

Shaken Baby Syndrome: Inadequate Logic, Unvalidated Theory, Insufficient Science

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Abstract:

This paper tells the ‘story’ of shaken baby syndrome/abusive head trauma, tracking the evolving medical literature and demonstrating the influences on some of the significant court cases in which the tenets of the diagnosis were shown to have been faulty. The author argues that current child protection approaches have turned a scientifically unvalidated medical hypothesis into a nearly unstoppable courtroom force. Parents of children with legitimate medical conditions are being targeted as abusers, and concerns have arisen that their children are denied the treatment they need. Child protection resources are being misdirected into prosecuting cases in which, arguably no wrongdoing may have occurred. In both the criminal and the public family jurisdictions, the cumulative effect is the wastage of public resources and miscarriages of justice. In many cases, human tragedy is the result, involving families being torn apart, wrongful imprisonment and the wrongful placement of children into state ‘Care’.

Introduction

For nearly 40 years, prosecutors have been winning convictions on charges ranging from child endangerment to murder based on the hypothesis of Shaken Baby Syndrome (SBS) (Committee on Child Abuse & Neglect, 1993), recently renamed Abusive Head Trauma (AHT) (Christian 2009). Doctors who accept the hypothesis believe that the syndrome is characterised by a triad of symptoms, consisting of retinal hemorrhages, cerebral hemorrhaging and encephalopathy (Richards *et al*, 2006; Harding, Risdon & Krous, 2004).

SBS has proved potent in the courtroom, but serious problems have arisen, particularly when the triad, or parts of it, have been assumed to indicate child abuse even if the child has no bruises, abrasions, grip marks, or other external signs of battering. Other problems and debates have arisen around timing; using evidence, for example from Willman *et al*, (1997) and Kirschner & Wilson (1994), the prosecution has tended to assume that the caretaker who is with the child when the symptoms arise, has caused the symptoms, whereas for example, Gilliland *et al* (1998) have provided evidence that the onset of symptoms may be delayed.

Accumulated evidence has now shown that the diagnosis of SBS is not scientifically supported (Findley *et al*, 2012) and therefore prone to be associated with Miscarriages of Justice (Geddes & Plunkett, 2004; National Registry, 2014). As yet though, many convicted caretakers remain convicted, broken and traumatized families remain broken and traumatized, and, with further tragic consequences, the prosecutions continue. As Tuerkheimer (2014a) explains, “We now know that the diagnostic triad alone does not prove beyond a reasonable doubt that an infant was abused, or that the last person with the baby was responsible for the baby's condition.” However, with still too few exceptions, “our legal system has failed to absorb this new consensus... innocent parents and caregivers remain incarcerated and, perhaps more perplexingly, triad-only prosecutions continue even to this day.” (Tuerkheimer, 2014a)

Historical Perspective

Although the foundations had been laid earlier (Caffey, 1946), child abuse as a medical diagnosis was conceptualised in the early 1960s with the publication of Dr. C. Henry Kempe's “The Battered-Child Syndrome” (Kempe *et al*, 1962), which triggered today's mandated-reporting laws for medical and educational professionals across the United States. The subspecialty of child abuse pediatrics was added to the medical-board exams just a few years ago (Giardino *et al*, 2011).

Under Kempe's guidelines, doctors began looking carefully for signs of abuse and soon noticed that children they believed to be battered often had retinal hemorrhages (RH), (Gilkes & Mann, 1967); some physicians came to believe RH occurred only in cases of abuse (Eisenbrey, 1979). Subdural hematoma (SDH, bleeding between the outer two layers of the meninges, the 3-ply membrane that encases the brain and spinal fluid) was historically associated with birth, impact injury, and meningeal infection (Guthkelch, 1953). In 1971, pediatric neurosurgery pioneer Dr. A. Norman Guthkelch in Britain proposed that shaking an infant could cause subdural bleeding without direct impact to the head (Guthkelch 1971). A later paper in the U.S. by pediatric radiologist Dr. John Caffey popularized the proposition (Caffey 1972), which was adopted on both sides of the Atlantic (Ludwig & Warman, 1984; Carty & Ratcliffe, 1995).

