findings in a consecutive series of AHT.

Exclusion Criteria

Studies were excluded if they did not include infants or children with AHT. With respect to study design, case reports and nonconsecutive case series were not included in the analysis, because the frequency and diagnostic accuracy of findings cannot be determined. Case reports of particular significance are, however, mentioned in the relevant sections.

Search Strategy

A comprehensive literature review was performed in September 2009. A broad range of terms encompassing AHT were searched including *shaken baby syndrome, battered child syndrome, child abuse, craniocerebral trauma, whiplash, shaken-impact syndrome, inflicted (traumatic) brain injury, non-accidental (head) injury,* and *abusive head trauma.* These were combined each with *retinal hemorrhage, retinoschisis, retinal fold,* and *optic nerve (sheath) hemorrhage.* The complete search string can be found in Appendix 1 (available on-line at http://aaojournal.org).

The following electronic databases were searched simultaneously via Ovid:

1. MEDLINE (1948–September 4, 2009, including in-process and non-indexed citations);
2. EMBASE (1947–September 4, 2009); and
3. Evidence Based Medicine Reviews, which included:

- Cochrane Collaboration;
- American College of Physicians Journal Club 1991 to September 2009;
- Database of Abstracts of Reviews of Effects; and
- National Health Service Economic Evaluation Database.

To reduce the likelihood that potentially relevant articles were missed, MEDLINE was also searched via PubMed and reference lists from relevant articles were checked. Foreign literature was reviewed for relevance by reading the English abstract. If thought to be relevant, the article was acquired and translation obtained. Foreign literature without an English abstract was not assessed.

Data Extraction

One author (GB) scanned the initial list of articles for relevance to the inclusion criteria, based on title and abstracts. All of the authors then independently assessed the relevant articles for inclusion. Disagreements were resolved by discussion. Grading of studies was performed with a checklist derived from a standard grading tool for systematic reviews of diagnostic accuracy.[10] A detailed explanation of the items that were graded is provided in Appendix 2 (available on-line at http://aaojournal.org).

For each study, tabulated data included (1) study design (i.e., prospective or retrospective, controlled, or case series); (2) study population; (3) cohort size; (4) data on intraocular and cerebral findings; and (5) grading items.

The level of evidence of included studies, and importance of clinical recommendations were rated according to the *Ophthalmology* guidelines. Level I evidence provides strong support for a statement, and is usually composed of well-performed, randomized controlled-trials or meta-analyses of randomized controlled-trials. Level II evidence provides substantial support for the statement but cannot be applied without qualification because the evidence lacks some qualities. This usually includes observational studies, such as cohort studies and case-control studies. Level III indicates a weak body of evidence relying on consensus statements, small noncomparative case series, and individual case reports. In terms of clinical recommendations, Level A is considered very important, level B moderately important, and level C possibly relevant to clinical outcome.

Results

The varied search terms for AHT combined with *retinal hemorrhage, retinal folds, retinoschisis* and *optic nerve sheath hemorrhage* returned a total of 971 unique articles. A total of 79 relevant articles from all search sets were reviewed in full. A further 9 articles were added from reference lists of relevant articles, making a total of 88 articles. Of these, 33 articles were excluded for various reasons (Fig 1), leaving 55 studies.

Grading was performed on all 55 eligible studies. Because we wished to assess the diagnostic accuracy of ocular findings for AHT, studies that did not adequately describe the method of ocular examination or used a method that was considered inadequate were also excluded. These were mainly retrospective reviews or prospective studies in which ocular findings were not a main focus.

The 20 studies remaining are the subject of this review (Table 1). Three of these studies only reported patients with retinal findings;

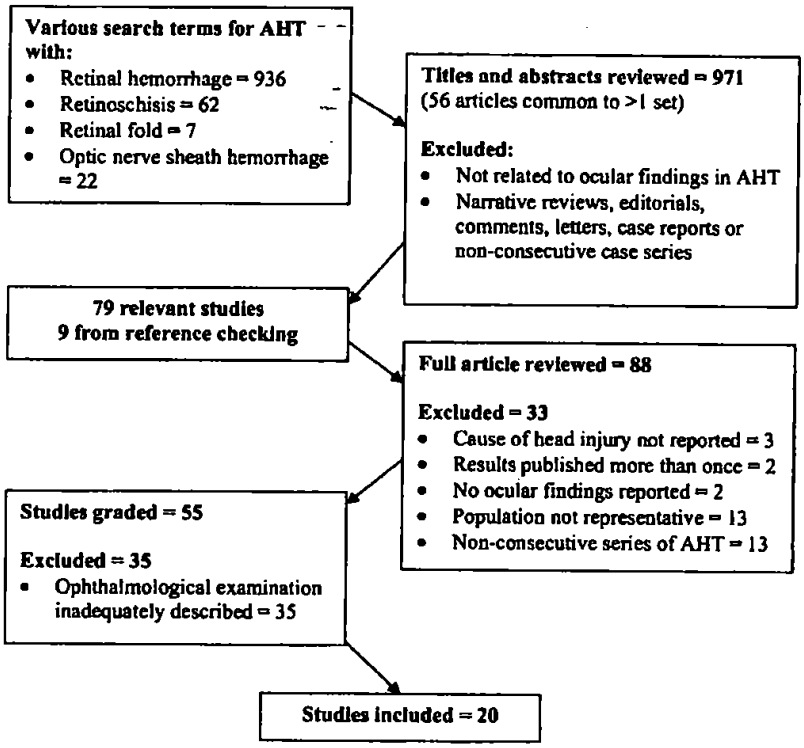

Figure 1. Results of search. AHT = abusive head trauma.

however, they were included because the cases were consecutively recruited and thus the pattern of findings could be analyzed. The overall quality of included studies, with respect to each of the graded items, is shown in Figure 2.

Several weaknesses were identified in the included studies. Only 2 studies mentioned that examiners were masked to the injury.[11,12] It is unlikely that examination was performed in a masked fashion in the other studies, because this detail was omitted. Confirmation of abuse remained unclear in more than half of studies owing to lack of detail regarding its determination. This also meant that there was the potential for circular logic in all but 4 studies,[12–15] because IOH are often used clinically as a diagnostic sign of abuse.

Individual results of grading for all studies and the references for excluded studies are available in Appendix 3 (available on-line at http://aaojournal.org).

Sources of Heterogeneity

Sources of heterogeneity in the studies included subgroups of patients, inclusion criteria, study design, and method of examination. Importantly, AHT is not a single diagnostic entity; therefore, significant variation in clinical presentation was apparent. Some studies recruited subgroups such as shaken baby syndrome, whereas other studies only recruited patients with subdural hematoma.

All but 1 study[16] included participants <4 years of age, and in this study only 3% were >3. Study designs comprised 3 main types, namely, autopsy series, retrospective chart reviews, and prospective clinical studies.

Intraocular Hemorrhages

The mean sensitivity of IOH for AHT in the included studies was 75% (Table 1). Clinical studies[12–15,17–20] found IOH in 74% (range, 51%–100%) of 560 combined cases and autopsy series found 82% with IOH (range, 63%–100%) in 84 combined cases.[6,7,21–25] This did not include the 3 studies that only reported cases with retinal findings and 2 large autopsy series of all forms of child abuse,[16,26] which found IOH in 46% (range, 45%–47%) of 555 combined cases.

The wide range is likely owing to the small sample sizes in some series[6,18,24] and intracranial hemorrhage as an inclusion criterion in others.[14] In 3 studies,[12–14] which were apparently free from circular logic and included confirmed cases of abuse, IOH were present in 80% of 151 cases of AHT.

Intraocular hemorrhages involved the retinal layers in almost all cases, in both clinical (94% of IOH) and autopsy series (99% of IOH). Most studies found evidence of hemorrhage in all of the retinal layers; however, numerous studies[16,21,23,26,27] reported the superficial layers—including sub-internal limiting membrane, nerve fiber and ganglion cell layers—to be most frequently and heavily involved. Preretinal hemorrhage was more frequently

Table 1. Summary of Studies in Review

Author (year, country)	Cohort Size	Age (yrs)	ICH %	IOH %	Comments
Retrospective chart reviews					
Wilkinson[11] 1989 U.S.A.	14 AHT (SBS)	0–2.3	100	100[‡]	Significant correlation found between diffuse IOH, large preretinal or vitreous hemorrhage and neurologic injury. Diagnostic accuracy not assessed. IOH part of diagnostic criteria for abuse.
Mills[28] 1998 U.S.A.	10 AHT (SBS)	0–1	100	100[†]	Circular retinal folds, retinoschisis, and fixed pupils correlated with fatal outcome. Study evaluated association between ocular findings and neurologic outcome, not diagnostic accuracy.
Kivlin[14] 2000 U.S.A.	111 AHT (SBS)	0–3	100*	83	SDH inclusion criterion, 95 clinical cases, none with peripheral IOH, posterior pole most heavily affected in 24% 16 autopsy cases, mostly diffuse IOH with 77% peripheral involvement. Higher rate of vitreous, subretinal and dome shaped hemorrhage under ILM compared with clinical cases.
Morad[17] 2002 Canada	75 AHT (SBS)	0–4	100	85	83% multiple confluent hemorrhages, and 87% bilateral. Hemorrhage confined to posterior pole in 20%, diffuse IOH 61%. IOH part of diagnostic criteria for abuse.
Gilles[12] 2003 U.S.A.	14 AHT	0–2.4	100	71	Retrospective review of fundus photographs. Laterality and severity of IOH correlated with cerebral hypoattenuation in 70% cases. Diffuse IOH in 50% cases in at least 1 eye.
Binenbaum[20] 2009 U.S.A.	49 AHT	0–1.4	—	51	Reported a positive correlation between incidence and severity of IOH and probability of abuse. Only studied cases which were investigated for AHT.
Prospective clinical studies					
Buys[18] 1992 Canada	3 AHT (SBS) 75 accidental HI	0–3	100 7	100 0	Majority of accidental head injuries mild, recruitment rate of 40%. Small number of cases of AHT for comparison. Excluded infants <1 month old.
Pierre-Kahn[15] 2003 France	224 AHT 7 accidental HI	0–3	100* 100*	68 0	SDH inclusion criterion. Small number of cases of accidental HI. Rate of IOH lower in cases with impact (28%) compared with no sign of impact (78%). No correlation between presence of IOH and age. No pattern of IOH identified.
Bechtel[13] 2004 U.S.A.	15 AHT 67 accidental HI	0–2	80* 27*	60 10	The following were significantly more frequent in AHT: IOH (60% vs 10%), bilateral IOH (40% vs 1%), preretinal hemorrhage (30% vs 0), peripheral IOH (27% vs 0; $P<0.001$ for all), premacular hemorrhage (20% vs 0; $P<0.005$). Vitreous hemorrhage no significant difference (13% vs 0%; $P = 0.03$).
Vinchon[19] 2005 France	57 AHT 93 accidental HI	0–2	81* 28*	75 7	IOH had positive predictive value for AHT 89%, negative predictive value for AHT 83%. IOH part of diagnostic criteria of abuse. Accidental head injury milder in severity.
Autopsy series					
Rao[21] 1988 U.S.A.	14 AHT (5 SBS) 16 controls	0–4 0–3	64 6	63 6	All retinal layers involved especially superficial; sub-ILM, nerve fiber, ganglion cell, inner nuclear. Controls included accidental trauma, SIDS, and others. Unclear whether control cases were consecutively obtained.
Elner[22] 1990 U.S.A.	10 suspected CA	0–4	60	70	Peripapillary scleral hemorrhage 50%. No details given on 3 cases without IOH.
Munger[27] 1993 U.S.A.	12 AHT (SBS)	0–2	92	100[§]	75% optic nerve sheath hemorrhage, 42% retinal folds. Hemorrhage most often in nerve fiber and ganglion cell layer.
Budenz[23] 1994 U.S.A.	13 AHT 6 SIDS	0–3 0–0.5	92 0	85 0	Nerve fiber, inner plexiform and inner nuclear layers most involved. Severe optic nerve sheath SDH in all cases of AHT.
Betz[6] 1996 Germany	7 AHT (SBS) 42 control	0–1 0–73	100 71	100 5	All retinal layers involved in AHT. Controls included 24 head injuries, 6 ICH, 2 intravital brain death, 10 SIDS. Two of 24 (8.3%) head injuries had IOH. Age-mismatched controls.
Green[7] 1996 U.K.	16 AHT 7 CA (not primarily HI)	0–3	94 0	81 14	Found hemorrhage at the following sites with increasing force: SDH, preretinal, intraretinal, optic nerve SDH, retinal detachment, choroidal, vitreous, SAH, intracerebral. One case of asphyxia had vitreous, preretinal and intraretinal hemorrhages.

(*Continued*)

Table 1. (Continued.)

Author (year, country)	Cohort size	Age (yrs)	ICH %	IOH %	Comments
Marshall[24] 2001 Canada	6 AHT (SBS)	0–3	100	100	Small series. Unclear as to how diagnosis of SBS was established. IOH part of diagnostic criteria of abuse.
Riffenburgh[26] 2005 U.S.A.	197 confirmed CA 240 suspected CA 401 control	1† 0.5† 0.3†	— — —	47 44 4	Bipolar and ganglion cell-nerve fiber layers most commonly affected in AHT. Controls were unexplained deaths including auto accidents, drowning, SIDS. Odds ratio for abuse ($P<0.001$) with: IOH = 18.9, optic nerve sheath hemorrhage = 32.5, IOH + ICH = 53.0, IOH + ICH + optic nerve sheath hemorrhage = 53.3 True incidence of ICH unknown.
Wygnanski-Jaffe[25] 2006 Canada	18 AHT (SBS) 18 accidental HI	0–3 0–14	94 67	89 17	Focused on optic nerve and orbital pathology. Optic nerve sheath hemorrhage found in 78% AHT and 39% accidental HI ($P<0.04$). Age mismatched controls. No extra details of IOH. IOH part of diagnostic criteria of abuse.
Emerson[16] 2007 U.S.A.	118 CA	0–9 94% <3	53	47	IOH severe in 68% and widespread, confluent in 9%. Associated with ICH or optic nerve sheath hemorrhage in 96% of cases. Peripapillary scleral hemorrhage in 38% always associated with perimacular folds and 98% with optic nerve sheath hemorrhage. Severe retinal involvement seen in 6 of 7 cases with admission of shaking. Nerve fiber layer involved in 97% of IOH, extending to deeper layers in 81%.

