

Research Article

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

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

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

1 Introduction

Shaken baby syndrome (SBS) has been defined as the presence of three specific findings: subdural hematoma (SDH), cerebral edema/brain swelling and retinal hemorrhage (RH) [8,25]. This injury cluster—sometimes referred to as “the triad”—has been presumed to occur as a result of abusive shaking but not as a result of household falls, even falls down stairs [7]. The shaking hypothesis, first proposed in the early 1970s [6,24], was seemingly accepted as settled science in 2001 in two documents: a position paper from the National Association of Medical Examiners

[7] and an updated position statement from the American Academy of Pediatrics (AAP) [2]. Although the hypothesis has never been scientifically proven, practitioners working with the accepted model have accumulated years of clinical experience that convinces them that the model is correct [5].

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

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

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

model (13, 252 rad/s² [28]) exceeded Duhaime's by an order of magnitude, although these figures seem to represent a different calculation strategy.

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

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

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

Depreitere et al. estimated the rupture threshold for adult bridging veins at 10,000 rad/s² [13], the same figure used by Duhaime et al. for concussion in infants [16]. Researchers have worked with even higher thresholds for infant SDH [11,16,40].

In the present study, researchers collected data from infant surrogates during abusive shaking simulations and various activities of daily living (ADLs) commonly experienced by infants. These ADLs would not be defined as abusive and would not be predicted to cause injury. Taking measurements during these activities establishes a baseline for commonly generated linear and angular head

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

Table 1: Anthropometry of infant surrogates.

motions, allowing researchers to compare these values to injury thresholds, an approach also used by researchers trying to understand whiplash accelerations in automobile accidents [1].

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

2 Materials and methods

2.1 Infant representatives

Researchers collected data from three infant representatives:

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

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

2.2 Subjects

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



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



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

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

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

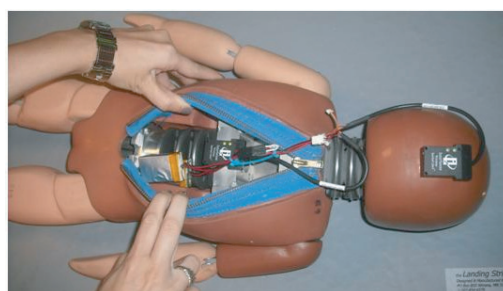


Figure 3: InterSense sensors on head and torso of CRABI-12 mannequin.



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

2.3 Test protocol

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

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

Data was collected in three sets:

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

2.3.1 Shaking

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



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



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

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

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

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

2.3.2 Pediatric activities of daily living

A subset of six adult volunteers, including two females and four males, manipulated the CRABI-12 mannequin

during various pediatric ADLs, whilst head motion data was acquired using the InterSense sensors, as previously described. The investigated ADLs included:

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

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

2.3.3 Infant playing

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

Figure 6 illustrates the subject ready to jump.

2.4 Analysis

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

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

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

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

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

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

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

3 Results

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

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

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

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

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

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

4 Limitations of the present study

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

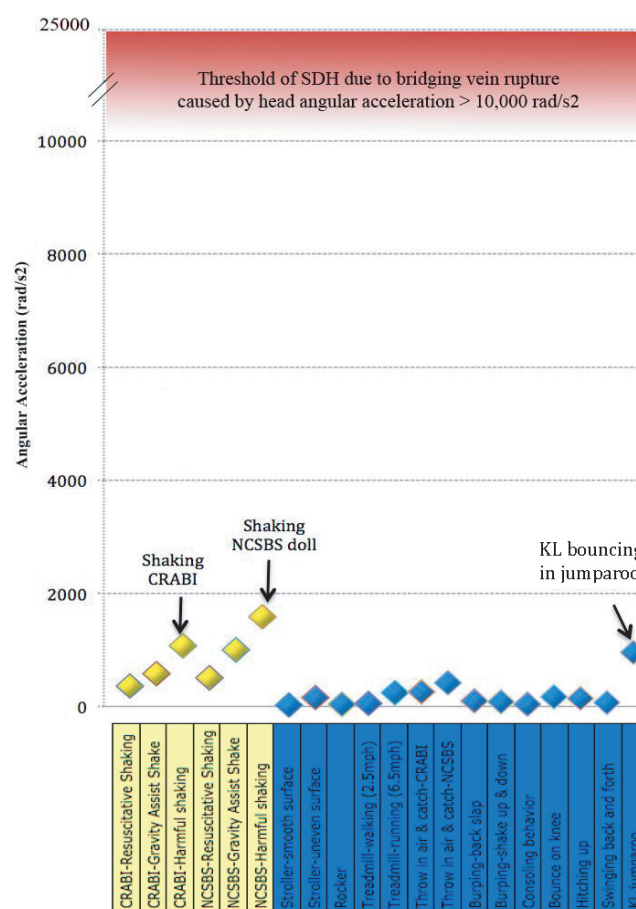


Figure 7: Head angular acceleration, in radians per second squared, during shaking (yellow) versus pediatric ADLs (blue).