Guthkelch had focused on SDH in the absence of impact injuries. Caffey first proposed that shaking would cause SDH, RH, and fractures of the long bones, adding cerebral edema later (Caffey, 1974). “Cerebral edema” is essentially “brain swelling,” although there is a medical distinction between excess fluid within the cells and excess fluid in the intercellular spaces.

The observation below in a 1984 paper illustrates that doctors had begun to believe that SDH, especially in the gap between the two hemispheres of the brain, resulted only from a violent event:

“Our findings indicate that the young infant manifesting respiratory alterations in the face of a normal pulmonary examination may have been shaken... The physical findings of tense or bulging fontanelle, head

circumference greater than 90th percentile for age, and retinal hemorrhage strengthen the diagnosis... Computer cranial tomography may specifically confirm SBS by showing acute interhemispheric subdural hematoma or cerebral contusion in the absence of external trauma.” (Ludwig & Warman, 1984)

A 1993 review assembled a definition of SBS much like that often used in court today:

“The full picture... comprises subdural haematoma, massive cerebral oedema, haemorrhagic retinopathy, fractured ribs and metaphyseal injury, but any combination of the above may occur. The classical picture occurs without any skull fracture, bruising of the scalp, oedema or evidence of direct head trauma.” (Brown & Minns, 1993)

“Metaphyseal injury” means a lesion at one edge of a bone’s growth plate, thought by Kempe to result only from abuse (Kempe *et al*, 1962).

Once doctors started interpreting SDH and RH as certain signs of Non Accidental Injury (NAI), the literature soon expanded with observational studies and case reports. By the turn of the century, hundreds of doctors in various specialties had published their efforts to learn more about the syndrome by studying the babies they treated or examined who had been diagnosed as shaken, based on the model of infant head injury then being taught at conferences and in medical schools (Block, 1999).

While the diagnosis was gaining acceptance in the field however, biomechanical research had started raising doubts. In 1987 the Duhaime team reported their findings that shaking by a normal adult does not generate the forces thought necessary to trigger concussion, SDH, or damage to brain cells from shearing or stretching (Duhaime *et al*, 1987). Noting that angular accelerations spiked when volunteers were told to throw the test mannequin down after shaking, the authors concluded:

“Although shaking may, in fact, be a part of the process, it is more likely that such infants suffer blunt impact. The most common scenario may be a child who is shaken, then thrown into or against a crib or other surface, striking the back of the head and thus undergoing a large, brief deceleration.” (Duhaime *et al*, 1987)

This research entered the discussion but had little effect on the thinking of most practitioners. Even in cases with signs of impact, doctors often conclude that SDH and RH indicate the child was shaken as well as slammed (Brown, 1993; Shapiro, 2012; National Registry, 2014).

Complicating the situation further, child-abuse experts teach that children seldom if ever receive fatal injuries in short falls, even falls down stairs (Joffe & Ludwig, 1988; Chadwick *et al*, 1991; Lyons & Oates, 1993; Chadwick *et al*, 2008), but other reports refute this conclusion (Hall, 1989; Denton, 2003; Lantz & Couture, 2011), including a fatal fall caught on videotape in which a toddler rolled off a post she was straddling,

with her feet 24 inches off the floor (Plunkett 2001). Similarly, recent research has acknowledged various non-traumatic causes of RH (Agrawal *et al*, 2012) and SDH (Squier & Mack, 2009) and non-abusive causes of interhemispheric subdurals (Tung *et al*, 2006), and bone development is a more complex process than previously understood (Seeman & Delman, 2006).