AHT = abusive head trauma; CA = child abuse (all forms); CE = cerebral edema; HI = head injury; ICH = intracranial hemorrhage; IOH = intraocular hemorrhage; SBS = shaken baby syndrome; — = not reported.
*Rate of SDH.
†Retinal hemorrhage was an inclusion criterion on.
‡Median ages reported only.

found in clinical than autopsy series (67% vs 27% of IOH). Vitreous hemorrhage was much more common in autopsy series than clinical series (72% vs 11% of IOH). Clinical studies did not often comment on subretinal or choroidal involvement; however, autopsy series found subretinal involvement in the majority (57%) of cases of AHT (Table 2). Intrascleral hemorrhages were found in 33% of 173 combined cases,[14,16,22,24,27] mainly in the peripapillary region involving the circle of Zinn.

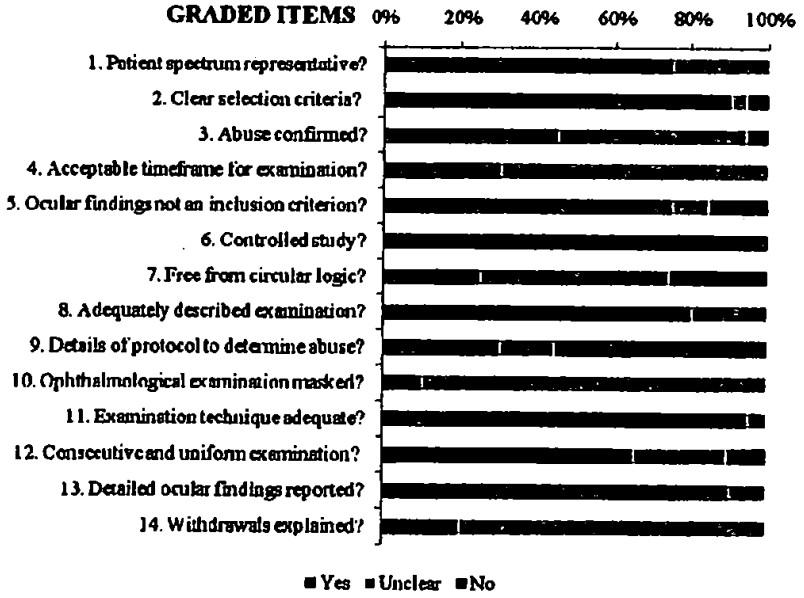

Figure 2. Graded items and overall quality of included studies.

Table 2. Location of Intraocular Hemorrhage in Abusive Head Trauma (AHT)

Clinical Studies			Layer Affected (# cases, % of IOH)			
Study (no. of cases)	*IOH, No. of Cases (%)*	*Age (mos)*	*Vitreous*	*Preretinal*	*Intraretinal*	*Subretinal*
Kivlin[14] (96)	77 (80)	0–36	13 (17)	36 (47)	76 (99)	10 (13)
Morad[17] (75)	64 (85)	2–48	—	55 (86)	55 (86)	44 (69)
Bechtel[13] (15)	9 (60)	0–24	2 (22)	5 (55)	9 (100)	—
Pierre-Kahn[15] (224)	152 (68)	0–29	15 (10)	106 (70)	142 (93)	—
Mills[28] (10)	10 (100)	2–10	3 (30)	7 (70)	10 (100)	—
Total (420)	312 (74)		33 (11)	209 (67)	292 (94)	54 (17)

Autopsy Studies			Layer Affected (# cases, % of IOH)				
Study (# cases)	*IOH (# cases, %)*	*Age (months)*	*Vitreous*	*Preretinal*	*Intraretinal*	*Subretinal*	*Choroid*
Emerson[16] (118)	55 (47)	0.3–108	47 (85)	—	53 (96)	37 (67)	8 (15)
Green[7] (16)	13 (81)	0.5–24	2 (15)	12 (92)	12 (92)	—	5 (38)
Munger[27*] (12)	12 (100)	1–21	10 (83)	3 (25)	12 (100)	11 (92)	6 (50)
Budenz[23] (13)	11 (85)	2–36	7 (54)	7 (54)	11 (100)	8 (73)	—
Kivlin[14] (27)	26 (97)	0–36	17 (65)	11 (42)	26 (100)	10 (38)	—
Total (186)	115 (62)		83 (72)	33 (27)	114 (99)	66 (57)	19 (17)

IOH = intraocular hemorrhage; — = not reported.
*Because IOH were an inclusion criteria, findings may be overrepresented.

The posterior pole of the retina (86% of IOH) was the most frequently reported area of IOH in AHT (Table 3). A similar proportion of hemorrhages (57% of IOH) were reported in the midperiphery and peripheral regions. Studies reporting laterality of ocular findings found, overall, 74% of IOH were bilateral in AHT.[6,11–15,17,18,20,21,24,27]

From the 4 prospective controlled studies, IOH were found in 6% of 242 combined cases of accidental head injuries,[13,15,18,19] and these were all reportedly mild in severity. Thus, when IOH are present after head injury, their specificity for abuse is 94% (range, 90%–100%). One study[19] found that moderate to severe IOH were only found in cases of AHT, and the sensitivity and specificity of these were 66% and 100%, respectively.

Other conditions studied were motor vehicle crashes (MVC),[21,26] sudden infant death syndrome,[6,21,23,26] central nervous system diseases, and other unexplained deaths.[21,26] The studies examining these were mostly small autopsy series and it is unclear whether the control groups were consecutively collected. Nevertheless in 1 large study,[26] IOH were found in just 4% of 401 control cases.

Meta-analysis of these data is unreliable because of the observational nature of studies and their heterogeneity with respect to design, age range, and inclusion criteria.

Retinal Folds and Retinoschisis

Seven studies[14–16,22,24,27,28] included in this review reported perimacular retinal folds in AHT. The frequency of this finding was higher in autopsy series (23%[16,22] of 128 combined cases) than in consecutive clinical studies[14,15] (3% of 347 combined cases). Their overall sensitivity for AHT was 8%. This was not inclusive of studies with retinal findings as an inclusion criterion,[24,27,28] which found retinal folds in 43% of 28 combined cases of AHT.

Traumatic retinoschisis was also reported in seven included studies[15–17,21,22,24,28] and its overall sensitivity for AHT (excluding studies which only reported positive retinal findings[24,28]) was 14%. Autopsy studies[16,21,22] once again demonstrated a higher proportion of cases with retinoschisis (23% of 142 combined cases) than clinical studies[15,17] (9% of 299 combined cases).

There were no studies in this review, which demonstrated retinal folds or retinoschisis in conditions other than AHT. These lesions have only been reported in a few case reports of other conditions relevant to infants, which will be discussed.

Table 3. Distribution of Intraocular Hemorrhage in Abusive Head Trauma (AHT)

Study (no. of cases)	Age (mos)	SDH %	IOH %	Bilateral IOH %	Posterior Pole (%)	Midperiphery (%)	Peripheral (%)
Emerson[16] (118)	0.3–108	53	47	80	85	78	61
Green[7] (16)	0.5–24	94	81	—	25	35	40
Marshall[24] (6)	1–34	100	100	100	100†	83†	67†
Morad[17] (75)	2–48	93	85	69	95	48	48
Gilles[12] (14)	1–54	100	71	57	90	—	90
Total % of IOH					86	57	57

IOH = intraocular hemorrhage; SDH = subdural hematoma; — = not reported.
*SDH was an inclusion criterion.
†Retinopathy of prematurity zones were used in the article. For the purpose of this table, Zone 1 was considered posterior pole, zone 2 midperiphery, and zone 3 peripheral (near ora serrata).

Optic Nerve Sheath Hemorrhage

Eleven included studies[6,7,16,21–28] examined this finding. The overall sensitivity of optic nerve sheath hemorrhage for AHT was 72% in 78 combined cases from consecutive autopsy series of AHT.[6,7,21–23,25] Subdural hematoma of the optic nerve sheath was found in 76% and subarachnoid hemorrhage in 46%. The frequency of involvement varied with inclusion criteria, and case series[16,26] including all forms of child abuse found only 50% of 311 combined cases with optic nerve sheath hemorrhage. Other case series of AHT,[24,27,28] with retinal findings as inclusion criteria, found 75% of cases had optic nerve sheath involvement.

Accidental head injury caused optic nerve sheath hemorrhage in 29% of 42 cases from 2 controlled autopsy studies.[6,25] Thus, its specificity for AHT can be estimated at 71%. The location of hemorrhage in the accidental cases was subdural in 92% and optic nerve subarachnoid hemorrhage was not seen. Riffenburgh[26] found that optic nerve sheath hemorrhage had a higher correlation with abuse than IOH, with an odds ratio of 32.5 ($P<0.001$) compared with 18.9 ($P<0.001$) for IOH.

One study[25] found all forms of orbital pathology more common in the AHT group. Orbital fat hemorrhage was observed in 50% of AHT, as opposed to 22% of accidental head injuries and extraocular muscle hemorrhage was observed in 28% of AHT, but not seen in accidental head injury. These findings were more marked in the posterior orbit.

Discussion

Since the first case report[2] of retinal trauma in an abused infant, level III retrospective studies[14,17] have confirmed that a significant proportion of infants with AHT have IOH. These studies have several potential drawbacks, including incomplete data and interobserver bias. The largest retrospective series[14] found that fatal cases had more severe and diffuse retinal findings extending to the periphery, whereas nonfatal cases had fewer IOH that were confined to the posterior pole.

Numerous level III autopsy series[6,7,16,21–27] have documented the full extent of injuries in severe cases of AHT. The reviewed studies found IOH much more frequently, and with greater severity, in AHT than in other studied conditions, including accidental head injury.

More recently, level II prospective controlled studies[13,15,19] have confirmed that IOH occur much more frequently in AHT than in accidental head injuries. The studies by Bechtel et al[13] and Vinchon et al[19] found that the specificity of IOH for AHT correlated more with the overall severity of IOH rather than their distribution or depth in the retina. Infants with AHT were also more likely to have multiple, bilateral IOH, involving the preretinal and intraretinal layers, covering the macula, and extending to the retinal periphery. This pattern of involvement was not specifically reported in any control cases in the included studies, but has been reported in fatal MVC[29] and crush head injury.[30–32]

The rate of IOH may be underestimated in most studies of AHT. This is because of delayed presentation in an unknown number of cases and delayed ophthalmologic examination after hospital admission. Only 5 studies[11,12,15,18,28] mentioned the timeframe within which examination was conducted.

The correlation between the incidence of IOH and age at presentation is unclear from the included studies. Riffenburgh et al[33] reported the greatest incidence of IOH in infants <1 year of age, whereas Kivlin et al[14] found that IOH significantly ($P<0.0063$) increased with age. Pierre-Kahn et al[15] found no correlation between age and presence of IOH in their large, prospective study.

Confirming the precise mechanism of injury is one of the major difficulties in studying AHT. Most of the included studies contained a proportion of cases that had evidence of direct cranial impact, in the form of scalp bruising or skull fractures. Pierre-Kahn et al[15] found that cases of AHT with evidence of direct impact had significantly lower rates of IOH compared with those with no evidence of impact. Morad et al[17] found no correlation between signs of direct impact and presence, or severity, of IOH. Budenz et al,[23] in a small autopsy series, found IOH in a higher proportion of cases with injury owing to blunt trauma. Evidence of direct impact, however, does not exclude shaking as a contributing factor. It thus remains difficult to confirm the relative contributions of direct impact and shaking to the overall picture of IOH. It is clear, however, that the full spectrum of ocular injuries can occur in cases without evidence of direct impact.

Emerson et al[16] proposed that peripapillary intrascleral hemorrhage may be characteristic of shaking injury, because evidence of blunt impact was absent in more than half of these cases in their series. Furthermore, all these cases had perimacular retinal folds, and 98% had optic nerve sheath hemorrhages, leading the authors to propose that they may result from torsion at the junction of the optic nerve and the globe during shaking, causing tears in dural bridging veins and the vessels near the circle of Zinn.

Retinal folds and traumatic retinoschisis in AHT were first reported in 1986 by Gaynon et al[3] and Greenwald et al,[4] respectively. Both lesions were reported in a minority of AHT cases in the included studies, although they were more commonly seen in autopsy series. They were also associated with more severe neurologic injury and a high mortality rate, in retrospective clinical series.[11,14,28] The rate of traumatic retinoschisis may be underestimated in clinical studies owing to similarity in appearance and difficulty in differentiation from preretinal hemorrhage.

None of the controlled studies in this review reported these lesions in conditions other than AHT, demonstrating a high specificity. They have previously been reported in 3 cases of crush head injuries.[30–32] A retrospective series[34] of 25 crush head injuries did not find any cases with retinal folds or retinoschisis, indicating that they are an uncommon finding. In the recent autopsy series by Kivlin et al,[29] elevated retinal folds were reported in 3 of 10 victims <3 years of age in fatal MVC. These patients reportedly experienced severe deceleration and rotational whiplash injury.

At our institution, we have also observed a hemorrhagic retinopathy with bilateral macular retinoschisis and elevated retinal folds in a 2-year-old infant after a fatal 11-meter fall onto concrete (Acta Paediatrica 2008;97:149). This fall resulted in multiple, bilateral parietal and occipital skull fractures, acute right subdural hematoma, and diffuse edema of the right cerebral hemisphere with mass effect and tentorial herniation. Police investigation was conducted and no crim-

inal charges were filed. Thus, these lesions have been rarely reported in other circumstances and the history should be the guiding factor in determining their etiology.

The data concerning retinal folds and retinoschisis are mostly derived from level III evidence. The only prospective, controlled study,[15] which mentioned these findings, reported a single case of AHT with a unilateral perimacular retinal fold. Further studies, which accurately and extensively document retinal findings in a wide variety of conditions, are required to better estimate the specificity of these signs for AHT.

Retinal folds and retinoschisis in AHT have been proposed to result from traction and shearing forces applied to the retina, at firm points of attachment to the vitreous during acceleration–deceleration.[3–5,24,28] In infants, the vitreous is especially well-attached to the retina at the posterior pole, over blood vessels and near the ora serrata.[33] This hypothesis does not readily explain the findings in crush head injury. Other authors have proposed that sustained venous stasis,[16] or an abrupt elevation in intracranial pressure transmitted to the optic nerve,[31,36] result in rupture or extravasation of fluid from intraretinal vessels causing separation of the internal limiting membrane[36] or outer nuclear layer[31] from the rest of the retina. Keithahn et al[36] hypothesized that perimacular folds were created as the internal limiting membrane separated from the macula, while remaining attached to the more peripheral retina.

