

Shaken Baby Syndrome and the Death of Matthew Eappen

A Forensic Pathologist's Response

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Subdural hemorrhage, retinal hemorrhage, and cerebral edema have been considered diagnostic for a "shaken infant" since the syndrome was described almost 30 years ago. However, the specificity of these findings has been disputed by defense witnesses in recent U.S. criminal prosecutions. This review examines the scientific basis for the shaken baby syndrome.

Key Words: Child abuse—Chronic subdural hematoma
Lucid interval—Shaken baby syndrome.

The problem isn't with what we don't know. The problem is with what we do know that isn't so.

Will Rogers

If the law has made you a witness remain a man of science. You have no victim to avenge, no guilty or innocent person to ruin or save. You must bear witness within the limits of science.

Paul H. Broussard
Chair of Forensic Medicine
Sorbonne, 1897

Scientific testimony in the U.S. Federal Court System must meet the threshold defined in *Daubert v. Merrell Dow*. Merrell Dow manufactures and markets the antinausea drug Bendectin, and Daubert alleged that the drug caused limb-reduction birth defects in their newborn. The Southern California Federal District Court granted summary judgment to the defendant, stating that in vitro studies, chemical structural analysis, and animal studies had no substantiated direct applicability to humans and that recalculation of epidemiologic data had not been submitted for peer review; therefore, neither type of testimony was admissible. The 9th Circuit U.S. Court of Appeals affirmed the California decision, and the U.S. Supreme Court denied review (1).

A portion of the Court of Appeals affirmation cited an earlier Court of Appeals decision (2) in stating that "courts must 'critically evaluate the reasoning process by which the experts connect data to their conclusions in order for courts to consistently and rationally resolve the disputes before them'." However, it is easier to determine whether a study has been published than to ascertain if the thought proc-

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ess was sound, and inability to apply the reasoning criterion may help to explain conflicting expert testimony in a number of recent court cases, including breast implant action and medical product liability suits. Unfortunately, if there is conflicting expert testimony, jurors may reject all medical or scientific opinions and base decisions on the personal characteristics of the opposing attorneys, personal life experiences, or likability of the defendant (3). In the United States, where 38 (76%) states allow the death penalty and where >3000 people are awaiting execution (4), the jury's decision may literally mean the difference between life and death.

Louise Woodward was convicted in 1997 of second-degree murder for the death of Matthew Eappen. The trial judge vacated the jury's verdict, found Ms. Woodward guilty of involuntary manslaughter, and sentenced her to time served. Following the judge's decision, a letter signed by more than 50 physicians specializing in the diagnosis and treatment of victims of child abuse was published on the Internet (5). The physicians stated, in response to criticism that prosecution witnesses lacked a scientific foundation for their testimony, that "many in the media and the public have failed to credit the jury in this case for having the intelligence to understand that...well-established medical evidence...overwhelmingly supported a violent shaking/impact episode on the day in question, when Matthew was in the sole custody of Ms. Woodward," and that the "rebleed" theory in infants is a courtroom diagnosis, not a medical diagnosis, and the jury properly rejected it." The authors continued, stating that Matthew had "massive head injuries...incompatible with any period of normal behavior subsequent to the injury" and the "shaken baby syndrome...is now a well characterized clinical and pathologic entity with diagnostic features in severe cases virtually unique to this type of injury," and challenging critics who would question the "specificity of these diagnostic features first do so in the peer-reviewed literature, before speculating on other causes in court (5)."

The medical witnesses for the prosecution during the trial gave specific testimony regarding four issues in infant head injury, stating that a massive amount of force, equivalent to a motor vehicle accident or a fall from a two-story building, is necessary to cause fatal head injury; retinal hemorrhage of a specific type is diagnostic for shaking; children with an ultimately fatal head injury never have a lucid interval; and a sudden change in symptoms in a child with a preexisting head injury requires newly inflicted trauma, not

simply a rebleed. Did the prosecution testimony meet the *Daubert V. Merrell Dow* threshold (1)?

Guthkelch (6) and Caffey (7) described the "shaken-whiplash syndrome" in the context of a battered child with multiple, multidirectional-force injuries and postulated that the weak neck muscles and relatively large head size of an infant made him or her particularly susceptible to cerebral injuries from shaking. There was no experimental model to prove or disprove their theory, and no disinterested witness had ever observed a shaking, but the theory was gradually accepted as fact: subdural hemorrhage and retinal hemorrhage in an unconscious or dead child is diagnostic for a shaken infant. Many years after these publications, Duhaime et al. (8,9) developed a model to demonstrate the particular susceptibility of the infant brain to shaking injury but were unable to generate the required force unless the head was impacted against a solid surface. The impact deceleration forces were greater than the shaking deceleration forces by nearly two orders of magnitude, and the authors concluded that "severe head injuries commonly diagnosed as shaking injuries require impact to occur and that shaking alone in an otherwise normal baby is unlikely to cause the shaken baby syndrome." This conclusion, from the first actual scientific study of shaking, cannot be reconciled with the basic premise of the shaken infant syndrome, and brings up the following question: if an impact is necessary, how can a "shaken-slam" be differentiated from a rotational fall?