Timing of the Injuries

The first formal statements about SBS emphasized the obscurity of the condition and cited slowly evolving symptoms. In 1993, the *American Academy of Pediatrics* wrote:

“Shaken baby syndrome is characterized as much by what is obscure or subtle as by what is immediately clinically identifiable. A shaken infant may suffer only mild ocular or cerebral trauma. The infant may have a history of poor feeding, vomiting, lethargy, and/or irritability occurring intermittently for days or weeks prior to the time of initial health care contact. The subtle symptoms are often minimized by physicians or attributed to mild viral illnesses, feeding dysfunction, or infant colic.” (Committee on Child Abuse & Neglect, 1993)

And in Britain:

“After a variable time, the infant will develop signs of cerebral irritation, cerebral oedema, or intracranial haemorrhage. Acute deterioration, convulsions, or respiratory or circulatory arrest may follow.” (Carty & Ratcliffe, 1995)

Physicians have recognized for centuries that a person might suffer a serious head injury followed by a “lucid interval,” a period of time when the patient appears to be normal, only to collapse later, after pressure inside the skull has built to debilitating levels. Child abuse doctors, however, often teach that the symptoms of a head injury are always immediate in a baby. A leading textbook in 1994, for example, argued that the infant’s immature brain reacts differently to trauma and offered this guideline, with no literature citations:

“Despite its frequency, deaths (sic) related to head trauma in infancy remain the most difficult to prosecute for two reasons: (1) lethal head trauma in infants is often produced without external evidence of injury, leading to speculation about mysterious natural causes or trivial accidental injury; and (2) many clinicians and pathologists equivocate about the timing of the lethal event because they are uncertain of the immediate relationship between the injury and its effect on the brain. It is best, therefore, to state immediately two axioms regarding fatal head trauma in infants:

1. Trivial events (i.e., falls from beds, couches, against coffee tables, etc.) produce trivial injuries; significant events (blows, shaking, forced impact) produce potentially lethal injuries.

2. Lethal injuries produce progressively more severe symptoms almost immediately; no significant “lucid” or asymptomatic period occurs.” (Kirschner & Wilson, 1994)

In an early review of the literature for guidance about timing, Nashelsky and Dix found only three published cases of pure shaking with no signs of impact:

“In two cases, there was onset of symptoms immediately after shaking. In one case, there was a delay of 4 days between shaking and onset of seizures although the child had several episodes of vomiting during the days after shaking. One wonders whether the infant was reshaken shortly before onset of seizure activity.” (Nashelsky & Dix, 1995)

The opinion of the authors aside, this seems to be a report of a lucid interval following a shaking injury.

Researchers at a San Diego children’s hospital tried to extrapolate timing from accidental injury. They identified 95 witnessed, fatal accidental injuries to children younger than 16 years—4 of them under the age of 2—almost all of them from motor vehicle accidents (Willman *et al*, 1997). The researchers looked only at cases where an emergency response team was sent to the scene. They tracked each child’s level of consciousness, starting with the assessments by the EMTs in the field. The only children who seemed to deteriorate after medical contact suffered from growing epidural hematomas (bleeding between the dura and the skull), so the authors concluded:

“Unless an epidural hematoma is present, children who die of blunt head injuries probably do not experience lucid intervals. In cases of fatal HI [head injury] where the history claims that the child looked well following the injury and only later began to act abnormal, the story must be questioned and nonaccidental trauma must be suspected.” (Willman *et al*, 1997)

The next year, however, forensic pathologist Dr. M.G.F. Gilliland published her observations based on the medical records of 76 fatal pediatric head-injury cases collected from her own and her colleagues’ experiences. A quarter of Gilliland’s cases featured a time lag between the trauma and an obvious deterioration in consciousness, with a definition of “brief” that differed significantly from Willman’s:

“Our data indicate that the interval is brief (less than 24 hours), in almost 3/4 of cases of head injury death, especially in shaking injuries. However, in more than 1/4 of the cases, the interval from injury to the onset of severe symptoms is longer. In all cases in which the children were seen by an independent observer after injury, they were described as not normal.” (Gilliland *et al*, 1998)

At that time, the medical literature contained at least two case reports of catastrophic infant head injuries with delayed symptoms (Frank, Zimmerman & Leeds, 1985; Nashelsky & Dix, 1995).