Only one large, controlled study[26] has documented optic nerve findings in a variety of conditions. The incidence of optic nerve sheath hemorrhage was significantly higher in AHT (odds ratio, 32.5; $P<0.001$) than in other conditions including accidental injury, sudden infant death syndrome, and drowning. Other studies[6,25] found almost one third of accidental head injuries also had optic nerve sheath hemorrhage. A recent autopsy series[29] of fatal MVC victims found optic nerve sheath hemorrhages in 9 of 10 cases, all of which were thought to have experienced significant whiplash injury.

The mechanisms underlying optic nerve sheath hemorrhage in AHT are still unresolved. Lambert et al[37] first described the presence of optic nerve sheath hemorrhages in an infant with presumed SBS. They proposed that expansion of the optic nerve sheath, owing to transmission of an acute elevation in intracranial pressure, resulted in rupture of bridging veins. In Riffenburgh's series,[26] however, optic nerve sheath hemorrhage often seemed to be a direct extension of intracranial submeningeal hemorrhage. The predilection of hemorrhage, in some studies,[25] to the optic nerve–globe junction and posterior orbit, have also lead to the hypothesis that these sites may act as fulcrums distributing force during acceleration–deceleration.

Investigation and Follow-up

Given that a diagnosis of abuse carries enormous social and legal ramifications, a comprehensive workup is required to exclude all potential mimics. This is usually undertaken by pediatricians trained in child abuse in collaboration with neurosurgeons, neurologists, radiologists, ophthalmologists, and other subspecialists, such as geneticists and endocrinologists.

A comprehensive history of the presenting illness, including the timeline of symptoms, and any episodes of prior trauma is taken. Detailed past medical history, including neurologic and hematologic disorders, and a family history, including any hereditary conditions, is sought. History of the pregnancy including labor, delivery, and neonatal vitamin K administration is also documented. If there is a history of trauma, a detailed reconstruction of the exact mechanism of injury is performed with assistance from the police.

As well as looking for evidence of cranial trauma, the infant is examined for evidence of extracranial injuries, such as skin, abdominal, and skeletal trauma. The relevant investigations for these injuries are undertaken.

Investigations to rule out other important mimics of child abuse are undertaken in all cases. A full coagulation screen and hematology consult are obtained. Metabolic and genetic conditions such as osteogenesis imperfecta,[38] Menkes,[39] and glutaric aciduria[40] are ruled out with appropriate advice from subspecialists. Cranial imaging is carried out according to recognized international guidelines comprising computed tomography scanning, magnetic resonance imaging, and magnetic resonance angiography, as well as bone scan and skeletal survey. Importantly, psychosocial stressors are not taken into account in making a diagnosis of abusive injury.

Once the diagnosis of AHT is confirmed, ophthalmologic follow-up varies depending on the findings. While a child is still admitted in hospital, weekly follow-up is appropriate. After discharge, the patient should be seen within 2 weeks to monitor the progress of retinal findings and assess the vision. Further follow-up can be lengthened to 4 weeks, 6 weeks, and then 6 months as long as the findings are adequately resolving. The patient's visual acuity, ocular alignment, retinae, and optic discs should be monitored at least until school entry age.

In most cases with extensive IOH, retinal folds, and retinoschisis, the prognosis is poor, and follow-up depends on future clinical management. If retinal detachments are present, referral is made to a vitreoretinal surgeon. If there are associated cranial nerve palsies, weekly follow-up is appropriate. Cortical blindness with severe intracranial injuries is assessed with visual-evoked potentials to estimate the visual outlook. Intracranial hemorrhage with no ocular signs is managed primarily by the neurology and neurosurgery teams with ophthalmologic consultation as required.

Clinical Recommendations

The significance of ocular findings in AHT has been the subject of great controversy. Previously identified[8] weaknesses in the evidence base were the lack of prospective, controlled studies, frequency of circular logic, and inconsistency in definition of cases. Currently, there is level II evidence from prospective controlled studies, supporting a significant relationship between IOH and AHT. Accurate classification of trauma as being from abuse remains difficult, because the trauma is generally unwitnessed and de-

nied by perpetrators. Level I evidence is impossible to achieve in this field, for obvious reasons.

Of the reviewed studies,[6,7,12–15,17–19,21–25] the most sensitive ocular sign for AHT, on clinical examination, is IOH, which is found in approximately 77% of infants (A, II). Combined data from prospective studies of head injury indicate that IOH have a specificity of 94% for abuse[13,15,18,19] (A, II). The specificity of IOH for AHT is further increased when there is bilateral involvement, preretinal hemorrhages, premacular and peripheral involvement, and moderate to severe IOH[13,19] (A, II). Intraocular hemorrhages are not pathognomonic of AHT, and have infrequently been reported in other conditions including severe, accidental head injury[13,19,21,25,26,29] (A, II).

Most frequently, IOH involve the posterior pole in AHT,[16,17,24] although this is not consistent between studies[7] (B, III). The intraretinal layers are most frequently affected in all studies (B, II). Clinical studies[13–15,17,28] report preretinal hemorrhage as the next most common site (B, II), whereas autopsy studies[14,16,23,27] find vitreous and subretinal involvement to be more common (B, III).

Perimacular retinal folds and traumatic retinoschisis have a low sensitivity for AHT, seen overall in approximately 8% and 14% of cases, respectively[14–17,21,22,24,27,28] (A, III). They have a high specificity for AHT and have not been demonstrated in other conditions in controlled studies[15,21] (A, III). They are not, however, pathognomonic for AHT, and have been reported in infants after fatal MVC,[29] crush head injuries,[30–32] and a high altitude fall (A, III). Apart from their diagnostic significance, they are a poor prognostic indicator and have been associated with more severe neurologic injury and mortality[14,28] (A, III).

Optic nerve sheath hemorrhages are found overall in approximately 72% of cases of AHT at autopsy[6,7,21–23,25] (A, III). In contrast, controlled studies have found optic nerve sheath hemorrhages in up to 29% of accidental head injury.[6,25] The diagnostic accuracy for abuse is improved when multiple pathologies including IOH, optic nerve sheath hemorrhage and intracranial hemorrhage are present[26] (A, III).

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Appendix Z

BRIEF COMMUNICATION

M.G.F. Gilliland,[1] *M.D.*

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

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

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

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

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

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Methods

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

Sample Selection

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

Symptom Onset Definition

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

Mechanism of Injury

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

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

Results

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

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

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

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

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

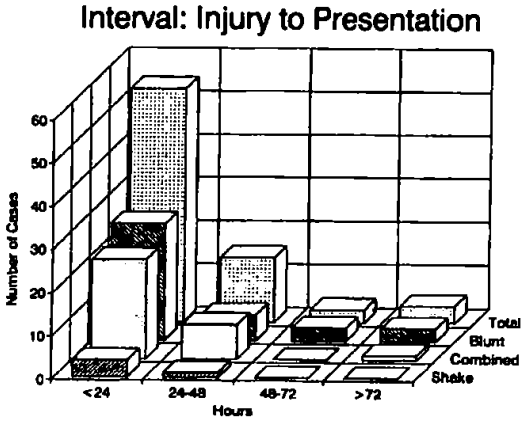

FIG. 1—*The graphic display confirms the impression that most of the children will present with severe symptoms in an interval of less than 24 hours after injury.*

Discussion

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

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

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

TABLE 1—*Interval from injury to severe symptoms.*

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

Conclusion

Enough variability in the interval between injury and the time of severe symptoms or presentation for medical care in fatally injured children exists to warrant circumspection in describing such an interval for investigators or triers of fact. Our data indicate that the interval is brief (less than 24 hours), in almost $^3/_4$ of cases of head injury death, especially in shaking injuries. However, in more than $^1/_4$ of the cases, the interval from injury to the onset of severe symptoms is longer. In all cases in which the children were seen by an independent observer after injury, they were described as not normal.

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Appendix AA

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Kristy B. Arbogast, Susan S. Margulies and Cindy W. Christian

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Initial Neurologic Presentation in Young Children Sustaining Inflicted and Unintentional Fatal Head Injuries

Kristy B. Arbogast, PhD*‡; Susan S. Margulies, PhD§; and Cindy W. Christian, MD‖

ABSTRACT. *Background.* It remains unclear if fatal brain injuries in young children are characterized by immediate rapid deterioration or can present after an initial period of lucidity. This issue has legal implications in child abuse, for which understanding the clinical course affects perpetrator identification.

Objective. To determine patterns of neurologic presentation on hospital admission in infants and toddlers who die of inflicted and unintentional injury.

Design/Methods. Data on children <48 months of age who sustained a fatal head injury from 1986–2002 were extracted from the Pennsylvania Trauma Outcomes Study. Only those with external-causes-of-injury codes for inflicted injury, falls, and motor vehicle crashes (MVCs) with a recorded Glasgow Coma Scale (GCS) on admission were included. The GCS was compared across mechanisms and age groups (0–11, 12–23, 24–35, and 36–47 months).

Results. Of the 314 fatally injured children, 37% sustained inflicted injury, 13% sustained a fall, and 49% sustained an MVC. At admission, 6.8% of all children had a GCS score of >7, and 1.9% presented with a GCS score of >12 (lucid). The incidence of admission a GCS score of >7 varied by mechanism. Overall, children with inflicted injury were 3 times more likely to present with a GCS score of >7 than those injured in MVCs (odds ratio [OR]: 3.6; 95% confidence interval [CI]: 1.2–10.3), but incidence of a GCS score of >7 did not differ between inflicted injuries and falls. Similarly, when considering only those children ≥24 months old, a GCS score of >7 did not differ by mechanism. In contrast, in those <24 months old, children who died as a result of inflicted injury were >10 times more likely to have a GCS score of >7 than those who died as a result of a MVC (OR: 9.36; 95% CI: 1.3–80.9).

Conclusions. The data suggest an age- and mechanism-dependent presentation of neurologic status in children with fatal head trauma. Although infrequent, young victims of fatal head trauma may present as lucid (GCS score: >12) before death. Furthermore, children <48 months old sustaining inflicted injury are 3 times more likely to be assessed with a moderate GCS score (>7) than those in MVCs. This effect is amplified in the youngest children (<24 months old): those with inflicted injury were 10 times more likely to present with moderate GCS scores than those in MVCs. In addition, this youngest age group seems to be overrepresented in those who present as lucid (GCS score: >12 [5 of 6]). It is unclear whether these differences are the result of inadequate tests to evaluate consciousness in younger children or differences in biomechanical mechanisms of inflicted trauma. *Pediatrics* 2005;116:180–184; *abuse, accidents, falls, head injuries, lucid interval.*

ABBREVIATIONS. PTOS, Pennsylvania Trauma Outcomes Study; ICD-9, *International Classification of Diseases, Ninth Revision*; MVC, motor vehicle crash; GCS, Glasgow Coma Scale; OR, odds ratio; CI, confidence interval.

Homicide is the leading cause of injury death in infancy, and half of infant homicides occur during the first 4 months of life.[1] The majority of infant homicides involve inflicted traumatic brain injury. It remains unclear if fatal head injuries in young children are characterized by immediate rapid deterioration or can present after an initial period of lucidity.[2–5] This issue has legal implications in child abuse, for which understanding the clinical course affects perpetrator identification. Because inflicted injuries are typically unwitnessed and perpetrators are unwilling to provide a truthful history, the timing between injury and first clinical presentation may be unknown, further confounding the description of the clinical course.

In an attempt to determine if fatal head injuries in abused children are obviously symptomatic immediately, previous researchers have focused on the presence of a lucid interval by studying children who sustained unintentional fatal head injuries, for which the injury event is typically well documented. Plunkett[3] reported 18 short falls that resulted in fatal head injuries and documented 12 lucid intervals. In this study the average age (5.2 years) was significantly higher than that associated with child abuse. It is notable that no children were <1 year of age, and for those children <4 years of age, the lucid intervals documented were limited to <15 minutes. Willman et al[2] reviewed 95 cases of accidental fatalities involving head injury through a retrospective chart review and identified 2 children with a lucid interval: 1 child died with an epidural hemorrhage, and 1 died as a result of abdominal exsanguination. In their study, as well, the average age (8.5 years) was significantly older than the typical child involved in

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abusive head injury. Only 2 infants were included in their study sample. Because of variability in myelination and the size of subdural spaces between infants and older children,[6] the generalizability of their results to the inflicted-head-injury population remains unclear.

In this study, we build on this previous work and study a younger population. Specifically, we identify patterns of neurologic presentation for children <4 years of age who died as a result of inflicted and unintentional head injury.

METHODS

For the period 1986–2003, the Pennsylvania Trauma Systems Foundation Trauma Outcomes Study (PTOS) was reviewed to extract data on children <48 months of age who sustained a fatal head injury. PTOS is a statewide registry of clinical data from all 26 accredited trauma centers (adult and pediatric) throughout the state of Pennsylvania. A standardized set of data elements for cases that meet specific criteria is collected by trained trauma registrars in individual institutions and submitted to a central database. Inclusion criteria for the PTOS study are patients (all ages) admitted for treatment of a diagnosis of trauma including all intensive care admissions, all patients who were dead on arrival, all trauma deaths, and all admissions for at least 48 hours, including transfers. The diagnosis of trauma is defined as *International Classification of Diseases, Ninth Revision* (ICD-9) codes of 800 through 995. Cases are enrolled regardless of specific injury patterns. Specific exclusions include those patients with an isolated hip fracture, isolated asphyxiation, drowning, and poisoning. To minimize data errors, registrars attend mandatory training sessions semiannually, 1 of which focuses on interabstractor reliability. Audits are also performed regularly to monitor coding accuracy of the data submitted.

For this study, we selected those children <48 months of age with fatal head injuries from this database. Determination of fatal head injuries was based on ICD-9 coding of injuries. Only children who died with external-causes-of-injury codes (E codes) for inflicted injury (E967), falls (E880–E888), and motor vehicle crashes (MVCs) (E810–E819) with a recorded Glasgow Coma Scale (GCS) score on hospital admission were included. All 3 injury events were studied to determine the neurologic presentation of young children who sustained fatal head injuries from 3 varied mechanisms. MVCs, in particular, were chosen as a comparison to inflicted-injury events because the timing of the MVC relative to hospital presentation is well defined; thus, the occurrence of lucidity, if present, would be more likely to be documented. GCS scores were compared across mechanisms and age groups (0–11, 12–23, 24–35, and 36–47 months) by using the χ^2-test method. For the purposes of this study, relevant categorization of GCS scores was determined a priori (poor neurologic status: GCS score 3–7; moderate neurologic status: GCS score 8–12; good neurologic status [lucid]: GCS score 13–15).

