IN THE CIRCUIT COURT, SEVENTH JUDICIAL CIRCUIT, IN AND FOR FLAGLER COUNTY, FLORIDA

CASE NO.: 18-00169-CFFA

STATE OF FLORIDA

V.

DEVIAUN ANTRIEL TOLER/

NOTICE OF FILING ARTICLES FOR DAUBERT HEARING

YOU ARE NOTIFIED that the Office of the State Attorney, by and through the undersigned Assistant State Attorney has on this date filed with the above Court the following articles:

1. "The New Science of Abusive Head Trauma," Daniel Lindberg, et. al, *International Journal on Child Maltreatment Research, Policy, and Practice*, July 2019.
2. "Acceptance of Shaken Baby Syndrome and Abusive Head Trauma as Medical Diagnosis," Sandeep Narang, M.D., *The Journal of Pediatrics*, July 2016.
3. "Biomechanical Response of the Infant Head to Shaking- An Experimental Investigation," Carole Jenny, M.D., *Journal of Neurotrauma*, 2016.

CERTIFICATE OF SERVICE

I HEREBY CERTIFY that a true and correct copy of the foregoing has been furnished via U.S. Mail to JOHN S HAGER, HAGER AND SCHWARTZ P.A., 140 SOUTH BEACH STREET SUITE 310, DAYTONA BEACH, FL 32114, on July 8, 2021.

Respectfully submitted:

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ORIGINAL PAPER

The "New Science" of Abusive Head Trauma

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Abstract

Claims that new science is changing accepted medical opinion about abusive head injury have been made frequently in the media, legal publications, and in legal cases involving abusive head trauma (AHT). This review analyzes recently published scientific articles about AHT to determine whether this new information has led to significant changes in the understanding, evaluation, and management of children with suspected AHT. Several specific topics are examined as follows: serious or fatal injuries from short falls, specificity of subdural hematoma for severe trauma, biomechanical explanations for findings, the specificity of retinal hemorrhages, the possibility of cerebral sinus thrombosis presenting with signs similar to AHT, and whether vaccines can produce such findings. We conclude (a) that the overwhelming weight of recent data does not change the fundamental consensus, (b) that abusive head trauma is a significant source of morbidity and mortality in children, (c) that subdural hematomas and severe retinal hemorrhages are commonly the results of severe trauma, (d) that these injuries should prompt an evaluation for abuse when identified in young children without a history of such severe trauma, and (e) that short falls, cerebral sinus thrombosis, and vaccinations are not plausible explanations for findings that raise concern for abusive head trauma.

Keywords Abusive head trauma · Retinal hemorrhages · Subdural hematoma

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> New scientific research has cast doubt on the forensic significance of this triad, thereby undermining the foundations of thousands of ... convictions. (Tuerkheimer 2009)

New scientific evidence that changes the generally accepted understanding of the medical community may justify an appeal in criminal cases where medical evidence was important in the court's conclusion. Recent claims that new science has changed the mainstream understanding of the forces and mechanisms of brain injuries in children, such as the quote above, are made in the popular media, legal articles, and criminal proceedings ("Cavazos 2011; Cenziper 2015; "Del Prete 2014; "State of Wisconsin v and Edmunds 2008; Tuerkheimer 2009, 2010). Because the medical diagnosis of abusive head trauma (AHT) also implies that a crime has occurred, scientific progress that substantially changes the way that AHT is understood or diagnosed can have profound effects for people accused or convicted of child physical abuse. However, in other areas relevant to child physical abuse, scientifically unsupported hypotheses such as *temporary brittle bone disease* or *infantile rickets* have also been submitted as new science, only to be widely discredited (Keller and Barnes 2008; Mendelson 2005; Paterson and Monk 2011; Slovis et al. 2012; Spivack and Otterman 2010; Sprigg 2011; Strouse 2013).

The objective of this review is to examine the concepts frequently submitted as "new science" in medical-legal cases of alleged abusive head trauma. The authors chose the topics based on their experience in legal settings. These include serious or fatal injuries from short falls, specificity of subdural hematoma for severe trauma, biomechanical explanations for findings, the specificity of retinal hemorrhages, the possibility of cerebral sinus thrombosis presenting with signs similar to AHT, and whether vaccines can produce such findings. With the exception of one author (RMR), who retired in 2010 after practicing child abuse pediatrics for more than 40 years, all authors are practicing child abuse physicians who have testified in US courts in cases of alleged AHT.

Serious or Fatal Injuries from Short falls

Case Presentation

A 4-month-old boy reportedly rolled off his parents' bed and was found crying on a hardwood floor. He was inconsolable, refused his bottle, and vomited. His parents called 911, and emergency medical personnel recorded that he had a seizure *en route* to the hospital. On arrival, he was comatose and swelling was noted on his scalp in the right parietal region. A head CT scan revealed a thin, bilateral subdural hematoma (SDH) with loss of gray-white differentiation. The retinal exam showed bilateral extensive retinal hemorrhages (RHs). The skeletal survey was negative. The patient died 3 days later. An autopsy confirmed the radiographic findings, and no other injuries were identified.

Analysis

The core question is whether the history of a short fall represents a plausible explanation for the injuries. Falls are the initial explanation in up to 70% of cases where children are ultimately diagnosed with abusive injuries (Duhaime et al. 1992; Leventhal et al. 1993; Scherl et al. 2000; Strait et al. 1995).

Three publications have suggested that short, accidental falls can be life-threatening or fatal. First, Plunkett and others (Plunkett 2001) examined records from the United States Consumer Product Safety Commission National Injury Information Clearinghouse to identify all head and neck injuries involving playground equipment from January 1988 through June 1999. This study identified 18 cases where fall-related head injuries led to fatalities, with 5 of them occurring at home. Cases included children aged 12 months–13 years and the height of falls (measured from the lowest part of the body to the ground) was 0.6–3 m (2–10 ft). The authors reported that 12 falls were witnessed by someone other than the caretaker, and 12 had a "lucid interval" (i.e., a period where the child appeared mostly well). Among six children with the retinal examination, 4 were reported to have had bilateral RHs (the nature of which was not described).

The authors noted several limitations to their study: six falls were not independently observed, eight falls did not have a reported height, and other included falls were from significant heights—at least 5 falls were from 5 ft or more, and the highest fall was from 10 ft. The study has also been criticized because none of the subjects was less than 12 months old, and only nine were less than 5 years old. None had formal retinal examinations, and only 1 of the 5 children less than 2 years of age had a witnessed fall.

The second commonly cited article (Hall, Reyes, Horvat, Meller, & Stein, 1989) was a retrospective study of pediatric deaths from the Cook County Medical Examiner's office over a 4-year period (1983–1986). Fatalities in 18 children with falls less than 3 ft and 18 children with falls between 3 ft and five stories were described, including one 8-month-old child who was dead on arrival after a reported fall from a couch to a hardwood floor, and who was found to have a large SDH.

There are several limitations to this study. The brief "Methods" section reveals that not all subjects had a full autopsy, that medical records were not available to the authors, and that radiographs were not used to evaluate for fractures. Further, because only the mean ages of subjects were given, it is not possible to know how many children were infants, and few details were provided about the reported mechanisms of injury.

Finally, a case report (Gardner 2007) involves an 11 month old who was reported to have fallen backwards from a sitting position to a carpeted floor. The child's 5-year-old brother reportedly witnessed this event. The infant was found to have a SDH requiring surgery and diffuse multi-layer RHs. A skeletal survey was negative. Treating physicians were divided about whether the injury could have been the result of the history provided. The author concluded that "if RHs can [ever] occur without shaking ... , they are of no value in determining etiology." (p. 435) The clear concern with this article is whether the account attributed to the young sibling was accurate. Some skepticism seems warranted. The fall described is particularly minor. If such falls resulted in serious injuries, one would expect this to be a rather common occurrence. It is not.

Although these publications are often offered as "new science," more recent data contradict these results. Chadwick and colleagues published data from 317 children

who presented after a reported fall to a single trauma center in San Diego (Chadwick et al. 1991). Among 65 children who fell from 5 to 9 ft, and 118 children who fell from 10 to 45 ft, there were zero and one fatality, respectively. However, among 100 children with falls reported from less than 4 ft, there were seven deaths. The child who died from the high fall died of sepsis after a prolonged hospital course. Conversely, the seven short fall deaths died of head injury and all had associated injuries or concern about the accuracy of the history provided. If death is a rare outcome from a "high" fall, it seems quite unlikely that short falls would be fatal. The authors concluded that deaths reported as the result of falls less than 4 ft raise the suspicion that the history is inaccurate.

In 2008, Chadwick and colleagues performed a systematic review to determine the risk of death resulting from falls less than 1.5 m (approximately 5 ft) in children younger than 5 years old. This review examined data from five book chapters, seven literature reviews, three public injury databases, and 177 peer-reviewed articles categorized according to sample characteristics and methodology. This included a review of California's statewide injury database, which reported a maximum of 13 short fall deaths among 2.5 million California children over a 5-year period. In this population, six fatalities were identified that might have been the result of short falls. Five articles described reliably witnessed falls in 560 children with hospital falls; none died. Twenty-five studies of children in large licensed daycares had only two fatalities attributed to falls in this setting. After data from all the above data sources, the authors concluded that the risk of death from a short fall is less than 1 in 2,000,000 per year.

Although science cannot prove that something is "impossible," these data suggest that death or serious injury in a young child from a short fall is *exceedingly* rare and that all such deaths should be carefully investigated with a detailed history, physical examination, radiographic and laboratory studies, and a full autopsy.

The Specificity of SDH

Proponents of "new science" use a straw man to argue that new science is changing mainstream opinion. This straw theory—"triad theory"—asserts that clinicians simplistically diagnose abuse in any case that has SDH, any RHs, and any cerebral edema. While this straw theory is ridiculous on its face (abuse is not raised when these findings are seen in children who present after being hit by a car, or with a known, severe crush injury), the underlying argument deserves to be addressed. Most doctors view most significant SDHs to be the result of significant trauma. New science advocates suggest that SDHs are commonly the result of minor trauma or other non-traumatic causes that doctors fail to consider.

Many theories have been postulated as to how SDHs can happen without trauma, and it is beyond the scope of this review to address all of these. While Ehlers-Danlos syndrome, dysphagic choking, and osteogenesis imperfecta (among others) are occasionally put forward as explanations for SDHs, the most common arguments are rooted in three concepts: (a) that children commonly have asymptomatic SDHs resulting from birth, (b) that SDHs can occur spontaneously in children with enlarged extra-axial

spaces, and (c) that sudden and catastrophic clinical decline can result from bleeding into an existing asymptomatic SDH.

Although some of these concepts are supported by the literature, ultimately, the conclusion that significant SDHs are commonly atraumatic is unsupported. To understand why, it is important to ask two questions. First, *can* SDH occur with little or no trauma? Second, when it does, what are the symptoms and prognosis?

SDH from Birth

With the evolution of fast magnetic resonance (MR) imaging techniques that image the infant brain without radiation or sedation, two groups of investigators have identified asymptomatic SDHs in a significant proportion of neonates. Looney et al. (2007) performed an MRI on 96 asymptomatic neonates. They identified SDH in 16 cases including two children who also had sub-arachnoid hemorrhages and five with intra-parenchymal hemorrhages. All affected children were born by vaginal deliveries and all hemorrhages were infra-tentorial or low in the occipital lobe.

A second group had similar results published 1 year later (Rooks et al. 2008). Of 101 asymptomatic neonates, 46 were found to have SDH on MRI. All were supra-tentorial and posterior, and < 3 mm in width. In contrast to Looney et al. (2007), this group did not identify other intracranial injury types. These authors also performed serial imaging to assess the natural history of these SDHs and showed that 94% had resolved by 1 month, and 100% resolved within 3 months. At 2-year follow-up examinations, six children (14%) had a speech delay and one was being evaluated for an autism spectrum disorder, but none had deficits clearly related to trauma. None had a sudden collapse.

Enlarged Extra-Axial Spaces

It has been well-described that the extra-axial space enlarges in older adults as the brain shrinks relative to the skull. That minor trauma can rarely cause SDH in elderly people led some to suggest that this could also be true in children with enlarged subdural spaces. If tearing of the bridging veins is the source of traumatic SDH, the theory goes; the widened extra-axial spaces may increase the risk for bridging veins to stretch and break, causing SDH.

Two articles have addressed this theory in children who had an MRI for macrocrania. The first, by McKeag et al. (2013), reviewed records from 177 children < 2 years old from the Children's Hospital of Philadelphia, where MRI for macrocrania showed enlarged extra-axial spaces. Of these, four (2.3%) had SDH. Each of these had a thorough evaluation for abuse. This was negative in three of the children. One was thought to have been abused based on the finding of multiple rib fractures in different stages of healing.

The second article, by Greiner et al. 2013a, identified 108 children less than 2 years old with enlarged extra-axial spaces among 168 children imaged for macrocrania at the Cincinnati Children's Hospital. Six children (3.6%) had SDH, all asymptomatic. Only two children with SDH were evaluated for abuse, and 1 was found to have characteristic RHs and was diagnosed as abuse.

An article from investigators in France (Vinchon et al. 2004), who prospectively collected data for several years in cases of pediatric SDH, found 16 cases in which they concluded that SDH had occurred without trauma. Twelve of these children had macrocrania, and others had illnesses such as severe dehydration. These authors conclude that "spontaneous" SDH does exist but it is rare and cannot be diagnosed without thorough evaluation for AHT. They state that the absence of traumatic features is insufficient to diagnose spontaneous SDH and conditions like macrocrania, severe dehydration, or arachnoidomegaly are necessary for the diagnosis.

The answer to the question "Is there new evidence that suggests that SDH can occur from birth, or without major trauma?" is "Yes." But this simple conclusion is not the end of the discussion. It bears emphasis that each child with a SDH in these series was asymptomatic: none presented in a coma, and no child was thought to have non-abusive SDH when other injuries or RHs were present. Each article cited contains statements emphasizing the need to consider AHT in cases with unexplained SDH. Small, asymptomatic SDHs in the absence of traumatic injuries are not the focus of most abuse investigations. In most cases of AHT, the child has sudden, obvious symptoms of traumatic brain injury or other signs of trauma. To change the accepted opinion of AHT, the onus is on "new science" proponents to show how asymptomatic SDHs can rapidly evolve, leading to sudden neurological collapse.

Re-Bleeding into SDH

Evidence of re-bleeding in adults and children has been known for decades and is based on follow-up imaging showing enlarged SDH or new hyperdense blood products in an existing or old SDH. Although recent studies disproved the dogma that CT is able to determine the age of a SDH by density (Bradford et al. 2013; Tung et al. 2006; Vinchon et al. 2004), it is accepted that re-bleeding can occur in a SDH without significant additional trauma. Whether this is because bridging veins are stretched or whether vascularized neomembranes bleed (or both) is currently unknown, but does not affect the understanding of whether re-bleeding is likely to produce the findings of AHT.

The key question is whether re-bleeding causes symptoms likely to be mistaken for AHT. There is less direct evidence to address this question. One article describes two cases where re-bleeding is suggested to have caused symptom progression (Hymel et al. 2002). In one case, a 20-month-old child with a known, 3-month-old SDH presented with 1 week of fussiness after hitting a windowsill. In the next, an 11 months old presented with vomiting and was found to have a SDH. Later that night, increased swelling and a more hyperdense SDH was seen on CT. These cases do not directly address the question at hand. In the first case, it is not clear that the child had increased symptoms, and in the second, it is not clear that the mildly increased symptoms were not the result of the simple progression of the acute SDH. In neither case did the child have sudden severe symptoms after being asymptomatic.

Although there are no new data that directly address the question of whether minor trauma into a known SDH results in re-bleeding and sudden collapse, this concept is implausible, given the natural history of known SDHs. While SDHs commonly resolve over weeks to months, children with known asymptomatic or mildly symptomatic SDH are discharged from the hospital after a few days. They are discharged without

protective helmets or other devices to prevent the minor trauma that is ubiquitous in the life of normal children. Yet children have not been reported to return with sudden, devastating symptoms after being discharged. Bradford et al. reported that, of 105 children with SDH, 17 (16%) were found to have re-bleeding, but none had symptoms (Bradford et al. 2013). The Pediatric Emergency Care Applied Research Network (PECARN) (Kuppermann et al. 2009) examined more than 10,000 children less than years old who presented to emergency departments for evaluation of traumatic brain injury. Robust follow-up procedures were employed to detect missed traumatic brain injuries. Results showed that asymptomatic children without loss of consciousness or severe mechanism of injury have < 0.02% chance of clinically important brain injury—including the potential for that brain injury to be identified days or weeks after presentation. In short, sudden symptoms as a result of re-bleeding into a SDH is not a known phenomenon among children with demonstrated SDH—it is only proposed for children where the original SDH is itself a theoretical entity.

Although there is new science about pediatric brain injury, it does not change the accepted understanding of AHT. In sum:

1. Small, asymptomatic SDH can occur from normal birth;
2. Small, asymptomatic, non-abusive SDH rarely occur in children with enlarged subarachnoid spaces:
3. CT imaging does not reliably estimate the age of SDH based on density.

Nevertheless, the vast majority of symptomatic SDH are the result of significant trauma; sudden, significant neurological symptoms in the setting of SDH imply a recent, significant traumatic event.

Biomechanics

It is sometimes suggested that biomechanical data offer a more pure and scientific approach than studies of human beings. Some proponents of "new science" have suggested that biomechanical evidence should change our understanding of the forces that are necessary or capable of causing traumatic brain injury.

In one of the first studies looking at the nature of the forces involved when a child is shaken, Duhaime et al. (1987) used two arguments to say that serious or fatal AHT injuries require more force than shaking alone. In the first phase, they carefully examined 13 consecutive dead infants diagnosed with the shaken baby syndrome (SBS) and found some evidence of head impact as well. They concluded that all cases of SBS must have some component of impact. In the second part, they constructed a doll model and measured peak accelerations. They first shook the doll (no impact) and compared peak accelerations to a trial in which they shook the doll and also allowed it to hit a bar (impact). Unsurprisingly, the sudden stop of an impact created a larger peak acceleration (deceleration). When the values were compared against monkey data and extrapolated, the authors concluded that shaking alone could not yield sufficient forces to cause serious brain injury.

Alexander et al. (1990) looked prospectively at 24 cases of diagnosed SBS. In addition, they were carefully examined for signs of trauma, often with MRI. Nine of the children died. There was not even marginal evidence of impact trauma in 12 of the cases, including five of the children who died. Shaking with impact (12 cases) and shaking without impact (12 cases) were both found. If impacts were significant in these AHT cases, as hypothesized by Duhaime et al. (1987), then all of the dead children should have shown impact. This was not found. Following this study, there has been no large study of children with AHT that has found all children to have signs of impact. Typically, about 50–70% of children have impact evidence along with shaking. Thus, the Duhaime hypothesis has been disproved. Shaking alone can cause serious and fatal injuries (Starling et al. 2004).

Duhaime et al.'s doll experiment, like others, have also been criticized for comparing only a single shake to a single impact—concluding that a single shake alone could not cause the injuries seen in AHT. However, perpetrators who confess to shaking have reported multiple shakes within seconds (Adamsbaum et al. 2010; Starling et al. 2004). The cumulative effect of these repetitive shakes was not modeled and would seem to be greater than that of a single shake.

Using a pig model, Raghupathi et al. (2004) shook 3–5-day-old anesthetized piglets once or twice with a 15-min gap. The mechanical shakes were rapid (< 15 m/sec) and did not involve impact. At 6 h post-injury, the brains were examined. More injury was seen with the double shake trial, showing that immature brains are vulnerable to repeated, relatively mild, non-impact loading conditions.

Bandak (2005) compared calculated structural failure limits of the cervical spine and deduced that the spine should be injured with less force than the brain. Clinically, this phenomenon is not seen; hence, the mechanism of shaking should be re-evaluated.

One response by Margulies and colleagues (Margulies et al. 2006) noted that Bandak made multiple numerical errors in his analysis, often off by a factor of 10. "There is no single, simple explanation responsible for the errors that appear in *every* value in Table 3." (Margulies et al. 2006, p. 278). When accurately calculated, they argued that the brain can indeed be injured without neck injury—even using Bandak's (2005) suppositions. They concluded that neck injury could occur during severe shaking without impact but that it would not necessarily occur if the shaking caused brain injury. Rangarajan and Shams (2006) also pointed out these numerical miscalculations and unclear assumptions. They noted that Bandak referenced three presentations at a conference in which the chair of the workshops said the material should not be used as references.

Using a mannequin, Wolfson et al. (2005) showed that concussion levels of energy could be achieved and raised concern about using models of single trauma to explain a phenomenon (shaking) that is often repetitive. "It is unlikely that further gross biomechanical investigation of the syndrome will be able to significantly contribute to the understanding of SBS." (p. 70) "Current injury criteria are based on high-energy, single-impact studies. Since this is not the type of loading in SBS it is suggested that their application here is inappropriate and that future studies should focus on injury mechanism in low-energy cyclic loading." (p. 70)

Biomechanical arguments asserted as part of the "new science" may also rest on extreme assumptions that do not seem to fit what we know of the natural world:

1. Heads are often treated as a single mathematical point. With 80–100 million neurons, layers of the brain of different densities, and a non-spherical shape, the human brain is over simplified when reduced to a simple point or shape.
2. Forces are complex. Repetitive shaking can result in translational movement in 3 dimensions, as well as spinning and shear forces, simultaneously. It is not clear that a measurement such as "peak acceleration" is the key factor to consider versus any number of forces acting over time.
3. Reductionist thinking often begins by quoting Newton's second law: force equals mass times acceleration. This basic concept considers only simple, solid shapes (instead of complex biological tissue), and non-complex forces (straight back and forth) and does not account for repetitive forces. A common history in cases of AHT is that the child fell off the sofa. Compare that to an adult falling off a sofa. Acceleration = gravity which is a constant on earth. The equation then reduces to force is proportional to mass. For a tumble off a sofa, this means the bigger the force the harder the fall. One would expect an adult with a far greater mass to hit the floor far harder. Yet no one claims sofa falls create adult fatalities, and suicidal adults would never choose a couch in favor of bridges or high heights.

Biomechanical modeling attempts to simplify and understand forces and consequences in nature. Such modeling can be useful but is limited as to whether it faithfully represents the real world. The "new science" proponents selectively cite some biomechanical hypotheses as representing what happens rather than adjusting those models to what is actually observed. At most, biomechanics is a long way from explaining the injuries seen with shaking and shaking with impact.

Retinal Hemorrhages

Just as SDH have been suggested to be non-specific for severe trauma, so too have RHs. Proponents of "new science" assert that RHs can be caused by a wide array of medical and traumatic injuries. Although this fact has been known for decades, it ignores the types and patterns of RH and their significance in determining their origin.

The contemporary understanding of RH is summarized in an article comparing the prevalence and types of RHs in victims of abuse and those with non-abusive traumatic brain injury. Bechtel et al. (2004) examined 15 children with AHT, and 67 with non-abusive brain injuries. Although RHs were present in nine (60%) abused children, they were only found in seven (10%) non-abused subjects. Furthermore, none of the non-abused cohort had RHs that extended to the periphery of the retina. These data suggest that while RHs can occur from non-abusive trauma, these RHs are mild. Mainstream opinion has therefore held that RHs that are multi-layered, numerous, and extend to the periphery of the retina are specific for abuse (Levin 2010).

The "new science" addressing RHs began with a case report of a child who was found to have severe RHs and perimacular folds after sustaining severe brain injury because of a crush injury from a heavy television (Lantz et al. 2004). Another case report from the same group reported a fatal SDH and severe RHs after an unwitnessed stairway fall (Lantz and Couture 2011). As discussed above, a fatality from such a stairway fall is at least very unusual, though this child was also noted to have a partial

thromboplastin time (PTT) > 200, implying a severe coagulopathy existed either before or after the severe brain injury.

The prevalence of RHs has also been addressed in children who are critically ill without trauma. Agrawal et al. (2012) examined 159 consecutive patients less than weeks old in a London pediatric intensive care unit. Subjects with AHT were excluded. The number of RHs was rated as mild (1–4), moderate (5–20), or severe (> 20). Of the159 subjects, 24 (15%) were found to have any RHs. Mild RHs were seen in 16 subjects, most of whom had sepsis. Two subjects had moderate RHs—one with sepsis and one with a television crush head injury. Six children were found to have severe RHs—three had leukemia and severe sepsis; one had hemorrhagic disease and a short fall and two had fatal traumatic brain injury (non-abusive).