Louise Woodward

Shaken baby syndrome hit the headlines in 1997, when “Boston nanny” Louise Woodward was accused of shaking to death 8-month-old Matthew Eappen. Like many children in shaking cases, Matthew had a skull fracture that could not be dated. He also had a healing wrist fracture that no one could explain and a fatal brain injury.

Prosecution experts testified that Matthew’s brain injury proved he had been shaken immediately before losing consciousness. The defense doctors argued that the boy suffered from an older brain injury that had started rebleeding while he was in the nanny’s care. To dispute the shaking diagnosis, the defense also called Prof. Lawrence Thibault, the biomechanical engineer who had designed the mannequins for the Duhaimé research, and Dr. Ayoub Ommaya, whose early biomechanical results Duhaimé relied on for her analysis (Ommaya et al, 1984). After a televised trial, the jury convicted Woodward, but then Judge Hiller Zobel reduced the charges to manslaughter and set the sentence to time served. Woodward returned to England, to something of a heroine’s welcome (Lyll, 1997).

Unhappy with the outcome, several dozen child abuse doctors published a statement dismissing the Woodward defense and decrying media coverage that treated it as credible. The doctors articulated their faith in both immediate symptoms and the reliability of three findings as proof of SBS:

“Infants simply do not suffer massive head injury, show no significant symptoms for days, then suddenly collapse and die. Whatever injuries Matthew Eappen may or may not have suffered at some earlier date, when he presented to the hospital in extremis he was suffering from proximately inflicted head injuries that were incompatible with any period of normal behavior subsequent to the injury. Such an injury would and did produce rapidly progressive, if not immediate, loss of consciousness.

The shaken baby syndrome (with or without evidence of impact) is now a well-characterized clinical and pathological entity with diagnostic features in severe cases virtually unique to this type of injury—swelling of the brain (cerebral edema) secondary to severe brain injury, bleeding within the head (subdural hemorrhage), and bleeding in the interior linings of the eyes (retinal hemorrhages). Let those who would challenge the specificity of these diagnostic features first do so in the peer-reviewed literature, before speculating on other causes in court.” (Chadwick *et al*, 1998)

Forensic pathologist John Plunkett responded by criticizing testimony at the Woodward trial that the force necessary to cause the child’s injuries would be equivalent to a fall from a two-story building or a car crash (Plunkett 1999), and he contested immediate symptoms, as he had previously in a response to the Willman paper. (Plunkett, 1998)

Also unsatisfied by the Woodward outcome was a handful of pioneers in the field of biomechanics, including Prof. Werner Goldsmith at Berkeley, who had devoted the last portion of a long and prolific career to studying helmet design, for which he focused on head and neck injuries. Drawn in by colleagues politicized by Woodward, Goldsmith collaborated with Dr. Ommaya and Prof. Thibault on their final paper, a biomechanical critique dismissing shaken baby syndrome (Ommaya et al, 2002). Later, a former student of Ommaya's published an analysis explaining his conclusion that an infant's neck would break from a shaking assault before the forces would be great enough to trigger subdural bleeding (Bandak, 2005). Critics of Bandak's work say he has made a miscalculation (Margulies, 2006, Rangarajan & Shams, 2006), but Bandak disputes their analysis (Bandak, 2006), and another commentator observes that even if Bandak made the alleged miscalculation, his point remains valid (Molina, 2009).

A number of other biomechanical researchers have revisited the Duhaime study. Most have replicated her results (Prange *et al*, 2003; Cory, 2004; Lloyd *et al* 2011), although one team has achieved angular accelerations more compatible with shaking as a mechanism (Jenny *et al*, 2002; Jenny, 2005).