RESULTS

Of the 314 fatally injured children identified, 37% (121) sustained inflicted injury, 13% (40) sustained falls, and 49% (153) were in MVCs. This distribution varied significantly by age ($P < .001$) (Fig 1).

Although most children had poor neurologic status on admission, 6.8% had an admission GCS score of >7, and 1.9% presented with a GCS score of 13–15 (lucid). Figure 2 demonstrates the distribution of GCS scores for each year of age.

The incidence of admission GCS scores varied by mechanism (Fig 3). Overall, children with inflicted injury were 3 times (odds ratio [OR]: 3.6; 95% confidence interval [CI]: 1.2–10.3) more likely to present with a GCS score of >7 than those in MVCs, but incidence of a GCS score of >7 did not differ between inflicted injury and falls (OR: 1.5; 95% CI: 0.4–5.5) (Table 1). When considering only children ≥24 months old, a GCS score of >7 did not differ by mechanism. In contrast, in those <24 months old, children who died as a result of inflicted injury were >10 times more likely to have a GCS score of >7 than those who died as a result of a MVC (OR: 10.6; 95% CI: 1.3–80.9). Thus, the influence of mechanism on the incidence of a GCS score of >7 varied by age.

Overall, 6 children were lucid (GCS score: >12) at admission, and children <24 months were overrepresented in this group (5 of 6). Moreover, 3 of 121 (2.5%) children with ultimately fatal inflicted injury were thought to be lucid at presentation to a Pennsylvania trauma hospital. All 3 were <24 months old (Table 2). Of the 6 children with a GCS score of >12 on admission, 4 sustained a subdural hematoma as part of their head injury.

DISCUSSION

Whether children can be lucid after sustaining a fatal head injury has important implications in the investigation of child abuse. Historically, infants and toddlers who have sustained fatal inflicted trauma are assumed to have been immediately and obviously symptomatic.[4,5] With a large sample of age-relevant cases, our data suggest that although infrequent, young victims of fatal head trauma may present as lucid (GCS score: 13–15) before death. In this series, ~2% of children <4 years of age were recorded as having a GCS score of 13–15 on arrival to a trauma hospital. By using PTOS data, our study only included children who were admitted to a Pennsylvania trauma hospital and did not capture data on children who died at the scene of the injury or at a local hospital before transfer to a trauma

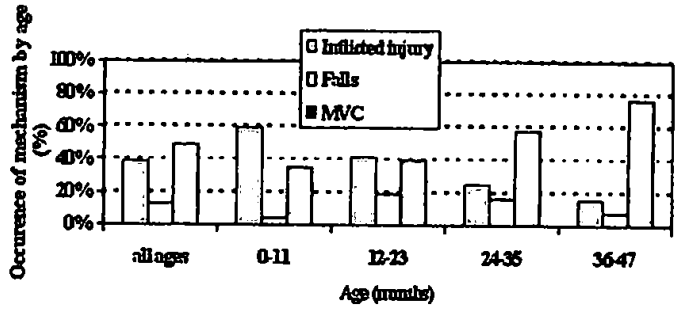

Fig 1. Distribution of injury mechanism (inflicted injury, falls, and MVCs) according to child age.

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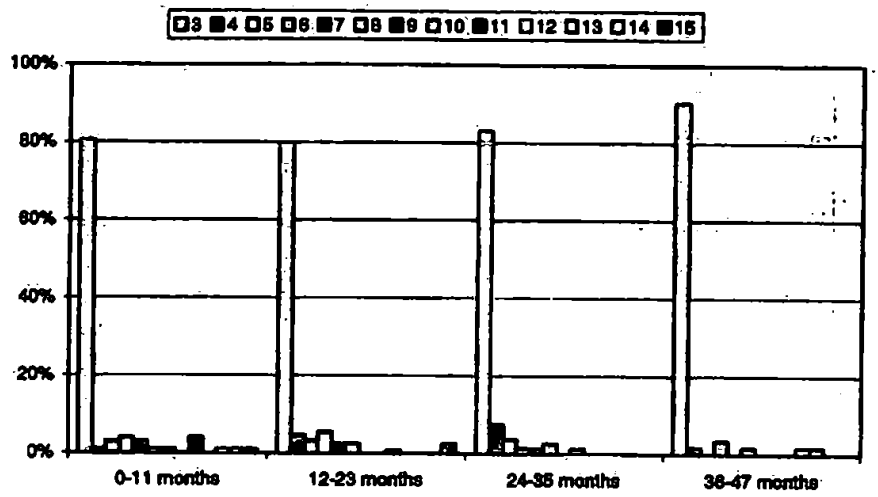

Fig 2. Distribution of GCS score according to child age.

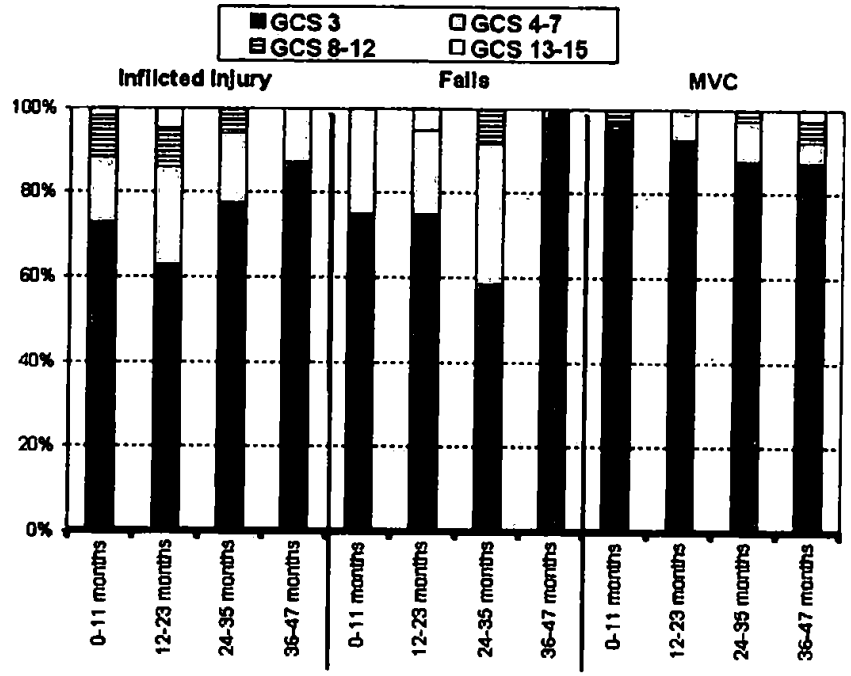

Fig 3. Distribution of GCS score according to child age and injury mechanism.

hospital. Therefore, our findings most likely overestimate the true percentage of children who seem to be lucid after fatal injury.

In our study, children <48 months old sustaining inflicted injury were 3 times more likely to be assessed with a moderate GCS score (>7) than those in MVCs. These results from a large sample support similar conclusions from single case reports.[4,5] In MVCs resulting in fatal injuries, the event is often a single, well-defined, high-energy impact. Inflicted injuries are typically more complex loading events that may include multiple purely inertial (shaking) as well as impact conditions, may encompass a wider magnitude range than MVCs, and may be repeated

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TABLE 1. Percentage of Children With GCS Scores of >7

Age Group	Inflicted Injury, % (n)	MVCs, % (n)	Falls, % (n)	Inflicted Injury vs MVCs/Inflicted Injury vs Falls, OR (95% CI)
Overall	10.7 (13/121)	3.3 (5/153)	7.5 (3/40)	3.6 (1.2–10.3)/1.5 (0.4–5.5)
0–23 mo	12.6 (12/95)	1.4 (1/72)	8.3 (2/24)	10.6 (1.3–80.9)/1.6 (0.3–7.6)
24–47 mo	3.8 (1/26)	4.9 (4/81)	6.2 (1/16)	0.8 (0.08–7.2)/0.6 (0.03–10.3)

TABLE 2. Children With GCS Scores of >12 (n = 6)

Mechanism (Age Group)	Head Injury	Hospital Length of Stay, d
MVCs		
36–47 mo	Closed skull fracture, SAH, SDH, EDH	3
Falls		
0–11 mo	Closed skull fracture, SAH, SDH, EDH	1
12–23 mo	Closed skull fracture, intracranial hemorrhage	9
Inflicted injury		
0–11 mo	Intracranial hemorrhage	1
12–23 mo	SDH	2
12–23 mo	SDH, MMA/intracranial hemorrhage	47

SAH indicates subarachnoid hematoma; SDH, subdural hematoma; EDH, epidural hematoma; MMA, middle meningeal artery.

at intervals of hours, days, or weeks. It remains unclear if repeated mild head injuries exacerbate or modulate the response compared with a single, more severe injury event.[7–9]

Our data indicate that the initial neurologic presentation of children with fatal head injury also depended on age. In the youngest children (<24 months old), those with inflicted injury were 10 times more likely to present with a moderate GCS score than those in MVCs. In addition, this youngest age group seems to be overrepresented in those who present as lucid (GCS score: 13–15 [5 of 6]). For those >24 months old, there was no difference in GCS scores among the 3 injury mechanisms. This result can be interpreted in several ways. It may suggest that inflicted injuries in infants occur more frequently, or that there are distinct responses of an infant's and toddler's brain to traumatic loading. Studies that examine the biomechanical, neurologic, and physiologic responses of the young child's brain across the age range are necessary to fully explain these results.

Alternatively, the overrepresentation of the youngest infants in the moderate-GCS-score group may simply reflect the inadequacy of the GCS in assessing neurologic damage or mental clarity in the very youngest patients. The GCS relies on motor and verbal skills that cannot be assessed accurately in infants and toddlers. For example, the motor component of the GCS assesses localization of pain, which does not typically develop in a child until ~18 months of age. The verbal response options are inappropriate for preverbal children, greatly limiting the reliability of the overall scale. Additionally, spontaneous eye opening and/or nonspecific movements in response to pain can be seen in infants who have suffered severe traumatic brain injury affecting the cortex, which can result in the assigning of an erroneously high GCS score.[10] To overcome these limitations, a number of pediatric-appropriate scales have been developed. Some of them do not overcome the challenges of finding practical but accurate ways to assess infant neurologic function and remain inappropriate for intubated patients.[11–13] Although appropriate scales have been developed for use in infants and toddlers, their prognostic ability has not been tested.[10] Despite progress to create and validate appropriate coma scales for young children, none have been broadly adopted by clinicians, and the GCS remains the scale used in most trauma centers, including those in Pennsylvania. However, because in this series the majority of patients who died as a result of inflicted injury were infants, the imprecision of GCS at younger ages may confound our findings.

Although our data suggest that a small percentage of infants and toddlers with fatal head injury can present as lucid to a trauma center, it does not imply that these children were completely asymptomatic. The GCS does not measure common symptoms of head injury such as vomiting, irritability, or subtle changes in alertness. It is possible that some children with GCS scores of 13–15 had clinical signs of head trauma not assessed by that scale.[14]

Specific clinical presentation among a sample of children with fatal head injuries is variable. Researchers have suggested that an infant's response to traumatic loading differs from that of an older child[6] and that the acceleration magnitude varies by the mechanism.[15,16] Little research has been conducted that correlates specific clinical presentations of young children with neuropathological findings and how that varies by age and injury mechanism. Future work should focus on making these linkages to determine if those fatal cases that present with a lucid interval demonstrate unique pathology from the overwhelming majority of children who are immediately obviously symptomatic.

These analyses were conducted on a specific population of young children with fatal head injuries from Pennsylvania's statewide trauma registry. This registry has been used previously to study injury events (the rates and injury characteristics of firearm

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injuries in adolescents and young adults[17] and risk of pulmonary embolism after head injury in adults[18]). Of Pennsylvania's 67 counties, 51 have a trauma center in the county or an adjoining county. These 51 counties include 84% of Pennsylvania's population. We hypothesize that the majority of those children with fatal head injuries not contained in the Pennsylvania Trauma Systems Foundation database died at the scene and did not enter the health care system. This consideration would only serve to emphasize the rarity of a lucid interval, because it would increase the underlying population of children with fatal head injuries from which our 6 children with GCS scores of 13–15 are drawn.

A population-based database such as that used in this study provides an appropriate sample to be able to conduct relevant statistical analysis. Data in the database were deidentified and abstracted from medical records, preventing the review of the actual medical details, confirmation of the injury mechanism, and determination of the nuances of the clinical time course of an individual patient. Future studies should utilize multicenter networks such as the Pediatric Emergency Care and Applied Research Network (PECARN) to identify substantial numbers of young children with fatal head injuries in which detailed clinical data and injury-mechanism information can be obtained.

CONCLUSIONS

Our data suggest that on rare occasion, an infant or toddler can sustain a fatal head injury yet present as lucid to hospital clinicians before death. Whether this is because of differences in pathologic injury, neurologic responses unique to the infant brain, or limitations of the bedside methods used to assess neurologic function in young children cannot be determined by this study. Because fatal head injury is a relatively rare event in infancy, improved understanding about the clinical course of infants and toddlers after fatal head injury will be served best by cooperative, multicenter work.

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Appendix BB

39

ABUSIVE HEAD TRAUMA

Kent P. Hymel, MD, and Katherine P. Deye, MD

Abusive pediatric head trauma begins with intense frustration and anger. In its aftermath, lives, relationships, families, and futures can be changed forever. Violent acts that result in traumatic brain injuries lead irrevocably to powerful, stressful experiences for involved family members and for many of the professionals who respond to treat, support, consult, investigate, protect, testify, prosecute, defend, and/or judge.

INCIDENCE AND EPIDEMIOLOGY

The incidence of inflicted head trauma during the first 1 or 2 years of life has been estimated in various studies to range from 16.1 to 33.8 cases per 100,000 infants per year.[1-3] A preponderance of evidence supports a conclusion that male infants are at greater risk.[1,5-7] Infants living in families experiencing socioeconomic or other acute stress are also at increased risk.[4,8] Race and ethnicity have not been found to be a significant predictor of inflicted pediatric neurotrauma.[9] Abusive head trauma appears to be the leading cause of infant homicide in the United States.[10]

HISTORICAL CONTEXT

The number and quality of published, peer-reviewed research studies regarding abusive head trauma have increased dramatically in recent years. Interpreting this expanding body of work is an ongoing challenge that requires an understanding of the historical context. Some individuals have played prominent roles by raising public awareness about abusive head trauma, by questioning widely held assumptions, or by redirecting our clinical or research priorities.