What force is necessary to cause the shaken-infant syndrome injury? Prosecution medical testimony in the Woodward trial and published reports state that it is equivalent to a fall from a two-story building or a motor vehicle accident (10-12). However, a fall from a two-story building is almost exclusively translational force, even if the child is spinning, and is unlikely to cause a subdural hemorrhage unless there is a skull fracture with tearing of a dural sinus. A motor vehicle accident involves low-strain rotational deceleration, again unlikely to cause a subdural hemorrhage, although it is certainly capable of causing death (13). (A major goal of automotive safety engineers is to increase deceleration time and distance; this is the reason children's safety seats are placed facing backward in the car. Most motor vehicle accidents are associated with constant velocity or deceleration prior to impact, and the collision or precollision deceleration will push the infant's head into the padded seat rather away from it.) There is no experimental data on immature skulls or brains; how-

ever, adult data from humans and other primates (13-17) and anecdotal evidence from children who have suffered clearly accidental head injuries from short-distance falls (12,18-20) indicate that an impact velocity of 4.6 m/second (15 feet/second) with a stopping time of 2 to 5 msec and a rotational deceleration of 100 radian/second² will cause a subdural hemorrhage. This is equivalent to a gravity rotational fall of 3 feet with the head striking a solid surface such as a linoleum floor, and rotating through a 60-degree arch during the few milliseconds prior to impact. The impact velocity and fall distance may be even less according to Aoki (19) and Howard (20).

Is retinal hemorrhage diagnostic for a shaking injury? Retinal bleeding is highly correlated with rotational deceleration injury/subdural hemorrhage in children, but the actual biomechanical mechanism is unknown, and retinal bleeding indistinguishable from that found in rotational deceleration may be found in association with ruptured vascular malformations (21-24), arachnoid cysts (25-27) and central nervous system infections. The pathognomonic sign has evolved from "retinal hemorrhage" to "flame-shaped retinal hemorrhage" to "multilayered flame-shaped retinal hemorrhage" (28) to, most recently, "multilayered flame-shaped retinal hemorrhage with macular folds" (29), with no data to support this progression other than the argument that the head injury must have been caused by shaking.

What is the evidence to support a conclusion that a child with ultimately fatal head trauma never has a lucid interval? The concept of diffuse axonal injury helps to explain prolonged unconsciousness in adult head-injury victims who have no mass lesion and to predict survivability from the injury (30-32), and experimental and clinical evidence validate this model for understanding many adult head injuries on a macroscopic and microscopic level. However, all of the data are on mature adult human, primate, and other animal brains, and there are no data on infant or immature primate brains. The theory, despite a lack of evidence that it can be extrapolated to children, is applied to conclude that shaken infants who die never have a lucid interval (33), and that the cause of death in those who die shortly after the shaking is axonal injury. However, at least some children with head injury, rotational and otherwise, have a documented lucid interval prior to the development of symptoms, including those who subsequently die (34-36). We simply do not know why some of these children die; it may be axonal injury, malignant cerebral edema, direct irritation of brain stem breathing centers, or

some other phenomenon not yet considered.

Does rebleeding or critical expansion of an established subdural hemorrhage require proximate trauma, and is the rebleed theory a courtroom rather than a medical diagnosis? The only successful experimental model for an expanding subdural hematoma requires cerebrospinal fluid to be mixed with blood and then injected into the subdural space (37). It is thought, but not proved, that the neomembrane surrounding an organizing subdural hematoma may itself bleed, that expansion of a subacute/chronic subdural hemorrhage may cause new bridging veins to rupture, or that disproportionate fibrinolytic activity complicating thrombin formation in the acute clot may predispose to new bleeding (38-39). A study by Gilliland and Folberg (40) concluded that "some shaken infants have no impact," based on autopsy evaluations of 80 children with head trauma, 9 of whom were determined to have died from shaking alone. This study does not mention the ages of the children, the nature of the subdural hemorrhage (acute, subacute, or chronic), or the time from hospital admission to removal of respirator support. If the initial trauma in these cases occurred 14 to ≥ 21 days prior to death, any soft tissue injury may have healed and been invisible to the unaided eye, and if these children had acute bleeding superimposed on older hemorrhage, the "no-impact" premise remains unproved. More significantly, the conclusion that rebleeding requires new trauma is unproved unless shaking alone can cause a subdural hemorrhage.