In 2001, the *American Academy of Pediatrics* updated its SBS position statement, taking a half-step back from the slowly evolving symptoms described in 1993. Combining old language with new, the statement said that parents would be immediately aware that their baby was behaving differently, although a doctor might not recognize a head injury:

“A victim of sublethal shaking may have a history of poor feeding, vomiting, lethargy, and/or irritability occurring for days or weeks. These clinical signs of shaken baby syndrome are immediately identifiable as problematic, even to parents who are not medically knowledgeable (19). However, depending on the severity of clinical signs, this may or may not result in caretakers seeking medical attention. These nonspecific signs are often minimized by physicians or attributed to viral illness, feeding dysfunction, or colic.” (Committee on Child Abuse & Neglect, 2001:207)

Footnote 19 is referring to an article by (Duhaime *et al*, 1998) that concludes:

“Thus, an alert, well-appearing child has not already sustained a devastating acute injury that will become clinically obvious hours to days later.”

Also in 2001, the National Association of Medical Examiners (NAME) released a committee statement that offered this description of assaultive head injuries:

“The mechanism of injury produced by inflicted head injuries in these children is most often rotational movements of the brain within the cranial cavity. Rotational movement of the brain damages the nervous system by creating shearing forces, which cause diffuse axonal injury with disruption of axons and tearing of bridging veins, which causes subdural and subarachnoid hemorrhages, and is very commonly associated with retinal schisis and hemorrhages. . . . The

pathologic findings of subdural hemorrhage, subarachnoid hemorrhage and retinal hemorrhages are offered as “markers” to assist in the recognition of the presence of shearing brain injury in young children.” (Case *et al*, 2001)

This definition does not mention cerebral edema but adds diffuse axonal injury (damage to neurons stretched or compacted beyond their physical limits rather than injured by a direct blow), one of the markers evaluated by the Duhaime team but not examined in the original primate research (Ommaya *et al*, 1968) cited by Guthkelch in his seminal paper (Guthkelch, 1971). “Subarachnoid hemorrhage” refers to bleeding underneath the arachnoid layer of the meninges, closer to the brain than subdural blood.

The 2001 NAME statement also argued that shearing of the neurons during the act of shaking would cause an immediate change in consciousness (Case *et al* 2001), a statement that triggered a third published case report of a lucid interval. Forensic Pathologist Dr. Huntington had autopsied a toddler who was brought to the hospital in the morning for ongoing vomiting. Hospital records described her as “fussy and clingy, but interactive and responsive,” and she was treated for gastrointestinal problems. The breathing difficulties began in the middle of the night, after she had spent more than 12 hours in the care of trained medical professionals, none of whom recognized the signs of an ultimately fatal brain injury (Huntington, 2002).

The lead author of the statement responded that the girl was in fact showing the signs of her brain injury:

“When a child has suffered a serious acceleration injury to the brain that will result in long-term neurologic impairment or cause death, the so-called lucid interval is a fiction. The change from “fine” to “not fine” may be lethargy or it may be unresponsiveness, but it is a neurologic change, and it occurs at the time of injury.” (Case *et al*, 2002)

A fourth case report soon followed, in which a 9-month-old collapsed on the third day after a reported fall from a bed (Denton & Mileusnic, 2003). The infant had been observed in the interim by his mother, his grandmother, and his babysitter, who knew about the fall and were looking for problems but had observed nothing alarming.

Finally, in 2005, researchers in Pennsylvania extracted cases of fatal head injuries in patients younger than 4 years from nearly two decades of state trauma records, and discovered that a small but positive number, about 2%, of children with ultimately fatal head injuries reached the emergency room with a score of 13-15 on the 15-point Glasgow Coma Scale (Arbogast, Margulies & Christian, 2005). Younger children were especially likely to appear normal at first medical contact.