In 1946, Dr. John Caffey[11] published a landmark paper titled, "Multiple fractures in the long bones of infants suffering from chronic subdural hematoma." In this paper, he described six children with subdural hematomas and multiple fractures. Caffey was the first to suggest that trauma explained this combination of findings. He advised physicians to look for fractures in children with subdural hematomas, and vice versa.

On November 14, 1956, Virginia Jaspars was sentenced to 10 to 22 years in prison. Investigations revealed evidence supporting a conclusion that, between 1948 and 1956, Ms. Jaspars killed or injured at least 15 infants left in her care. New Haven, Connecticut physicians Drs. Paul Goldstein, Steve Downing, Michael Kashgarian, and (most importantly) Robert Salinger uncovered vital clues that ultimately revealed the nature of Ms. Jaspars' abusive actions. When Ms. Jaspars confessed to the death of Jennifer Malkan, she reported that she was irritated by the child's persistent crying and therefore, "... shook the child to cause her head to bob back and forth." She added, "The baby lost her breath and her eyes were funny in her head."[12]

In 1971, Dr. Norman Guthkelch, the first pediatric neurosurgeon in Great Britain, published an article in the *British Medical Journal* titled "Infantile subdural haematoma and its relationship to whiplash injuries."[13] He described multiple infants with subdural hematomas whose caregivers described violently shaking the children. Dr. Guthkelch's manuscript represents the first clear reference to shaking as a mechanism for intracranial injuries in infants and young children.

In 1987, pediatric neurosurgeon Dr. Ann-Christine Duhaime and colleagues published a landmark paper in the *Journal of Neurosurgery*.[14] Their retrospective analysis of 48 cases of shaken baby syndrome led Duhaime and her colleagues to conduct a series of biomechanical experiments. They concluded, "... severe head injuries commonly diagnosed as shaking injuries require impact to occur," and, "... shaking alone in an otherwise normal baby is unlikely to cause the shaken baby syndrome."

Recently, some professionals have opined that the shaken baby syndrome is an unproven myth. Widely held theories of shaking-whiplash injury are now directly challenged.[15,16] On the other hand, a considerable body of literature supports a conclusion that isolated shaking can injure or kill babies.[13,17-20] The debate continues—in the medical literature, in the lay press, and in courtrooms. Chapter 41, "The Case for Shaking," provides a more detailed overview of this topic.

NOMENCLATURE

In response to this ongoing debate, clinicians and researchers have assigned a variety of labels to the constellation of clinical signs and cranial injuries that they have interpreted to be the result of abusive actions. These labels have included shaken baby syndrome, whiplash-shaken infant syndrome, shaking-impact injury, shaking-slam injury, abusive head trauma, inflicted head trauma, inflicted pediatric neurotrauma, and inflicted traumatic brain injury. In recent years, more and more clinicians and researchers are choosing to use more inclusive and less specific labels (e.g., "abusive head trauma" or "inflicted head trauma") when referring to these cases, avoiding labels that imply unrealistic certainty regarding the specific injury mechanisms (e.g., "shaken baby

syndrome"). In 2009, the American Academy of Pediatrics Committee on Child Abuse and Neglect recommended use of the phrase "abusive head trauma" when describing an inflicted injury to the head and its contents.[21]

RESPONSIBILITIES OF THE CHILD ABUSE MEDICAL SPECIALIST

Child abuse medical specialists who play a role in these real-life dramas are expected to do all of the following competently and consistently:

- To recognize cases of abusive head trauma masquerading as accidental trauma
- To recognize cases of accidental head trauma misinterpreted as abusive
- To direct an appropriately thorough child abuse diagnostic evaluation
- To assemble and integrate the relevant historical, clinical, laboratory and radiological information
- To actively consider, confirm, and/or exclude *nontraumatic* etiologies for the child's clinical presentation, physical examination findings, and radiological findings
- To formulate objective, forensic impressions of reasonable medical certainty regarding the nature, extent, severity, mechanisms, and timing of the child's traumatic injuries, and
- To communicate those impressions clearly, consistently, and without bias—to family members, to other health care providers, to child protection case workers, to police investigators, and later, to attorneys, judges, and/or juries.

In this chapter, we summarize an approach to accomplish these tasks reliably.

THE CLINICAL SPECTRUM

A majority of infant victims of inflicted head trauma present with recent, sudden onset of clear and persistent clinical signs, attributed to an accidental cranial impact (e.g., a fall). A few will present initially for medical treatment with acute and severe *but delayed* clinical deterioration following an earlier, unrecognized, or unreported head injury event. Only rare cases of inflicted pediatric neurotrauma will present with a thorough caregiver accounting of his or her abusive acts that preceded the victim's acute clinical deterioration.

Abusive acts can involve isolated contact injury mechanism(s), isolated inertial injury mechanism(s), or combined contact and inertial injury mechanisms. The resulting primary and secondary traumatic cranial injuries can be closed or penetrating, superficial or deep, and focal or diffuse. The associated clinical manifestations can be mild, moderate, life-threatening or fatal; acute, delayed, or late; and recognized or missed. The caregiver's specific explanation for the child's traumatic cranial injuries and clinical presentation can be complete, incomplete, or absent; consistent or inconsistent (with repetition over time); adequate or inadequate (to explain the child's clinical, laboratory, and/or radiological findings); and truthful, partially truthful, or fabricated.

In recognition of this extraordinary clinical variability and the potential for historical inconsistencies, researchers have used a wide variety of definitional criteria to categorize and to compare cases of accidental versus inflicted pediatric head trauma. In hindsight, it appears that some of the earliest definitional criteria used by researchers to categorize cases as accidental or inflicted were fundamentally flawed with inherent biases or by circular reasoning. Despite these limitations, our child maltreatment medical literature provides overwhelming evidence that adults' abusive actions can cause devastating or fatal traumatic brain injuries in infants and young children.[22-51]

Abusive acts have been linked to subdural, subarachnoid, and retinal hemorrhages;[7,22,32-33] localized axonal injury in the region of the craniocervical junction; acute respiratory compromise or arrest; loss of consciousness; hypotension;[36-40] and secondary, diffuse, hypoxic-ischemic brain injury with swelling.[23,39,41-42] Young victims of inflicted head trauma frequently present with associated cutaneous injuries[32,43] and fractures of the skull,[27,44,45] ribs, or extremities.[11,46] At the time of initial neuroimaging, many young victims of abusive head trauma reveal radiological evidence of prior (and therefore repetitive) traumatic, intracranial injuries.[24]

Acute clinical signs (appearing within seconds, minutes, or hours) can include craniofacial soft tissue injuries, inconsolability, loss of appetite, vomiting, altered sleep patterns, seizures, alteration or loss of consciousness, and cardiorespiratory compromise and/or arrest. *Delayed* clinical signs (appearing later, but within hours, days, or weeks) can include inconsolability, recurrent vomiting, loss of appetite, altered sleep patterns, seizures, alteration or loss of consciousness, and cardiorespiratory compromise or arrest. *Late* clinical signs (appearing weeks, months, or years after injury) can include feeding difficulties, sensory deficits, motor impairments, macrocephaly, microcephaly, behavioral dysfunctions, developmental delays, intellectual deficits, attention deficits, and educational dysfunctions. For a more complete discussion of outcomes, see Chapter 48, "Outcome of Abusive Head Trauma."

MISSED CASES

Considered in isolation, each of these clinical manifestations of traumatic brain injury is nonspecific. Each can result from inflicted head trauma, from noninflicted head trauma, or from other nontraumatic etiologies. Young victims of abusive head trauma who present for medical evaluation and treatment with isolated, less severe, nonspecific clinical signs (e.g., irritability, lethargy, recurrent vomiting, loss of appetite, and/or altered sleep patterns) are frequently misdiagnosed. In their 1999 analysis, Jenny and colleagues[47] reported that physicians recognized less than one in five cases of inflicted pediatric head trauma if the victim presented with normal respirations, no seizures, and no facial or scalp soft tissue injuries, and if the victim came from a Caucasian family with two adult caregivers.

A high index of suspicion is warranted. Without a high index of suspicion, cases will be missed, victims will be returned to their abusive environment, and children will be reinjured. In their 1999 study, Jenny and colleagues[47] reported that 27.8% of victims of inflicted head trauma were reinjured after a missed diagnosis.

REPORTING SUSPECTED ABUSE

Medical providers are not trained or mandated to investigate a crime scene or to assess psychosocial risk of child abuse or neglect. We *are* mandated by law in all 50 states to report any *suspicion* of abuse. Defining precisely what constitutes a reasonable suspicion of abusive head trauma is difficult, if not impossible. Yet, experienced clinicians have come to recognize numerous specific examples of clinical presentations (involving an infant or young child) that create a reasonable suspicion of abusive head trauma and prompt a report of suspected abuse to child protective services. These examples include the following:

- Unexplained loss of consciousness, lethargy, hypotonia, seizures, coma, respiratory distress, apnea, persistent irritability, recurrent vomiting, and/or poor feeding when infectious, metabolic, or toxic causes have been excluded
- Clear and persistent clinical signs that prompt diagnostic evaluation revealing evidence of traumatic brain injury, nonfocal subdural or subarachnoid hemorrhage and/or extensive, dense, multilayered retinal hemorrhages—unexplained by traumatic childbirth, a motor vehicle accident, a complex accidental fall (e.g., down stairs in an infant walker or in the arms of a falling adult), a fall from an elevated height (usually greater than 6 feet) or other equivalent accidental trauma
- Facial or scalp soft tissue injuries not overlying bony prominences, involving the external ears or associated with intraoral trauma or bleeding (e.g., torn frenulum or gingival laceration)
- Facial or scalp soft tissue injuries or skull fractures in a preambulatory infant, or
- Skull fractures that are multiple, complex, or diastatic —attributed to a short-distance fall (usually less than 6 feet).

THE RELEVANT FORENSIC ISSUES

In cases of suspected inflicted head trauma, the relevant forensic issues to be addressed by the child abuse medical consultant include the following:

- Have *nontraumatic* etiologies for the child's clinical, laboratory, and radiological findings been reasonably excluded?
- Does the caretaker's account or explanation of the child's head injury event adequately explain the child's clinical presentation and traumatic cranial injuries?
- What is the best estimate of the timing of the child's head injury event?

THE DIFFERENTIAL DIAGNOSIS

To identify inflicted injuries masquerading as accidental trauma, a high index of suspicion for inflicted pediatric head trauma is essential. To identify accidental injuries misinterpreted as abusive, and to identify nontraumatic etiologies for the child's clinical, laboratory, and/or radiological findings, *objectivity* is equally important. Birth trauma, accidental head trauma, and other nontraumatic explanations for intracranial bleeding and/or brain injury must be *actively* considered. These differential diagnostic considerations fall into the following general categories:

- Nonabusive trauma (e.g., forceps delivery, vacuum extraction, breech delivery, motor vehicle crash, a complex accidental fall, a fall from an elevated height)
- Congenital or metabolic condition or variation of normal (e.g., aneurysm, arteriovenous malformation, benign expansion of the extra-axial spaces, glutaric acidemia)
- Neoplastic diseases (e.g., brain tumor, acute leukemia)
- Bleeding diathesis (e.g., hemorrhagic disease of the newborn, hemophilia, idiopathic thrombocytopenic purpura, von Willebrand's disease)
- Acquired causes (e.g., meningitis, superior sagittal sinus thrombosis, obstructive hydrocephalus), and
- Connective tissue disease (e.g., osteogenesis imperfecta, Ehlers-Danlos syndrome)

For a more complete discussion of conditions confused with abusive head trauma, see Chapter 47, "Conditions Confused with Head Trauma."

INJURY MECHANISMS

Does the account or explanation of the child's head injury event adequately explain the child's clinical presentation and traumatic cranial injuries? To answer this question, a biomechanics-based approach is recommended.[48] Work backwards, from injury to history: (1) What are the child's primary cranial injuries? (2) What mechanisms of injury (i.e., contact and/or inertial) are required to explain each of these primary cranial injuries? and (3) Are these specific, required mechanisms of injury evident in the history provided by the caregiver? If not, child abuse must be considered.

Primary traumatic cranial injuries are those that occur at the moment of injury and result from mechanical distortions and strains. All primary traumatic cranial injuries derive from one of two mechanisms of injury: contact or inertial. A *contact* cranial injury results solely from skull deformation and requires cranial impact. Examples of contact cranial injuries include the following: (1) craniofacial bruising, abrasions, lacerations, or soft tissue swelling, (2) subgaleal hemorrhage or cephalohematoma, (3) skull fractures, and, (4) any associated epidural, subarachnoid, or subdural hemorrhage; brain contusions;or brain lacerations resulting solely from skull deformation. Contact cranial injuries can be viewed as those injuries that result from cranial impact if/when the head is fixed and prevented from accelerating (or decelerating) as a consequence of that impact.

An *inertial* cranial injury results solely from whole-head cranial acceleration or deceleration. Because the head is tethered to the body by the neck, cranial acceleration (or deceleration) is usually rotational rather than straight-line. Inertial cranial injuries can be induced in two ways: (1) impulsively, via forces transmitted through the neck (e.g., a whiplash injury), or (2) by cranial impact, if the impact causes cranial acceleration (or deceleration). Examples of inertial cranial injuries include the following: (1) acute concussion, (2) diffuse traumatic axonal injury, (3) nonfocal, acute subdural hematoma that results from tearing of one or more bridging veins, and (4) any associated subarachnoid

hemorrhage, brain contusions, or brain lacerations resulting solely from rotational cranial acceleration. Inertial mechanisms of cranial injury have been linked to worse outcomes.[50]

Because inertial cranial injuries can result from cranial impact, victims of inflicted pediatric head trauma can reveal a combination of contact and inertial primary injuries. However, some cranial impacts that produce clinically significant inertial cranial injuries do *not* produce visible, external, soft tissue injuries at the site of impact. In fatal cases of pediatric neurotrauma, scalp reflection at the time of autopsy might reveal evidence of cranial impact that did not produce visible, external, soft tissue injuries.[14] It follows that in living children with isolated inertial cranial injuries, impact can never be definitively excluded (or shaking confirmed) as the mechanism of that child's inertial cranial injuries. For a more complete discussion of head injury biomechanics, see Chapter 40, "Biomechanics of Head Trauma in Infants and Young Children."