Similarly, Longmuir et al. (2014) reported results of retinal examinations in 85 intubated PICU patients. Six patients (7%) had any RHs, including four children with AHT; all of whom had severe RHs. The other two children with RHs included one with moderate RHs from a TV crush injury, and one with mild RHs after a cardiac arrest attributed to SIDS.

Without evidence, the hypothesis has been raised that vaccines may cause retinal hemorrhages in the absence of trauma (Clemetson 2004; Gardner 2005; Squier 2011). Binenbaum et al. (2015) reasoned that if vaccines were a cause of RHs, then RHs would be seen frequently and would be temporally associated with immunization. They conducted a retrospective cohort study from June 1, 2009 through August 30, 2012, at the Pediatric Ophthalmology Clinics of the Children's Hospital of Philadelphia. They examined 5177 children 1–23 months old who were undergoing dilated funduscopic exam for any reason.

The outcomes and measures they examined were the prevalence and cause of RHs and the temporal association between vaccine injections within 7, 14, and 21 days preceding these examinations and RHs. Reasons for dilated examinations in these children included strabismus, amblyopia, red eye, trauma, tear duct obstruction, poor visual behavior, or systemic diseases with ocular findings. The inclusion criterion was the availability of vaccine records. Excluded from the study were a history of direct eye trauma, intraocular surgery, and retinopathy of prematurity stage 3 or worse.

Among the 5177 children included (with 7675 fundus examinations), nine (0.17%) had RHs and all of these were victims of abusive head trauma (AHT) diagnosed with non-ocular findings including intracranial hemorrhage (9), skull fracture (5), bruises (3), hypoxic-ischemic brain injury (2), and spinal fracture, spinal hematoma, or perpetrator confession (1 each).

Vaccination records were available for 2210 (with 3425 fundus examinations). These vaccines included pneumococcus; diphtheria, tetanus, and pertussis (DTP); Haemophilus influenza type B (HIB); polio; hepatitis B; measles-mumps-rubella (MMR); and varicella. Four of the 2210 children (0.18%) with vaccination records had RHs. None of these 4 had vaccinations within 7 days preceding the diagnosis of RHs. One had vaccination within 14 days.

The conclusion of the investigators was that there was no association between receiving a vaccination injection and the presence of RHs in the subsequent 7, 14, or 21 days.

"New science" has not changed the understanding of RHs and its association with AHT. As has been known for decades, RHs can be caused by a host of traumatic and medical causes. However, severe RHs—numerous (> 20), multi-layered, and extending to the *ora serrata*—are very specific for severe traumatic brain injury. Although AHT is undoubtedly the most common source of severe RHs, serious crush injuries and children with leukemia and coagulopathy can also have severe RHs. Vaccination, however, does not cause RHs.

Cerebral Sinus Thrombosis

Not a single peer-reviewed paper has been identified linking cerebral sinovenous thrombosis (CSV) with other signs of AHT. Nevertheless, this hypothesis is suggested in legal proceedings. CSV is a rare disorder in children (0.67/100,000). The single largest age group with CSV is hospitalized neonates, comprising 43% of all cases up to 19 years of age. Less than 10% of cases have extra-cerebral hemorrhages. CSV is usually associated with systemic illness (84%) such as dehydration, metabolic acidosis, central nervous system infection, cyanotic heart disease, head injuries, craniotomy, renal, and thromboembolic disorders.

Choudhary et al. (2015) reported data from all children at their center younger than 36 months of age who were diagnosed with abusive head trauma (AHT) and who had both magnetic resonance imaging and MRI venography. The purpose of the study was to define the incidence and characteristics of venous and sinus abnormalities in abusive head trauma cases. The study was conducted from 2001 to 2012. Neuroradiologists independently analyzed MRI and MRI venography.

Forty-five children with a median age of 3 months (range 15 days to 31 months) were included. Sixty-two percent were boys. RHs were seen in 71%, extra-cranial fractures in 55%, and in 91%, a CT or MRI showed SDH. AHT was diagnosed by consensus of the treating medical team, perpetrator confession, and/or a judicial ruling. An experienced pediatric radiologist reviewed all of the skeletal surveys.

Thrombosis was defined as the absence of veins or sinuses on 3-D phase contrast MRI venography. MRI venography showed a mass effect on the venous sinuses or cortical draining veins in 69% (31/45). This mass effect was either displacement or partial or complete effacement of the venous structures from an adjacent SDH or brain swelling. The "lollipop" sign occurs when the bridging vein terminates in a sub-arachnoid blood clot and consequently does not drain into the sinus. This represents direct trauma to the cortical bridging veins and was seen in 44% (20/45) of the children.

The authors describe the limitations of the study, including technical limitations of MRI venography, the variations in venous anatomy, and in the appearance of venous thrombosis as well as the imaging peculiarities in children, mainly in terms of size of vessels.

The authors conclude that primary cortical sinus and venous thrombosis is a rare disorder, occurring in two to seven cases per million people. In children, predisposing factors are present in up to 95%. Outside the perinatal period, contributing factors include dehydration, malignancy, chemotherapy, iron-deficiency anemia, infection or sepsis, thrombophilia, gene mutations, and oral contraceptives. The clinical

presentation of venous thrombosis is one of progressive, sub-acute decline, often over several days, usually in the context of another identifiable illness.

The Real New Science

For the reasons above, the understanding of AHT has not significantly changed in recent years. One recent survey of hundreds of pediatric specialists at leading children's hospitals directly assessed which causes were considered most likely to cause the findings associated with AHT. Short falls, vaccines, or choking remained fringe theories as explanations for SDH, RH, and coma or death (Narang et al. 2016). But with the relatively recent recognition of child abuse pediatrics as a new subspecialty within pediatrics, it would be surprising to conclude that the science of AHT has not advanced. Indeed, remarkable progress has been made, especially in the recognition, prognosis, and treatment of AHT.

Centers have identified sentinel injuries that should routinely prompt consideration of AHT, including bruises in young infants, unexplained oral injuries, long-bone fractures, and abdominal injuries (Lindberg et al. 2015; Pierce et al. 2016; Sheets et al. 2013). Multi-center networks and systematic reviews have identified features of brain injuries that are most concerning for abuse (Hymel et al. 2014; Kemp et al. 2011). Recently, a decision rule to identify subtle signs of abusive and non-abusive brain injuries in young infants was validated in four pediatric emergency departments (Berger et al. 2016). Other studies have identified broad variability in screening practices for occult abusive injuries and have recommended best practices to move toward an object ive, standardized approach to diagnosing abuse (Greiner et al. 2013b; Harper et al. 2013; Lindberg et al. 2013; Wood et al. 2015a, 2012, 2015b, 2010). Although confidence is waning in CT estimates of the age of SDHs, preliminary work suggests that someday estimation of the timing of RH may improve (Binenbaum et al. 2016). Development of new, fast MRI techniques suggests that screening for AHT will soon be done without the risks of radiation or sedation (Berger 2014; Cohen et al. 2015).

There is much new science with respect to AHT. Without exception, valid, and reproducible methods support the commonly held understanding of AHT that children who present with severe symptoms and who are found to have SDH and RHs are very likely to have been victims of significant trauma (Choudhary et al. 2018).

In a world of finite research resources, the real shame of this review is that so much time and effort is being devoted to research whose only purpose is to counter fringe theories. In any other field, hypotheses and theories based only on case reports, limited biomechanical theory, and aberrant interpretations of radiographs would have self-limited effects. Without reproduction, citations or enduring influence on the field, they would join tens of thousands of forgotten pieces of medical scholarship. It is only the fact that these articles are used in court gives them continued relevance. Ideally, this could be countered by broadly increasing the data literacy of lawyers, judges, jurors, and the public. However, until there is a reliable method by which courts can distinguish the validity of scientific data and thereby differentiate valid methods from fringe theories, it is left to the individual ethics of expert witnesses to communicate correctly the current state of the science.

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Compliance with Ethical Standards

Conflict of Interest The authors have each provided expert witness testimony in cases with alleged child physical abuse.

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Acceptance of Shaken Baby Syndrome and Abusive Head Trauma as Medical Diagnoses

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Objective To assess the current general acceptance within the medical community of shaken baby syndrome (SBS), abusive head trauma (AHT), and several alternative explanations for findings commonly seen in abused children.

Study design This was a survey of physicians frequently involved in the evaluation of injured children at 10 leading children's hospitals. Physicians were asked to estimate the likelihood that subdural hematoma, severe retinal hemorrhages, and coma or death would result from several proposed mechanisms.

Results Of the 1378 physicians surveyed, 682 (49.5%) responded, and 628 were included in the final sample. A large majority of respondents felt that shaking with or without impact would be likely or highly likely to result in subdural hematoma, severe retinal hemorrhages, and coma or death, and that none of the alternative theories except motor vehicle collision would result in these 3 findings. SBS and AHT were comsidered valid diagnoses by 88% and 93% of the respondents, respectively.

Conclusions Our empirical data confirm that SBS and AHT are still generally accepted by physicians who frequently encounter suspected child abuse cases, and are considered likely sources of subdural hematoma, severe retinal hemorrhages, and coma or death in young children. Other than a high-velocity motor vehicle collision, no alternative theories of causation for these findings are generally accepted. *(J Pediatr 2016;■■:■■-■■).*

Although shaking, with or without impact, has been recognized as a dangerous form of child physical abuse since the early 1970s,[1,2] the validity of shaken baby syndrome (SBS) and abusive head trauma (AHT) has recently been called into question in prominent national newspapers such as the *New York Times* and *Washington Post*,[3,4] judicial decisions,[5,6] and some medical literature.[7,8] In fact, a US Supreme Court Justice recently commented in a dissenting opinion that there is widespread "controversy" within the medical community regarding the concepts of AHT and SBS.[9,10] Not surprisingly, this has resulted in confusion in the courts and a chilling effect on child protection hearings and criminal prosecutions.[11]

Legal interventions are an important part of primary safety determinations and secondary prevention for victims of maltreatment. In that process, courts frequently rely on medical expert testimony to opine on the most likely source of a child's injuries. To determine the admissibility of scientific testimony, courts must assess whether concepts are "generally accepted" in the medical community. In approximately one-half of the US jurisdictions, known as Frye jurisdictions, "general acceptance" is the sole criterion for admitting expert testimony on a certain concept.[11] In the remainder of US jurisdictions, known as Daubert jurisdictions, "general acceptance" is one of several criteria used to assess reliability, but is still afforded significant weight.[12] In addition, several professional medical society ethical guidelines for expert testimony state that testimony should reflect generally accepted opinions, and/or that an expert who endorses a minority opinion should volunteer that information.[13-16]

In courts, evidence of what is generally accepted in the medical community has typically been adduced by the opinion of a solitary expert or a small cadre of experts. This approach is susceptible to the biases and knowledge base of the testifying physicians, and leaves open the possibility that a small group could create an incorrect impression about whether or not any particular concept is generally accepted. Courts are ill-equipped to measure the broad opinion of the wider medical field or to assess the validity of a single physician's assessment of that broad opinion. Although SBS has historically been considered a valid medical diagnosis,[17] to date no well-conducted study has measured the acceptance of SBS or AHT as diagnoses, or of the likelihood that shaking will result in subdural hematoma (SDH), retinal hemorrhages (RH), or coma or death, the findings commonly associated with SBS and AHT.[18,19]

Given the importance of this issue to child protection and legal outcomes, we aimed to attain empirical data on the acceptance of SBS and AHT as valid medical

AHT	Abusive head trauma
MVC	Motor vehicle collision
REDCap	Research Electronic Data Capture
RH	Retinal hemorrhages
SBS	Shaken baby syndrome
SDH	Subdural hematoma

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diagnoses by the physicians most commonly involved in those cases. We also sought to determine whether shaking, with or without impact, and other mechanisms of injury are generally accepted as reasonable explanations for SDH, RH, and coma or death.

Methods

This observational survey study was reviewed and approved by the University of Texas-Houston Institutional Review Board, and was conducted between March and October 2015. To identify a feasible sample size and limit enrollment or response bias, we surveyed hospitals identified from the 2014-15 *US News & World Report* Honor Roll of Children's Hospitals.[20] From the 10 leading children's hospitals, we identified faculty physicians (MD, DO) within the specialty departments most commonly involved in suspected AHT cases: Emergency Medicine, Critical Care, Child Abuse Pediatrics, Pediatric Ophthalmology, Pediatric Radiology, Pediatric Neurosurgery, and Child Neurology. Because forensic pathologists are not typically located within children's hospitals, we contacted the medical examiners' offices that jurisdictionally comported with the surveyed hospitals and offered participation in the survey. If no medical examiner's office comported with a particular jurisdiction, we contacted the responsible coroner's office and offered participation in the survey.

We obtained contact information (e-mail and mailing addresses) from hospital websites or physician collaborators. In March 2015, physicians were invited to participate by e-mail, and were informed that the survey was voluntary and anonymous. Using a modified Dillman method,[21] the lead investigator (S.N.) sent an e-mail to eligible physicians, providing a summary of the study's objective and methods, along with a unique, anonymous online link to the survey. After the initial e-mail, nonresponders were sent a reminder e-mail (with survey links) every 2 weeks on 2 separate occasions. If a physician had not completed the survey after 3 e-mail attempts, then a hard copy of the survey (with $1 attached) was mailed to the physician's office address on 2 separate occasions at 2-week intervals. After this, if the participant still had not responded, he or she was logged as a nonresponder, and his or her contact information was permanently deleted. Data collection efforts were completed in October 2015. As an incentive to improve response rates, participants were entered into up to 5 randomized, biweekly drawings for a $200 gift card (depending on the time of response, with earlier responders being eligible for and entered into more drawings).

To minimize the potential for bias, we did not approach nonresponders and used no additional methods to encourage recruitment by any respondent. To ensure an appropriate sampling frame, we asked each respondent to report his or her specialty on the survey, and those who reported specialties other than those being sought to be surveyed excluded.

Study data were collected and managed using REDCap (Research Electronic Data Capture) tools hosted at the University of Texas at Houston.[22] REDCap is a secure, web-based application designed to support data capture for research studies. No identifying information was recorded in REDCap, and once a physician completed the survey, his or her contact information was permanently deleted, thereby preserving anonymity.

Survey

Each participant reported his or her age (20-30, 31-40, 41-50, 51-60, 61+ years), board certification status, and years in practice (0-5, 6-10, 11-20, 20-30, 31-40, or 41+ years). Each participant was also asked to choose his or her field of specialty from the list of specialties sought (ie, Emergency Medicine, Critical Care, Child Abuse Pediatrics, Pediatric Ophthalmology, Pediatric Radiology, Pediatric Neurosurgery, and Child Neurology), or to report another specialty. Those reporting more than 1 surveyed specialty (n = 8) were included under each specialty for the report of respondent characteristics, but were only counted once in the remainder of the survey. Those reporting a specialty that was included in the sampling frame and a specialty that was not included (eg, Pediatric Emergency Medicine, General Pediatrics) were counted within the included specialty. Those identified within a division of pediatric emergency medicine who listed their specialty as "urgent care" were included with Emergency Medicine. Those listing only exclusion specialties (eg, General Pediatrics, Allergy and Immunology, Anesthesia, Pulmonology) were excluded.

Respondents rated the likelihood of each finding (SDH, RH, coma or death) to result from several proposed mechanisms in a child aged <3 years using a 5-point Likert scale (from "highly unlikely" to "highly likely"). "Severe RH" was defined as too numerous to count, multilayered hemorrhages extending to the periphery. Proposed mechanisms included shaking without impact, shaking with impact against a soft surface (eg, a bed), a very short fall (<3 feet) with impact against a hard surface, a high-velocity motor vehicle collision (MVC), hypoxia, dysphagic choking, vitamin D deficiency rickets, and adverse reaction to vaccines.

Finally, respondents were asked whether they believed SBS to be a valid medical diagnosis (yes, no, don't know/unsure), whether they believed AHT to be a valid medical diagnosis (yes, no, don't know/unsure), and the basis for those opinions (clinical experience, medical literature, both, or neither). Respondents were offered the chance to ask questions or to comment on the survey or the study as a whole by contacting the principal investigator.

For analysis, we defined a "fringe opinion" as one in which <5% of respondents deemed a given mechanism for a finding as likely/highly likely or unlikely/highly unlikely (**Table I**). For analysis of shaking with impact versus shaking without impact results, we defined "discordance" as a rating that changed from highly unlikely or unlikely to likely or highly likely (or vice versa), depending on whether or not impact was present. Descriptive statistics were used to determine the prevalence of each response along with associated 95% CIs. Comparisons were conducted using OR with 95% CI.

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Table I. Fringe opinions

	Likely/highly likely	%	Unlikely/highly unlikely	%
SDH	Vaccines	0.0	Shake WITH impact	3.2
	Vitamin D	2.3		
	Choking	2.7		
	Hypoxia	4.0		
RH	Vaccines	0.0	Shake WITH impact	1.0
	Vitamin D	0.8	Shake NO impact	1.8
	Short fall	3.2		
Coma/death	Vitamin D	0.6	Shake NO impact	3.7
	Vaccines	1.0	Shake WITH impact	4.8
	Short fall	3.1	MVC	3.5
	SBS invalid		4.8	
	AHT invalid		1.0	

A causative mechanism was considered a fringe opinion if the combined percentage of respondents rating it as likely or highly unlikely or as unlikely or highly unlikely was <5%.

Results

The survey was sent to 1378 clinicians, of whom 682 (49.5%) responded. A department of child neurology (n = 22) at 1 institution declined as a block to participate, and were counted as nonresponders. We excluded 54 (8%) survey respondents because they either did not list their specialty (n = 9) or listed only specialties that were not included in our sampling frame (23 general, primary, or hospitalist pediatricians and 22 other pediatric subspecialists). The remaining 628 respondents composed the main cohort for this analysis. Characteristics of the respondents are summarized in **Table II**. Among the respondents, the most common specialties listed were Emergency Medicine, Critical Care, Neurology, and Radiology. The large majority (88.2%) of respondents reported being board-certified in their specialty.

Table II. Respondent characteristics

Characteristics	n (%)
Specialty*	
Emergency Medicine	192 (30.9)
Critical Care	108 (17.4)
Neurology	101 (16.3)
Radiology	96 (15.5)
Ophthalmology	45 (7.2)
Neurosurgery	30 (4.8)
Child Abuse	30 (4.8)
Pathology	27 (4.3)
Board-certified†	548 (88.2)
Age, y‡	
20-30	4 (0.6)
31-40	240 (38.6)
41-50	180 (29.0)
51-60	128 (20.6)
61+	68 (11.0)
Years in practice§	
0-5	148 (23.8)
6-10	135 (21.7)
11-20	164 (26.4)
21-30	106 (17.1)
31-40	48 (7.7)
41+	16 (2.6)

*Sums to 629 because 8 respondents listed 2 specialties.
†Six respondents did not report board certification status.
‡One respondent did not report age.
§Four respondents did not report years in practice.

Ninety-nine respondents (15.8%) omitted answers for at least 1 question. The most common scenarios in which more than 3 responses were omitted were nonophthalmologists omitting questions about RH and, conversely, ophthalmologists exclusively answering questions related to RH. No question was omitted by more than 22 respondents.

Respondents' opinions about the most likely source of SDH, severe RH, and coma or death are shown in the **Figure**. More than 80% of respondents felt that shaking with or without impact was likely or highly likely to produce SDH, more than 90% reported that it was likely or highly likely to produce RH, and more than 78% reported that it was likely or highly likely to result in coma or death. The corresponding results for a short fall were 18%, 3%, and 3%, respectively.

Either SBS or AHT was characterized as a valid diagnosis by 607 respondents (96.7%; 95% CI, 94.9%-97.9%). SBS was endorsed as valid by 554 respondents (88.1%; 95% CI, 85.3%-90.5%); AHT, by 584 respondents (93.0%; 95% CI, 90.7%-94.9%). Pathologists were statistically significantly more likely to be divergent with respect to the validity of AHT and SBS, with 8 of 27 stating that SBS is not a valid diagnosis, but that AHT is valid (OR, 13.5; 95% CI, 4.7-38.1, relative to other specialties) (**Table III**). Two pathologists responded that SBS is valid, but AHT is not.

Among the respondents stating that SBS or AHT is a valid diagnosis, 545 (89.7%) reported that they were informed by both the scientific literature and their own clinical experience, 48 (8%) were informed only by their clinical experience, and 11 (1.8%) were informed only by the scientific literature. One respondent did not answer the question, and 2 respondents listed "other" as the reason for considering the diagnosis valid. With respect to specific findings (SDH, RH, coma or death), the respondents showed very little discordance in their responses according to the presence or absence of impact.

Using our definition of "fringe opinion," 165 respondents (26.6%) reported at least 1 fringe opinion. We also included respondents who stated that either SBS (n = 30; 4.8%) or AHT (n = 6, 1.0%) were not valid. Of the 6 respondents who stated that they thought AHT was not a valid diagnosis, 5 agreed that shaking with or without impact was likely or highly likely to result in SDH and RH. All 5 of these respondents agreed that shaking with impact was likely or highly likely to result in coma or death; 2 of the 5 were neutral about the likelihood of shaking without impact resulting in coma or death. One respondent reported that AHT was invalid, and that shaking with or without impact is unlikely or highly unlikely to result in SDH, RH, or coma or death. This respondent reported that only a MVC or a short fall were likely to result in SDH, no option was likely to result in RH, and only a MVC was likely to result in coma or death.

Discussion

Our survey results represent national, multidisciplinary physician opinions on the validity of SBS and AHT, and of the

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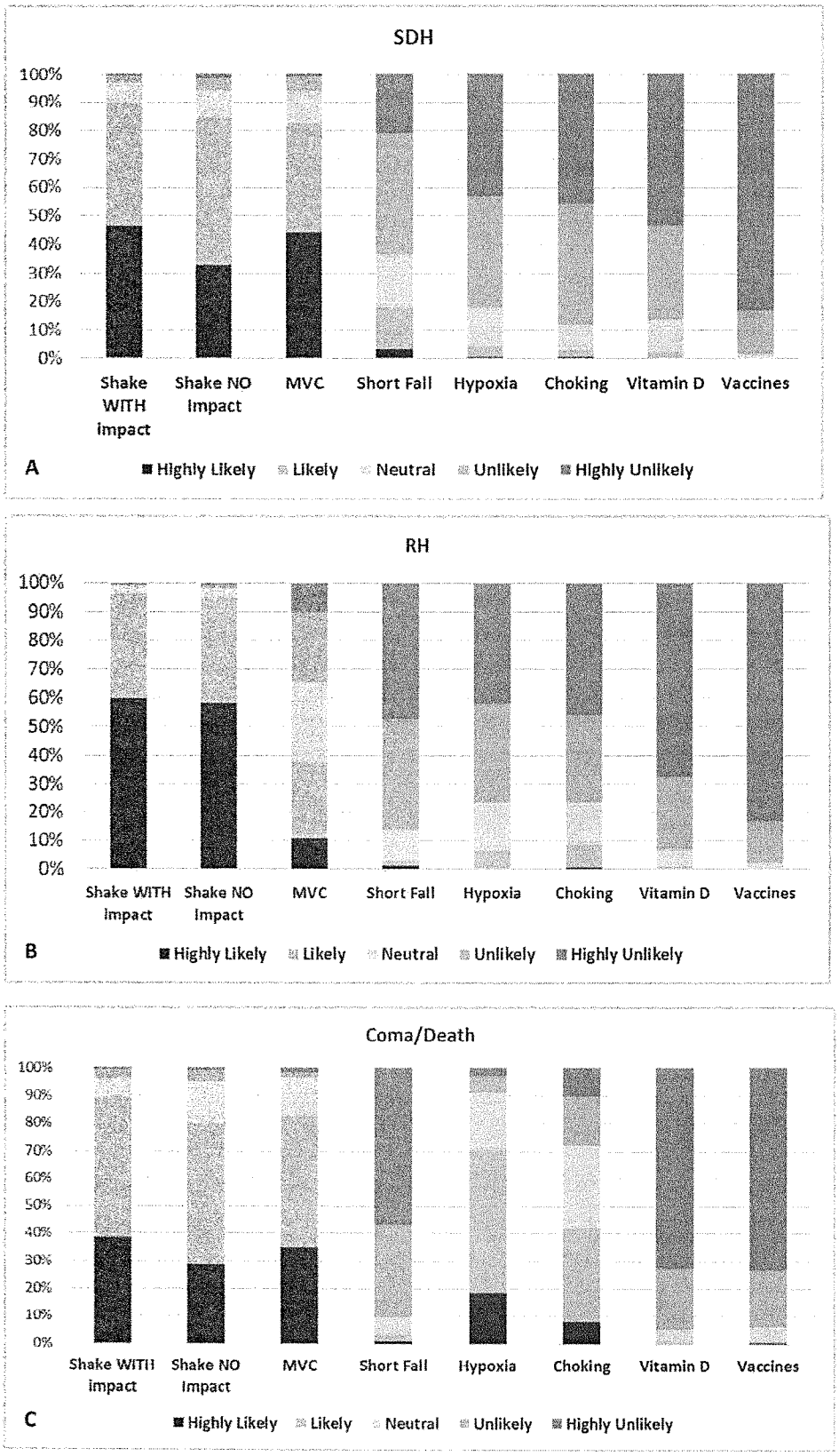

Figure. Percentage of respondents who believe that SDHs, severe RHs, and coma/death would result from the above events.