In 2003, meanwhile, Dr. Mark Donohoe had applied the criteria of evidence-based medicine to the early SBS literature, identifying articles through a Medline search on the phrase “shaken baby syndrome” (Donohoe, 2003). He concluded that the hypothesis had not been proven, noting that much of the literature consisted of

“... opinions that shed no new light upon SBS and did not add to knowledge about SBS. Many of the authors repeated the logical flaw that if RH and SDH are almost always seen in SBS, the presence of RH and SDH “prove” that a baby was shaken intentionally...

Thus, the data available in the medical literature by the end of 1998 were inadequate to support any standard case definitions, or any standards for diagnostic assessment.” (Donohoe, 2003)

In fairness, setting up double blinded experiments with valid control groups in this arena is challenging, because there is no objective standard for establishing that a child has been abused. Still, the circular reasoning Donohoe identified continues in today’s literature. An attempt by Bechtel *et al* (2004) to identify the characteristics that distinguish abused children from children with accidental injury, for example, identified children with subdural hematoma (*SDH*) with no history of a traumatic event as “inflicted”. The resulting paper has since been incorporated into a number of meta-analyses that pool the data from a collection of similar studies, e.g., Maguire *et al* (2011) and Piteau *et al* (2012).

Ten years later, Donohoe’s work continues to receive harsh criticism from child abuse experts (Greeley, 2011; Holmgren, 2013), but no one has yet identified the research that has established shaken baby syndrome is more than a hypothesis. Direct experimentation is unthinkable, obviously, and the occasional witnessed or videotaped shaking has yet to result in the symptoms now diagnosed as shaking injury (Luttner, 2012).

Researchers in Britain also began raising questions about some of the neurological tenets of shaken baby syndrome. In 2001 neuropathologist Dr. Jennian Geddes concluded that much of the brain damage in these cases comes not from direct trauma but from a lack of oxygen to the tissues, as bleeding and swelling interfere with normal systems (Geddes *et al*, 2001, parts I & II). Working from a foundation laid by Geddes (Geddes *et al*, 2003), neuropathologist Dr. Waney Squier and her colleagues proposed that the very thin subdural collections often seen in presumed shaking cases represent seepage of blood from the dura itself, rather than spillage from torn bridging veins (Squier & Mack, 2009). Rejected at first, Squier’s proposed non-traumatic origin for thin-film subdurals is now gaining acceptance (Slovis *et al*, 2012).

Audrey Edmunds

The 2001 statement from the *National Association of Medical Examiners* (Case *et al*, 2001) ultimately brought another highly contested case into the debate. Dr. Robert Huntington, who had responded with the letter documenting delayed symptoms in a hospital setting, had testified several years earlier in the trial of Audrey Edmunds, a care provider convicted of murder for the presumed shaking death of an infant. After his

personal experience, Dr. Huntington knew he had been wrong when he told the Edmunds jury that the symptoms of a fatal pediatric head injury would be immediately obvious.

When they learned of Dr. Huntington's change of heart, law professor Keith Findley and his students at the Wisconsin Innocence Project began researching both the timing of the symptoms and the larger questions around shaken baby. They successfully reopened the Edmunds case, with the argument that opinion within the medical community had changed, and the consensus around SBS was dissolving. A state appeals court vacated Edmunds' conviction in 2008 (*State v. Edmunds 2008*).

The Edmunds decision attracted the attention of law professor Deborah Tuerkheimer, who followed up the footnotes in the appeal and reached the same conclusion as Findley's team: Not only had shaken baby syndrome never been proven, but biomechanical research and accumulated experience were both eroding its credibility (Tuerkheimer, 2009; Tuerkheimer, 2011).