TIMING OF INJURY

Child protective services and police investigators—not medical providers—investigate child abuse. On the other hand, when a medical provider can reasonably identify the *timing* of a victim's inflicted head injury event, the victims' caregiver at that time will appropriately become the subject of a child abuse investigation. Under ideal circumstances, this investigation will lead to court actions that protect an abused child from re-injury. For these reasons, accurate assessment of the timing of abusive head trauma is an essential skill of the child abuse medical specialist.

Serial cranial imaging studies that reveal an expected progression of posttraumatic findings (e.g., the evolution and resolution of brain or scalp swelling, the degradation of posttraumatic subdural collections, or the evolution of posttraumatic encephalopathic changes) facilitate only very broad estimates of the timing of a child's head injury event. The vast majority of young victims of moderate to severe (accidental or inflicted) pediatric neurotrauma become rapidly, clearly, and persistently ill.[49-51] It follows that precise documentation of the timing of onset of the victim's most severe and persistent clinical signs (of traumatic brain injury) facilitates an estimation of the timing of the child's head injury event. Stated more simply, to estimate the timing of head injury, answer this question: When did the child first become clearly and persistently ill with clinical signs linked to his or her traumatic cranial injuries?[49]

Severe *and delayed* acute clinical deterioration is possible after accidental or inflicted childhood neurotrauma.[50,51,53-60] The published literature regarding this phenomenon suggests that delayed clinical deterioration will occur if and when a child with focal intracranial injuries (e.g., epidural hematoma) experiences secondary systemic hypoxemia and/or ischemia, brain swelling, increased intracranial pressure, herniation, and/or brainstem compression. The vast majority of children who experience delayed and severe clinical deterioration after closed head trauma are not completely asymptomatic before their subsequent clinical deterioration, but manifest persistent or recurrent, less severe, nonspecific clinical signs of their initial head injury (e.g., recurrent vomiting, persistent irritability, prolonged loss of appetite, abnormal sleep patterns, seizures, accelerating head growth).

THE DIAGNOSTIC EVALUATION

In the absence of visible craniofacial soft tissue injuries, alteration or loss of consciousness, seizures, or cardiorespiratory compromise, abusive head trauma is frequently missed, misdiagnosed, or not even considered.[47] Diagnostic uncertainty increases if/when the caretaker of the head-injured child delays seeking medical care and/or denies any history of closed head trauma to explain the child's acute clinical signs. Victims of closed head trauma sometimes fail to reveal visible, external signs of their accidental or abusive cranial impacts.[14] Some infants with traumatic intracranial injuries will have a normal neurological examination.[61-67] Vigilance and a high index of suspicion are required. To *diagnose* inflicted head trauma, we must first *consider* the diagnosis.[47]

In a majority of cases, a diagnosis of abusive head trauma will be challenged, appropriately, by other medical providers, by family members, and/or later in a courtroom. To avoid over- and/or under-diagnosis of abusive head trauma, objectivity is essential. To ensure objectivity, a *consistently thorough* diagnostic evaluation is warranted. This evaluation must include a comprehensive history; meticulous physical examination; and specific laboratory testing, imaging studies, and/or consultations by medical specialists that facilitate *active* consideration of inflicted head trauma *and* alternate diagnoses. In some cases, and in some jurisdictions, you will find it necessary to order tests specifically to "rule out" an alternate diagnosis.

History

Obtain an independent *history of the present illness* from any and all potential caretakers, ideally in isolation from all other potential perpetrators of inflicted trauma (Table 39-1). How, when, and where did the child first became clearly and persistently ill? Document verbatim any and all statements made by caregiver(s) to explain the child's presenting clinical signs. Meticulously document the specific circumstances of any alleged, accidental, head injury event *and* the subsequent, acute clinical consequences of that event. If the infant or child suffered an accidental cranial impact but did not become clearly and persistently symptomatic thereafter, the injury event was likely inconsequential. Use direct quotations whenever possible. Document when and to whom the statements are made. A thorough medical history will allow you to ascertain the nature and the clinical severity of the child's injury or condition and may capture information that will prove to be forensically significant.

Obtain a *past medical history*, *family medical history* and a *review of systems* that are sufficiently comprehensive to allow you to reasonably exclude (or to elucidate) alternate potential explanations for the child's presenting clinical signs, laboratory, and/or radiological findings (Tables 39-2, 39-3, and 39-4). *Actively* seek information regarding alternative diagnoses, including congenital brain defects, variations, neoplasia, or acquired conditions; a bleeding diathesis; connective tissue disease; birth asphyxiation or trauma; and previous, clinically significant, traumatic, or hypoxic-ischemic brain injuries. Take a thorough *social history* to

Table 39-1 History of Present Illness

- When was the last time that your child was acting, eating, and interacting in a completely normal way?
- When did your child first become clearly and persistently ill or symptomatic?
- What was the very first thing that you noticed about your child that concerned you?
- What did you observe next?
- What other clinical signs appeared thereafter?
- How long did these clinical signs last?
- Did your child have an alteration or a loss of consciousness?
- Was he ever completely limp like a rag doll?
- Could you awaken him?
- Did you try to awaken him?
- What did you try to do to awaken him?
- Did he stop breathing?
- Did he have difficulty breathing?
- What were his respiratory efforts like?
- Did he turn blue?
- Did he stiffen his entire body?
- Did his eyes roll backwards?
- Did he have a seizure?
- Did his arms or legs jerk repeatedly?
- What did these arm or leg movements look like?
- Were his eyes or his gaze deviated to one side?
- Which adult(s) first observed your child's abnormal behaviors?
- When, where, and how did this adult discover or first observe these abnormal behaviors?
- What happened?
- Did your child fall?
- Who witnessed this fall?
- How far did he fall?
- Onto what kind of surface?
- Precisely how did your child land when he fell?
- What part of his body impacted first?
- What part of his body impacted next?
- How did he act immediately after the fall?
- How did he act over the next several minutes (and hours) after the fall?

identify past and current stressors and risk factors for abuse (Table 39-5). Supplement your medical history by reviewing the child's birth and outpatient medical records thoroughly (Table 39-6).

Physical Examination

Practitioners should follow these guidelines during physical examination:

Supplement your understanding of the child's acute clinical condition and course by reviewing his or her EMT and emergency department records. Then perform a meticulous initial physical examination. Carefully measure and plot growth parameters for potential evidence of acute or chronic malnutrition, macrocephaly (e.g., potentially related to a chronic subdural collection, posttraumatic hydrocephalus, or acute brain swelling) or microcephaly (e.g., potentially related to posttraumatic cerebral atrophy). Assess vital signs, skin color and perfusion, respirations, and overall responsiveness for evidence of diffuse or deep brain injury associated with respiratory insufficiency and/or compensated shock. Look for asymmetrical movements and functional deficits.

Visually inspect the child's entire body. Accidental bruising of the knees, shins, elbows, and forehead are common in cruising or ambulatory children, but very uncommon in infants who are not yet cruising.[60] Some bruises are easily overlooked (e.g., in the scalp or involving the external ears). A majority of pediatric victims of blunt abdominal trauma will not reveal abdominal wall bruising. Document, measure, and describe all traumatic skin injuries in the medical record.

Carefully palpate the head and scalp, extremities, and ribs for tenderness, swelling, induration, deformity or crepitus—signs that might indicate an underlying fracture. Palpate the sutures and fontanels for signs of increased intracranial pressure. Carefully inspect the external ears, tympanic membranes, nose, mouth, and hypopharynx. Inflicted traumatic injuries are common in these locations. Inspect the child's retina bilaterally in a darkened room. Be patient, because the examination requires time. Consider a short-acting mydriatic to improve retinal visualization. For a more complete discussion of eye injuries in child abuse, see Chapter 44, "Eye Injuries in Child Abuse."

Compare and contrast respiratory movements, effort, and breath sounds to help confirm or exclude clinically significant traumatic thoracic injuries. Examine the abdomen for signs of tenderness, rigidity, rebound, guarding, or loss of bowel sounds. Inspect the genitalia and anus for bleeding or other potential signs of penetrating trauma.

Perform a careful neurological examination. Objectively document the child's mental status, responsiveness, clinical injury severity, and coma scores. Compare your observations to similar assessments made at the scene of injury, during transport, and/or in the emergency department. Remember that intracranial injuries can be clinically subtle or silent.[69]

Re-examine the child serially. Some bruises evolve over time or appear only after treatment for shock. Serial physical examinations can reveal evolution of patterned bruises, delayed soft-tissue swelling, signs of an acute abdomen, bony deformities, evolving losses of function and/or evolving neurological deficits. Serial photo documentation of traumatic soft tissue injuries is strongly recommended.

Neuroimaging

In an infant or young child with *acute*, unexplained neurological signs or symptoms and suspected child abuse, obtain an initial, unenhanced cranial CT scan (with brain and bone window settings) and a standard radiographic (four-view) skull series. If the initial cranial CT scan is positive (or if the initial CT scan is negative but suspicion of child abuse remains high), obtain cranial MRI with T1- and T2-weighted conventional or fast spin echo imaging, different susceptibility-weighted sequences (such as gradient echo and diffusion-weighted), and FLAIR sequences. Consider magnetic resonance angiography (MRA) and venography (MRV) to help confirm or exclude vascular lesions, especially if/when parenchymal lesions are discovered.

In an infant or young child with *chronic* neurological signs and suspected child abuse, obtain cranial MRI with T1- and

Table 39-2 Past Medical History

- Did you (mother) experience complications during your pregnancy?
- What complications did you experience?
- Did you (mother) experience post-partum hemorrhage?
- Was your child born prematurely?
- How prematurely?
- Do you know *why* your child was born prematurely?
- How was your child delivered? Vaginally? By caesarean section?
- Did the doctor have to use forceps or a vacuum machine to help deliver your baby?
- Did your child have to be delivered urgently or emergently?
- Do you know *why* he had to be delivered urgently or emergently?
- During labor, did your child's heart rate drop too low prior to his/her birth?
- In the delivery room, immediately after his birth ...
  - ... did your child cry vigorously right away?
  - ... did his/her body become pink quickly?
  - ... did your child quickly begin to move his/her arms and legs actively?
  - ... did his/her body turn blue after his birth?
  - ... for how long did he/she look blue?
  - ... was he/she ever limp and floppy like a rag doll?
  - ... for how long was he/she limp and floppy like a rag doll?
  - ... did somehow have to help your child to start breathing?
  - ... what interventions were required to help your child start breathing?
  - ... did someone perform chest compressions?
- During his/her stay in the newborn nursery, did your child ...
  - ... ever have a seizure?
  - ... become lethargic, limp, or unresponsive?
  - ... receive treatment with intravenous antibiotics?
  - ... receive treatment with oxygen?
  - ... receive rescue breathing?
  - ... require a breathing tube?
  - ... require a ventilator?
  - ... require chest compressions?
  - ... undergo circumcision?
  - ... experience prolonged bleeding from his circumcision site?
- How long did your baby stay in the hospital after birth?
- Since your child's discharge from the newborn nursery, has he/she ever ...
  - ... been hospitalized?
  - ... had surgery?
  - ... had a broken bone?
  - ... had a seizure?
  - ... suffered a concussion?
  - ... become unconscious?
  - ... become completely limp like a rag doll?
  - ... become lethargic?
  - ... become completely unresponsive?
  - ... stopped breathing?
  - ... turned blue?
  - ... received treatment with oxygen?
  - ... required rescue breathing?
  - ... required a breathing tube?
  - ... required a ventilator?
  - ... required chest compressions?
  - ... been inconsolable?
  - ... experienced prolonged, recurrent vomiting?
  - ... required treatment for a head injury?
  - ... experienced frequent or prolonged nasal or oral bleeding?
  - ... manifested frequent or easy bruising, especially in "unusual" places?
  - ... passed blood in his urine or stools?
  - ... experienced prolonged, post-operative bleeding?
  - ... had oozing, prolonged bleeding, or hematoma formation at the site of immunizations?

Table 39-3 Family Medical History

Is there a family medical history of ...
- ... easy bruising?
- ... a blood clotting or bleeding disorder?
- ... menorrhagia?
- ... prolonged post-operative bleeding?
- ... severe or recurrent nosebleeds?
- ... excessive bleeding with dental procedures?
- ... mental retardation?
- ... cerebral palsy?
- ... seizures?
- ... developmental delays?
- ... hearing loss?
- ... blindness?
- ... frequent fractures?
- ... dental problems?
- ... brittle bone disease or osteogenesis imperfecta?
- ... any other connective tissue or bone disease?
- ... sudden, unexpected infant death or crib death?
- ... kidney disease?
- ... mental illness?
- ... any other genetic or hereditary condition affecting young children?

Table 39-4 Review of Systems

"Has your child ever ..."
- ... experienced unexplained weight loss?
- ... become very lethargic or very hard to wake up?
- ... become completely floppy like a rag doll?
- ... been completely unresponsive?
- ... had problems with vision or hearing?
- ... experienced bleeding from the nose, ears, or mouth?
- ... experienced shortness of breath?
- ... stopped breathing?
- ... turned blue or appeared dusky around the lips?
- ... experienced recurrent vomiting?
- ... demonstrated prolonged loss of appetite?
- ... had blood in the urine or stools?
- ... had unexplained bruises, abrasions, or cuts?
- ... had a seizure?
- ... seemed continually irritable or inconsolable?
- ... had very swollen lymph glands?
- ... had a fracture or broken bone?
- ... appeared to not be using one extremity like the one on the opposite side?

T2-weighted conventional or fast spin echo imaging, along with gradient echo and FLAIR sequences to exclude (or to confirm) older injuries. For a more complete discussion of imaging of abusive head trauma, see Chapter 42.

Laboratory Studies, Medical Consultations and Secondary Diagnostic Evaluations

Young victims of moderate or severe closed head trauma deserve broad laboratory screening and additional medical

Table 39-5 Social History

- What are the names and ages of the biological parents?
- How long have they known each other?
- Are they currently married to each other?
- If so, for how long?
- If not, were they ever married to each other?
- For how long?
- When did they separate or divorce?
- Why did they separate or divorce?
- Is either biological parent now remarried?
- Was the pregnancy for this child planned?
- Who now lives in the home with this child?
- Has this child (or any other child living in this home) ever been the subject of a previous report to child protective services?
- Which of the adults who live with this child provides primary care for this child?
- Do these adults also work outside the home?
- Have these adults had to deal with issues of poverty or unemployment?
- Domestic violence?
- Alcohol abuse?
- Drug abuse?
- Incarceration?
- Depression?
- Other mental health problems?
- What other adults also provide child care for this child?
- Does the child attend day care?
- How often?