The shaken infant syndrome is well established, generally accepted, and reported in the peer-reviewed literature. However, is the syndrome theory, or is it fact? Did medical testimony for the prosecution in Ms. Woodward's trial meet the reasoning criterion emphasized in *Daubert v. Merrell Dow* (1)? "Diagnostic errors have been made in the past when confidently stated dogma has been repeated and built upon. Increasing experience may merely allow the same mistake to be made with increasing confidence" (41). The plural of anecdote is not data, and the sum of "vast clinical experience" is not science.

Objective evidence strongly suggests that we should abandon the term "shaken-infant syndrome" or "shaken-slammed infant syndrome" and instead use an actual description of the injury mechanism (i.e., "rotational deceleration") and admit that we do not know the force required to cause the injury. We must recognize that "retinal hemorrhage," regardless of its characteristics, is at best an external marker for a probable head injury. Our testimony needs to ac-

knowledge that there are very limited data regarding a lucid interval in a child following rotational head injury, and that the concept of diffuse axonal injury may not explain why some children die very quickly after an injury whereas others appear to have a symptom-free or relatively symptom-free interval prior to death. We need to differentiate an old or subacute subdural hemorrhage from an acute bleed and admit that we know little about the actual cause or causes of a re-bleed. We should cautiously interpret a caretaker's story that is inconsistent with the physical findings, because the caretaker may not know the true history, especially if a lucid interval has occurred. We must abandon simplistic, inaccurate, and unproven phrases such as "globally changed", "immediately unconscious," "fall from a two-story building," and "force so great the perpetrator must have known that he or she would cause injury." (How can it ever be known scientifically what another human being knew or intended?) Modern medicine has many examples of concepts and technologies accepted as proved and promoted as a standard of care that were subsequently determined to have no value and in fact to be wrong (e.g., routine use of fetal monitors, routine skull radiographs in children with head injuries, Papanicolaou smears in woman who have had had a hysterectomy for non-malignant disease, indications for blood component therapy) (42). We need to differentiate between what we know to be true and what we think or hope to be true. The concept of a shaken infant syndrome deserves to be examined and re-examined, even when we think we finally have it right.

Forensic pathologists especially must be cautious in their testimony and critical in their evaluation of the literature. Too many of my colleagues, as well as most other physicians and much of the general public, think of our profession as the "whodunit" discipline. It is not. Forensic pathology is the "what happened" specialty. When our focus is on the "who," we forget the "what" or may consider it unimportant. Worse, we may alter our explanation or interpretation of the "what" to make it conform to our opinion of the "who." The need to consider alternative explanations ceases, and doors to further inquiry close. Objectivity fails because we are forced to defend an advocate's role, be it for the government or for the defendant. We must not forget that our only responsibility is to bear witness within the limits of science.

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Shaken Baby Syndrome

To the Editor:

Dr. John Plunkett has made an important and significant contribution to the medicolegal and forensic pathology literature regarding the so-called "shaken baby syndrome" (SBS). His well-written, objective, and succinct article hopefully will be read by all practicing forensic pathologists in coroner and medical examiner offices throughout the United States (1). I also hope that this article will be brought to the attention of pediatric neurosurgeons, neurologists, ophthalmologists, and emergency department physicians, especially those practicing in children's hospitals.

The actual existence of SBS as a pure pathologic entity unassociated with blunt force head trauma must be seriously questioned. This diagnosis is made much too frequently and is definitely much too zealously and dramatically pursued by forensic pathologists and clinicians. It has developed a scientifically attractive appeal that has lured law enforcement officers and prosecutors into its web.

In any event, whether our colleagues accept or reject SBS as a pure traumatic event leading to specific pathophysiologic manifestations, we should all refrain from the kinds of dramatic, unscientific remarks that have permeated testimony to the point of becoming trite, such as the standard phrases "the equivalent of a fall from a two-story building," and "the worst case of child abuse I have ever seen." Forensic pathologists functioning as coroners and medical examiners should remember that they are not an integral part of the prosecutor's team, and they should not consciously or unwittingly use language that is clearly designed to inflame and prejudice a jury. Forensic pathologists must constantly strive to be objective, rational, fair, and even-handed in expressing their conclusions and opinions in trial testimony and all other formal legal proceedings

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Shaken Baby Syndrome

To the Editor:

We read with interest the paper by Plunkett (1) entitled "Shaken Baby Syndrome and the Death of Matthew Eappen." Dr. Plunkett makes several points in his paper.

First, he casts doubt on the existence of whiplash shaking syndrome. He cites a lack of experimental evidence and a lack of disinterested witnesses observing the shaking. He states that experimental evidence has shown an inability of an individual to shake an infant with sufficient force to cause fatal head injuries. He alleges that early descriptions of whiplash shaking syndrome by Caffey (2) and Guthkelch (3) began myths that have been perpetrated throughout the years without additional evidence.