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Table III. Validity of AHT and SBS by specialty

Specialties	n	Yes, n (%)	No	Don't know/ unsure	Blank
AHT valid					
Emergency Medicine	196	184 (93.9)	0	10	2
Critical Care	108	102 (94.4)	2	3	1
Neurology	103	95 (92.2)	1	5	2
Radiology	96	82 (88.5)	2	8	4
Ophthalmology	46	44 (95.7)	0	2	0
Neurosurgery	30	30 (100.0)	0	0	0
Child Abuse Pediatrics	30	30 (100.0)	0	0	0
Pathology	27	25 (92.6)	1	1	0
SBS valid					
Emergency Medicine	196	175 (89.3)	7	11	3
Critical Care	108	99 (91.7)	2	7	0
Neurology	103	96 (93.2)	4	1	2
Radiology	96	84 (87.5)	2	6	4
Ophthalmology	46	45 (97.8)	0	1	0
Neurosurgery	30	23 (76.6)	5	2	0
Child Abuse Pediatrics	30	28 (93.3)	2	0	0
Pathology	27	11 (40.7)	8	8	0

Totals sum to 636 because 8 respondents listed 2 specialties: 4 for Child Abuse Pediatrics and Emergency Medicine, 2 for Critical Care and Emergency Medicine, and 2 for Critical Care and Neurology.

likelihood that findings commonly seen in those cases—SDH, severe RH, and coma or death—result from various causal mechanisms. Although "general acceptance" is not defined by a definitive numerical threshold in legal settings (although acceptance by <50% of field clearly would not meet the criterion for "general acceptance"), our results provide empirical data that clearly support the conclusion that SBS and AHT are still generally accepted as valid medical diagnoses across a broad range of specialties. Furthermore, our data show that shaking with or without impact (in contradistinction to several other alternative theories) is generally accepted to be a dangerous form of child physical abuse and capable of producing SDH, RH, and coma or death. Several alternative explanations that have been proposed to cause SDH, RH, and coma or death are not generally accepted. This high degree of consensus, irrespective of specialty, experience, or age, refutes recent reports in the lay press and legal commentary of a substantial controversy within the medical community regarding SBS and AHT. Other authors have discussed the various motivations for those media sources to proffer such assertions.[22,23]

As a specialty, forensic pathologists were discordant from other respondents, being more likely to question the validity of SBS as a diagnosis, although not more likely to question the validity of AHT (**Table III**). In this respect, our results are similar to the results of a survey of forensic pathologists that showed 35% questioning SBS.[23] That survey did not address the topic of AHT separately from SBS, however.

Our survey results demonstrate that physicians, irrespective of specialty, viewed the risks of shaking, with or without impact, to be similar to a high-velocity MVC and dissimilar to a very short fall. Although this finding may seem unremarkable to clinicians, it is important in light of some biomechanical literature arguing that shaking without impact cannot generate sufficient forces to cause SDH,[24,25] and biomechanical[24] and pathology[26] literature suggesting very short falls as a reasonable explanation for those findings. We believe the divergence of our results from this literature represents a recognition of the limitations of biomechanical data, a primacy of clinical literature and experience in relation to that literature, or both.

Our study has several limitations. First, we did not include general pediatricians in our sampling frame, even though some general pediatricians have substantial experience caring for children who have sustained physical abuse. Thus, our results are susceptible to selection bias. However, we chose to include only those specialties with the greatest likelihood of evaluating and treating pediatric traumatic brain injury. Our results could be different if general pediatricians with high rates of exposure to traumatic brain injury had systematically different opinions about the risks and injuries associated with shaking or other suggested mechanisms.

Second, as with all survey studies, ours might have been subject to response bias if respondents held systematically different opinions from nonrespondents. If present, this could have affected our results by increasing or decreasing the true proportion of clinicians who accept SBS or AHT. We do not feel that this limitation significantly affected our results, however, for several reasons. First, our sampling frame was chosen to reflect practicing clinicians from 10 leading hospitals, rather than groups that are most active in legal proceedings involving child abuse and neglect (and thus more motivated to respond). Second, our relatively high response rate (nearly 50% of those surveyed, with more than 600 clinicians) limits the potential that a small cadre of clinicians with divergent opinions would significantly affect results. Finally, our results show remarkable unanimity. Thus, nearly all nonresponders would have to harbor opinions that are diametrically opposed to responders for AHT or SBS to have an acceptance rate of <50% or for fringe opinions to be generally accepted.

The limitations of the *US News & World Report* hospital rankings have been discussed elsewhere.[27] Our intention in using these rankings was not to endorse a ranking of any particular children's hospital; rather, we sought to identify a relatively large and diverse cohort of clinicians likely to care for child victims of trauma, and to decrease the possibility that the survey would be preferentially distributed to clinicians whose opinion regarding AHT or SBS was known to the authors. It is possible that our results would differ if we were to use different hospitals or a different ranking system; however, given the degree of consensus, we believe it unlikely that such different choices would change the conclusion regarding whether SBS, AHT, or the other alternative hypotheses are generally accepted.

Finally, some respondents indicated confusion about the questions. For example, 1 respondent (who contacted the lead investigator) noted that there are important developmental and anatomic differences between infants aged <12 months and young children aged <3 years that could significantly impact the likelihood of the resulting findings. Another respondent noted that it would have been more appropriate to ask about the likely mechanism, given a particular finding, than to ask about the likely findings resulting from a given mechanism.

Although we recognize both points, we believe that any ambiguity in the survey design would bias against a high level of consensus. ■

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Biomechanical Response of the Infant Head to Shaking - An Experimental Investigation

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Using an infant surrogate with improved biofidelity, we found higher angular acceleration and higher angular velocity than previously reported during infant surrogate shaking experiments. Findings highlight the importance of surrogate biofidelity when investigating shaking.

Key words: head injury, pediatric injury, child abuse, shaking, biomechanics

INTRODUCTION

Violent shaking of infants has been thought to cause serious brain damage since Guthkelch documented the injuries of two shaken infants in 1971.[1] In 1987, Duhaime and colleagues challenged this theory by constructing an infant surrogate and subjecting it to violent shaking and impacts.[2] They measured the rotational acceleration and velocity generated during shaking and impact and concluded that shaking alone did not reach expected biomechanical injury thresholds to cause concussion, subdural hematomas, or diffuse axonal injury. When the infant surrogate's head was struck against a hard surface, however, head injury thresholds were exceeded. They concluded that shaking alone would not be likely to cause serious head injury to an infant. This hypothesis has been tested by others using different surrogates, as well as by computer modeling, with varying results.[3-5]

Duhaime's work has led to a longstanding controversy as to whether it is possible to harm infants by violently shaking them. The concept that "shaking doesn't hurt babies" has been promoted in the popular media.[6-8] These sources highlight stories of people accused of abusing their children by shaking, and quote defense experts who claim that biomechanical studies have shown that shaking an infant cannot cause subdural hemorrhages, encephalopathy, and retinal hemorrhages. Yet, extensive clinical experience resulting from a number of well-documented cases has demonstrated the harmful effects of shaking children.[9-14] In a number of cases, adults have voluntarily reported violently shaking babies, sometimes associated with impact, and sometimes not.[15,16] These infants often are found to have serious or fatal brain injuries, and in survivors, a poor prognosis.[17-20] In addition, a program of intensive education of new parents about the dangers of shaking led to a substantial decrease in the incidence of serious infant head trauma in Buffalo, New York.[21]

We are left with a situation in which clinical experience is not consistent with the biomechanical data from previous laboratory experiments.[2,3] We used an instrumented anthropomorphic test device (ATD, often referred to as a 'crash test dummy' or surrogate) scaled to the size of a human infant to characterize infant head kinematics during shaking. The ATD was specifically designed to have biomechanical responses similar to that of a human infant, especially as it relates to the spine and head-neck regions. We used an instrumented ATD to acquire kinematic and temporal-spatial data during shaking by an adult male.

MATERIALS AND METHODS

The purpose of our testing was to characterize head-neck kinematics associated with violent shaking. Events were simulated in a laboratory setting using an instrumented infant anthropomorphic test device (ATD) and videography.

Shaking Scenario

A 50th percentile Japanese adult male (172 cm; 65.3 kg) volunteer was used to shake the infant ATD in a manner consistent with accounts given by adults who confessed to infant shaking. Five repeat trials consisting of multiple shaking cycles (minimum of 12 cycles/trial) were conducted. The volunteer was advised to grasp the infant ATD with both hands about the upper torso just below the axillary region, suspending it in an upright posture facing them. The volunteer was instructed to "violently" shake the ATD fore and aft for a period of 3 to 4 seconds. Videography was used to capture shaking.

Infant Anthropomorphic Test Device (ATD) or Surrogate

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An *Aprica 2.5* infant ATD was used to conduct shaking experiments (Figure 1). The *Aprica 2.5* is a customized twelve segment, instrumented infant ATD that represents a 5th percentile Japanese newborn (GESAC Inc; Boonesboro, MD).[22] The *Aprica 2.5* has a mass of 2.6 kg and an overall length of 450 mm. Additional anthropometric and inertial properties of the ATD are provided in Table 1. As shown in Table 1, nearly 1/3 of the ATD's mass (0.77 kg) is contained within the head, consistent with the ratio found in the human infant.[23]

Because of the paucity of biomechanical data describing infant response to mechanical phenomena, biofidelity of the *Aprica 2.5* was established by scaling response during head impact tests, head-neck pendulum tests, chest impact tests, and lumbar flexion tests. Data were scaled from existing pediatric ATDs ranging from 6 months to 10 years of age.[22] Since neck response is expected to greatly influence head injury outcome measures in shaking, particular attention was given to development of the *Aprica 2.5* neck. Little data exist to define the biomechanical response of a human infant neck. The ATD neck was constructed to produce an infant neck response to dynamic loading based upon scaling of adult ATD response characteristics.[24] The target neck stiffness for the *Aprica 2.5* neck was determined to be 17 Newton-meter/radian (Nm/rad).[24,25] A head/neck pendulum test was used to verify the dynamic neck response.[26] Rangarajan, et al has provided an overview of the development of the *Aprica 2.5* infant ATD. [22]

Anthropomorphic Test Device Instrumentation

The *Aprica 2.5* ATD was instrumented with tri-axial accelerometers (Kyowa ASM-200BA) positioned at the center of mass of the head and at the apex of the head. Head accelerometers were oriented to allow for determination of angular acceleration in the sagittal plane.

Accelerometers were calibrated following standardized procedures in accordance with SAE J211.[27]

Data Acquisition and Analysis

Data were sampled and collected at 10,000 Hz according to SAE J211.[27] Accelerometer data were filtered using a low pass Butterworth filter with a channel filter class of 1000 Hz.

Angular acceleration in the sagittal plane was derived from linear accelerometer data, because at critical levels, angular acceleration of the head has been shown correlate with concussion, diffuse axonal injury, and subdural hematomas.[28,29] Assuming that primary motion of the head was in the sagittal plane, an estimate of head angular acceleration can be determined by the difference of the linear accelerations in the fore/aft direction measured at two points in the same plane divided by the distance between them (Equation 1).

Equation 1

$$\alpha = \frac{a_2 - a_1}{r}$$

where: α = angular acceleration

a_1 = linear acceleration measured at head center of mass

a_2 = linear acceleration measured at superior aspect of head

r = distance between the accelerometers

The distance between the accelerometers positioned at the center of mass of the head and the apex of the head was 0.0432 meter.

Angular velocity of the head was determined by integrating the angular acceleration time history data. Peak values of head angular acceleration, angular velocity, maximum change in angular velocity, and time of exposure were determined for each shaking cycle, and mean peaks with 95% confidence intervals are reported for a given trial and across all trials. Mean and 95% confidence intervals of sagittal plane angular displacement time histories for a representative test series were also described to assess repeatability in shaking events.

RESULTS

Five shaking test series, each 3 to 4 seconds in duration, were completed using the *Aprica 2.5* ATD surrogate and volunteer shaker. Figure 2 illustrates the sequence of a representative shaking event. As the volunteer flexed his elbows pulling the ATD toward himself, the neck/head of the ATD extended rearward in the sagittal plane. As the volunteer extended his elbows pushing the ATD away from himself, the neck/head flexed forward in the sagittal plane. This visual sequence also illustrates that the ATD chin impacted the chest at peak neck flexion. Given the flexibility of the torso, the occiput did not contact the posterior upper torso at peak neck extension since the torso arched forward.

Mean peak neck flexion was approximately 1.98 radians [rad] (113 degrees [deg]) (95% CI: 1.68-2.28) and mean peak neck extension was approximately 2.16 rad (123 deg) (95% CI: 1.83-2.49) during a typical shaking cycle (Figure 3). Relatively tight confidence intervals illustrate the repeatability of the shaking cycles. As shown in Figure 3, one cycle occurs over approximately 0.25 second, yielding a shaking frequency of 4 hertz.

Mean peak angular head accelerations in the sagittal plane fell within a range of 7,035 rad/sec^2 (radians/second^2) (95% CI: 6,168-7,902) to 10,379 rad/sec^2 (95% CI: 9,304-11,452) across the five test series (Figure 4). The maximum angular head acceleration across all shaking events was 13,260 rad/sec^2 (Table 2), occurring in Test Series 4.

Mean peak angular head velocity measured in the sagittal plane ranged from 71.2 radians/second (rad/sec) (95% CI: 68.5-73.9) to 98.4 rad/sec (95% CI: 95.71-101.1) across the test series (Figure 5). The maximum angular velocity occurred in Test Series 2, and was 105.7 rad/sec (Table 2).

The mean peak change in angular velocity was between 132 rad/sec (95% CI: 128.6-136.4) and 167 rad/sec (95% CI: 164.3-170.0) across all test series (Figure 6). The maximum peak change in angular head velocity was 174 rad/sec in Test Series 2 and 3 (Table 2). Figure 7 presents the combinations of mean angular accelerations and mean peak change in angular velocities resulting from each test series.

The mean time duration of acceleration/deceleration across the five Test Series ranged from 98.5 msec (95% CI: 93.2-103.8) to 137.1 msec (95% CI: 132.9-141.3) (Figure 8). The shortest duration (72.1 msec) occurred in Test Series 1, while the longest duration (168.2 msec) occurred in Test Series 5.

DISCUSSION

The controversy over whether shaking alone can lead to severe head injury in infants began with the study by Duhaime et al. in 1987.[2] Using an infant surrogate constructed from a doll, Duhaime et al. found that lower angular and linear accelerations of the head were generated with

shaking of the surrogate than by inflicted impact. They reported a mean angular head acceleration of 1,138 rad/sec^2, with a mean angular velocity of 61 rad/sec and a mean acceleration pulse duration of 107 msec in their shaking experiments.

Prange et al. repeated this study, attempting to improve the biofidelity of the infant surrogate, and found that inflicted impacts against hard surfaces were more likely to be associated with angular accelerations reaching injury thresholds than shaking or falls from 1.5 m or less.[3] Compared to the Prange et al. study, a recent study by Coats et al. found markedly *lower* peak angular acceleration and velocity, and increased pulse durations during similar impact events using an ATD with a more biofidelic neck design, a deformable, sutured skull and a more biofidelic body mass distribution.[30] The Coats et al. study did not investigate shaking, but their differing findings compared to those of Prange et al. for head impacts highlights the marked influence ATD design can have on injury related outcomes.

The findings of our study differed substantially from those of Duhaime and Prange. Our maximum angular head acceleration was found to be 13,260 rad/sec^2, a 10-fold increase over Duhaime's findings and more than twice that reported by Prange et al. Further, peak angular head accelerations across all test series in our study exceeded those reported by Prange and Duhaime. Similarly, peak change in angular velocity measured in our study (174 rad/sec) was almost 3 times greater than that measured by Prange and Duhaime. The mean angular acceleration pulse duration for shaking measured in our study ranged from 98 msec to 137 msec, and was similar to that found by Duhaime and Prange.

Differences between our findings and those of Duhaime and Prange are likely due to differences in the surrogates used to represent an infant, and possibly differences in the delivery of shaking by volunteers. When considered in isolation, differences in surrogate design that would lead to increases or decreases in head accelerations include the following:

Body design: The ATD used by Prange employed a torso that combined the mass of arms and legs of an infant, represented as wooden structure, along with a neck and a head. The surrogate used in our study consisted of a segmented flexible torso/spine, along with a head, neck and upper and lower extremities. A less than complete ATD body, such as that used by Prange would reduce the overall inertial resistance to motion during shaking and could increase torso acceleration. That is, the lack of upper and lower extremities would offer less resistance to the back and forth motion of the torso during shaking allowing for greater ease of torso motion.

Surrogate mass: The mass of our ATD was 2.6 kg, whereas the mass of the surrogates used by Prange and Duhaime were 4.83 kg and 3.0 to 4.0 kg, respectively. Increased mass increases the inertial resistance of the surrogate body to motion and tends to decrease torso acceleration.

Head mass: The mass of the surrogate head used in our study was 0.77 kg, while the head mass of ATDs used by Prange and Duhaime was 1.13 kg and 0.77-0.87 kg, respectively. The lower head mass used in our study would offer less inertial resistance leading to higher head accelerations with shaking.

Neck structure and stiffness: Neck structure and stiffness play a critical role in the head's response to shaking. Duhaime, et al. examined the effects of various neck designs and found that a resistance-free hinged neck design was associated with the highest head accelerations with shaking.[2] Prange et al. utilized a negligible-resistance hinge neck to generate a worst-case head acceleration scenario.[3] The neck assembly used in our surrogate consisted of urethane tubing with a centered safety cable joining superior and inferior aluminum plates that allowed for head-

neck rotation in the sagittal, coronal and transverse planes. The safety cable was adjusted to provide desired neck bending properties (Figure 8).[22] Duhaime and Prange did not report neck properties of their surrogates, but given the negligible resistance offered by their hinge structures, we estimate that our surrogate neck was likely stiffer and provided greater resistance to head-neck motion during shaking. Both surrogates used by Duhaime and Prange constrained head-neck motions to the sagittal plane (anterior-posterior directions) by utilizing a hinged neck (worst-case scenario), while our surrogate's neck was free to move in three planes. When considering the constraint of head-neck motion in the sagittal plane in isolation, it would tend to increase head accelerations during shaking, since out-of-plane lateral motion is not permitted.

While it is interesting to study surrogate design characteristics in isolation of each other, one characteristic may outweigh or nullify the influence of another characteristic when the surrogate is considered as a whole (i.e. all characteristics together). Such is the case with head motion constraint or lack thereof. That is, even though the lack of head motion constraint tends to decrease head acceleration (as compared to constrained head motion), other design characteristics had a greater bearing on the resulting head acceleration in our experiments. It is the combination of surrogate design characteristics (e.g. head mass, neck length, torso stiffness, surrogate mass, neck stiffness, etc.), along with severity of shaking that dictate the resulting head acceleration. Together these factors drive key kinematic features of the head, such as chin-to-chest contact which can generate large peaks in head acceleration. Chin-to-chest contact occurred in our experiments and was associated with peak head accelerations, but was not reported in experiments conducted of Duhaime and Prange. This kinematic feature likely contributed to head accelerations exceeding those measured by Duhaime and Prange during shaking experiments.

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Another study examining biomechanical outcomes during shaking of an infant surrogate was conducted by Cory, et al.[4] They reproduced the Duhaime's 1987 experiments using an adjustable replica of their surrogate to determine the influence of surrogate parameters on head response outcomes during shaking, varying head-neck joint location (i.e., occipital condyle location), neck construction (rubber neck vs. hinged neck), torso padding (cotton wool versus silicone), and location of the surrogate center of gravity (CG). Under a combination of worst-case parameters, Cory measured a peak angular head acceleration of 10,217 rad/sec^2 and a peak angular head velocity of 61 rad/sec. Cory's results exceeded peak angular accelerations reported by both Duhaime and Prange, and approach those measured in our experiments.[2,3] Cory found the combination of a high surrogate CG, hinged neck and cotton wool padded torso produced worst-case angular head acceleration values.

One of the differences between the *Aprica 2.5* ATD and the surrogate used by Cory is the flexibility of the thoracic spine and torso stiffness. During shaking, Cory et al. described both chin-to-chest and posterior head-to-posterior torso contact (likely contributing to higher accelerations than those measured by Duhaime and Prange). The *Aprica 2.5* ATD also displayed chin-to-chest contact, but the posterior aspect of the head did not contact the torso during shaking. The *Aprica 2.5* ATD has a flexible thoracic spine allowing for arching of the torso during the extension portion of shaking which diminishes the likelihood of head contact with the posterior torso. Cory et al. did not quantify neck properties of the surrogate used in their study, and thus a direct comparison with the *Aprica 2.5* ATD neck was not possible.

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The location of the center of rotation of the head also influences the rotational response of the head to shaking. That is, for a given shaking event, as the distance to the center of rotation increases in length, the angular acceleration would decrease assuming all other parameters were held constant. Although the *Aprica 2.5* ATD neck joins the torso at a point 5 cm below the base of the skull, the design of the neck allows for a moving center of rotation as the head rotates. This can effectively provide a radius of rotation that is less than 5 cm in length at various points in the head's rotation about the neck. In contrast, the surrogates used by Duhaime, Cory and Prange that led to worst-case head accelerations had fixed centers of rotation that were located 3.3 cm, 3.3 cm and 4.5 cm, respectively, below the base of the skull. In contrast, the *Aprica 2.5* ATD neck allowed for a moving center of rotation mimicking that of a human infant. Given this moving center of rotation (i.e. the center of rotation can change during neck flexion/extension) it is difficult to evaluate how the neck center of rotation would affect angular head acceleration in comparison to the surrogates used by Duhaime, Cory and Prange. In general, if the *Aprica 2.5* ATD center of head rotation was located such that the radius of rotation was less than that of the other surrogates, there would be a tendency towards increased angular head acceleration for a given shaking input.

