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SPECIAL ISSUE: ABUSIVE HEAD TRAUMA

Update on injury mechanisms in abusive head trauma - shaken baby syndrome

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Abstract Violently shaking a baby leads to clinical presentations ranging from seizures to cardiopulmonary arrest. The main injuries sustained are retinal hemorrhages, subdural hemorrhages, and sometimes fractures and spine injury. It is important to have a global view of the injuries sustained by the infant to correctly discuss the biomechanical aspects of abusive head trauma. Recent works based on finite element models have shown that whiplash-shaking alone is enough to generate vitreo-retinal traction leading to retinal hemorrhage and to cause the rupture of bridging veins leading to subdural hemorrhage. We will review the main papers dealing with the mechanisms of shaken baby syndrome and present the most relevant hypothesis concerning the biomechanical aspects of injuries related to shaken baby syndrome.

Keywords Abusive head trauma · Biomechanics · Pathophysiology · Infant · Child

Introduction

A recent public hearing [1] made a review of the diagnosis of shaken baby syndrome, differential diagnosis and its legal consequences. Indeed, when dealing with an abusive head trauma case, the possibility of shaken baby syndrome must be considered. The incidence of shaken baby syndrome is between 15 and 28.7 per 100,000 infants in France and is certainly underestimated. Most cases occur before 6 months of age. With an average mortality of 21.6%, consequences of shaken baby syndrome consist also of physical and mental disorders: delayed psychomotor development, behavior disorder, learning difficulties, speech and language difficulties [2, 3].

Linked to the act of shaking by Guthkelch in 1971 [4], with the first injuries and mechanisms explained by Caffey in 1972 and 1974 [5, 6], this syndrome went through several names and labels (whiplash shaken baby, non-accidental head injury) and is sometimes subjected to high controversy in certain countries outside Europe. Indeed, few authors question the validity of the mechanisms thought to be engaged and others argue shaking only could not produce such injuries [7–10], and thus conclude it does not exist [11–13].

We will review the main papers dealing with the mechanisms of shaken baby syndrome and present the most relevant hypothesis explaining the injuries related to it.

Injuries related to shaken baby syndrome

The main injuries that raise high suspicion for shaken baby syndrome are:

- Subdural hematomas, which are more likely to be bilateral [4], but can be also unilateral. Subdural hematomas are the milestone in the shaken baby syndrome odyssey: indeed, contrary to other types of child abuse, infants with shaken baby syndrome can have this life-threatening injury without any external evidence of trauma.
- Retinal hemorrhages, which are also usually bilateral; unilateral ones do not rule out the diagnosis of shaken baby syndrome, though [14]. They are said to be flame-shaped, multilayered and diffuse.

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- Bone fractures: Due to the pressure of the perpetrator's hands, victims can have ribs or long bone (usually humerus) fracture; and if the shaking is very violent, it can also induce metaphyseal fractures [5] or joint fissuring due to the motion of the arms and legs during the shaking.
- Injuries to the spine: For a long time, diagnosis of shaken baby syndrome was done by looking mainly for subdural hematomas, retinal hemorrhages and fractures; the main progress in imaging techniques and especially the easier access to MRI, and the systematic dissection of the spine at autopsy after an infant death have shown the prevalence of these spinal injuries affecting not only the cervical spine but also the lumbar region. The cervical trauma may lead to cardiorespiratory arrest and explains the sometimes seen thin-layered subdural hematomas [15].

Shaken baby syndrome diagnosis is sometimes discussed because the above cited injuries may have other origins than shaking but what finally leads to the diagnosis is their association. A violent shaking event may lead to all these injuries at the same time. We will not deal with the bone fractures for which the mechanism of injury is clear. We will focus on the scientific evolution regarding the mechanism of the other injuries seen in shaken baby syndrome cases that have been the subject for controversies.

Mechanism of injury

The act of shaking seems to be benign and innocent, though the injuries are worse than in domestic or road accidents. "This extraordinary contradiction" noted by Caffey between the causes and the consequences, is counterintuitive [6]. One must not forget that we are talking about violent shaking and therefore from the beginning Caffey and Guthkelch used the word "whiplash" [4, 6]. It is important to understand this aspect when dealing with the mechanism of injury.

Retinal hemorrhage

Retinal hemorrhages had been used as evidence for child abuse [16] many years before Caffey described this injury in shaken baby syndrome cases. Their origins were suspected to be linked to increased intracranial pressure and/or obstruction of the retinal vasculature. This idea was supported by Duhaime et al. [7], although Greenwald et al. [17] in 1986 excluded those mechanisms and gave this explanation: "When an infant is shaken, its head is subjected to repetitive accelerations and decelerations due to the back and forth motion. … Transmission of force through these connections (lens, vitreous gel, retina) could result momentarily in significant traction on the retina, particularly in the posterior pole."

More recently, using optical coherence tomography, Forbes stated that "vein occlusion are uncommon in SBS" and that retinal hemorrhages are not observed in increased intracranial pressure cases [18]. Optical coherence tomography also revealed the existence of persistent attachment of vitreous at the apices of the perimacular region, which could explain the role of shearing forces at the vitreo-retinal interface in retinal hemorrhages.