In 2009, the American Academy of Pediatrics updated its position statement again, this time making no comment on timing but abandoning the term "Shaken Baby Syndrome" in favor of "Abusive Head Trauma," with this explanation:

Legal challenges to the term "shaken baby syndrome" can distract from the more important questions of accountability of the perpetrator and/or the safety of the victim. The goal of this policy statement is not to detract from shaking as a mechanism of AHT but to broaden the terminology to account for the multitude of primary and secondary injuries that result from AHT. (Christian, 2009)

Shirley Ree Smith

SBS resurfaced in the news in 2011, when the U.S. Supreme Court reinstated the 1997 conviction of grandmother Shirley Ree Smith, vacating a 2006 reversal by the Ninth Circuit Court of Appeals, which had declared the evidence against Smith "constitutionally insufficient." The Supreme Court's decision to reverse did not address the question of Smith's guilt or innocence, but turned on the court's opinion that the Ninth Circuit did not have the power to overrule the jury's decision (*Cavazos v. Smith (2011) 132 S. Ct. 2.*). As in Woodward, the legal outcome supported the prosecution, but the defendant went free amid a whirl of sympathetic publicity, this time after Governor Jerry Brown issued a pardon. The Smith decision also contained a strongly worded dissent written by Justice Ruth Bader Ginsberg, who cited some of the research casting doubt on shaken baby syndrome and noted that the child's symptom list (subdural hemorrhage, subarachnoid hemorrhage, and optic nerve sheath hemorrhage) did not match any known profile of the condition.

Again, the outcome irked the community of child abuse experts, who denounced the decision at conferences (Greeley, 2012; Alexander & Holmgren, 2012) and in print (Moreno & Holmgren, 2013). "Despite all the ballyhoo, there has been no paradigm shift

in the scientific support for the diagnosis of AHT/SBS,” attorneys Moreno and Holmgren wrote, attributing the “false controversy” to scientists in it for the money:

... the outlier views expressed by defense medical witnesses and parroted in law review articles is self-validating (sic). The academics cite the same handful of defense medical witnesses, the media cites both, the defense medical witnesses benefit from the publicity and are hired in more cases, and the cycle begins anew. (Moreno & Holmgren, 2013)

This presumed cadre of defense-witnesses-for-hire receives recurring grief and ridicule at shaken baby conferences: In 2010, in front of 600 people, a guitar-playing pediatrician led a karaoke-style sing-along with the chorus “If I only get ten grand” to the tune of “If I Only Had a Brain” from the *Wizard of Oz* (Holmgren, 2010).

Speakers at that conference also characterized “the so-called triad” as a straw man invented by defense experts to make the diagnosis look overly simple. “Only people who are NOT active physicians working with children, naïve journalists, and professors with a biased agenda would propose that only three signs and symptoms support a diagnosis,” declared Dr. Robert Block, at the time president pro tem of the American Academy of Pediatrics (Block 2010). Another frequent presenter at SBS/AHT conferences has recently published an editorial declaring the AHT controversy “fabricated,” with this observation:

“The complex features of AHT are often disparagingly distilling (sic) simply to “The Triad”; a term devoid of any real clinical meaning and not used at all in practice.” (Greeley, 2014)

For the first decade of SBS conferences, however, the “triad” was taught as a matter of course. From the executive summary published in 2001, for example, after the third conference:

“As in prior conferences, a major focus of this conference was the constellation of injuries which are present in almost every shaken baby case, but which are rarely seen in any other accidental form of trauma (sic). Often referred to as the “triad,” the consensus continues to be that a collection of (1) damage to the brain, evidenced by severe brain swelling and/or diffuse traumatic axonal injury; (2) bleeding under the membranes which cover the brain, usually subdural and/or subarachnoid bleeding; and, (3) bleeding in the layers of the retina, often accompanied by other ocular damage, when seen in young children or infants, is virtually diagnostic of severe, whiplash shaking of the head.” (Parrish, 2001)

Also as mentioned earlier, dozens of child abuse experts signed a statement endorsing a triad—a more restrictive triad than Parrish’s, but the same one articulated in the *British Medical Journal* a few years later (Harding, 2004)—after the Woodward sentencing (Chadwick *et al*, 1998).