To determine the likelihood of injury from exposure to a given phenomenon (i.e. impact or shaking), outcome measures known to be associated with injury risk are typically compared to published injury thresholds. During *Aprica 2.5* ATD shaking experiments, injury threshold levels for concussion reported for primates with 400 g brains (similar in mass to a young infant) were exceeded,[31] while published injury thresholds for diffuse axonal injury were not exceeded.[29] To date however, there are no validated infant brain injury thresholds. Scaling thresholds from adult cadaver and primate studies to infants based on brain mass alone can

produce misleading pediatric head injury thresholds.[32] Other factors such as brain material properties and geometry must also be considered when attempting to assess brain injury risk.[33] Furthermore, published pediatric head injury thresholds fail to account for repetitive exposure to acceleration or deceleration that occurs in shaking, but instead consider only exposure to a single event. Repetitive head injuries in animal models have been shown to cause greater injury at lower peak rotational velocities than do single impulse loads.[34] The effects of repetitive cyclic events such as those involved in shaking have not been systematically studied in animal models other than mice and piglets.[35,36]

The duration of exposure to acceleration is also an important factor when attempting to predict the risk of and type of brain injury that can result from an event. In our experiments, duration of exposure (i.e. acceleration pulse duration) ranged from 72.1 msec to 168.2 msec. Löwenhielm et al described accelerations and associated pulse durations of 15 to 44 msec leading to subdural hematomas, substantially shorter pulse durations than experienced by our ATD during shaking.[37] Ommaya found that lower levels of acceleration are capable of producing injury in cases of longer pulse duration.[38] Genaralli and Thibault suggested that longer acceleration pulse durations permit brain tissue strains resulting from accelerations to propagate deeper into the brain leading to functional damage found in cerebral concussion or structural damage found in axonal injury.[28]

Other studies have noted the extreme vulnerability of the infant brain to the metabolic effects of brain injury when compared with the adult brain.[39,40] This would suggest that the infant brain is more susceptible to injury and might account for the poor outcomes noted in children surviving abusive head trauma (AHT).[18,19] Another worrisome aspect of infant abuse that could account

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for the hypoxic injury to the brain is the finding that in a large percentage of infant abuse cases resulting in death, subtle injury to the high cervical cord and lower brain stem is seen on autopsy.[41] It is postulated that these cord injuries could lead to apnea or changes in autoregulation of cerebral blood flow, causing the typical hypoxic changes seen in infant victims of AHT.

The influence of shaking on the infant brain and the pathophysiology of infant brain injury is more complicated than can be represented in existing surrogate models used in biomechanical shaking experiments. For example, the immature brain could be more vulnerable to angular acceleration than the adult brain. Raghupathi and colleagues found the immature piglet brain to be more vulnerable to a single inertial load than that of the adult pig brain.[42] They also found that repeating an inertial load on the piglet head twice, 15 minutes apart, led to more diffuse axonal injury in the piglet brain and to a significant decrease in arterial blood pressure 60 minutes post injury.[36] Since violent shaking of infants involves repetitive inertial events occurring within a few seconds, the mechanical phenomena necessary to cause injury in the immature brain could be less severe than would be necessary to cause a single impact injury. In addition, surrogate experimental outcomes do not take into account the well-known deficits in cerebral autoregulation that occur after infant brain injury leading to profound cerebral hypotension and hypoperfusion.[43]

All of these factors suggest that the infant brain is more susceptible to injury than the adult brain. Taking these factors into account, predictions of risk based upon comparison with published brain injury thresholds are not likely to be reliable given the limitations inherent in these thresholds.

Another factor that complicates the estimation of infant brain injury thresholds is the fact that infants who experience abusive head trauma are often subjected to multiple bouts of trauma over days to weeks. Several studies have shown that infants presenting with AHT are likely to have suffered previous abusive head trauma.[44-46] Using an animal model, Huh, et al demonstrated a graded pathological response to repetitive mild injury in immature rats.[34] Rats experiencing three mild impacts to the head (not resulting in fracture) over 15 minutes developed axonal injury and brain atrophy, compared to rat pups receiving only a single mild impact. When an immature brain experiences multiple injuries, some 'priming' might occur that makes the brain more susceptible to damage from subsequent injuries. Additionally, vulnerable infant axons within the brain may not be capable of repair between bouts of trauma. These phenomena may effectively lower infant brain injury thresholds, which are used to predict probability of injury when compared to experimental outcomes such as angular acceleration.

In addition to the lack of data on the biomechanical properties of the infant neck, animal models may not adequately represent the lack of supporting musculature found in the human infant neck.[47] However, the neck of a newborn goat has been estimated to be equivalent in strength to the neck of a one-year-old human.[48] When studying the effects of shaking, the lack of protective infant neck musculature is a key factor that must be represented in any model.

This study is limited by the inability of *any* available ATD to represent the biomechanical characteristics of an infant with complete accuracy. The *Aprica 2.5* ATD, however, was designed to match a newborn infant's anthropometrics and to be biofidelic within the limits of the technology and published human response data. In addition, only one volunteer was used to

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induce shaking of the ATD. Using multiple individuals for ATD shaking may produce varying inputs leading to a different biomechanical response. However, our intent was to investigate shaking response generated by a nominal representative average (50^{th} percentile) male, providing the first step towards understanding the influence of improved ATD biofidelity. Similarly, one's interpretation of the instruction to "violently" shake the ATD is subjective and may vary across individual shakers. However, the volunteer shaker used in this study was educated on perpetrator actions when abusively shaking a child and was asked to replicate shaking that would occur during rage or anger. Finally, the *Aprica 2.5* ATD represents a small newborn infant. Shaking an ATD representing an older infant would undoubtedly lead to a differing biomechanical response due to differences in mass, neck flexibility and torso/spine flexibility.

Higher levels of angular acceleration and angular velocity measured using the instrumented *Aprica 2.5* ATD during shaking suggest that more significant injuries may be caused by shaking alone than previously reported. These findings are consistent with documented clinical experience showing that violent shaking of infants is potentially harmful or even lethal.[49-51] This study also demonstrated the importance of biofidelic torso/spine and head-neck regions when investigating kinematics associated with shaking. While biofidelic ATDs can improve our understanding of kinematics during shaking, infant neuropathological response to measured accelerations and velocities still remains unclear.

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AUTHOR DISCLOSURE STATEMENT

Drs. Jenny, Bertocci, and Rangarajan have testified in courts of law regarding infant head trauma.

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FIGURE LEGENDS

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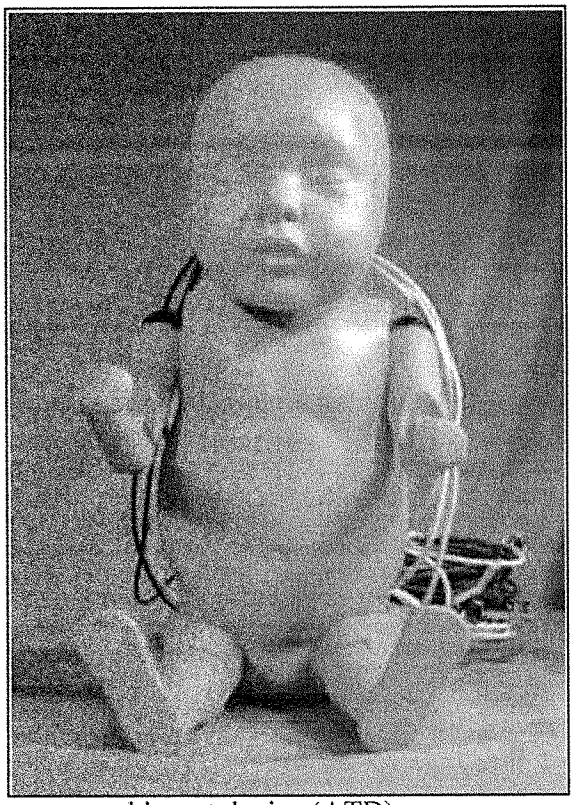

Figure 1. *Aprica 2.5* anthropomorphic test device (ATD).

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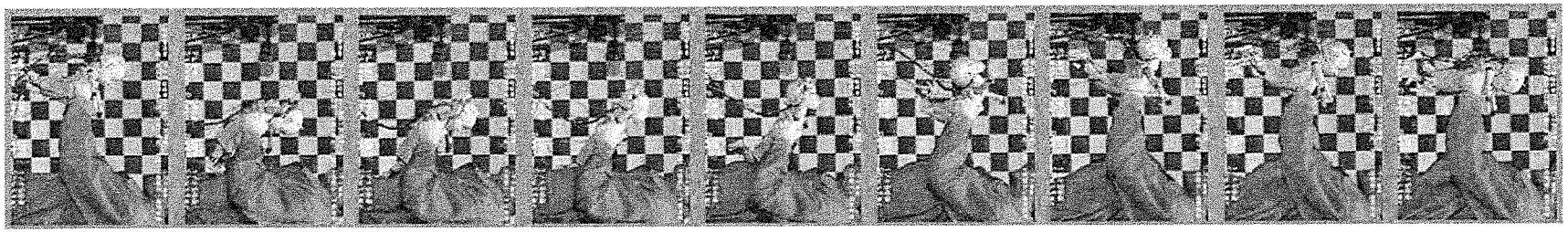

Figure 2. Kinematic sequence of one shaking cycle.

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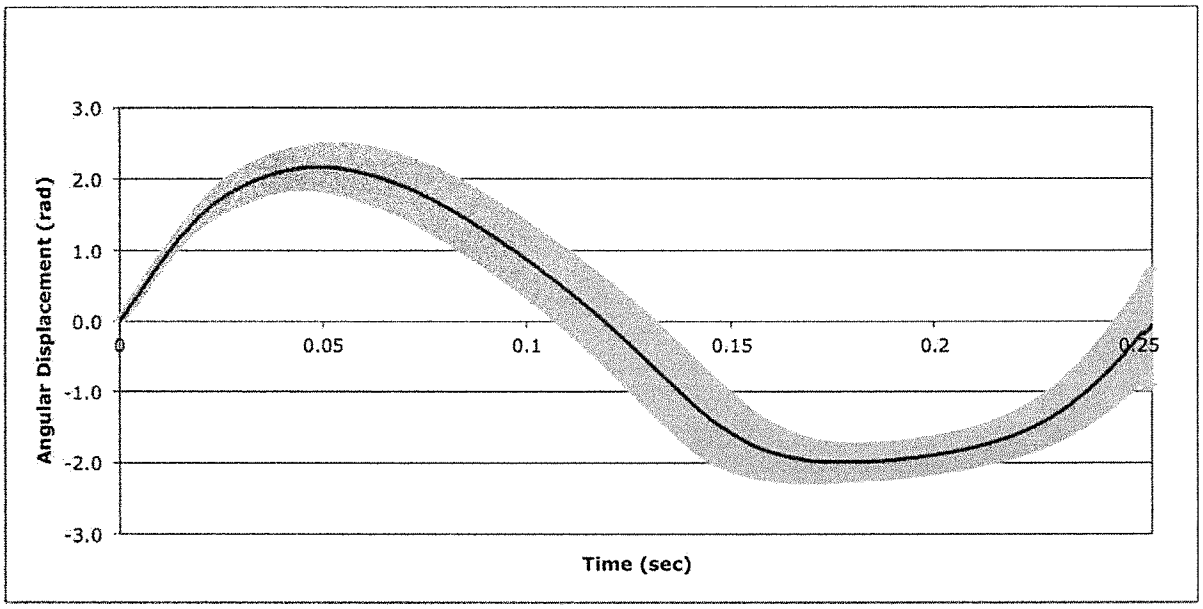

Figure 3. Mean angular head displacement time history for individual cycles of shaking for a representative test series (Test Series No. 3). One cycle occurs over approximately 0.25 sec (4 Hz). Positive values represent extension and negative values represent flexion. (Error bars – appear as shading - represent 95% confidence intervals.) (Note: 1 radian = 57.3°)

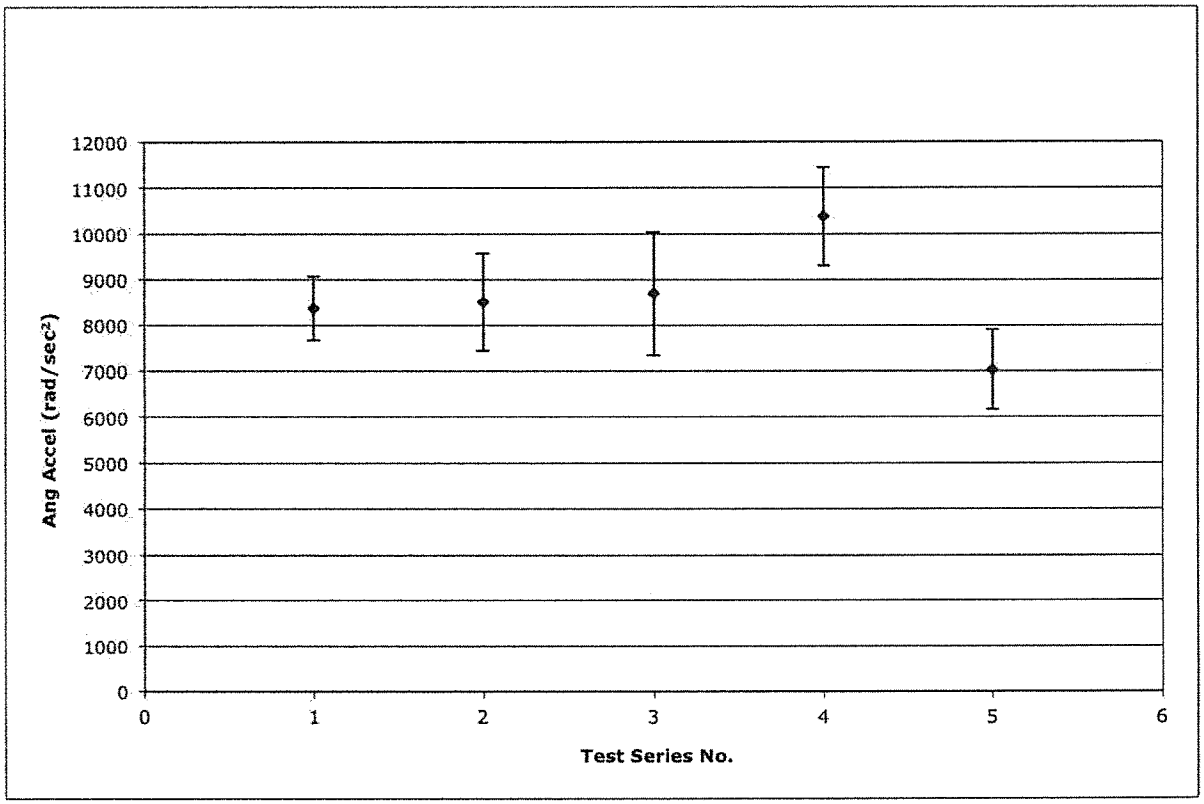

Figure 4. Mean peak sagittal plane angular head accelerations for each test series. (Error bars represent 95% confidence intervals.)

Biomechanical Response of the Infant Head to Shaking - An Experimental Investigation
(doi: 10.1089/neu.2016.4687)
This paper has been peer-reviewed and accepted for publication, but has yet to undergo copyediting and proof correction. The final published version may differ from this proof.

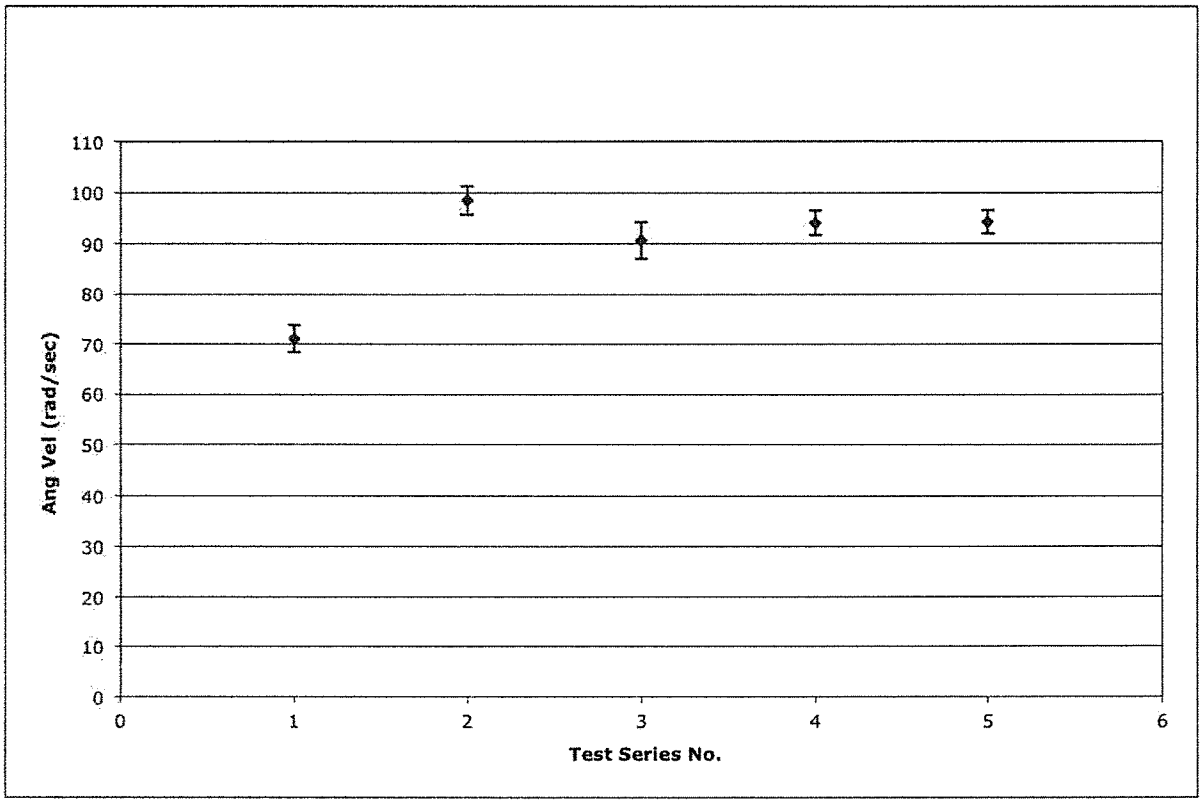

Figure 5. Mean sagittal plane peak angular head velocity for each test series. (Error bars represent 95% confidence intervals.)

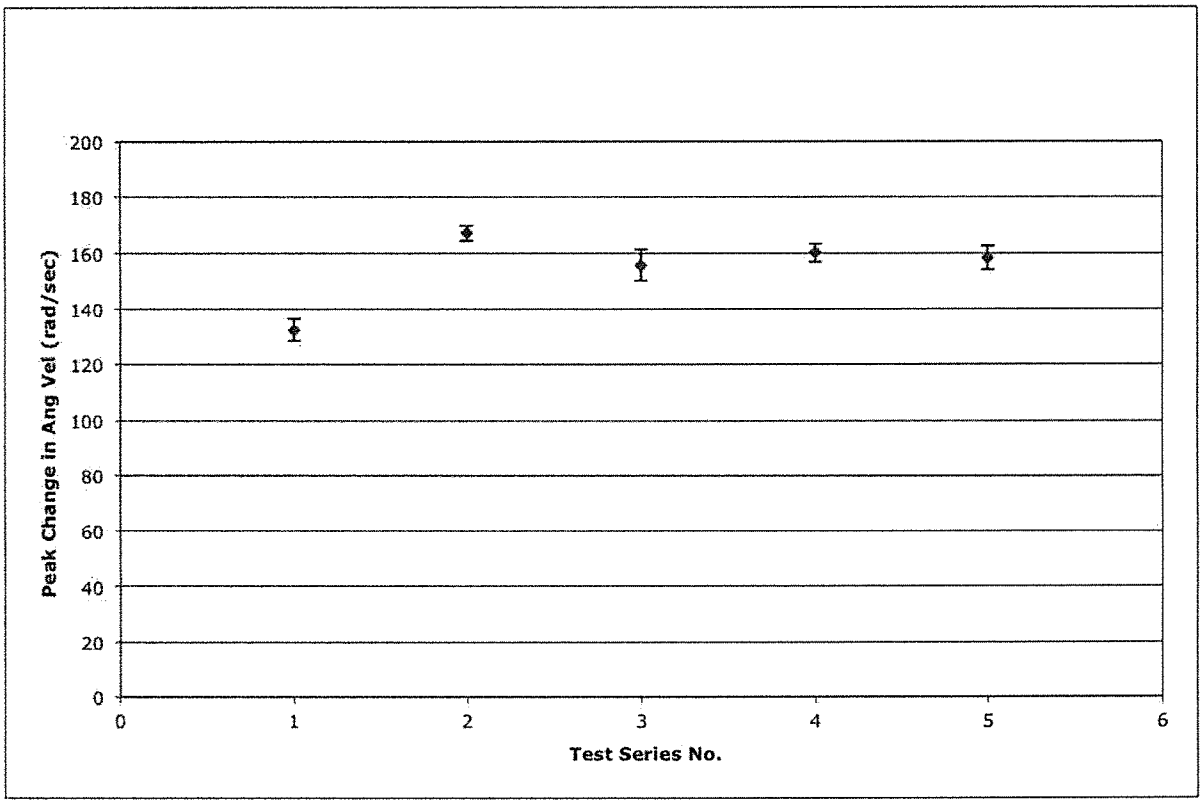

Figure 6. Mean peak change in sagittal plane angular head velocity for each test series. (Error bars represent 95% confidence intervals.)

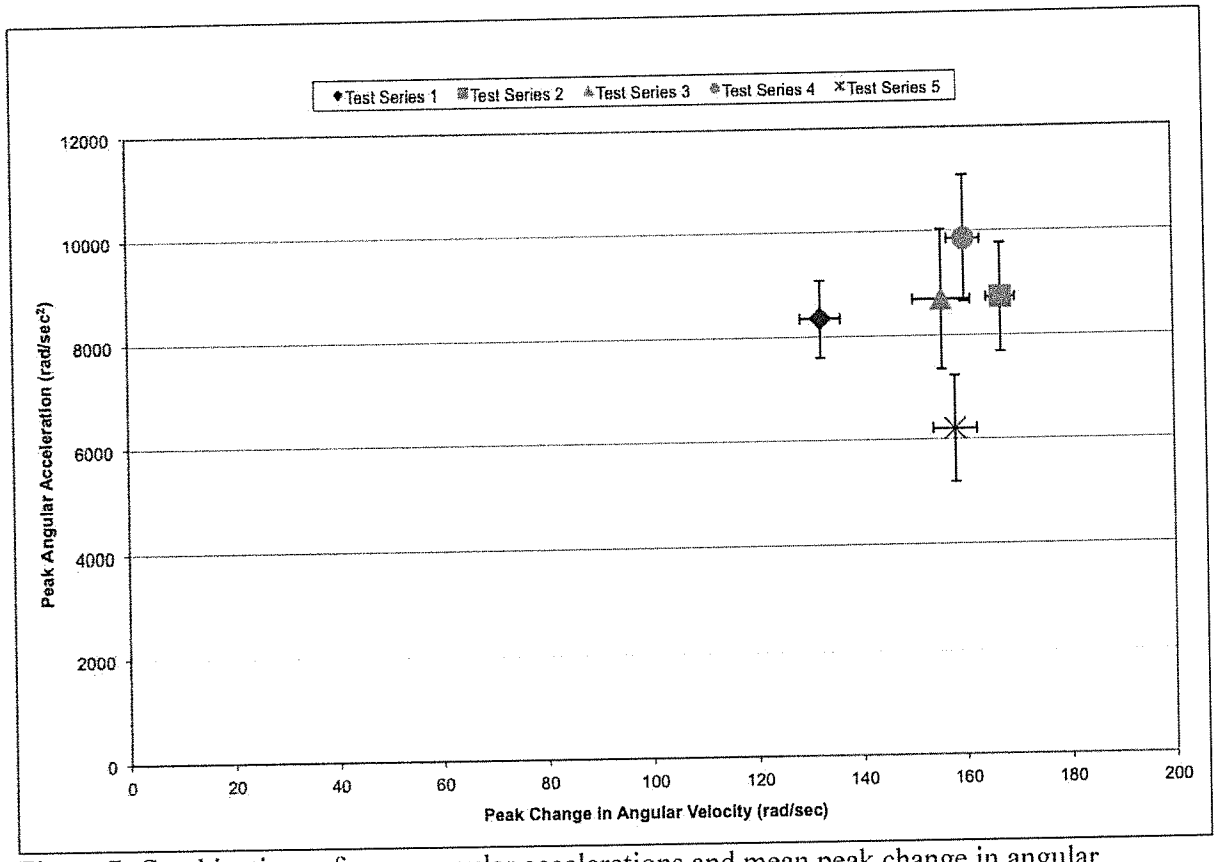

Figure 7. Combinations of mean angular accelerations and mean peak change in angular velocities for each test series. (Error bars represent 95% confidence intervals.)

This paper has been peer-reviewed and accepted for publication, but has yet to undergo copyediting and proof correction. The final published version may differ from this proof.

Table 2. Peak angular head acceleration, peak change in angular head velocity and peak angular head velocity for each test series.