Table 39-6 Review of Birth and Outpatient Records

- Do the child's birth records document ...
  - ... prolonged late decelerations?
  - ... cord around the neck?
  - ... a premature delivery?
  - ... a spontaneous vaginal delivery?
  - ... forceps extraction?
  - ... vacuum-assisted delivery?
  - ... neonatal asphyxiation?
  - ... neonatal resuscitation?
  - ... prolonged bleeding with circumcision?
  - ... vitamin K prophylaxis?
  - ... neonatal seizures?
  - ... neonatal lethargy?
  - ... neonatal sepsis?
- Do the child's outpatient medical records document ...
  - ... immunization status?
  - ... current medications?
  - ... current allergies?
  - ... normal growth and development?
  - ... developmental delays?
  - ... macrocephaly or microcephaly?
  - ... excessive bruising?
  - ... bruising before cruising?
  - ... bruising in unexpected places?
  - ... patterned bruising?
  - ... sensory deficits?
  - ... apparent life-threatening events?
  - ... prior fractures?
  - ... prior seizures?
  - ... other unexplained trauma?
  - ... failure to thrive?

consultations as required to help confirm or exclude occult trauma and its potential complications (e.g., hypovolemic shock, peritonitis associated with an occult ruptured hollow viscous, acquired consumptive coagulopathy). At a minimum, in cases of suspected physical abuse involving a child less than 2 years of age, the diagnostic evaluation should include the following: (1) CBC with platelet count, (2) coagulation screening tests, (3) serum chemistries, liver, and pancreatic function tests, (4) urinalysis, (5) pediatric ophthalmology evaluation, and (6) an initial radiographic skeletal survey. Definitive confirmation (or exclusion) of specific diagnoses may require serial laboratory testing, additional screening (or definitive) laboratory testing, additional medical specialty consultations, and/or ancillary radiographic studies (Table 39-7).

Table 39-7 Laboratory Studies, Medical Consultations, and Secondary Diagnostic Evaluations

- Obtain CBC with platelet count
- Obtain screening coagulation tests
- Obtain serum chemistry panel, liver and pancreatic function tests
- Obtain urinalysis
- Obtain an initial radiographic skeletal survey
- Obtain pediatric ophthalmology consultation
- In the presence of shock or possible internal traumatic injuries, obtain trauma services consultation
- In the presence of possible or probable seizures, obtain pediatric neurology consultation
- In a child with abnormal coagulation screening tests, an abnormal bleeding history, or an abnormal initial cranial imaging study:
  - Obtain initial and serial PT, PTT, CBC with platelet count, thrombin time, fibrinogen level, fibrin split products and D-dimer to exclude (or to confirm) an acquired consumptive coagulopathy (e.g., DIC)
  - Consider von Willebrand's screening tests
  - Consider pediatric hematology consultation
- In a child with an unexplained alteration in mental status:
  - Consider urine and serum toxicology screening
  - Consider pediatric neurology consultation
- In the presence of cranial imaging results compatible with glutaric academia (type 1), obtain serum amino acid and urine organic acid testing
- When connective tissue, metabolic, or nutritional bone disease is a diagnostic consideration:
  - Obtain serum calcium, phosphorus, and alkaline phosphatase
  - Consider testing for 1,25-OH vitamin D and its precursors
  - Consider skin biopsy or genetic testing to confirm (or to exclude) connective tissue disease
  - Consider metabolic disease consultation
- After stabilization and/or before hospital discharge:
  - Consider nuclear medicine bone scan
  - Consider physical therapy consultation
  - Consider audiological evaluation
  - Consider speech therapy consultation
  - Consider occupational therapy consultation
  - Plan a neurodevelopmental evaluation
  - Order a follow-up radiographic skeletal survey

DIAGNOSTIC OBJECTIVITY

To ensure objectivity, a *consistently thorough* diagnostic approach is essential. A comprehensive medical history, meticulous physical examination(s), and the appropriate use of laboratory tests, imaging studies, and medical specialty consultations—coupled with *active* consideration of alternate diagnoses—will frequently (although not inevitably) lead to a reasonable degree of medical certainty regarding the nature, extent, severity, mechanisms, and timing of pediatric neurotrauma. In many cases of suspected pediatric abusive head trauma, it is the discovery of associated *noncranial* traumatic injuries suspicious for abuse (e.g., patterned bruises, bruises in a pre-ambulatory infant, rib fractures) that provides the most compelling evidence that an infant's *cranial* injuries were also inflicted.

COMMUNICATION

Communicating your diagnostic impressions—clearly and effectively; orally and in writing; in a hospital room or in a courtroom; to parents, investigators, attorneys, judges, and jurors—can be extraordinarily challenging. Communication failures can lead to failures to protect children at risk. When testifying, keep it simple. Make eye contact when speaking. Maintain a professional demeanor. Remember that jurors tend to remember what they hear first and last. Experience helps.

STRENGTH OF THE MEDICAL EVIDENCE

Abusive head trauma is a well-recognized clinical entity. Our substantial (and growing) collective clinical experience—summarized in our published, peer-reviewed, medical literature regarding this subject—leads us to one incontrovertible conclusion: abusive actions directed toward an infant or young child can cause devastating traumatic intracranial injuries. This conclusion derives largely from Class 3 scientific evidence (i.e., retrospective case reports and clinical series). Higher (Class 1 or 2) scientific evidence is virtually nonexistent, since direct testing of hypotheses (e.g., is shaking dangerous?) is impossible in humans.

Current controversies regarding abusive head trauma derive almost exclusively from the inevitable requirement to interpret specific historical, clinical, laboratory, and/or radiological findings in a *forensic* context. Does the caregiver's account of the child's head injury event reasonably explain the child's clinical, physical, laboratory, and radiological findings? If not, why not? Have nontraumatic etiologies been reasonably excluded? Can we reasonably conclude that the child was abused?

Preliminary studies of our initial *forensic* impressions regarding hypothetical cases of pediatric head trauma reveal significant variations that require further analysis.[70] In many cases, reasonable doubt arises if/when a clinical, physical, laboratory, or radiological finding is considered in isolation, rather than within the context of the child's entire presentation. What is the best unifying explanation for the child's clinical, physical, laboratory, and radiological findings?

Prospective, multicentered, comparative studies of clinical presentations, mechanisms, injuries, and outcomes following accidental versus inflicted pediatric head trauma are limited in number. Meta-analyses of these studies are difficult, because researchers have used highly variable definitional criteria to categorize cases as accidental versus inflicted. Prospective, *long-term* outcome studies following abusive head trauma are nonexistent.

The practice recommendations that appear in this chapter (e.g., active consideration of alternate diagnoses) derive from our own past professional experiences or "lessons learned."

SUGGESTED DIRECTIONS FOR FUTURE RESEARCH

We need objective, widely accepted, definitional criteria for categorizing cases of pediatric head trauma as accidental versus inflicted. We need to use these definitional criteria uniformly in future, multicentered, prospective, comparative studies of clinical presentations, injuries, mechanisms, and outcomes following (accidental versus inflicted) pediatric head trauma. We need research studies that clarify how—and why—medical professionals reach specific *forensic* conclusions. Finally, and perhaps most urgently, because perpetrators rarely disclose their abusive actions and head-injured infants can have normal neurological examinations, we need rapid, noninvasive, sensitive, and specific screening tests that will facilitate detection of subtle, easily missed, traumatic brain injuries in infants and young children. For a more complete discussion of biochemical markers of head trauma in children, see Chapter 46.

CONCLUSION

Medical providers with child abuse expertise enter and exit the real life dramas that surround cases of abusive head trauma at two predictable times—shortly after a head-injured child is admitted to a hospital, and much later, in a courtroom. Both entrances can be highly emotionally charged. To function effectively, a child abuse medical specialist must listen carefully, remain objective, execute a thorough diagnostic evaluation, actively consider alternate potential explanations, and communicate results and forensic impressions clearly. If you can accomplish these tasks consistently, you will speak with conviction. More importantly, you will help to protect children from repetitive abusive trauma.

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Appendix W

Evidence-Based Medicine and Shaken Baby Syndrome

Part I: Literature Review, 1966–1998

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(*Am J Forensic Med Pathol* 2003;24: 239–242)

In recent years, there has been a clear move toward basing medical practice and opinions on the best available medical and scientific evidence. This process has been termed *evidence-based medicine* (EBM) and involves a review of the quality of evidence that is available in various diseases and fields of inquiry within medicine.

This is the first of 2 articles that attempts to formally rank the available medical scientific evidence by internationally accepted methods, to determine the degree of confidence that can be held on various claims about the condition termed *shaken baby syndrome* (SBS). Areas with good scientific evidence are identified, and shortcomings in the research and publications on the subject are addressed.

Approximately half of all indexed medical publications on the subjects of SBS and shaken-impact syndrome were published before 1999 and half since that time. Given that 1998/1999 is regarded as the turning point in acceptance of the tenets and practice of EBM, it seemed reasonable to assess the quality of evidence before 1999 and compare it with the quality of evidence on the same subject matter since that time. At the conclusion of Part II, the 2 periods are compared to determine the extent to which EBM has affected the field of SBS in terms of quality of available evidence.

The aim of this review is to be neutral on the subject of SBS. Neutrality is difficult to define in this field, in part because of the polarization of opinions on the highly emotional subject of infant injury and death and in part because of clear data deficiencies arising from difficulties in performing experiments. It is clearly unethical to intentionally shake infants to induce trauma, and there is an obvious problem with studies and reports that rely on either indirect or disputed evidence of the occurrence, severity, or type of trauma.

Many studies lacking these critical data make the obvious logical error of selecting cases by the presence of the very clinical findings and test results they seek to validate as diagnostic. Not surprisingly, such studies tend to find their own case selection criteria pathognomonic of SBS.

Neutrality in this review simply means that there is no selective quotation of the available literature, and literature is not chosen to support any particular view. The assessment is of the methods and quality of the actual research, and until this assessment is complete, the content, findings, and recommendations are irrelevant. At the end of the ranking, those studies that achieve the highest QER scores are reviewed for their content, findings, and recommendations. Their outcomes are collated, and the entire published data set is then reviewed as a whole to determine the summarized recommendations, noting areas of agreement, conflict, or controversy. From this, the problems with the published evidence are noted, and data gaps are identified. Recommendations can then be made according to the summarized data.

In assessment of the quality of the available scientific evidence, the author has taken an approach recently defined worldwide as an appropriate scale for review of quality of evidence. This approach has been described recently in context of setting Australian clinical guidelines.

Genuine hypothesis testing requires use of appropriate research methodologies, including collection of relevant control data, and suitable statistical analysis. The interpretation of individual study findings may be constrained by factors such as whether the cohort examined was adequately representative of the patient population in general. Replication across studies and in independent research centers is a key factor in the reliability of evidence.

Compelling evidence comes from consistent findings in 2 or more well-constructed, controlled trials or population-based epidemiologic studies (i.e., level I or level II evidence). By contrast, clinical practice guidelines with level IV evidence represent consensus statements of the expert panel according to clinical experience and limited scientific data. Although these statements may influence current practice,

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they are likely to be modified in response to further research findings. Data from a single case series without control subjects provide little more than a stimulus for subsequent hypothesis testing.

Quality of Evidence Ratings

I: Consistent evidence obtained from more than 2 independent, randomized, and controlled studies or from 2 independent, population-based epidemiologic studies. Studies included here are characterized by sufficient statistical power, rigorous methodologies, and inclusion of representative patient samples. Meta-analysis of smaller, well-characterized studies may support key findings.

II: Consistent evidence from 2 randomized controlled studies from independent centers, a single multicenter randomized controlled study, or a population-based epidemiologic study. Data included here have sufficient statistical power, rigorous methodologies, and the inclusion of representative patient samples.

III-1: Consistent evidence obtained from 2 or more well-designed and controlled studies performed by a single research group.

III-2: Consistent evidence obtained from more than 1 study but in which such studies have methodologic constraints, such as limited statistical power, or the inclusion of patient samples that may be nonrepresentative.

III-3: Evidence obtained from a single case study or a selected cohort study.

III-4: Conflicting evidence obtained from 2 or more well-designed and controlled studies.

IV: Consensus opinions of authorities according to clinical experience or descriptive reports.

SBS: Literature Review (1966–1998)

Overview and Methods

The entire Biomednet Medline database (http://www.biomednet.com/db/medline) was searched by using the search term *shaken baby syndrome* and Internet Explorer in late November 1998. Other articles identified that had not yet been indexed on MEDLINE but had been published were also included.

The entire set of retrieved articles was reviewed, and those in which SBS was only peripherally mentioned or in which SBS was unrelated to the original article were omitted. Letters and brief correspondence were also discarded, unless they added new information or data on SBS. Articles in non-English journals that lacked an English abstract were also generally excluded from assessment.

These exclusions reduced the initial list of 71 articles to 54, which were reviewed, categorized, and ranked according to the QER above. To these was added the important study by Jayawant et al. from *BMJ* of December 5, 1998. The editorial was omitted because it added nothing to the original article.

It was impossible to review the full original article in many cases, although all of the major articles were reviewed in full. The remainder was assessed for categorization by using the authors' abstracts.

Each article was assigned to 1 of 4 categories: (1) randomized controlled trial; (2) case series with or without controls (with date, series size recorded); (3) single case reports; and (4) other, including review articles, opinion pieces, and articles on social implications.

Results of Quality of Evidence Ratings

Fifty-four articles or abstracts were reviewed. One was a randomized controlled trial.[34] This trial was not relevant to the general topic of SBS because it assessed a diagnostic technique (electroretinograph) that proved unsuccessful in diagnosis. Twenty-six were case series.[1,2,7,9,11,13–15,18,19,26,28–30,33,36,39,42,45–50,52,53] Twenty-five were retrospective studies, and 1 was prospective.

In total, 307 SBS cases were claimed to have been assessed among the 23 articles in which numbers of SBS patients were provided, with a mean study size of 13 cases and a median of 7 cases per series.