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

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

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

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

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

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

5 Discussion

It has been assumed for decades that aggressive manual shaking, with or without impact, produces the characteristic “triad” of SBS symptoms (subdural hemorrhages, cerebral edema/brain swelling and retinal hemorrhages) in an infant. This model is based on the hypothesis that uncontrolled motion of the infant head during shaking causes the damage directly, by tearing bridging veins to produce subdural

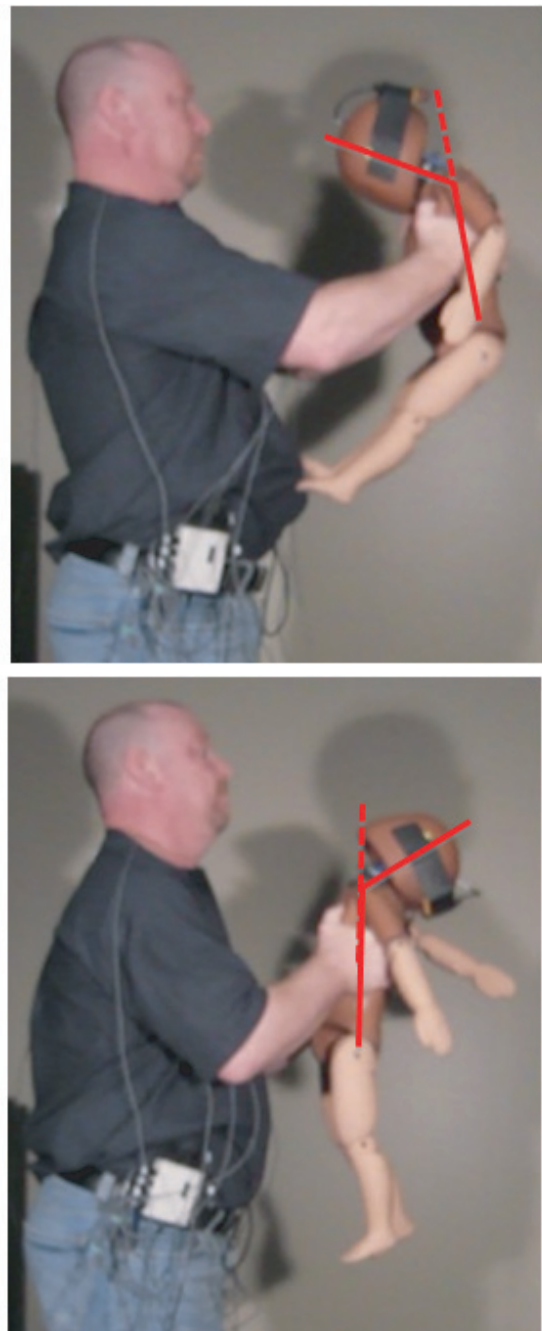


Figure 8: CRABI neck in the sagittal plane shows possible endpoint impact during shaking.

hematoma, stretching neurons to produce diffuse axonal injury, and causing vitreous traction on the retina to produce retinal hemorrhages, schisis and folds.

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

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

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

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

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

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

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

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

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

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

6 Conclusions

This study demonstrates that angular acceleration of the head during aggressive shaking of the CRABI biofidelic mannequin (1068.3 rad/s^2) is statistically indistinguishable

($P \leq .05$) from angular head kinematics experienced by a 7-month-old infant fervently playing in his Jumparoo (954.4 rad/s^2). Other pediatric ADLs, such as being burped or bounced on a knee, are clearly negligible. Furthermore, measured angular accelerations fall 84% below the scientifically accepted biomechanical threshold for bridging-vein rupture of $10,000 \text{ rad/s}^2$.