Test Series No.	Peak Angular Head Acceleration (rad/sec^2)	Peak Change in Angular Head Velocity (rad/sec)	Peak Angular Head Velocity (rad/sec)
1	10,630	143	80
2	12,150	174	106
3	12,030	174	104
4	13,260	173	102
5	9,613	167	100

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IN THE CIRCUIT COURT, SEVENTH JUDICIAL CIRCUIT, IN AND FOR FLAGLER COUNTY, FLORIDA

CASE NO.: 18-00169-CFFA

STATE OF FLORIDA

V.

DEVIAUN ANTRIEL TOLER/

NOTICE OF FILING ARTICLES FOR DAUBERT HEARING

YOU ARE NOTIFIED that the Office of the State Attorney, by and through the undersigned Assistant State Attorney has on this date filed with the above Court the following articles:

1. "The New Science of Abusive Head Trauma," Daniel Lindberg, et. al, *International Journal on Child Maltreatment Research, Policy, and Practice*, July 2019.
2. "Acceptance of Shaken Baby Syndrome and Abusive Head Trauma as Medical Diagnosis," Sandeep Narang, M.D., *The Journal of Pediatrics*, July 2016.
3. "Biomechanical Response of the Infant Head to Shaking- An Experimental Investigation," Carole Jenny, M.D., *Journal of Neurotrauma*, 2016.

CERTIFICATE OF SERVICE

I HEREBY CERTIFY that a true and correct copy of the foregoing has been furnished via U.S. Mail to JOHN S HAGER, HAGER AND SCHWARTZ P.A., 140 SOUTH BEACH STREET SUITE 310, DAYTONA BEACH, FL 32114, on July 8, 2021.

Respectfully submitted:

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ORIGINAL PAPER

The "New Science" of Abusive Head Trauma

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Abstract

Claims that new science is changing accepted medical opinion about abusive head injury have been made frequently in the media, legal publications, and in legal cases involving abusive head trauma (AHT). This review analyzes recently published scientific articles about AHT to determine whether this new information has led to significant changes in the understanding, evaluation, and management of children with suspected AHT. Several specific topics are examined as follows: serious or fatal injuries from short falls, specificity of subdural hematoma for severe trauma, biomechanical explanations for findings, the specificity of retinal hemorrhages, the possibility of cerebral sinus thrombosis presenting with signs similar to AHT, and whether vaccines can produce such findings. We conclude (a) that the overwhelming weight of recent data does not change the fundamental consensus, (b) that abusive head trauma is a significant source of morbidity and mortality in children, (c) that subdural hematomas and severe retinal hemorrhages are commonly the results of severe trauma, (d) that these injuries should prompt an evaluation for abuse when identified in young children without a history of such severe trauma, and (e) that short falls, cerebral sinus thrombosis, and vaccinations are not plausible explanations for findings that raise concern for abusive head trauma.

Keywords Abusive head trauma · Retinal hemorrhages · Subdural hematoma

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> New scientific research has cast doubt on the forensic significance of this triad, thereby undermining the foundations of thousands of ... convictions. (Tuerkheimer 2009)

New scientific evidence that changes the generally accepted understanding of the medical community may justify an appeal in criminal cases where medical evidence was important in the court's conclusion. Recent claims that new science has changed the mainstream understanding of the forces and mechanisms of brain injuries in children, such as the quote above, are made in the popular media, legal articles, and criminal proceedings ("Cavazos 2011; Cenziper 2015; "Del Prete 2014; "State of Wisconsin v and Edmunds 2008; Tuerkheimer 2009, 2010). Because the medical diagnosis of abusive head trauma (AHT) also implies that a crime has occurred, scientific progress that substantially changes the way that AHT is understood or diagnosed can have profound effects for people accused or convicted of child physical abuse. However, in other areas relevant to child physical abuse, scientifically unsupported hypotheses such as *temporary brittle bone disease* or *infantile rickets* have also been submitted as new science, only to be widely discredited (Keller and Barnes 2008; Mendelson 2005; Paterson and Monk 2011; Slovis et al. 2012; Spivack and Otterman 2010; Sprigg 2011; Strouse 2013).

The objective of this review is to examine the concepts frequently submitted as "new science" in medical-legal cases of alleged abusive head trauma. The authors chose the topics based on their experience in legal settings. These include serious or fatal injuries from short falls, specificity of subdural hematoma for severe trauma, biomechanical explanations for findings, the specificity of retinal hemorrhages, the possibility of cerebral sinus thrombosis presenting with signs similar to AHT, and whether vaccines can produce such findings. With the exception of one author (RMR), who retired in 2010 after practicing child abuse pediatrics for more than 40 years, all authors are practicing child abuse physicians who have testified in US courts in cases of alleged AHT.

Serious or Fatal Injuries from Short falls

Case Presentation

A 4-month-old boy reportedly rolled off his parents' bed and was found crying on a hardwood floor. He was inconsolable, refused his bottle, and vomited. His parents called 911, and emergency medical personnel recorded that he had a seizure *en route* to the hospital. On arrival, he was comatose and swelling was noted on his scalp in the right parietal region. A head CT scan revealed a thin, bilateral subdural hematoma (SDH) with loss of gray-white differentiation. The retinal exam showed bilateral extensive retinal hemorrhages (RHs). The skeletal survey was negative. The patient died 3 days later. An autopsy confirmed the radiographic findings, and no other injuries were identified.

Analysis

The core question is whether the history of a short fall represents a plausible explanation for the injuries. Falls are the initial explanation in up to 70% of cases where children are ultimately diagnosed with abusive injuries (Duhaime et al. 1992; Leventhal et al. 1993; Scherl et al. 2000; Strait et al. 1995).

Three publications have suggested that short, accidental falls can be life-threatening or fatal. First, Plunkett and others (Plunkett 2001) examined records from the United States Consumer Product Safety Commission National Injury Information Clearinghouse to identify all head and neck injuries involving playground equipment from January 1988 through June 1999. This study identified 18 cases where fall-related head injuries led to fatalities, with 5 of them occurring at home. Cases included children aged 12 months–13 years and the height of falls (measured from the lowest part of the body to the ground) was 0.6–3 m (2–10 ft). The authors reported that 12 falls were witnessed by someone other than the caretaker, and 12 had a "lucid interval" (i.e., a period where the child appeared mostly well). Among six children with the retinal examination, 4 were reported to have had bilateral RHs (the nature of which was not described).

The authors noted several limitations to their study: six falls were not independently observed, eight falls did not have a reported height, and other included falls were from significant heights—at least 5 falls were from 5 ft or more, and the highest fall was from 10 ft. The study has also been criticized because none of the subjects was less than 12 months old, and only nine were less than 5 years old. None had formal retinal examinations, and only 1 of the 5 children less than 2 years of age had a witnessed fall.

The second commonly cited article (Hall, Reyes, Horvat, Meller, & Stein, 1989) was a retrospective study of pediatric deaths from the Cook County Medical Examiner's office over a 4-year period (1983–1986). Fatalities in 18 children with falls less than 3 ft and 18 children with falls between 3 ft and five stories were described, including one 8-month-old child who was dead on arrival after a reported fall from a couch to a hardwood floor, and who was found to have a large SDH.

There are several limitations to this study. The brief "Methods" section reveals that not all subjects had a full autopsy, that medical records were not available to the authors, and that radiographs were not used to evaluate for fractures. Further, because only the mean ages of subjects were given, it is not possible to know how many children were infants, and few details were provided about the reported mechanisms of injury.

Finally, a case report (Gardner 2007) involves an 11 month old who was reported to have fallen backwards from a sitting position to a carpeted floor. The child's 5-year-old brother reportedly witnessed this event. The infant was found to have a SDH requiring surgery and diffuse multi-layer RHs. A skeletal survey was negative. Treating physicians were divided about whether the injury could have been the result of the history provided. The author concluded that "if RHs can [ever] occur without shaking ... , they are of no value in determining etiology." (p. 435) The clear concern with this article is whether the account attributed to the young sibling was accurate. Some skepticism seems warranted. The fall described is particularly minor. If such falls resulted in serious injuries, one would expect this to be a rather common occurrence. It is not.

Although these publications are often offered as "new science," more recent data contradict these results. Chadwick and colleagues published data from 317 children

who presented after a reported fall to a single trauma center in San Diego (Chadwick et al. 1991). Among 65 children who fell from 5 to 9 ft, and 118 children who fell from 10 to 45 ft, there were zero and one fatality, respectively. However, among 100 children with falls reported from less than 4 ft, there were seven deaths. The child who died from the high fall died of sepsis after a prolonged hospital course. Conversely, the seven short fall deaths died of head injury and all had associated injuries or concern about the accuracy of the history provided. If death is a rare outcome from a "high" fall, it seems quite unlikely that short falls would be fatal. The authors concluded that deaths reported as the result of falls less than 4 ft raise the suspicion that the history is inaccurate.

In 2008, Chadwick and colleagues performed a systematic review to determine the risk of death resulting from falls less than 1.5 m (approximately 5 ft) in children younger than 5 years old. This review examined data from five book chapters, seven literature reviews, three public injury databases, and 177 peer-reviewed articles categorized according to sample characteristics and methodology. This included a review of California's statewide injury database, which reported a maximum of 13 short fall deaths among 2.5 million California children over a 5-year period. In this population, six fatalities were identified that might have been the result of short falls. Five articles described reliably witnessed falls in 560 children with hospital falls; none died. Twenty-five studies of children in large licensed daycares had only two fatalities attributed to falls in this setting. After data from all the above data sources, the authors concluded that the risk of death from a short fall is less than 1 in 2,000,000 per year.

Although science cannot prove that something is "impossible," these data suggest that death or serious injury in a young child from a short fall is *exceedingly* rare and that all such deaths should be carefully investigated with a detailed history, physical examination, radiographic and laboratory studies, and a full autopsy.

The Specificity of SDH

Proponents of "new science" use a straw man to argue that new science is changing mainstream opinion. This straw theory—"triad theory"—asserts that clinicians simplistically diagnose abuse in any case that has SDH, any RHs, and any cerebral edema. While this straw theory is ridiculous on its face (abuse is not raised when these findings are seen in children who present after being hit by a car, or with a known, severe crush injury), the underlying argument deserves to be addressed. Most doctors view most significant SDHs to be the result of significant trauma. New science advocates suggest that SDHs are commonly the result of minor trauma or other non-traumatic causes that doctors fail to consider.

Many theories have been postulated as to how SDHs can happen without trauma, and it is beyond the scope of this review to address all of these. While Ehlers-Danlos syndrome, dysphagic choking, and osteogenesis imperfecta (among others) are occasionally put forward as explanations for SDHs, the most common arguments are rooted in three concepts: (a) that children commonly have asymptomatic SDHs resulting from birth, (b) that SDHs can occur spontaneously in children with enlarged extra-axial

spaces, and (c) that sudden and catastrophic clinical decline can result from bleeding into an existing asymptomatic SDH.

Although some of these concepts are supported by the literature, ultimately, the conclusion that significant SDHs are commonly atraumatic is unsupported. To understand why, it is important to ask two questions. First, *can* SDH occur with little or no trauma? Second, when it does, what are the symptoms and prognosis?

SDH from Birth

With the evolution of fast magnetic resonance (MR) imaging techniques that image the infant brain without radiation or sedation, two groups of investigators have identified asymptomatic SDHs in a significant proportion of neonates. Looney et al. (2007) performed an MRI on 96 asymptomatic neonates. They identified SDH in 16 cases including two children who also had sub-arachnoid hemorrhages and five with intra-parenchymal hemorrhages. All affected children were born by vaginal deliveries and all hemorrhages were infra-tentorial or low in the occipital lobe.

A second group had similar results published 1 year later (Rooks et al. 2008). Of 101 asymptomatic neonates, 46 were found to have SDH on MRI. All were supra-tentorial and posterior, and < 3 mm in width. In contrast to Looney et al. (2007), this group did not identify other intracranial injury types. These authors also performed serial imaging to assess the natural history of these SDHs and showed that 94% had resolved by 1 month, and 100% resolved within 3 months. At 2-year follow-up examinations, six children (14%) had a speech delay and one was being evaluated for an autism spectrum disorder, but none had deficits clearly related to trauma. None had a sudden collapse.

Enlarged Extra-Axial Spaces

It has been well-described that the extra-axial space enlarges in older adults as the brain shrinks relative to the skull. That minor trauma can rarely cause SDH in elderly people led some to suggest that this could also be true in children with enlarged subdural spaces. If tearing of the bridging veins is the source of traumatic SDH, the theory goes; the widened extra-axial spaces may increase the risk for bridging veins to stretch and break, causing SDH.

Two articles have addressed this theory in children who had an MRI for macrocrania. The first, by McKeag et al. (2013), reviewed records from 177 children < 2 years old from the Children's Hospital of Philadelphia, where MRI for macrocrania showed enlarged extra-axial spaces. Of these, four (2.3%) had SDH. Each of these had a thorough evaluation for abuse. This was negative in three of the children. One was thought to have been abused based on the finding of multiple rib fractures in different stages of healing.

The second article, by Greiner et al. 2013a, identified 108 children less than 2 years old with enlarged extra-axial spaces among 168 children imaged for macrocrania at the Cincinnati Children's Hospital. Six children (3.6%) had SDH, all asymptomatic. Only two children with SDH were evaluated for abuse, and 1 was found to have characteristic RHs and was diagnosed as abuse.

An article from investigators in France (Vinchon et al. 2004), who prospectively collected data for several years in cases of pediatric SDH, found 16 cases in which they concluded that SDH had occurred without trauma. Twelve of these children had macrocrania, and others had illnesses such as severe dehydration. These authors conclude that "spontaneous" SDH does exist but it is rare and cannot be diagnosed without thorough evaluation for AHT. They state that the absence of traumatic features is insufficient to diagnose spontaneous SDH and conditions like macrocrania, severe dehydration, or arachnoidomegaly are necessary for the diagnosis.

The answer to the question "Is there new evidence that suggests that SDH can occur from birth, or without major trauma?" is "Yes." But this simple conclusion is not the end of the discussion. It bears emphasis that each child with a SDH in these series was asymptomatic: none presented in a coma, and no child was thought to have non-abusive SDH when other injuries or RHs were present. Each article cited contains statements emphasizing the need to consider AHT in cases with unexplained SDH. Small, asymptomatic SDHs in the absence of traumatic injuries are not the focus of most abuse investigations. In most cases of AHT, the child has sudden, obvious symptoms of traumatic brain injury or other signs of trauma. To change the accepted opinion of AHT, the onus is on "new science" proponents to show how asymptomatic SDHs can rapidly evolve, leading to sudden neurological collapse.

Re-Bleeding into SDH

Evidence of re-bleeding in adults and children has been known for decades and is based on follow-up imaging showing enlarged SDH or new hyperdense blood products in an existing or old SDH. Although recent studies disproved the dogma that CT is able to determine the age of a SDH by density (Bradford et al. 2013; Tung et al. 2006; Vinchon et al. 2004), it is accepted that re-bleeding can occur in a SDH without significant additional trauma. Whether this is because bridging veins are stretched or whether vascularized neomembranes bleed (or both) is currently unknown, but does not affect the understanding of whether re-bleeding is likely to produce the findings of AHT.

The key question is whether re-bleeding causes symptoms likely to be mistaken for AHT. There is less direct evidence to address this question. One article describes two cases where re-bleeding is suggested to have caused symptom progression (Hymel et al. 2002). In one case, a 20-month-old child with a known, 3-month-old SDH presented with 1 week of fussiness after hitting a windowsill. In the next, an 11 months old presented with vomiting and was found to have a SDH. Later that night, increased swelling and a more hyperdense SDH was seen on CT. These cases do not directly address the question at hand. In the first case, it is not clear that the child had increased symptoms, and in the second, it is not clear that the mildly increased symptoms were not the result of the simple progression of the acute SDH. In neither case did the child have sudden severe symptoms after being asymptomatic.

Although there are no new data that directly address the question of whether minor trauma into a known SDH results in re-bleeding and sudden collapse, this concept is implausible, given the natural history of known SDHs. While SDHs commonly resolve over weeks to months, children with known asymptomatic or mildly symptomatic SDH are discharged from the hospital after a few days. They are discharged without

protective helmets or other devices to prevent the minor trauma that is ubiquitous in the life of normal children. Yet children have not been reported to return with sudden, devastating symptoms after being discharged. Bradford et al. reported that, of 105 children with SDH, 17 (16%) were found to have re-bleeding, but none had symptoms (Bradford et al. 2013). The Pediatric Emergency Care Applied Research Network (PECARN) (Kuppermann et al. 2009) examined more than 10,000 children less than years old who presented to emergency departments for evaluation of traumatic brain injury. Robust follow-up procedures were employed to detect missed traumatic brain injuries. Results showed that asymptomatic children without loss of consciousness or severe mechanism of injury have < 0.02% chance of clinically important brain injury—including the potential for that brain injury to be identified days or weeks after presentation. In short, sudden symptoms as a result of re-bleeding into a SDH is not a known phenomenon among children with demonstrated SDH—it is only proposed for children where the original SDH is itself a theoretical entity.

Although there is new science about pediatric brain injury, it does not change the accepted understanding of AHT. In sum:

1. Small, asymptomatic SDH can occur from normal birth;
2. Small, asymptomatic, non-abusive SDH rarely occur in children with enlarged subarachnoid spaces:
3. CT imaging does not reliably estimate the age of SDH based on density.

Nevertheless, the vast majority of symptomatic SDH are the result of significant trauma; sudden, significant neurological symptoms in the setting of SDH imply a recent, significant traumatic event.

Biomechanics

It is sometimes suggested that biomechanical data offer a more pure and scientific approach than studies of human beings. Some proponents of "new science" have suggested that biomechanical evidence should change our understanding of the forces that are necessary or capable of causing traumatic brain injury.

In one of the first studies looking at the nature of the forces involved when a child is shaken, Duhaime et al. (1987) used two arguments to say that serious or fatal AHT injuries require more force than shaking alone. In the first phase, they carefully examined 13 consecutive dead infants diagnosed with the shaken baby syndrome (SBS) and found some evidence of head impact as well. They concluded that all cases of SBS must have some component of impact. In the second part, they constructed a doll model and measured peak accelerations. They first shook the doll (no impact) and compared peak accelerations to a trial in which they shook the doll and also allowed it to hit a bar (impact). Unsurprisingly, the sudden stop of an impact created a larger peak acceleration (deceleration). When the values were compared against monkey data and extrapolated, the authors concluded that shaking alone could not yield sufficient forces to cause serious brain injury.

Alexander et al. (1990) looked prospectively at 24 cases of diagnosed SBS. In addition, they were carefully examined for signs of trauma, often with MRI. Nine of the children died. There was not even marginal evidence of impact trauma in 12 of the cases, including five of the children who died. Shaking with impact (12 cases) and shaking without impact (12 cases) were both found. If impacts were significant in these AHT cases, as hypothesized by Duhaime et al. (1987), then all of the dead children should have shown impact. This was not found. Following this study, there has been no large study of children with AHT that has found all children to have signs of impact. Typically, about 50–70% of children have impact evidence along with shaking. Thus, the Duhaime hypothesis has been disproved. Shaking alone can cause serious and fatal injuries (Starling et al. 2004).

Duhaime et al.'s doll experiment, like others, have also been criticized for comparing only a single shake to a single impact—concluding that a single shake alone could not cause the injuries seen in AHT. However, perpetrators who confess to shaking have reported multiple shakes within seconds (Adamsbaum et al. 2010; Starling et al. 2004). The cumulative effect of these repetitive shakes was not modeled and would seem to be greater than that of a single shake.

Using a pig model, Raghupathi et al. (2004) shook 3–5-day-old anesthetized piglets once or twice with a 15-min gap. The mechanical shakes were rapid (< 15 m/sec) and did not involve impact. At 6 h post-injury, the brains were examined. More injury was seen with the double shake trial, showing that immature brains are vulnerable to repeated, relatively mild, non-impact loading conditions.

Bandak (2005) compared calculated structural failure limits of the cervical spine and deduced that the spine should be injured with less force than the brain. Clinically, this phenomenon is not seen; hence, the mechanism of shaking should be re-evaluated.

One response by Margulies and colleagues (Margulies et al. 2006) noted that Bandak made multiple numerical errors in his analysis, often off by a factor of 10. "There is no single, simple explanation responsible for the errors that appear in *every* value in Table 3." (Margulies et al. 2006, p. 278). When accurately calculated, they argued that the brain can indeed be injured without neck injury—even using Bandak's (2005) suppositions. They concluded that neck injury could occur during severe shaking without impact but that it would not necessarily occur if the shaking caused brain injury. Rangarajan and Shams (2006) also pointed out these numerical miscalculations and unclear assumptions. They noted that Bandak referenced three presentations at a conference in which the chair of the workshops said the material should not be used as references.

Using a mannequin, Wolfson et al. (2005) showed that concussion levels of energy could be achieved and raised concern about using models of single trauma to explain a phenomenon (shaking) that is often repetitive. "It is unlikely that further gross biomechanical investigation of the syndrome will be able to significantly contribute to the understanding of SBS." (p. 70) "Current injury criteria are based on high-energy, single-impact studies. Since this is not the type of loading in SBS it is suggested that their application here is inappropriate and that future studies should focus on injury mechanism in low-energy cyclic loading." (p. 70)

Biomechanical arguments asserted as part of the "new science" may also rest on extreme assumptions that do not seem to fit what we know of the natural world:

1. Heads are often treated as a single mathematical point. With 80–100 million neurons, layers of the brain of different densities, and a non-spherical shape, the human brain is over simplified when reduced to a simple point or shape.
2. Forces are complex. Repetitive shaking can result in translational movement in 3 dimensions, as well as spinning and shear forces, simultaneously. It is not clear that a measurement such as "peak acceleration" is the key factor to consider versus any number of forces acting over time.
3. Reductionist thinking often begins by quoting Newton's second law: force equals mass times acceleration. This basic concept considers only simple, solid shapes (instead of complex biological tissue), and non-complex forces (straight back and forth) and does not account for repetitive forces. A common history in cases of AHT is that the child fell off the sofa. Compare that to an adult falling off a sofa. Acceleration = gravity which is a constant on earth. The equation then reduces to force is proportional to mass. For a tumble off a sofa, this means the bigger the force the harder the fall. One would expect an adult with a far greater mass to hit the floor far harder. Yet no one claims sofa falls create adult fatalities, and suicidal adults would never choose a couch in favor of bridges or high heights.

Biomechanical modeling attempts to simplify and understand forces and consequences in nature. Such modeling can be useful but is limited as to whether it faithfully represents the real world. The "new science" proponents selectively cite some biomechanical hypotheses as representing what happens rather than adjusting those models to what is actually observed. At most, biomechanics is a long way from explaining the injuries seen with shaking and shaking with impact.

Retinal Hemorrhages

Just as SDH have been suggested to be non-specific for severe trauma, so too have RHs. Proponents of "new science" assert that RHs can be caused by a wide array of medical and traumatic injuries. Although this fact has been known for decades, it ignores the types and patterns of RH and their significance in determining their origin.

The contemporary understanding of RH is summarized in an article comparing the prevalence and types of RHs in victims of abuse and those with non-abusive traumatic brain injury. Bechtel et al. (2004) examined 15 children with AHT, and 67 with non-abusive brain injuries. Although RHs were present in nine (60%) abused children, they were only found in seven (10%) non-abused subjects. Furthermore, none of the non-abused cohort had RHs that extended to the periphery of the retina. These data suggest that while RHs can occur from non-abusive trauma, these RHs are mild. Mainstream opinion has therefore held that RHs that are multi-layered, numerous, and extend to the periphery of the retina are specific for abuse (Levin 2010).

The "new science" addressing RHs began with a case report of a child who was found to have severe RHs and perimacular folds after sustaining severe brain injury because of a crush injury from a heavy television (Lantz et al. 2004). Another case report from the same group reported a fatal SDH and severe RHs after an unwitnessed stairway fall (Lantz and Couture 2011). As discussed above, a fatality from such a stairway fall is at least very unusual, though this child was also noted to have a partial

thromboplastin time (PTT) > 200, implying a severe coagulopathy existed either before or after the severe brain injury.