Caffey (2) developed the concept of whiplash shaking syndrome after interviewing a nurse working in a hospital newborn nursery in which an inordinate number of children died or were found subsequently to be mentally retarded. The nurse stated that she did not like the infants crying and would shake them until they ceased. Others (4) have had cases in which there was a confession of whiplash shaking.

Plunkett, citing experiments conducted by Duhaime et al. (5), suggests that it is impossible for an individual to shake an infant with sufficient force to cause craniocerebral injuries. However, Byard and Cohle (4) and Alexander et al. (6) have specifically sought evidence for blunt trauma in babies fitting the profile for whiplash shaking syndrome and have not found it, suggesting that shaking alone can cause fatal head injury. Furthermore, experimental models in Duhaime et al.'s (5) research used doll heads stuffed with cotton, which may not closely resemble an infant's skull and brain.

Plunkett cites "anecdotal evidence of children who have suffered clearly accidental head injuries from short distance falls" to emphasize the point that children may suffer such severe head injuries with minimal trauma. Curiously, he also states that a review of the literature of short falls (7-14) shows a total of 3,056 cases of short falls with 9 fatalities. Seven of these were reported by Chadwick et al., (12) who stated that these deaths were suspicious and that the history was probably falsified. Aoki and Masuzawa (13) reported 2 deaths out of 26 falls allegedly occurring at home, but there were no witnesses to these falls other than the caretaker. Additionally, Chadwick and Salerno (15) studied 338 children who experienced short falls, each of which occurred in a situation in which multiple witnesses were present (most of the cases were

from day care centers with multiple caretakers). The most serious injury occurred in a 2½-year-old who fell 5 feet onto a concrete walk and was unconscious for approximately 1 minute. A computed tomography scan of the head showed no injury, and he recovered fully in 1 hour.

Reiber (16) reported 19 cases of injuries from short falls (5-6 feet or less). In no cases were there witnesses other than the caretaker. He felt that 14 (74%) represented an inflicted trauma, whereas 3 had unexplained retinal hemorrhages or other evidence of battering. He opined that in two cases the history of an accidental fall was genuine but did not state why, because, as in the other cases, there were no corroborative witnesses.

Hall et al. (17) also reported a series of severe and fatal injuries from short falls. Only two of the falls in their series were witnessed by someone besides the caretakers (medical personnel in each case). These children appeared well after the fall and did not receive medical care until they returned subsequently with fatal head injuries. One of their conclusions was that short falls in children may be fatal. However, even in the two falls witnessed by medical personnel, because there was no immediate treatment, there was a subsequent opportunity for an assailant to fatally injure the children. Joffe and Diamond (18) pointed out that although Hall and co-authors stated that the falls were less than 3 feet, they admitted they did not have prehospital and hospital records in order to make this conclusion. In Hall's series there was a high frequency of delay in seeking medical care for the fatally injured children. In their letter of response, Hall et al. (19) concluded that despite the history of a trivial fall, a child should be observed for evidence of a severe head injury.

Plunkett states that a lucid interval may occur after development of an injury that becomes fatal. The three papers that he cites to support this concept include one (20) in which one of three cases cited allegedly had a 4-day lucid interval before being admitted to the hospital. However, there was no reason to believe that the baby-sitter could not have assaulted the child at any period subsequent to the 4 days, including immediately prior to admission. The other two citations by Plunkett used to support his lucid interval theory were a letter to the editor (21) that he wrote disputing the conclusion of a study published by Willman et al. (22) who found 1 "lucid interval" in 95 fatally head-injured children. This one case involved an epidural hematoma. The final citation by Plunkett is a court case, and in none of his three citations is there scientific evidence for a lucid interval (23).

In their response to Dr. Plunkett's letter to the editor, Willman et al. (24) emphasized that in cases

of child abuse the histories are obviously unreliable, and therefore the only reliable data to determine whether a lucid interval exists after a fatal head injury are accidental injuries in which the time and circumstances are known. Dr. Plunkett in his letter had stated that biomechanics of motor vehicle crashes and abusive injuries (shaking, direct impact, and falls) were different. Willman and associates pointed out, and this has been my experience, that the pattern of injuries seen in children fatally injured in motor vehicle accidents is often quite similar to that seen in abusive injuries. A demonstration of diffuse axonal injury in shaken babies (25) is further evidence for the immediate loss of consciousness in shaken babies.

To quote Plunkett (1), "Forensic pathologists especially must be cautious in their testimony and critical in their evaluation of the literature."

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AUTHOR'S REPLY

I thank Dr. Cohle and his colleagues for their comments. The majority opinion (the specificity of retinal and subdural hemorrhage for inflicted trauma, nonlethality of short distance falls, and absence of a lucid interval in ultimately fatal head injury) is certainly on their side. I wrote the article to encourage consideration of a minority view supported by biomechanical analysis and nontautologic reasoning.

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