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

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

References

- [1] M. Allen, I. Weir-Jones, D. Motiuk, K. Flewin, R. Goring, R. Kobetitch, et al., *Acceleration perturbations of daily living. A comparison to 'whiplash'*, Spine, 19 (1994), 1285–1290.
- [2] American Academy of Pediatrics: Committee on Child Abuse and Neglect, *Shaken baby syndrome: rotational cranial injuries—technical report*, Pediatrics, 108 (2001), 206–210.
- [3] F. A. Bandak, *Shaken baby syndrome: A biomechanics analysis of injury mechanisms*, Forensic Sci Int, 151 (2005), 71–79.
- [4] P. D. Barnes, *Imaging of nonaccidental injury and the mimics: issues and controversies in the era of evidence-based medicine*, Radiol Clin North Am, 49 (2011), 205–229.
- [5] R. W. Block, *SBS/AHT 2010: What we know, what we must learn, what we must do to move forward*, in Eleventh International Conference on Shaken Baby Syndrome/Abusive Head Trauma, National Center On Shaken Baby Syndrome, Atlanta, GA, 2010.
- [6] J. Caffey, *On the theory and practice of shaking infants: Its potential residual effects of permanent brain damage and mental retardation*, Am J Dis Child, 124 (1972), 161–169.
- [7] M. E. Case, M. A. Graham, T. C. Handy, J. M. Jentzen, and J. A. Monteleone, *Position paper on fatal abusive head injuries in infants and young children*, Am J Forensic Med Pathol, 22 (2001), 112–122.
- [8] D. L. Chadwick, R. H. Kirschner, R. M. Reece, L. R. Ricci, R. Alexander, M. Amaya, et al., *Shaken baby syndrome—a forensic pediatric response*, Pediatrics, 101 (1998), 321–323.
- [9] C. W. Christian and R. Block, *Abusive head trauma in infants and children*, Pediatrics, 123 (2009), 1409–1411.
- [10] Commonwealth v Ann Power, 2005, *Report to the Middlesex County District Attorney Office Cambridge Massachusetts by Carole Jenny*. December 29, 2005.
- [11] C. Z. Cory and B. M. Jones, *Can shaking alone cause fatal brain injury? A biomechanical assessment of the Duhaime shaken baby syndrome model*, Med Sci Law, 43 (2003), 317–333.
- [12] T. J. David, *Non-accidental head injury—the evidence*, Pediatr Radiol, 38 (2008), S370–S377.
- [13] B. Depreitere, C. Van Lierde, J. V. Sloten, R. Van Audekercke, G. Van der Perre, C. Plets, et al., *Mechanics of acute subdural hematomas resulting from bridging vein rupture*, J Neurosurg, 104 (2006), 950–956.
- [14] K. Desantis Klinich, G. M. Hulbert, and L. W. Schneider, *Estimating infant head injury criteria and impact response using crash reconstruction and finite element modeling*, Stapp Car Crash J, 46 (2002), 165–194.