The prevalence of RHs has also been addressed in children who are critically ill without trauma. Agrawal et al. (2012) examined 159 consecutive patients less than weeks old in a London pediatric intensive care unit. Subjects with AHT were excluded. The number of RHs was rated as mild (1–4), moderate (5–20), or severe (> 20). Of the159 subjects, 24 (15%) were found to have any RHs. Mild RHs were seen in 16 subjects, most of whom had sepsis. Two subjects had moderate RHs—one with sepsis and one with a television crush head injury. Six children were found to have severe RHs—three had leukemia and severe sepsis; one had hemorrhagic disease and a short fall and two had fatal traumatic brain injury (non-abusive).

Similarly, Longmuir et al. (2014) reported results of retinal examinations in 85 intubated PICU patients. Six patients (7%) had any RHs, including four children with AHT; all of whom had severe RHs. The other two children with RHs included one with moderate RHs from a TV crush injury, and one with mild RHs after a cardiac arrest attributed to SIDS.

Without evidence, the hypothesis has been raised that vaccines may cause retinal hemorrhages in the absence of trauma (Clemetson 2004; Gardner 2005; Squier 2011). Binenbaum et al. (2015) reasoned that if vaccines were a cause of RHs, then RHs would be seen frequently and would be temporally associated with immunization. They conducted a retrospective cohort study from June 1, 2009 through August 30, 2012, at the Pediatric Ophthalmology Clinics of the Children's Hospital of Philadelphia. They examined 5177 children 1–23 months old who were undergoing dilated funduscopic exam for any reason.

The outcomes and measures they examined were the prevalence and cause of RHs and the temporal association between vaccine injections within 7, 14, and 21 days preceding these examinations and RHs. Reasons for dilated examinations in these children included strabismus, amblyopia, red eye, trauma, tear duct obstruction, poor visual behavior, or systemic diseases with ocular findings. The inclusion criterion was the availability of vaccine records. Excluded from the study were a history of direct eye trauma, intraocular surgery, and retinopathy of prematurity stage 3 or worse.

Among the 5177 children included (with 7675 fundus examinations), nine (0.17%) had RHs and all of these were victims of abusive head trauma (AHT) diagnosed with non-ocular findings including intracranial hemorrhage (9), skull fracture (5), bruises (3), hypoxic-ischemic brain injury (2), and spinal fracture, spinal hematoma, or perpetrator confession (1 each).

Vaccination records were available for 2210 (with 3425 fundus examinations). These vaccines included pneumococcus; diphtheria, tetanus, and pertussis (DTP); Haemophilus influenza type B (HIB); polio; hepatitis B; measles-mumps-rubella (MMR); and varicella. Four of the 2210 children (0.18%) with vaccination records had RHs. None of these 4 had vaccinations within 7 days preceding the diagnosis of RHs. One had vaccination within 14 days.

The conclusion of the investigators was that there was no association between receiving a vaccination injection and the presence of RHs in the subsequent 7, 14, or 21 days.

"New science" has not changed the understanding of RHs and its association with AHT. As has been known for decades, RHs can be caused by a host of traumatic and medical causes. However, severe RHs—numerous (> 20), multi-layered, and extending to the *ora serrata*—are very specific for severe traumatic brain injury. Although AHT is undoubtedly the most common source of severe RHs, serious crush injuries and children with leukemia and coagulopathy can also have severe RHs. Vaccination, however, does not cause RHs.

Cerebral Sinus Thrombosis

Not a single peer-reviewed paper has been identified linking cerebral sinovenous thrombosis (CSV) with other signs of AHT. Nevertheless, this hypothesis is suggested in legal proceedings. CSV is a rare disorder in children (0.67/100,000). The single largest age group with CSV is hospitalized neonates, comprising 43% of all cases up to 19 years of age. Less than 10% of cases have extra-cerebral hemorrhages. CSV is usually associated with systemic illness (84%) such as dehydration, metabolic acidosis, central nervous system infection, cyanotic heart disease, head injuries, craniotomy, renal, and thromboembolic disorders.

Choudhary et al. (2015) reported data from all children at their center younger than 36 months of age who were diagnosed with abusive head trauma (AHT) and who had both magnetic resonance imaging and MRI venography. The purpose of the study was to define the incidence and characteristics of venous and sinus abnormalities in abusive head trauma cases. The study was conducted from 2001 to 2012. Neuroradiologists independently analyzed MRI and MRI venography.

Forty-five children with a median age of 3 months (range 15 days to 31 months) were included. Sixty-two percent were boys. RHs were seen in 71%, extra-cranial fractures in 55%, and in 91%, a CT or MRI showed SDH. AHT was diagnosed by consensus of the treating medical team, perpetrator confession, and/or a judicial ruling. An experienced pediatric radiologist reviewed all of the skeletal surveys.

Thrombosis was defined as the absence of veins or sinuses on 3-D phase contrast MRI venography. MRI venography showed a mass effect on the venous sinuses or cortical draining veins in 69% (31/45). This mass effect was either displacement or partial or complete effacement of the venous structures from an adjacent SDH or brain swelling. The "lollipop" sign occurs when the bridging vein terminates in a sub-arachnoid blood clot and consequently does not drain into the sinus. This represents direct trauma to the cortical bridging veins and was seen in 44% (20/45) of the children.

The authors describe the limitations of the study, including technical limitations of MRI venography, the variations in venous anatomy, and in the appearance of venous thrombosis as well as the imaging peculiarities in children, mainly in terms of size of vessels.

The authors conclude that primary cortical sinus and venous thrombosis is a rare disorder, occurring in two to seven cases per million people. In children, predisposing factors are present in up to 95%. Outside the perinatal period, contributing factors include dehydration, malignancy, chemotherapy, iron-deficiency anemia, infection or sepsis, thrombophilia, gene mutations, and oral contraceptives. The clinical

presentation of venous thrombosis is one of progressive, sub-acute decline, often over several days, usually in the context of another identifiable illness.

The Real New Science

For the reasons above, the understanding of AHT has not significantly changed in recent years. One recent survey of hundreds of pediatric specialists at leading children's hospitals directly assessed which causes were considered most likely to cause the findings associated with AHT. Short falls, vaccines, or choking remained fringe theories as explanations for SDH, RH, and coma or death (Narang et al. 2016). But with the relatively recent recognition of child abuse pediatrics as a new subspecialty within pediatrics, it would be surprising to conclude that the science of AHT has not advanced. Indeed, remarkable progress has been made, especially in the recognition, prognosis, and treatment of AHT.

Centers have identified sentinel injuries that should routinely prompt consideration of AHT, including bruises in young infants, unexplained oral injuries, long-bone fractures, and abdominal injuries (Lindberg et al. 2015; Pierce et al. 2016; Sheets et al. 2013). Multi-center networks and systematic reviews have identified features of brain injuries that are most concerning for abuse (Hymel et al. 2014; Kemp et al. 2011). Recently, a decision rule to identify subtle signs of abusive and non-abusive brain injuries in young infants was validated in four pediatric emergency departments (Berger et al. 2016). Other studies have identified broad variability in screening practices for occult abusive injuries and have recommended best practices to move toward an object ive, standardized approach to diagnosing abuse (Greiner et al. 2013b; Harper et al. 2013; Lindberg et al. 2013; Wood et al. 2015a, 2012, 2015b, 2010). Although confidence is waning in CT estimates of the age of SDHs, preliminary work suggests that someday estimation of the timing of RH may improve (Binenbaum et al. 2016). Development of new, fast MRI techniques suggests that screening for AHT will soon be done without the risks of radiation or sedation (Berger 2014; Cohen et al. 2015).

There is much new science with respect to AHT. Without exception, valid, and reproducible methods support the commonly held understanding of AHT that children who present with severe symptoms and who are found to have SDH and RHs are very likely to have been victims of significant trauma (Choudhary et al. 2018).

In a world of finite research resources, the real shame of this review is that so much time and effort is being devoted to research whose only purpose is to counter fringe theories. In any other field, hypotheses and theories based only on case reports, limited biomechanical theory, and aberrant interpretations of radiographs would have self-limited effects. Without reproduction, citations or enduring influence on the field, they would join tens of thousands of forgotten pieces of medical scholarship. It is only the fact that these articles are used in court gives them continued relevance. Ideally, this could be countered by broadly increasing the data literacy of lawyers, judges, jurors, and the public. However, until there is a reliable method by which courts can distinguish the validity of scientific data and thereby differentiate valid methods from fringe theories, it is left to the individual ethics of expert witnesses to communicate correctly the current state of the science.

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Compliance with Ethical Standards

Conflict of Interest The authors have each provided expert witness testimony in cases with alleged child physical abuse.

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Acceptance of Shaken Baby Syndrome and Abusive Head Trauma as Medical Diagnoses

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Objective To assess the current general acceptance within the medical community of shaken baby syndrome (SBS), abusive head trauma (AHT), and several alternative explanations for findings commonly seen in abused children.

Study design This was a survey of physicians frequently involved in the evaluation of injured children at 10 leading children's hospitals. Physicians were asked to estimate the likelihood that subdural hematoma, severe retinal hemorrhages, and coma or death would result from several proposed mechanisms.

Results Of the 1378 physicians surveyed, 682 (49.5%) responded, and 628 were included in the final sample. A large majority of respondents felt that shaking with or without impact would be likely or highly likely to result in subdural hematoma, severe retinal hemorrhages, and coma or death, and that none of the alternative theories except motor vehicle collision would result in these 3 findings. SBS and AHT were comsidered valid diagnoses by 88% and 93% of the respondents, respectively.

Conclusions Our empirical data confirm that SBS and AHT are still generally accepted by physicians who frequently encounter suspected child abuse cases, and are considered likely sources of subdural hematoma, severe retinal hemorrhages, and coma or death in young children. Other than a high-velocity motor vehicle collision, no alternative theories of causation for these findings are generally accepted. *(J Pediatr 2016;■■:■■-■■).*

Although shaking, with or without impact, has been recognized as a dangerous form of child physical abuse since the early 1970s,[1,2] the validity of shaken baby syndrome (SBS) and abusive head trauma (AHT) has recently been called into question in prominent national newspapers such as the *New York Times* and *Washington Post*,[3,4] judicial decisions,[5,6] and some medical literature.[7,8] In fact, a US Supreme Court Justice recently commented in a dissenting opinion that there is widespread "controversy" within the medical community regarding the concepts of AHT and SBS.[9,10] Not surprisingly, this has resulted in confusion in the courts and a chilling effect on child protection hearings and criminal prosecutions.[11]

Legal interventions are an important part of primary safety determinations and secondary prevention for victims of maltreatment. In that process, courts frequently rely on medical expert testimony to opine on the most likely source of a child's injuries. To determine the admissibility of scientific testimony, courts must assess whether concepts are "generally accepted" in the medical community. In approximately one-half of the US jurisdictions, known as Frye jurisdictions, "general acceptance" is the sole criterion for admitting expert testimony on a certain concept.[11] In the remainder of US jurisdictions, known as Daubert jurisdictions, "general acceptance" is one of several criteria used to assess reliability, but is still afforded significant weight.[12] In addition, several professional medical society ethical guidelines for expert testimony state that testimony should reflect generally accepted opinions, and/or that an expert who endorses a minority opinion should volunteer that information.[13-16]

In courts, evidence of what is generally accepted in the medical community has typically been adduced by the opinion of a solitary expert or a small cadre of experts. This approach is susceptible to the biases and knowledge base of the testifying physicians, and leaves open the possibility that a small group could create an incorrect impression about whether or not any particular concept is generally accepted. Courts are ill-equipped to measure the broad opinion of the wider medical field or to assess the validity of a single physician's assessment of that broad opinion. Although SBS has historically been considered a valid medical diagnosis,[17] to date no well-conducted study has measured the acceptance of SBS or AHT as diagnoses, or of the likelihood that shaking will result in subdural hematoma (SDH), retinal hemorrhages (RH), or coma or death, the findings commonly associated with SBS and AHT.[18,19]

Given the importance of this issue to child protection and legal outcomes, we aimed to attain empirical data on the acceptance of SBS and AHT as valid medical

AHT	Abusive head trauma
MVC	Motor vehicle collision
REDCap	Research Electronic Data Capture
RH	Retinal hemorrhages
SBS	Shaken baby syndrome
SDH	Subdural hematoma

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diagnoses by the physicians most commonly involved in those cases. We also sought to determine whether shaking, with or without impact, and other mechanisms of injury are generally accepted as reasonable explanations for SDH, RH, and coma or death.

Methods

This observational survey study was reviewed and approved by the University of Texas-Houston Institutional Review Board, and was conducted between March and October 2015. To identify a feasible sample size and limit enrollment or response bias, we surveyed hospitals identified from the 2014-15 *US News & World Report* Honor Roll of Children's Hospitals.[20] From the 10 leading children's hospitals, we identified faculty physicians (MD, DO) within the specialty departments most commonly involved in suspected AHT cases: Emergency Medicine, Critical Care, Child Abuse Pediatrics, Pediatric Ophthalmology, Pediatric Radiology, Pediatric Neurosurgery, and Child Neurology. Because forensic pathologists are not typically located within children's hospitals, we contacted the medical examiners' offices that jurisdictionally comported with the surveyed hospitals and offered participation in the survey. If no medical examiner's office comported with a particular jurisdiction, we contacted the responsible coroner's office and offered participation in the survey.

We obtained contact information (e-mail and mailing addresses) from hospital websites or physician collaborators. In March 2015, physicians were invited to participate by e-mail, and were informed that the survey was voluntary and anonymous. Using a modified Dillman method,[21] the lead investigator (S.N.) sent an e-mail to eligible physicians, providing a summary of the study's objective and methods, along with a unique, anonymous online link to the survey. After the initial e-mail, nonresponders were sent a reminder e-mail (with survey links) every 2 weeks on 2 separate occasions. If a physician had not completed the survey after 3 e-mail attempts, then a hard copy of the survey (with $1 attached) was mailed to the physician's office address on 2 separate occasions at 2-week intervals. After this, if the participant still had not responded, he or she was logged as a nonresponder, and his or her contact information was permanently deleted. Data collection efforts were completed in October 2015. As an incentive to improve response rates, participants were entered into up to 5 randomized, biweekly drawings for a $200 gift card (depending on the time of response, with earlier responders being eligible for and entered into more drawings).

To minimize the potential for bias, we did not approach nonresponders and used no additional methods to encourage recruitment by any respondent. To ensure an appropriate sampling frame, we asked each respondent to report his or her specialty on the survey, and those who reported specialties other than those being sought to be surveyed excluded.

Study data were collected and managed using REDCap (Research Electronic Data Capture) tools hosted at the University of Texas at Houston.[22] REDCap is a secure, web-based application designed to support data capture for research studies. No identifying information was recorded in REDCap, and once a physician completed the survey, his or her contact information was permanently deleted, thereby preserving anonymity.

Survey

Each participant reported his or her age (20-30, 31-40, 41-50, 51-60, 61+ years), board certification status, and years in practice (0-5, 6-10, 11-20, 20-30, 31-40, or 41+ years). Each participant was also asked to choose his or her field of specialty from the list of specialties sought (ie, Emergency Medicine, Critical Care, Child Abuse Pediatrics, Pediatric Ophthalmology, Pediatric Radiology, Pediatric Neurosurgery, and Child Neurology), or to report another specialty. Those reporting more than 1 surveyed specialty (n = 8) were included under each specialty for the report of respondent characteristics, but were only counted once in the remainder of the survey. Those reporting a specialty that was included in the sampling frame and a specialty that was not included (eg, Pediatric Emergency Medicine, General Pediatrics) were counted within the included specialty. Those identified within a division of pediatric emergency medicine who listed their specialty as "urgent care" were included with Emergency Medicine. Those listing only exclusion specialties (eg, General Pediatrics, Allergy and Immunology, Anesthesia, Pulmonology) were excluded.

Respondents rated the likelihood of each finding (SDH, RH, coma or death) to result from several proposed mechanisms in a child aged <3 years using a 5-point Likert scale (from "highly unlikely" to "highly likely"). "Severe RH" was defined as too numerous to count, multilayered hemorrhages extending to the periphery. Proposed mechanisms included shaking without impact, shaking with impact against a soft surface (eg, a bed), a very short fall (<3 feet) with impact against a hard surface, a high-velocity motor vehicle collision (MVC), hypoxia, dysphagic choking, vitamin D deficiency rickets, and adverse reaction to vaccines.

Finally, respondents were asked whether they believed SBS to be a valid medical diagnosis (yes, no, don't know/unsure), whether they believed AHT to be a valid medical diagnosis (yes, no, don't know/unsure), and the basis for those opinions (clinical experience, medical literature, both, or neither). Respondents were offered the chance to ask questions or to comment on the survey or the study as a whole by contacting the principal investigator.

For analysis, we defined a "fringe opinion" as one in which <5% of respondents deemed a given mechanism for a finding as likely/highly likely or unlikely/highly unlikely (**Table I**). For analysis of shaking with impact versus shaking without impact results, we defined "discordance" as a rating that changed from highly unlikely or unlikely to likely or highly likely (or vice versa), depending on whether or not impact was present. Descriptive statistics were used to determine the prevalence of each response along with associated 95% CIs. Comparisons were conducted using OR with 95% CI.

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Table I. Fringe opinions

	Likely/highly likely	%	Unlikely/highly unlikely	%
SDH	Vaccines	0.0	Shake WITH impact	3.2
	Vitamin D	2.3		
	Choking	2.7		
	Hypoxia	4.0		
RH	Vaccines	0.0	Shake WITH impact	1.0
	Vitamin D	0.8	Shake NO impact	1.8
	Short fall	3.2		
Coma/death	Vitamin D	0.6	Shake NO impact	3.7
	Vaccines	1.0	Shake WITH impact	4.8
	Short fall	3.1	MVC	3.5
	SBS invalid		4.8	
	AHT invalid		1.0	

A causative mechanism was considered a fringe opinion if the combined percentage of respondents rating it as likely or highly unlikely or as unlikely or highly unlikely was <5%.

Results

The survey was sent to 1378 clinicians, of whom 682 (49.5%) responded. A department of child neurology (n = 22) at 1 institution declined as a block to participate, and were counted as nonresponders. We excluded 54 (8%) survey respondents because they either did not list their specialty (n = 9) or listed only specialties that were not included in our sampling frame (23 general, primary, or hospitalist pediatricians and 22 other pediatric subspecialists). The remaining 628 respondents composed the main cohort for this analysis. Characteristics of the respondents are summarized in **Table II**. Among the respondents, the most common specialties listed were Emergency Medicine, Critical Care, Neurology, and Radiology. The large majority (88.2%) of respondents reported being board-certified in their specialty.

Table II. Respondent characteristics

Characteristics	n (%)
Specialty*	
Emergency Medicine	192 (30.9)
Critical Care	108 (17.4)
Neurology	101 (16.3)
Radiology	96 (15.5)
Ophthalmology	45 (7.2)
Neurosurgery	30 (4.8)
Child Abuse	30 (4.8)
Pathology	27 (4.3)
Board-certified†	548 (88.2)
Age, y‡	
20-30	4 (0.6)
31-40	240 (38.6)
41-50	180 (29.0)
51-60	128 (20.6)
61+	68 (11.0)
Years in practice§	
0-5	148 (23.8)
6-10	135 (21.7)
11-20	164 (26.4)
21-30	106 (17.1)
31-40	48 (7.7)
41+	16 (2.6)

*Sums to 629 because 8 respondents listed 2 specialties.
†Six respondents did not report board certification status.
‡One respondent did not report age.
§Four respondents did not report years in practice.

Ninety-nine respondents (15.8%) omitted answers for at least 1 question. The most common scenarios in which more than 3 responses were omitted were nonophthalmologists omitting questions about RH and, conversely, ophthalmologists exclusively answering questions related to RH. No question was omitted by more than 22 respondents.

Respondents' opinions about the most likely source of SDH, severe RH, and coma or death are shown in the **Figure**. More than 80% of respondents felt that shaking with or without impact was likely or highly likely to produce SDH, more than 90% reported that it was likely or highly likely to produce RH, and more than 78% reported that it was likely or highly likely to result in coma or death. The corresponding results for a short fall were 18%, 3%, and 3%, respectively.

Either SBS or AHT was characterized as a valid diagnosis by 607 respondents (96.7%; 95% CI, 94.9%-97.9%). SBS was endorsed as valid by 554 respondents (88.1%; 95% CI, 85.3%-90.5%); AHT, by 584 respondents (93.0%; 95% CI, 90.7%-94.9%). Pathologists were statistically significantly more likely to be divergent with respect to the validity of AHT and SBS, with 8 of 27 stating that SBS is not a valid diagnosis, but that AHT is valid (OR, 13.5; 95% CI, 4.7-38.1, relative to other specialties) (**Table III**). Two pathologists responded that SBS is valid, but AHT is not.

Among the respondents stating that SBS or AHT is a valid diagnosis, 545 (89.7%) reported that they were informed by both the scientific literature and their own clinical experience, 48 (8%) were informed only by their clinical experience, and 11 (1.8%) were informed only by the scientific literature. One respondent did not answer the question, and 2 respondents listed "other" as the reason for considering the diagnosis valid. With respect to specific findings (SDH, RH, coma or death), the respondents showed very little discordance in their responses according to the presence or absence of impact.

Using our definition of "fringe opinion," 165 respondents (26.6%) reported at least 1 fringe opinion. We also included respondents who stated that either SBS (n = 30; 4.8%) or AHT (n = 6, 1.0%) were not valid. Of the 6 respondents who stated that they thought AHT was not a valid diagnosis, 5 agreed that shaking with or without impact was likely or highly likely to result in SDH and RH. All 5 of these respondents agreed that shaking with impact was likely or highly likely to result in coma or death; 2 of the 5 were neutral about the likelihood of shaking without impact resulting in coma or death. One respondent reported that AHT was invalid, and that shaking with or without impact is unlikely or highly unlikely to result in SDH, RH, or coma or death. This respondent reported that only a MVC or a short fall were likely to result in SDH, no option was likely to result in RH, and only a MVC was likely to result in coma or death.

Discussion

Our survey results represent national, multidisciplinary physician opinions on the validity of SBS and AHT, and of the

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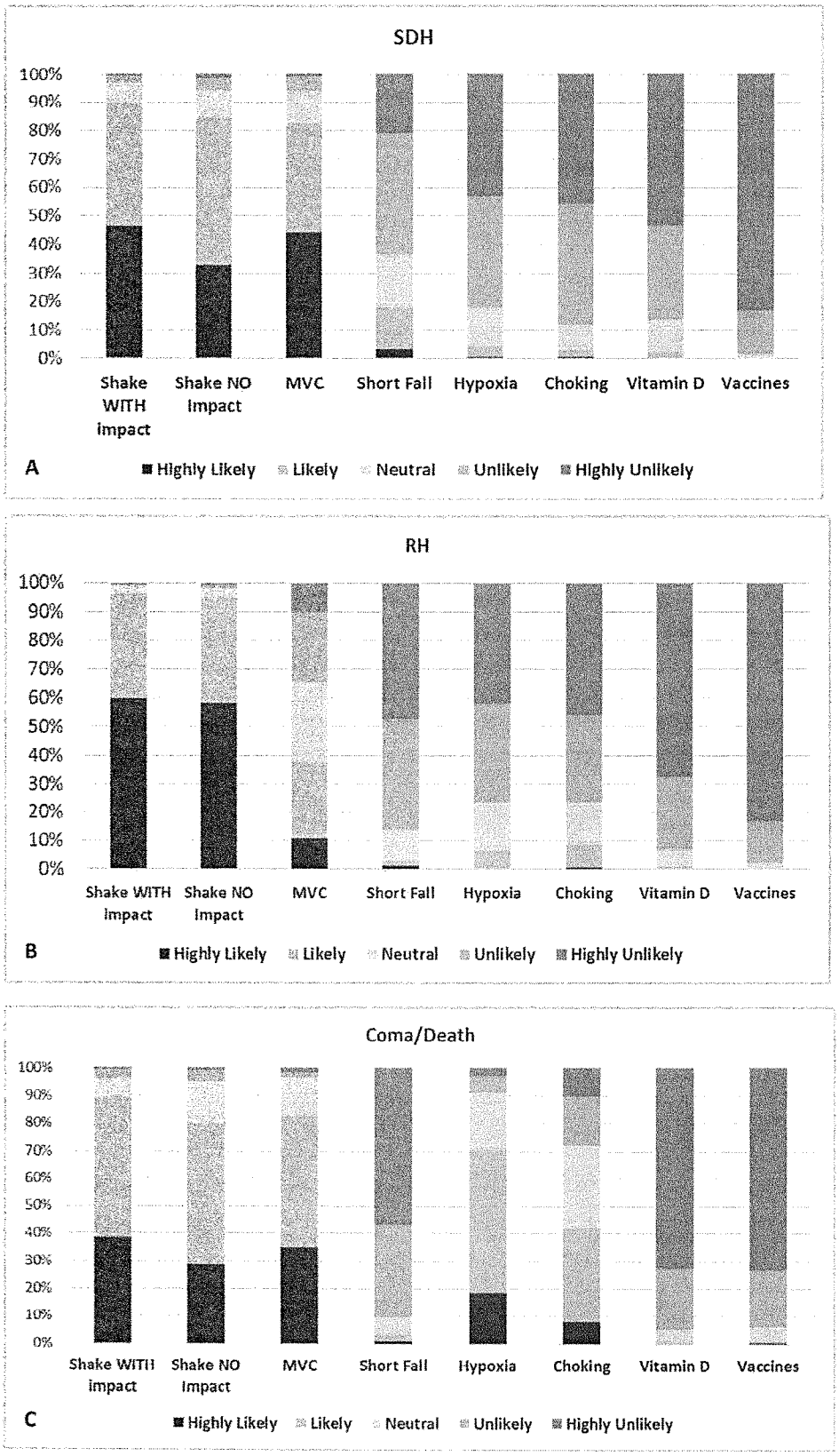

Figure. Percentage of respondents who believe that SDHs, severe RHs, and coma/death would result from the above events.