The medical literature now contains a growing body of case reports and opinions about other conditions and mechanisms that can produce the same symptoms (Piatt, 1999; Pittman, 2003; Clemetson, 2004; Orient, 2005; Talbert, 2005; Talbert, 2009; Barnes, Galaznik, Gardner & Shuman, 2010; Stray-Pedersen *et al*, 2010; Barnes, 2011; Squier, 2011; Talbert, 2012; De Leeuw *et al*, 2013 and Scheimberg *et al*, 2013). The recent editorial dismissing “The Triad” as a defense construct also criticizes papers like these as “unsupported and fringe theories... created not with a clinical gap to fill, but explicitly to be used in court” (Greeley, 2014). Ultimately though, Tuerkheimer (2014b) reports that U.S. District Judge Matthew Kennelly had finally declared Shaken baby Syndrome, “an Article of Faith” in the Del Prete case.

The *National Registry of Exonerations* in the U.S. meanwhile, documents a number of convictions that have been overturned after re-examination of the medical records revealed other explanations for the child’s collapse, so far including seizure disorder, stroke, sickle cell disease, sepsis infection, short fall and urinary tract infection and hypoxia (National Registry, 2014).

One of the ongoing problems though, is that many states have Child Protection laws which mandate that medical and educational professionals report any suspicion of child abuse, to be investigated by the designated authorities. There are no sanctions for a mistaken report and malice is hard to prove, but there are sanctions for a mandated reporter who sees signs of abuse and fails to act. In an effort to meet their mandated duties, hospitals have started coordinating their efforts with the state through child protection teams, which bring together law enforcement, social workers, and physicians from a range of specialties such as radiology, ophthalmology, and child abuse pediatrics. However, the investigations can be extremely harmful to members of families, including to the children, raising considerable ethical dilemmas (Barry & Redleaf, 2014). Social workers remove children from contact with the presumed abuser and pressure other family members to accept the diagnosis, heedless of the financial and emotional consequences (Barry & Redleaf, 2014) and police begin building a case against the caretaker who was on the scene when the problems started. On occasion, police have employed high-pressure, confrontational tactics on parents who were already emotionally distressed and confused because their child is critically ill (Leestma, 2006; Babcock & Hadaech, 2014; Boeri, 2011).

Conclusion

Faced with a growing chorus of critics and scant biomechanical evidence to support their model, proponents of the shaken baby syndrome hypothesis are now relying on their own clinical experience (Gill *et al*, 2009) and confessions (Adamsbaum *et al*, 2010) in an attempt to validate their opinions. However, confessions obtained under the pressure of plea agreements do not prove guilt. Even if the confession evidence were taken to imply guilt, this might help support the proposition that shaking an infant can cause brain bleeding and swelling, but it does nothing to prove that the triad of symptoms has always resulted from assault. As Donohoe (2003) pointed out, the logical flaw in the theory of

SBS is in assuming that since it is argued that a triad of symptoms appear in SBS, then the presence of the symptoms proves that the baby must have been intentionally shaken.

This paper is not arguing that shaking a baby is safe, or that infants do not suffer terrible injuries at the hands of parents and caretakers. It is pointing out that the hypothesis of the shaken baby/shaking impact syndrome is scientifically questionable and that the consequences of applying it in the courtroom have involved the human tragedy of miscarriages of justice.

Individual families affected by the diagnosis of infant shaking have been defeated by a united force against them. The prosecution in these cases has the full power of the state on its side, as well as the star witnesses. The accused parents may have limited medical knowledge and inadequate resources to hire an effective defense team for a trial that hinges on expert testimony. How do you prove you didn't shake your baby?

The physicians who have seen their opinions confirmed by decades of trial outcomes now believe that their model of infant head injury has been proven. The problem is that the theory has been entrenched through legal decision, rather than proven through scientific investigation. Historically, courts have endorsed the theory. Fortunately more recently, courts have been more questioning, bringing judicial decision-making more closely in line with science. The challenge ahead is how to unravel three decades of questionable convictions, and how to restructure child protection policies to stop the bulldozer.

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