Selection criteria for SBS cases were unstated in 12 articles, based on presumption or suspicion in 10, and confirmed in 4 by confession or conviction. Two studies had appropriate control groups, 3 had inappropriate control groups, and 21 were case series without control groups. Twelve studies were case reports:[3–5,8,10,17,22,24,31,32,51,55]

Retinal pathology in suspected SBS, 5 cases

Blunt head injury at autopsy, 1

Subdural hemorrhage (SDH) and retinal hemorrhage (RH) as a result of fall and chest compression, 1 case

Shaking causing traumatic aneurism, 1 case

Arteriovenous malformation, not SBS, as cause, 1 case

Intentional asphyxia and shaking of 15-week-old baby, 1 case

Magnetic resonance imaging value in diagnosis, 1 case

Raised intracranial pressure as cause of RH, 1 case

Fifteen were "other" articles[6,12,16,20,21,23,25,27,37,38,40,41,43,44,54]

Historical reviews of SBS,10 articles

Opinion papers without original material, 3 articles

Social issues and SBS, 1 article

Review of imaging in SBS (computed tomography versus magnetic resonance imaging), 1 article

RESULTS

The randomized controlled trial was unrelated in any aspect of interest in this review and addressed a method of assessment of the retinae that proved to be unsuccessful.

Of the case series, the flaws noted above are relevant. All but 1 was retrospective, and all but 5 had no control population to compare cases with. Three of those that did

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include controls chose insufficient or inappropriate controls (head impact trauma, without healthy controls or other illness unrelated to head injury). This shortcoming would normally have excluded these studies from the literature review (because they do not fulfill criteria for inclusion). Given the difficulties inherent in assessment of SBS and of identifying appropriate control groups, however, these articles have been included as QER III-2.

In studies with confirmed (i.e., admitted or observed) trauma and SBS, there were few common findings, apart from the presence of SDH accompanied by RH in 80% of examined cases. Some articles attempt to measure other risk factor, and the study by Jayawant et al.[42] is noteworthy in this respect.

Finally, the "other" articles do little but summarize opinions and summarize past data. Such articles do not add to the quality of understanding of the condition, nor are they necessarily accurate in what has become a rather emotionally charged area of research and polarized opinion.

In this article, the quality of evidence, rather than the predominance of findings, is being assessed. The issue of the evidence for SBS appears 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 evidence.

Data Gaps Identified

There exist major data gaps in the medical literature about SBS. There is a very obvious lack of clear definition of cases. For valid studies, some method of determining cases of actual proven shaking must be found, and appropriate control groups (trauma without shaking, other illness, healthy controls) must be defined and assessed blindly. This gold standard has yet to be achieved in even a single study in the field of SBS. There is a lack of useful and specific laboratory or other markers proven to identify SBS. There is poor definition and quantification of the social and family risk factors to provide guidance on likelihood of abuse for a given set of circumstances. Last, there is a strong need for a checklist or other diagnostic or management tool to assess cases and to quantify index of suspicion of shaking.

CONCLUSIONS

There was no evidence on the subject of SBS that exceeded QER III-2 by the end of 1998, which means that there was inadequate scientific evidence to come to a firm conclusion on most aspects of causation, diagnosis, treatment, or any other matters pertaining to SBS.

The majority of evidence achieved only a level of QER IV, opinions that shed no new light upon SBS and did not add to knowledge about SBS. Many of the authors repeated the logical flaw that if RH and SDH are nearly always seen in SBS, the presence of RH and SDH "prove" that a baby was shaken intentionally. Many other studies assumed that the presence of RH and SDH was sufficient to make the diagnosis of SBS in terms of case selection.

The remainder of articles are QER III-3, and as noted above, the inclusion of case series without controls would normally not occur. Thus, the data available in the medical literature by the end of 1998 were inadequate to support *any* standard case definitions, or *any* standards for diagnostic assessment.

Before 1999, there existed serious data gaps, flaws of logic, inconsistency of case definition, and a serious lack of tests capable of discriminating NAI cases from natural injuries. By 1999, there was an urgent need for properly controlled, prospective trials into SBS, using a variety of controls. Without published and replicated studies of that type, the commonly held opinion that the finding of SDH and RH in an infant was strong evidence of SBS was unsustainable, at least from the medical literature.

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Appendix CC

STATE OF WASHINGTON
SNOHOMISH COUNTY SUPERIOR COURT

STATE OF WASHINGTON, Plaintiff, v. MICHAEL MORRIS Defendant.	NO. 09-1-01071-9 TRIAL MEMORANDUM IN SUPPORT OF DEFENDANT'S MOTION TO DISMISS PURSUANT TO STATE V. GREEN

The Defendant Cannot be Convicted of Assault of a Child in the First Degree because the State has not provided sufficient evidence to link the Defendant to A.M.'s injuries.

Where no rational trier of fact could find the essential elements of the crime beyond a reasonable doubt, a trial court must dismiss. State v. Green, 94 Wn.2d 216, 616 P.2d 628 (1980).

In order to convict the Defendant of the charge of Assault of a Child in the First Degree, the State must prove that Mr. Morris intentionally assaulted A.M. Even accepting the State's evidence as true and construing it in the light most favorable to the State, there has not been sufficient evidence presented that, assuming someone intentionally harmed A.M., that person was Mr. Morris.

The evidence has shown that Mr. Morris was alone with A.M. for approximately 10 minutes. When Brittany Morris left the house, Mr. Morris was preparing to feed A.M. a bottle. Mrs. Morris testified that, within 10 minutes of leaving the house, she received a call from Cheralyn Orkiz informing her that Mr. Morris was on his way to the hospital with A.M. This was confirmed by the testimony of both Mrs. Orkiz and her husband, Cristian.

Although A.M. started to exhibit symptoms in Mr. Morris's care, the State has not presented

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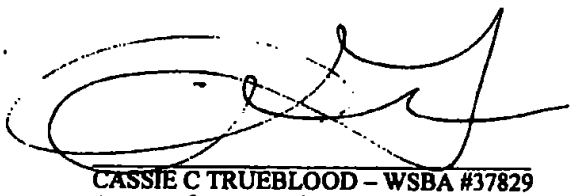
sufficient evidence that Mr. Morris intended to cause, or even *did* cause, the harm that occurred. Dr. Feldman testified that, although in his belief the symptoms caused by shaking are immediate in most cases, it is possible for infants to have a symptom-free "lucid interval" in which they seem normal (or near-normal) before symptoms present themselves. Upon cross-examination, Dr. Feldman recognized the validity of the article by Adamsbaum, which indicates that 16 of 29 admitted cases of child shaking were the product of repeated, violent shaking over a period of weeks or months. He also admitted the validity of the portion of the study which indicated that in the 13 cases of one-time shaking, immediate symptoms were only reported in four of the cases. This indicates immediate symptoms in four of 29 cases.

Dr. Feldman's exact testimony with regards to the possibility of lucid interval in the case of A.M. indicates that it is "highly unlikely" such a thing occurred. This doesn't rise to the level required to allow this case to proceed. Again the standard in Green is whether any reasonable trier of fact can find all the elements *beyond a reasonable doubt*. State v. Vladovic, 99 Wash.2d 413 (1983, *en banc*) (emphasis in the original). Even Dr. Feldman himself cannot definitively say that the harm occurred in the 10 minutes in which Mr. Morris was alone with A.M. Further, the article by Adamsbaum, which was propagated by Dr. Feldman himself, indicates that lucid intervals are not rare and are, in fact, present more often than not.

CONCLUSION

The State has not met its burden to allow this case to proceed to the jury and the charge must be dismissed.

Dated this 6th day of June, 2011.



CASSIE C TRUEBLOOD – WSBA #37829
Attorney for Accused

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FILED
2010 AUG 11 PM 3:30
SONYA KRASKI
COUNTY CLERK
SNOHOMISH CO. WASH

FILED
2010 AUG 11 PM 3:29
SONYA KRASKI
COUNTY CLERK
SNOHOMISH CO. WASH

CL14423643

ORIGINAL

IN THE SUPERIOR COURT OF THE STATE OF WASHINGTON

IN AND FOR THE COUNTY OF SNOHOMISH

STATE OF WASHINGTON Plaintiff, vs. MICHEAL J. MORRIS, Defendant.	NO. 09-1-01071-9 ORDER AUTHORIZING EXPENDITURE OF PUBLIC FUNDS

THIS MATTER having come on before the undersigned and finding that the expenses requested are necessary for the effective representation of the defendant in this case and are reasonable, NOW THEREFORE

IT IS HEREBY ORDERED that the court authorizes payment to reimburse the Snohomish County Public Defender Association for fees paid to Harborview Medical Center, for services related to providing medical records on file with Harborview Medical Center, in an amount not to exceed **$210.89.**

IT IS FURTHER ORDERED that these expenditures will be paid upon presentation of proper invoice (attached).

SNOHOMISH COUNTY PUBLIC DEFENDER
1721 HEWITT AVENUE - SUITE 200
EVERETT, WASHINGTON 98201
(425) 339-6300

DONE IN OPEN COURT this 12th day of Feb, 2010

JUDGE/COURT ADMINISTRATOR

Presented by:

CASSIE C. LITTLE - WSBA #37829
Attorney for Defendant

SNOHOMISH COUNTY PUBLIC DEFENDERS
1721 HEWITT AVENUE - SUITE 200
EVERETT, WASHINGTON 98201
(425) 339-6300

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2010 AUG 11 PM 3:20

SONYA KRASKI
COUNTY CLERK
SNOHOMISH CO. WASH

CL14423642

ORIGINAL

SUPERIOR COURT

2010 FEB 10 AM 9:37

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IN THE SUPERIOR COURT OF THE STATE OF WASHINGTON

IN AND FOR THE COUNTY OF SNOHOMISH

STATE OF WASHINGTON Plaintiff, vs. MICHAEL J. MORRIS, Defendant,	NO. 09-1-01071-9 MOTION AND DECLARATION FOR ORDER AUTHORIZING EXPENDITURE OF PUBLIC FUNDS

MOTION

COMES NOW the Defendant, by and through his attorney, and moves this Court for an order authorizing expenditure of public funds in the amount of **$210.89** to obtain copies of medical records on file with Harborview Medical Center This motion is supported by the attached declaration of counsel.

DATED this 10th day of February, 2010

Respectfully submitted,

CASSIE C. LITTLE - WSBA# 37829
Attorney for Defendant

SNOHOMISH COUNTY PUBLIC DEFENDERS
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(425) 339-6300

48

DECLARATION

I, CASSIE C. LITTLE, am the attorney for the above-named defendant and make the following declaration in support of the above motion:

1) I represent that the services stated below are necessary in presenting an adequate defense of the defendant in this case.

2) In order to adequately represent Mr. Morris, it is necessary to obtain copies of medical records on file with Harborview Medical Center regarding Alexis Morris (DOB: 04-15-09).

3) The defendant is financially unable to obtain these services.

4) I do not request the Court to hear further support of this motion on the record. If the Court determines that a further hearing is necessary, this hearing should be in-camera.

5) I request the limit of authority be fixed by the Court in an amount not to exceed **$210.89**.

I declare under penalty of perjury under the laws of the State of Washington that the above is true and correct.

SIGNED in Everett, Washington, this 10th day of February, 2010.

CASSIE C. LITTLE

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2009 OCT 28 PM 3:24

SONYA KRASKI
COUNTY CLERK
SNOHOMISH CO. WASH

ORIGINAL

IN THE SUPERIOR COURT OF THE STATE OF WASHINGTON

IN AND FOR THE COUNTY OF SNOHOMISH

STATE OF WASHINGTON Plaintiff, vs. MICHAEL J. MORRIS, Defendant.	NO. 09-1-01071-9 ORDER AUTHORIZING EXPENDITURE OF PUBLIC FUNDS

THIS MATTER having come on before the undersigned and finding that the expenses requested are necessary for the effective representation of the defendant in this case and are reasonable, NOW THEREFORE

IT IS HEREBY ORDERED that the court authorizes payment to reimburse the Snohomish County Public Defender Association for fees paid to IOD, Inc., for services related to providing medical records on file with Seattle Children's Hospital, in an amount not to exceed **$545.16.**

IT IS FURTHER ORDERED that these expenditures will be paid upon presentation of proper invoice (attached).

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DONE IN OPEN COURT this 22nd day of September, 2017.

JUDGE/COURT ADMINISTRATOR

Presented by:

HALEY DEBELL - WSBA #35946
Attorney for Defendant

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FILED

2009 SEP 11 PM 3:44 SNOHOMISH COUNTY SUPERIOR COURT

2009 OCT 28 PM 3:24 SONYA KRASKI COUNTY CLERK SNOHOMISH CO. WASH

ORIGINAL

IN THE SUPERIOR COURT OF THE STATE OF WASHINGTON

IN AND FOR THE COUNTY OF SNOHOMISH

STATE OF WASHINGTON Plaintiff, vs. MICHAEL J. MORRIS, Defendant,	NO. 09-1-01071-9 MOTION AND DECLARATION FOR ORDER AUTHORIZING EXPENDITURE OF PUBLIC FUNDS

MOTION

COMES NOW the Defendant, by and through his attorney, and moves this Court for an order authorizing expenditure of public funds in the amount of **$545.16** to obtain copies of medical records on file with Seattle Children's Hospital. This motion is supported by the attached declaration of counsel.

DATED this 11 day of September, 2009

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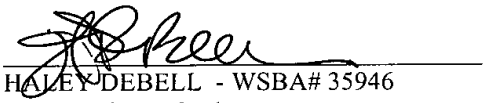
22

Respectfully submitted,



HALEY DEBELL - WSBA# 35946
Attorney for Defendant

DECLARATION

I, HALEY DEBELL, am the attorney for the above-named defendant and make the following declaration in support of the above motion:

1) I represent that the services stated below are necessary in presenting an adequate defense of the defendant in this case.

2) In order to adequately represent Mr. Morris, it is necessary to obtain copies of medical records on file for A.M., (DOB: 04/15/09) with Seattle Children's Hospital.

3) The defendant is financially unable to obtain these services.

4) I do not request the Court to hear further support of this motion on the record. If the Court determines that a further hearing is necessary, this hearing should be in-camera.

5) I request the limit of authority be fixed by the Court in an amount not to exceed **$545.16**.

I declare under penalty of perjury under the laws of the State of Washington that the above is true and correct.

SIGNED in Everett, Washington, this 11 day of September, 2009.

HALEY DEBELL

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