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Table III. Validity of AHT and SBS by specialty

Specialties	n	Yes, n (%)	No	Don't know/ unsure	Blank
AHT valid					
Emergency Medicine	196	184 (93.9)	0	10	2
Critical Care	108	102 (94.4)	2	3	1
Neurology	103	95 (92.2)	1	5	2
Radiology	96	82 (88.5)	2	8	4
Ophthalmology	46	44 (95.7)	0	2	0
Neurosurgery	30	30 (100.0)	0	0	0
Child Abuse Pediatrics	30	30 (100.0)	0	0	0
Pathology	27	25 (92.6)	1	1	0
SBS valid					
Emergency Medicine	196	175 (89.3)	7	11	3
Critical Care	108	99 (91.7)	2	7	0
Neurology	103	96 (93.2)	4	1	2
Radiology	96	84 (87.5)	2	6	4
Ophthalmology	46	45 (97.8)	0	1	0
Neurosurgery	30	23 (76.6)	5	2	0
Child Abuse Pediatrics	30	28 (93.3)	2	0	0
Pathology	27	11 (40.7)	8	8	0

Totals sum to 636 because 8 respondents listed 2 specialties: 4 for Child Abuse Pediatrics and Emergency Medicine, 2 for Critical Care and Emergency Medicine, and 2 for Critical Care and Neurology.

likelihood that findings commonly seen in those cases—SDH, severe RH, and coma or death—result from various causal mechanisms. Although "general acceptance" is not defined by a definitive numerical threshold in legal settings (although acceptance by <50% of field clearly would not meet the criterion for "general acceptance"), our results provide empirical data that clearly support the conclusion that SBS and AHT are still generally accepted as valid medical diagnoses across a broad range of specialties. Furthermore, our data show that shaking with or without impact (in contradistinction to several other alternative theories) is generally accepted to be a dangerous form of child physical abuse and capable of producing SDH, RH, and coma or death. Several alternative explanations that have been proposed to cause SDH, RH, and coma or death are not generally accepted. This high degree of consensus, irrespective of specialty, experience, or age, refutes recent reports in the lay press and legal commentary of a substantial controversy within the medical community regarding SBS and AHT. Other authors have discussed the various motivations for those media sources to proffer such assertions.[22,23]

As a specialty, forensic pathologists were discordant from other respondents, being more likely to question the validity of SBS as a diagnosis, although not more likely to question the validity of AHT (**Table III**). In this respect, our results are similar to the results of a survey of forensic pathologists that showed 35% questioning SBS.[23] That survey did not address the topic of AHT separately from SBS, however.

Our survey results demonstrate that physicians, irrespective of specialty, viewed the risks of shaking, with or without impact, to be similar to a high-velocity MVC and dissimilar to a very short fall. Although this finding may seem unremarkable to clinicians, it is important in light of some biomechanical literature arguing that shaking without impact cannot generate sufficient forces to cause SDH,[24,25] and biomechanical[24] and pathology[26] literature suggesting very short falls as a reasonable explanation for those findings. We believe the divergence of our results from this literature represents a recognition of the limitations of biomechanical data, a primacy of clinical literature and experience in relation to that literature, or both.

Our study has several limitations. First, we did not include general pediatricians in our sampling frame, even though some general pediatricians have substantial experience caring for children who have sustained physical abuse. Thus, our results are susceptible to selection bias. However, we chose to include only those specialties with the greatest likelihood of evaluating and treating pediatric traumatic brain injury. Our results could be different if general pediatricians with high rates of exposure to traumatic brain injury had systematically different opinions about the risks and injuries associated with shaking or other suggested mechanisms.

Second, as with all survey studies, ours might have been subject to response bias if respondents held systematically different opinions from nonrespondents. If present, this could have affected our results by increasing or decreasing the true proportion of clinicians who accept SBS or AHT. We do not feel that this limitation significantly affected our results, however, for several reasons. First, our sampling frame was chosen to reflect practicing clinicians from 10 leading hospitals, rather than groups that are most active in legal proceedings involving child abuse and neglect (and thus more motivated to respond). Second, our relatively high response rate (nearly 50% of those surveyed, with more than 600 clinicians) limits the potential that a small cadre of clinicians with divergent opinions would significantly affect results. Finally, our results show remarkable unanimity. Thus, nearly all nonresponders would have to harbor opinions that are diametrically opposed to responders for AHT or SBS to have an acceptance rate of <50% or for fringe opinions to be generally accepted.

The limitations of the *US News & World Report* hospital rankings have been discussed elsewhere.[27] Our intention in using these rankings was not to endorse a ranking of any particular children's hospital; rather, we sought to identify a relatively large and diverse cohort of clinicians likely to care for child victims of trauma, and to decrease the possibility that the survey would be preferentially distributed to clinicians whose opinion regarding AHT or SBS was known to the authors. It is possible that our results would differ if we were to use different hospitals or a different ranking system; however, given the degree of consensus, we believe it unlikely that such different choices would change the conclusion regarding whether SBS, AHT, or the other alternative hypotheses are generally accepted.

Finally, some respondents indicated confusion about the questions. For example, 1 respondent (who contacted the lead investigator) noted that there are important developmental and anatomic differences between infants aged <12 months and young children aged <3 years that could significantly impact the likelihood of the resulting findings. Another respondent noted that it would have been more appropriate to ask about the likely mechanism, given a particular finding, than to ask about the likely findings resulting from a given mechanism.

Although we recognize both points, we believe that any ambiguity in the survey design would bias against a high level of consensus. ■

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Biomechanical Response of the Infant Head to Shaking - An Experimental Investigation

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Using an infant surrogate with improved biofidelity, we found higher angular acceleration and higher angular velocity than previously reported during infant surrogate shaking experiments. Findings highlight the importance of surrogate biofidelity when investigating shaking.

Key words: head injury, pediatric injury, child abuse, shaking, biomechanics

INTRODUCTION

Violent shaking of infants has been thought to cause serious brain damage since Guthkelch documented the injuries of two shaken infants in 1971.[1] In 1987, Duhaime and colleagues challenged this theory by constructing an infant surrogate and subjecting it to violent shaking and impacts.[2] They measured the rotational acceleration and velocity generated during shaking and impact and concluded that shaking alone did not reach expected biomechanical injury thresholds to cause concussion, subdural hematomas, or diffuse axonal injury. When the infant surrogate's head was struck against a hard surface, however, head injury thresholds were exceeded. They concluded that shaking alone would not be likely to cause serious head injury to an infant. This hypothesis has been tested by others using different surrogates, as well as by computer modeling, with varying results.[3-5]

Duhaime's work has led to a longstanding controversy as to whether it is possible to harm infants by violently shaking them. The concept that "shaking doesn't hurt babies" has been promoted in the popular media.[6-8] These sources highlight stories of people accused of abusing their children by shaking, and quote defense experts who claim that biomechanical studies have shown that shaking an infant cannot cause subdural hemorrhages, encephalopathy, and retinal hemorrhages. Yet, extensive clinical experience resulting from a number of well-documented cases has demonstrated the harmful effects of shaking children.[9-14] In a number of cases, adults have voluntarily reported violently shaking babies, sometimes associated with impact, and sometimes not.[15,16] These infants often are found to have serious or fatal brain injuries, and in survivors, a poor prognosis.[17-20] In addition, a program of intensive education of new parents about the dangers of shaking led to a substantial decrease in the incidence of serious infant head trauma in Buffalo, New York.[21]

We are left with a situation in which clinical experience is not consistent with the biomechanical data from previous laboratory experiments.[2,3] We used an instrumented anthropomorphic test device (ATD, often referred to as a 'crash test dummy' or surrogate) scaled to the size of a human infant to characterize infant head kinematics during shaking. The ATD was specifically designed to have biomechanical responses similar to that of a human infant, especially as it relates to the spine and head-neck regions. We used an instrumented ATD to acquire kinematic and temporal-spatial data during shaking by an adult male.

MATERIALS AND METHODS

The purpose of our testing was to characterize head-neck kinematics associated with violent shaking. Events were simulated in a laboratory setting using an instrumented infant anthropomorphic test device (ATD) and videography.

Shaking Scenario

A 50th percentile Japanese adult male (172 cm; 65.3 kg) volunteer was used to shake the infant ATD in a manner consistent with accounts given by adults who confessed to infant shaking. Five repeat trials consisting of multiple shaking cycles (minimum of 12 cycles/trial) were conducted. The volunteer was advised to grasp the infant ATD with both hands about the upper torso just below the axillary region, suspending it in an upright posture facing them. The volunteer was instructed to "violently" shake the ATD fore and aft for a period of 3 to 4 seconds. Videography was used to capture shaking.

Infant Anthropomorphic Test Device (ATD) or Surrogate

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An *Aprica 2.5* infant ATD was used to conduct shaking experiments (Figure 1). The *Aprica 2.5* is a customized twelve segment, instrumented infant ATD that represents a 5th percentile Japanese newborn (GESAC Inc; Boonesboro, MD).[22] The *Aprica 2.5* has a mass of 2.6 kg and an overall length of 450 mm. Additional anthropometric and inertial properties of the ATD are provided in Table 1. As shown in Table 1, nearly 1/3 of the ATD's mass (0.77 kg) is contained within the head, consistent with the ratio found in the human infant.[23]

Because of the paucity of biomechanical data describing infant response to mechanical phenomena, biofidelity of the *Aprica 2.5* was established by scaling response during head impact tests, head-neck pendulum tests, chest impact tests, and lumbar flexion tests. Data were scaled from existing pediatric ATDs ranging from 6 months to 10 years of age.[22] Since neck response is expected to greatly influence head injury outcome measures in shaking, particular attention was given to development of the *Aprica 2.5* neck. Little data exist to define the biomechanical response of a human infant neck. The ATD neck was constructed to produce an infant neck response to dynamic loading based upon scaling of adult ATD response characteristics.[24] The target neck stiffness for the *Aprica 2.5* neck was determined to be 17 Newton-meter/radian (Nm/rad).[24,25] A head/neck pendulum test was used to verify the dynamic neck response.[26] Rangarajan, et al has provided an overview of the development of the *Aprica 2.5* infant ATD. [22]

Anthropomorphic Test Device Instrumentation

The *Aprica 2.5* ATD was instrumented with tri-axial accelerometers (Kyowa ASM-200BA) positioned at the center of mass of the head and at the apex of the head. Head accelerometers were oriented to allow for determination of angular acceleration in the sagittal plane.

Accelerometers were calibrated following standardized procedures in accordance with SAE J211.[27]

Data Acquisition and Analysis

Data were sampled and collected at 10,000 Hz according to SAE J211.[27] Accelerometer data were filtered using a low pass Butterworth filter with a channel filter class of 1000 Hz.

Angular acceleration in the sagittal plane was derived from linear accelerometer data, because at critical levels, angular acceleration of the head has been shown correlate with concussion, diffuse axonal injury, and subdural hematomas.[28,29] Assuming that primary motion of the head was in the sagittal plane, an estimate of head angular acceleration can be determined by the difference of the linear accelerations in the fore/aft direction measured at two points in the same plane divided by the distance between them (Equation 1).

Equation 1

$$\alpha = \frac{a_2 - a_1}{r}$$

where: α = angular acceleration

a_1 = linear acceleration measured at head center of mass

a_2 = linear acceleration measured at superior aspect of head

r = distance between the accelerometers

The distance between the accelerometers positioned at the center of mass of the head and the apex of the head was 0.0432 meter.

Angular velocity of the head was determined by integrating the angular acceleration time history data. Peak values of head angular acceleration, angular velocity, maximum change in angular velocity, and time of exposure were determined for each shaking cycle, and mean peaks with 95% confidence intervals are reported for a given trial and across all trials. Mean and 95% confidence intervals of sagittal plane angular displacement time histories for a representative test series were also described to assess repeatability in shaking events.

RESULTS

Five shaking test series, each 3 to 4 seconds in duration, were completed using the *Aprica 2.5* ATD surrogate and volunteer shaker. Figure 2 illustrates the sequence of a representative shaking event. As the volunteer flexed his elbows pulling the ATD toward himself, the neck/head of the ATD extended rearward in the sagittal plane. As the volunteer extended his elbows pushing the ATD away from himself, the neck/head flexed forward in the sagittal plane. This visual sequence also illustrates that the ATD chin impacted the chest at peak neck flexion. Given the flexibility of the torso, the occiput did not contact the posterior upper torso at peak neck extension since the torso arched forward.

Mean peak neck flexion was approximately 1.98 radians [rad] (113 degrees [deg]) (95% CI: 1.68-2.28) and mean peak neck extension was approximately 2.16 rad (123 deg) (95% CI: 1.83-2.49) during a typical shaking cycle (Figure 3). Relatively tight confidence intervals illustrate the repeatability of the shaking cycles. As shown in Figure 3, one cycle occurs over approximately 0.25 second, yielding a shaking frequency of 4 hertz.

Mean peak angular head accelerations in the sagittal plane fell within a range of 7,035 rad/sec^2 (radians/second^2) (95% CI: 6,168-7,902) to 10,379 rad/sec^2 (95% CI: 9,304-11,452) across the five test series (Figure 4). The maximum angular head acceleration across all shaking events was 13,260 rad/sec^2 (Table 2), occurring in Test Series 4.

Mean peak angular head velocity measured in the sagittal plane ranged from 71.2 radians/second (rad/sec) (95% CI: 68.5-73.9) to 98.4 rad/sec (95% CI: 95.71-101.1) across the test series (Figure 5). The maximum angular velocity occurred in Test Series 2, and was 105.7 rad/sec (Table 2).

The mean peak change in angular velocity was between 132 rad/sec (95% CI: 128.6-136.4) and 167 rad/sec (95% CI: 164.3-170.0) across all test series (Figure 6). The maximum peak change in angular head velocity was 174 rad/sec in Test Series 2 and 3 (Table 2). Figure 7 presents the combinations of mean angular accelerations and mean peak change in angular velocities resulting from each test series.

The mean time duration of acceleration/deceleration across the five Test Series ranged from 98.5 msec (95% CI: 93.2-103.8) to 137.1 msec (95% CI: 132.9-141.3) (Figure 8). The shortest duration (72.1 msec) occurred in Test Series 1, while the longest duration (168.2 msec) occurred in Test Series 5.

DISCUSSION

The controversy over whether shaking alone can lead to severe head injury in infants began with the study by Duhaime et al. in 1987.[2] Using an infant surrogate constructed from a doll, Duhaime et al. found that lower angular and linear accelerations of the head were generated with

shaking of the surrogate than by inflicted impact. They reported a mean angular head acceleration of 1,138 rad/sec^2, with a mean angular velocity of 61 rad/sec and a mean acceleration pulse duration of 107 msec in their shaking experiments.

Prange et al. repeated this study, attempting to improve the biofidelity of the infant surrogate, and found that inflicted impacts against hard surfaces were more likely to be associated with angular accelerations reaching injury thresholds than shaking or falls from 1.5 m or less.[3] Compared to the Prange et al. study, a recent study by Coats et al. found markedly *lower* peak angular acceleration and velocity, and increased pulse durations during similar impact events using an ATD with a more biofidelic neck design, a deformable, sutured skull and a more biofidelic body mass distribution.[30] The Coats et al. study did not investigate shaking, but their differing findings compared to those of Prange et al. for head impacts highlights the marked influence ATD design can have on injury related outcomes.

The findings of our study differed substantially from those of Duhaime and Prange. Our maximum angular head acceleration was found to be 13,260 rad/sec^2, a 10-fold increase over Duhaime's findings and more than twice that reported by Prange et al. Further, peak angular head accelerations across all test series in our study exceeded those reported by Prange and Duhaime. Similarly, peak change in angular velocity measured in our study (174 rad/sec) was almost 3 times greater than that measured by Prange and Duhaime. The mean angular acceleration pulse duration for shaking measured in our study ranged from 98 msec to 137 msec, and was similar to that found by Duhaime and Prange.

Differences between our findings and those of Duhaime and Prange are likely due to differences in the surrogates used to represent an infant, and possibly differences in the delivery of shaking by volunteers. When considered in isolation, differences in surrogate design that would lead to increases or decreases in head accelerations include the following:

Body design: The ATD used by Prange employed a torso that combined the mass of arms and legs of an infant, represented as wooden structure, along with a neck and a head. The surrogate used in our study consisted of a segmented flexible torso/spine, along with a head, neck and upper and lower extremities. A less than complete ATD body, such as that used by Prange would reduce the overall inertial resistance to motion during shaking and could increase torso acceleration. That is, the lack of upper and lower extremities would offer less resistance to the back and forth motion of the torso during shaking allowing for greater ease of torso motion.

Surrogate mass: The mass of our ATD was 2.6 kg, whereas the mass of the surrogates used by Prange and Duhaime were 4.83 kg and 3.0 to 4.0 kg, respectively. Increased mass increases the inertial resistance of the surrogate body to motion and tends to decrease torso acceleration.

Head mass: The mass of the surrogate head used in our study was 0.77 kg, while the head mass of ATDs used by Prange and Duhaime was 1.13 kg and 0.77-0.87 kg, respectively. The lower head mass used in our study would offer less inertial resistance leading to higher head accelerations with shaking.

Neck structure and stiffness: Neck structure and stiffness play a critical role in the head's response to shaking. Duhaime, et al. examined the effects of various neck designs and found that a resistance-free hinged neck design was associated with the highest head accelerations with shaking.[2] Prange et al. utilized a negligible-resistance hinge neck to generate a worst-case head acceleration scenario.[3] The neck assembly used in our surrogate consisted of urethane tubing with a centered safety cable joining superior and inferior aluminum plates that allowed for head-

neck rotation in the sagittal, coronal and transverse planes. The safety cable was adjusted to provide desired neck bending properties (Figure 8).[22] Duhaime and Prange did not report neck properties of their surrogates, but given the negligible resistance offered by their hinge structures, we estimate that our surrogate neck was likely stiffer and provided greater resistance to head-neck motion during shaking. Both surrogates used by Duhaime and Prange constrained head-neck motions to the sagittal plane (anterior-posterior directions) by utilizing a hinged neck (worst-case scenario), while our surrogate's neck was free to move in three planes. When considering the constraint of head-neck motion in the sagittal plane in isolation, it would tend to increase head accelerations during shaking, since out-of-plane lateral motion is not permitted.

While it is interesting to study surrogate design characteristics in isolation of each other, one characteristic may outweigh or nullify the influence of another characteristic when the surrogate is considered as a whole (i.e. all characteristics together). Such is the case with head motion constraint or lack thereof. That is, even though the lack of head motion constraint tends to decrease head acceleration (as compared to constrained head motion), other design characteristics had a greater bearing on the resulting head acceleration in our experiments. It is the combination of surrogate design characteristics (e.g. head mass, neck length, torso stiffness, surrogate mass, neck stiffness, etc.), along with severity of shaking that dictate the resulting head acceleration. Together these factors drive key kinematic features of the head, such as chin-to-chest contact which can generate large peaks in head acceleration. Chin-to-chest contact occurred in our experiments and was associated with peak head accelerations, but was not reported in experiments conducted of Duhaime and Prange. This kinematic feature likely contributed to head accelerations exceeding those measured by Duhaime and Prange during shaking experiments.

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Biomechanical Response of the Infant Head to Shaking - An Experimental Investigation
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Another study examining biomechanical outcomes during shaking of an infant surrogate was conducted by Cory, et al.[4] They reproduced the Duhaime's 1987 experiments using an adjustable replica of their surrogate to determine the influence of surrogate parameters on head response outcomes during shaking, varying head-neck joint location (i.e., occipital condyle location), neck construction (rubber neck vs. hinged neck), torso padding (cotton wool versus silicone), and location of the surrogate center of gravity (CG). Under a combination of worst-case parameters, Cory measured a peak angular head acceleration of 10,217 rad/sec^2 and a peak angular head velocity of 61 rad/sec. Cory's results exceeded peak angular accelerations reported by both Duhaime and Prange, and approach those measured in our experiments.[2,3] Cory found the combination of a high surrogate CG, hinged neck and cotton wool padded torso produced worst-case angular head acceleration values.

One of the differences between the *Aprica 2.5* ATD and the surrogate used by Cory is the flexibility of the thoracic spine and torso stiffness. During shaking, Cory et al. described both chin-to-chest and posterior head-to-posterior torso contact (likely contributing to higher accelerations than those measured by Duhaime and Prange). The *Aprica 2.5* ATD also displayed chin-to-chest contact, but the posterior aspect of the head did not contact the torso during shaking. The *Aprica 2.5* ATD has a flexible thoracic spine allowing for arching of the torso during the extension portion of shaking which diminishes the likelihood of head contact with the posterior torso. Cory et al. did not quantify neck properties of the surrogate used in their study, and thus a direct comparison with the *Aprica 2.5* ATD neck was not possible.

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The location of the center of rotation of the head also influences the rotational response of the head to shaking. That is, for a given shaking event, as the distance to the center of rotation increases in length, the angular acceleration would decrease assuming all other parameters were held constant. Although the *Aprica 2.5* ATD neck joins the torso at a point 5 cm below the base of the skull, the design of the neck allows for a moving center of rotation as the head rotates. This can effectively provide a radius of rotation that is less than 5 cm in length at various points in the head's rotation about the neck. In contrast, the surrogates used by Duhaime, Cory and Prange that led to worst-case head accelerations had fixed centers of rotation that were located 3.3 cm, 3.3 cm and 4.5 cm, respectively, below the base of the skull. In contrast, the *Aprica 2.5* ATD neck allowed for a moving center of rotation mimicking that of a human infant. Given this moving center of rotation (i.e. the center of rotation can change during neck flexion/extension) it is difficult to evaluate how the neck center of rotation would affect angular head acceleration in comparison to the surrogates used by Duhaime, Cory and Prange. In general, if the *Aprica 2.5* ATD center of head rotation was located such that the radius of rotation was less than that of the other surrogates, there would be a tendency towards increased angular head acceleration for a given shaking input.