- [15] A. C. Duhaime, C. W. Christian, L. B. Rorke, and R. A. Zimmerman, *Nonaccidental head injury in infants—the “shaken-baby syndrome”*, *N Engl J Med*, 338 (1998), 1822–1829.
- [16] A. C. Duhaime, T. A. Gennarelli, L. E. Thibault, D. A. Bruce, S. S. Margulies, and R. Wiser, *The shaken baby syndrome. A clinical, pathological, and biomechanical study*, *J Neurosurg*, 66 (1987), 409–415.
- [17] J. M. Duncan, *Laboratory note: On the tensile strength of the fresh adult foetus*, *Br Med J*, 2 (1874), 763–764.
- [18] J. R. Funk, S. M. Duma, S. J. Manoogian, and S. Rowson, *Biomechanical risk estimates for mild traumatic brain injury*, *Annu Proc Assoc Adv Automot Med*, 51 (2007), 343–361.
- [19] J. F. Geddes, A. K. Hackshaw, G. H. Vowles, C. D. Nickols, and H. Whitwell, *Neuropathology of inflicted head injury in children. I. Patterns of brain damage*, *Brain*, 124 (2001), 1290–1298.
- [20] J. F. Geddes and J. Plunkett, *The evidence base for shaken baby syndrome*, *Br Med J*, 328 (2004), 719–720.
- [21] J. F. Geddes, R. C. Tasker, A. K. Hackshaw, C. D. Nickols, G. G. Adams, H. Whitwell, et al., *Dural haemorrhage in non-traumatic infant deaths: does it explain the bleeding in ‘shaken baby syndrome’?*, *Neuropathol Appl Neurobiol*, 29 (2003), 14–22.
- [22] J. F. Geddes, G. H. Vowles, A. K. Hackshaw, C. D. Nickols, I. S. Scott, and H. Whitwell, *Neuropathology of inflicted head injury in children. II. Microscopic brain injury in infants*, *Brain*, 124 (2001), 1299–1306.
- [23] M. G. Gilliland, *Interval duration between injury and severe symptoms in nonaccidental head trauma in infants and young children*, *J Forensic Sci*, 43 (1998), 723–725.
- [24] A. N. Guthkelch, *Infantile subdural haematoma and its relationship to whiplash injuries*, *Br Med J*, 2 (1971), 430–431.
- [25] B. Harding, R. A. Risdon, and H. F. Krous, *Shaken baby syndrome*, *Br Med J*, 328 (2004), 720–721.
- [26] R. W. Huntington III, *Symptoms following head injury*, *Am J Forensic Med Pathol*, 23 (2002), 105–106.
- [27] N. G. Ibrahim and S. S. Margulies, *Biomechanics of the toddler head during low-height falls: an anthropomorphic dummy analysis*, *J Neurosurg Pediatr*, 6 (2010), 57–68.
- [28] C. Jenny, *Junk Medical Science in the Courtroom*. Rhode Island Hospital Pediatric Grand Rounds. Providence, RI (July 23, 2010), <http://lifespan.mediasite.com/mediasite/Viewer/?peid=d237bed531df42e49223ccdb685c48741d>.
- [29] A. M. Kemp, N. Stoodley, C. Cobley, L. Coles, and K. W. Kemp, *Apnoea and brain swelling in non-accidental head injury*, *Arch Dis Child*, 88 (2003), 472–476.
- [30] J. E. Leestma, *Case analysis of brain-injured admittedly shaken infants: 54 cases, 1969–2001*, *Am J Forensic Med Pathol*, 26 (2005), 199–212.
- [31] J. F. Luck, R. W. Nightingale, A. M. Loyd, M. T. Prange, A. T. Dibb, Y. Song, et al., *Tensile mechanical properties of the perinatal and pediatric PMHS osteoligamentous cervical spine*, *Stapp Car Crash J*, 52 (2008), 107–134.
- [32] J. Mack, W. Squier, and J. T. Eastman, *Anatomy and development of the meninges: implications for subdural collections and CSF circulation*, *Pediatr Radiol*, 39 (2009), 200–210.
- [33] D. K. Molina, A. Clarkson, K. L. Farley, and N. J. Farley, *A review of blunt force injury homicides of children aged 0 to 5 years in Bexar County, Texas, from 1988 to 2009*, *Am J Forensic Med Pathol*, (2011).
- [34] J. Ouyang, Q. Zhu, W. Zhao, Y. Xu, W. Chen, and S. Zhong, *Biomechanical assessment of the pediatric cervical spine under bending and tensile loading*, *Spine*, 30 (2005), E716–E723.
- [35] M. Prange, W. Newberry, T. Moore, D. Peterson, B. Smyth, and C. Corrigan, *Inertial neck injuries in children involved in frontal collisions*. Society of Automotive Engineers, SAE 2007-01-1170 (presented at the 2007 SAE World Congress, Detroit, MI), 2007.
- [36] M. T. Prange, B. Coats, A. C. Duhaime, and S. S. Margulies, *Anthropomorphic simulations of falls, shakes, and inflicted impacts in infants*, *J Neurosurg*, 99 (2003), 143–150.
- [37] A. Sirotnak, *Medical disorders that mimic abusive head trauma*, in *Trauma in Infants and Children*, L. S. Frasier, R. Alexander, K. Rauth-Farley, and R. N. Parrish, eds., GW Medical Publishing, St. Louis, 2006, 191–226.
- [38] W. Squier, E. Lindberg, J. Mack, and S. Darby, *Demonstration of fluid channels in human dura and their relationship to age and intradural bleeding*, *Childs Nerv Syst*, 25 (2009), 925–931.
- [39] C. Van Ee, B. Moroski-Browne, D. Raymond, K. Thibault, W. Hardy, and J. Plunkett, *Evaluation and refinement of the CRABI-6 anthropomorphic test device injury criteria for skull fracture*, in *Proceedings of the ASME 2009 International Mechanical Engineering Congress & Exposition*, Lake Buena Vista, FL, 2009.
- [40] D. R. Wolfson, D. S. McNally, M. J. Clifford, and M. Vloeberghs, *Rigid-body modelling of shaken baby syndrome*, *Proc Inst Mech Eng H*, 219 (2005), 63–70.
- [41] L. Zhang, K. H. Yang, and A. I. King, *A proposed injury threshold for mild traumatic brain injury*, *J Biomech Eng*, 126 (2004), 226–236.