To determine the likelihood of injury from exposure to a given phenomenon (i.e. impact or shaking), outcome measures known to be associated with injury risk are typically compared to published injury thresholds. During *Aprica 2.5* ATD shaking experiments, injury threshold levels for concussion reported for primates with 400 g brains (similar in mass to a young infant) were exceeded,[31] while published injury thresholds for diffuse axonal injury were not exceeded.[29] To date however, there are no validated infant brain injury thresholds. Scaling thresholds from adult cadaver and primate studies to infants based on brain mass alone can

produce misleading pediatric head injury thresholds.[32] Other factors such as brain material properties and geometry must also be considered when attempting to assess brain injury risk.[33] Furthermore, published pediatric head injury thresholds fail to account for repetitive exposure to acceleration or deceleration that occurs in shaking, but instead consider only exposure to a single event. Repetitive head injuries in animal models have been shown to cause greater injury at lower peak rotational velocities than do single impulse loads.[34] The effects of repetitive cyclic events such as those involved in shaking have not been systematically studied in animal models other than mice and piglets.[35,36]

The duration of exposure to acceleration is also an important factor when attempting to predict the risk of and type of brain injury that can result from an event. In our experiments, duration of exposure (i.e. acceleration pulse duration) ranged from 72.1 msec to 168.2 msec. Löwenhielm et al described accelerations and associated pulse durations of 15 to 44 msec leading to subdural hematomas, substantially shorter pulse durations than experienced by our ATD during shaking.[37] Ommaya found that lower levels of acceleration are capable of producing injury in cases of longer pulse duration.[38] Genaralli and Thibault suggested that longer acceleration pulse durations permit brain tissue strains resulting from accelerations to propagate deeper into the brain leading to functional damage found in cerebral concussion or structural damage found in axonal injury.[28]

Other studies have noted the extreme vulnerability of the infant brain to the metabolic effects of brain injury when compared with the adult brain.[39,40] This would suggest that the infant brain is more susceptible to injury and might account for the poor outcomes noted in children surviving abusive head trauma (AHT).[18,19] Another worrisome aspect of infant abuse that could account

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for the hypoxic injury to the brain is the finding that in a large percentage of infant abuse cases resulting in death, subtle injury to the high cervical cord and lower brain stem is seen on autopsy.[41] It is postulated that these cord injuries could lead to apnea or changes in autoregulation of cerebral blood flow, causing the typical hypoxic changes seen in infant victims of AHT.

The influence of shaking on the infant brain and the pathophysiology of infant brain injury is more complicated than can be represented in existing surrogate models used in biomechanical shaking experiments. For example, the immature brain could be more vulnerable to angular acceleration than the adult brain. Raghupathi and colleagues found the immature piglet brain to be more vulnerable to a single inertial load than that of the adult pig brain.[42] They also found that repeating an inertial load on the piglet head twice, 15 minutes apart, led to more diffuse axonal injury in the piglet brain and to a significant decrease in arterial blood pressure 60 minutes post injury.[36] Since violent shaking of infants involves repetitive inertial events occurring within a few seconds, the mechanical phenomena necessary to cause injury in the immature brain could be less severe than would be necessary to cause a single impact injury. In addition, surrogate experimental outcomes do not take into account the well-known deficits in cerebral autoregulation that occur after infant brain injury leading to profound cerebral hypotension and hypoperfusion.[43]

All of these factors suggest that the infant brain is more susceptible to injury than the adult brain. Taking these factors into account, predictions of risk based upon comparison with published brain injury thresholds are not likely to be reliable given the limitations inherent in these thresholds.

Another factor that complicates the estimation of infant brain injury thresholds is the fact that infants who experience abusive head trauma are often subjected to multiple bouts of trauma over days to weeks. Several studies have shown that infants presenting with AHT are likely to have suffered previous abusive head trauma.[44-46] Using an animal model, Huh, et al demonstrated a graded pathological response to repetitive mild injury in immature rats.[34] Rats experiencing three mild impacts to the head (not resulting in fracture) over 15 minutes developed axonal injury and brain atrophy, compared to rat pups receiving only a single mild impact. When an immature brain experiences multiple injuries, some 'priming' might occur that makes the brain more susceptible to damage from subsequent injuries. Additionally, vulnerable infant axons within the brain may not be capable of repair between bouts of trauma. These phenomena may effectively lower infant brain injury thresholds, which are used to predict probability of injury when compared to experimental outcomes such as angular acceleration.

In addition to the lack of data on the biomechanical properties of the infant neck, animal models may not adequately represent the lack of supporting musculature found in the human infant neck.[47] However, the neck of a newborn goat has been estimated to be equivalent in strength to the neck of a one-year-old human.[48] When studying the effects of shaking, the lack of protective infant neck musculature is a key factor that must be represented in any model.

This study is limited by the inability of *any* available ATD to represent the biomechanical characteristics of an infant with complete accuracy. The *Aprica 2.5* ATD, however, was designed to match a newborn infant's anthropometrics and to be biofidelic within the limits of the technology and published human response data. In addition, only one volunteer was used to

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induce shaking of the ATD. Using multiple individuals for ATD shaking may produce varying inputs leading to a different biomechanical response. However, our intent was to investigate shaking response generated by a nominal representative average (50^{th} percentile) male, providing the first step towards understanding the influence of improved ATD biofidelity. Similarly, one's interpretation of the instruction to "violently" shake the ATD is subjective and may vary across individual shakers. However, the volunteer shaker used in this study was educated on perpetrator actions when abusively shaking a child and was asked to replicate shaking that would occur during rage or anger. Finally, the *Aprica 2.5* ATD represents a small newborn infant. Shaking an ATD representing an older infant would undoubtedly lead to a differing biomechanical response due to differences in mass, neck flexibility and torso/spine flexibility.

Higher levels of angular acceleration and angular velocity measured using the instrumented *Aprica 2.5* ATD during shaking suggest that more significant injuries may be caused by shaking alone than previously reported. These findings are consistent with documented clinical experience showing that violent shaking of infants is potentially harmful or even lethal.[49-51] This study also demonstrated the importance of biofidelic torso/spine and head-neck regions when investigating kinematics associated with shaking. While biofidelic ATDs can improve our understanding of kinematics during shaking, infant neuropathological response to measured accelerations and velocities still remains unclear.

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AUTHOR DISCLOSURE STATEMENT

Drs. Jenny, Bertocci, and Rangarajan have testified in courts of law regarding infant head trauma.

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FIGURE LEGENDS

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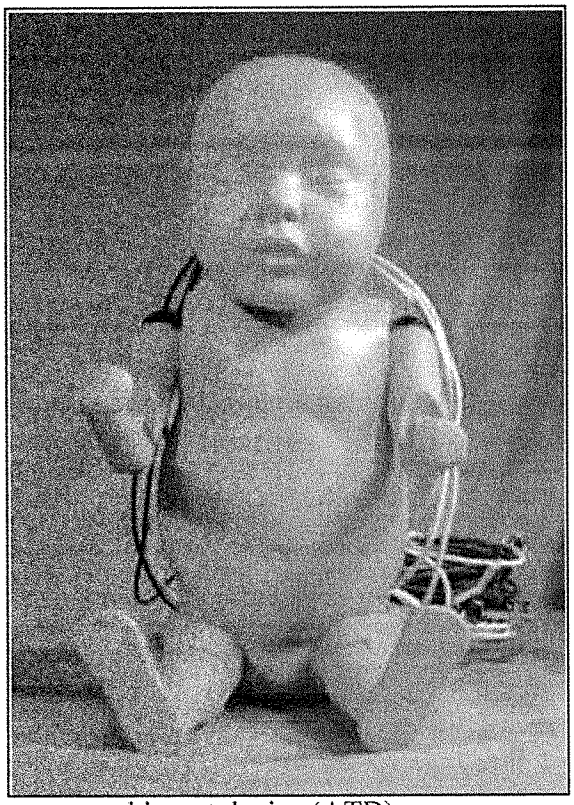

Figure 1. *Aprica 2.5* anthropomorphic test device (ATD).

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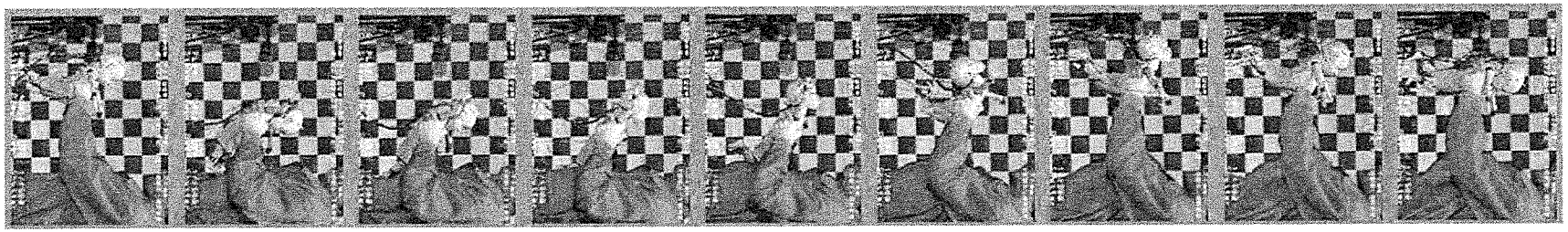

Figure 2. Kinematic sequence of one shaking cycle.

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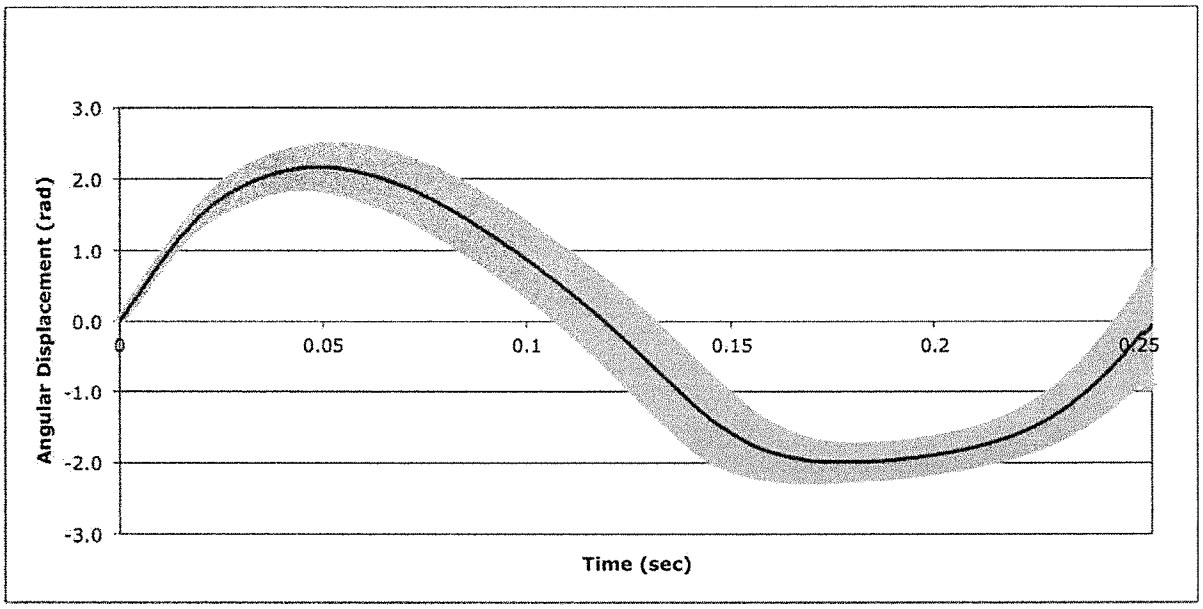

Figure 3. Mean angular head displacement time history for individual cycles of shaking for a representative test series (Test Series No. 3). One cycle occurs over approximately 0.25 sec (4 Hz). Positive values represent extension and negative values represent flexion. (Error bars – appear as shading - represent 95% confidence intervals.) (Note: 1 radian = 57.3°)

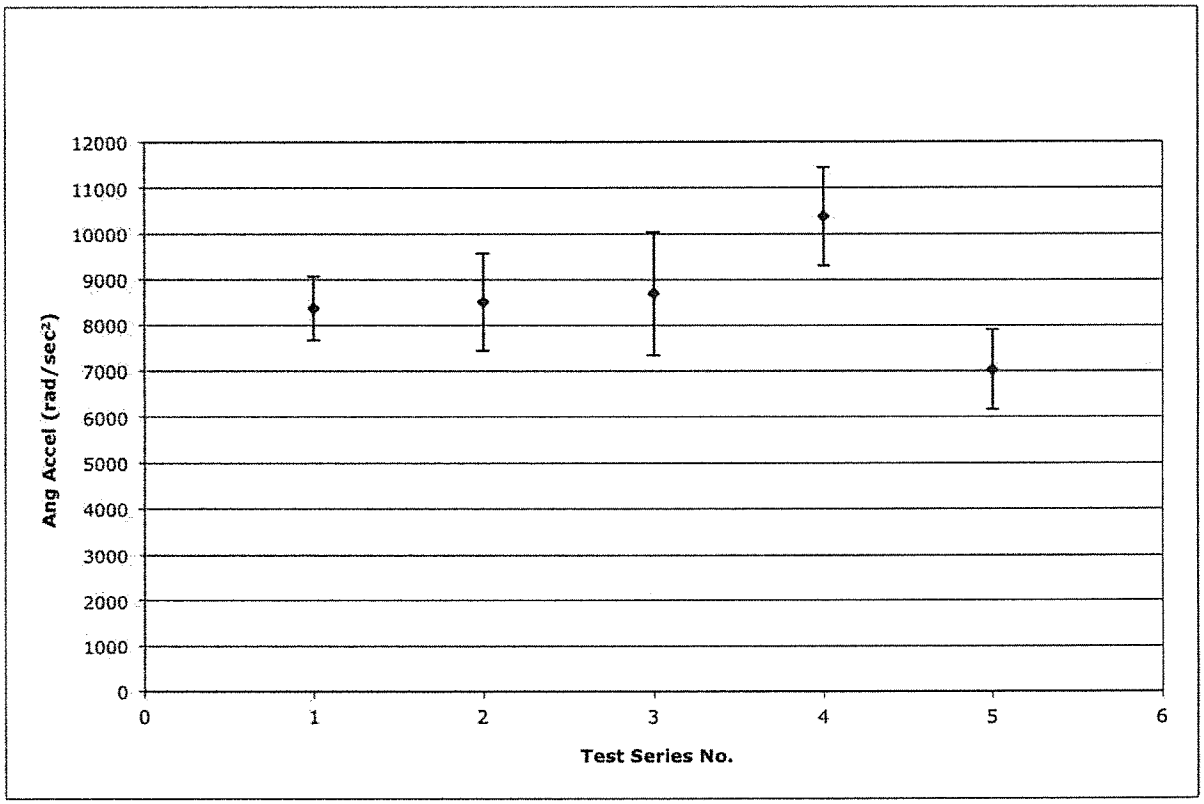

Figure 4. Mean peak sagittal plane angular head accelerations for each test series. (Error bars represent 95% confidence intervals.)

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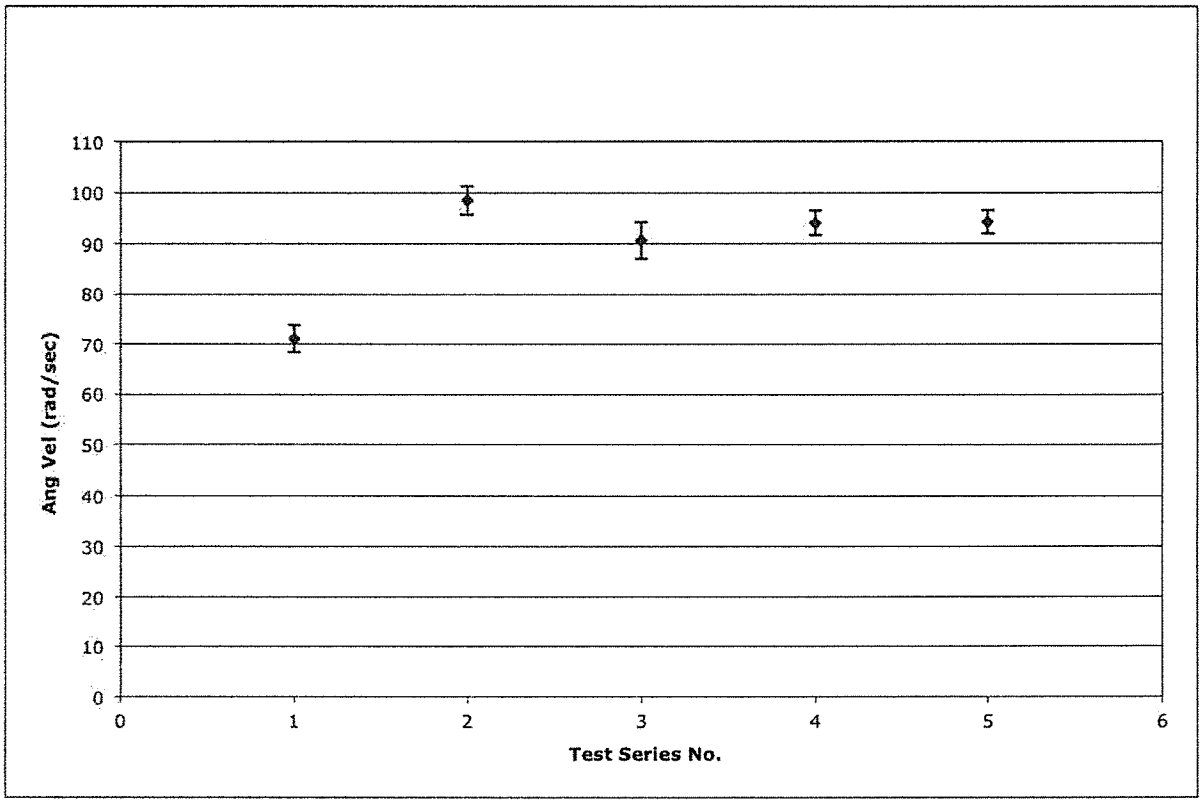

Figure 5. Mean sagittal plane peak angular head velocity for each test series. (Error bars represent 95% confidence intervals.)

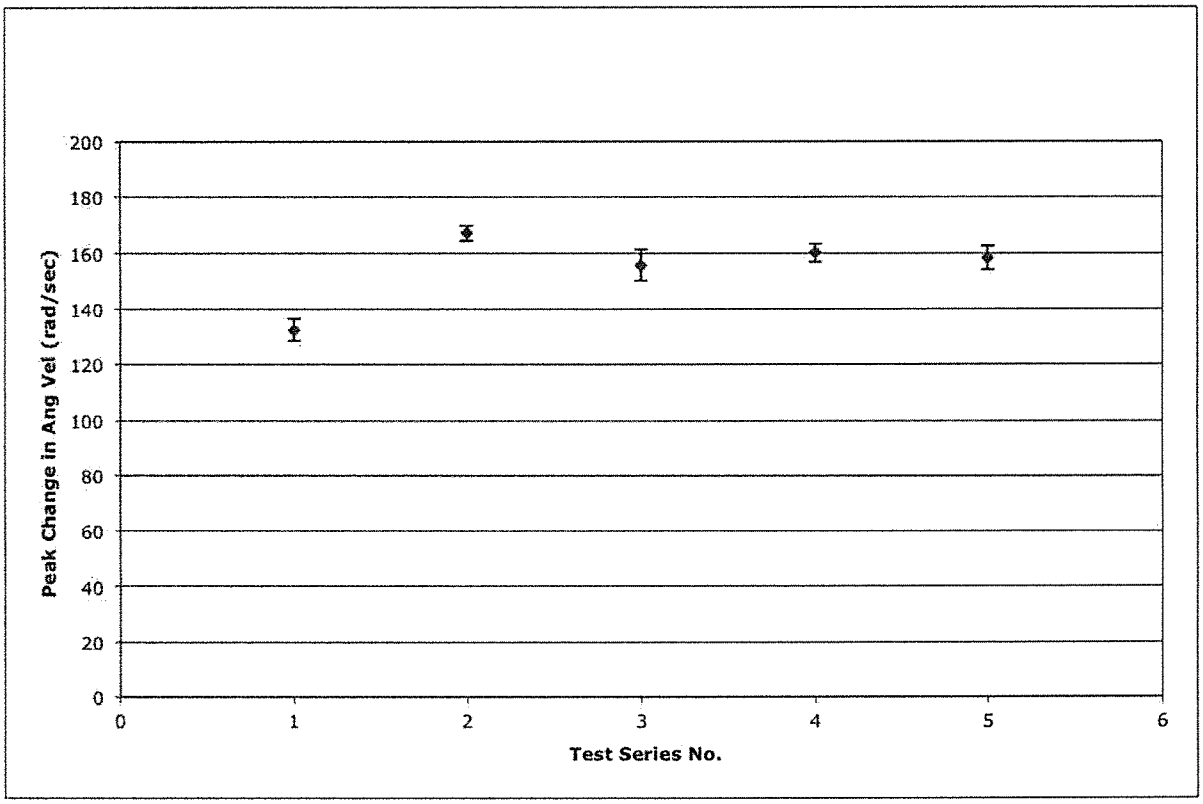

Figure 6. Mean peak change in sagittal plane angular head velocity for each test series. (Error bars represent 95% confidence intervals.)

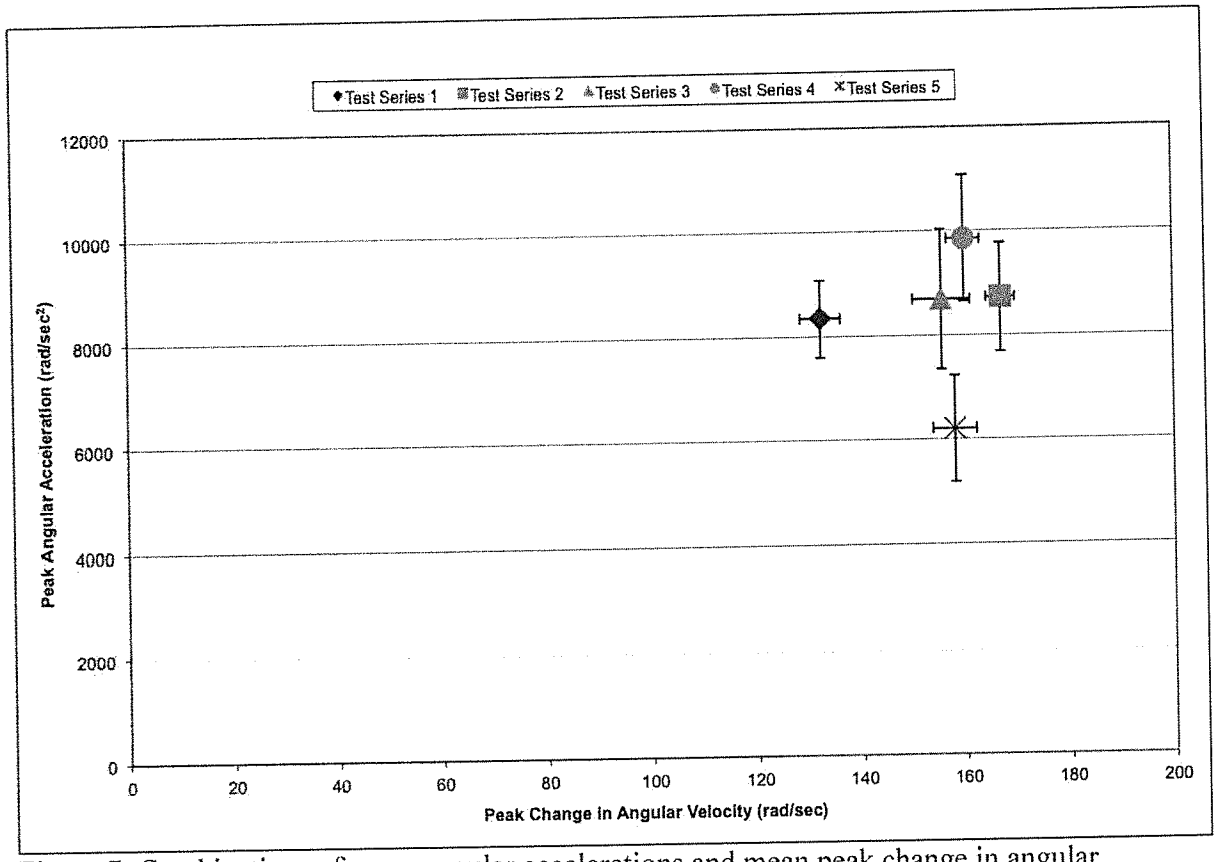

Figure 7. Combinations of mean angular accelerations and mean peak change in angular velocities for each test series. (Error bars represent 95% confidence intervals.)

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Table 2. Peak angular head acceleration, peak change in angular head velocity and peak angular head velocity for each test series.

Test Series No.	Peak Angular Head Acceleration (rad/sec^2)	Peak Change in Angular Head Velocity (rad/sec)	Peak Angular Head Velocity (rad/sec)
1	10,630	143	80
2	12,150	174	106
3	12,030	174	104
4	13,260	173	102
5	9,613	167	100