Another study on postmortem orbital findings was conducted by Wygnanski-Jaffe et al. [19] on 18 cases of shaken baby syndrome and 18 cases of fatal accidental head trauma. Authors looked for retinal hemorrhages but also noted other hemorrhages (optic nerve intradural, subdural, subarachnoid hemorrhage). There was a striking gap in retinal hemorrhage percentages between shaken baby syndrome cases (78%) and accidental cases (17%); in those latter cases, retinal hemorrhages were "few in number" inside the eyes, whereas in shaken baby syndrome cases, they were diffuse, multilayered and too numerous to count [20].

These postmortem findings strengthen the theory explaining injuries in shaken baby syndrome. Yet the confirmation of the possible origin of retinal hemorrhages in shaken baby syndrome needs further experimentation. Experiments on animals or cadavers may be useful, but there is, of course, a difference between living human babies and animals and, thus, an analogy cannot be easily made.

Considering these limitations, scientists have turned towards mathematical modeling (finite element modeling) and simulation combined with ever-increasing technological power and means. Several eye models have been created to understand different injuries to the eye such as globe rupture [21] or blunt impact [22–25]. Models were also created to study the effect of shaking on the eye. Cirovic et al. [21] were the first in 2005, to our knowledge, to have conducted such work. Their eye model was composed of the orbital bones, the orbital fat, the sclera and the vitreous. They ran two types of simulations: impact and oscillation. From impact simulation (about 100 G, orbital bone amplitude of 1 mm), authors hypothesized that there should be suction between the eyeshell and orbital fat, and that extraocular muscles have a secondary role in holding the eye. This hypothesis was also corroborated by Schutte et al. [22] with their finite element simulations: Orbital fat not only has a main role in holding the eyeshell, but it also stabilizes extraocular muscles paths during eye motion.

From oscillatory movement simulation (200 Hz, orbital bone amplitude of 0.1 mm), in which they took into account this suction role of orbital fat, authors showed that the amplitude of the eye increases over time, thus increasing stresses at the poles of the eye.

The authors gave an important insight of the resonant effects during shaking, which may explain the disproportionate injuries in shaken baby syndrome. However, as they claim, the results of this first study are preliminary and provisional as

further work is needed, especially in creating a more accurate model with better material properties and giving realistic shaking frequency.

A few years later, Hans et al. [23] built up an acute and complete eye finite element model with all the relevant anatomical elements: lens, zonules, aqueous, vitreous, retina, choroid, sclera, optic nerve, ocular muscles and tendons and orbital fat. This eye model was then put in a baby skull model. The authors simulated a 4-Hz oscillation of the head around the neck pivot (the neck and neck muscles were not modeled), from −60° to +60°. The results showed a half-sine pattern for the four shaking cycles, with a peak force at each +60° amplitude.

They also simulated a 45-cm fall with a 9-month-old child dummy, impacting the occipital area of the head; the acceleration was about 60 G's.

By comparing the retinal nodal forces in these two tests, it can be seen that not only are the forces lower in the fall cases, but that they are also sustained for less time: Mean forces to the posterior eye of 0.08 N sustained for 150 ms, compared to 0.025 N for 26 ms in impact simulation. By keeping in mind that the reported fall caused left cerebral hemisphere hemorrhage and unilateral posterior retinal hemorrhages, one can imagine what could result from the 4-Hz shaking motion.

Another important point is the comparison they made with the monkey retinal adhesion force gathered from Kita et al. [24], which is of 0.14 N and by far exceeded in the third shaking cycle. Although the eye model components' mechanical properties are taken from human adult cadavers and sometimes modified from animals, the authors quoted that results are "suspected not to change too much if more compliant sclera, choroid, or retina is used." They concluded that shaking motion alone can induce higher forces in the posterior retina than impact.

The most recent work on shaking was done by Rangarajan et al. [25]. By modeling the orbit, the fat, the four rectus muscles, the sclera, the vitreous and the retina, the authors studied how changes in material properties of fat (elastic, viscoelastic solid) and vitreous (viscoelastic solid, Newtonian fluid) can affect retinal stress. Indeed, as the fat controls the motion of the globe in the orbit and as the vitreous motion is thought to pull the retina, their behaviors have to be understood correctly. For simulations, they choose an input frequency of 5 Hz, taken from tests on a 2.5-kg biofidelic doll.

Results revealed that contrary to fat, which did not show much effect on maximum stress and stress distribution when material property were changed, vitreous material property modifications showed two effects. First, increasing vitreous bulk modulus from 0.7 to 7 decreased maximum stress by 50%; second, switching the material type from viscoelastic to fluid increased the stress buildup and maximum stress.

Furthermore, results correlated with clinical manifestation of retinal hemorrhages in shaken baby syndrome, as maximum stress occurred in the posterior pole, where the vitreous is firmly attached to the retina. Last but not least, authors highlighted the stress accumulation during repetitive acceleration-deceleration motion and therefore stated that biomechanical calculations including only one single acceleration-deceleration motion, as during impact, cannot be used in the modeling of this form of abuse.

Limitations as for all finite element models are numerous and almost the same: eye components modeling (lens, zonules, choroid), better material properties, a well-refined mesh, shaking frequency. That is why these simulation works need to be continued and improved to better investigate eye trauma with biofidelic models.

It is interesting to notice that even though the hypothesis of vitreo-retinal traction had been submitted early in the shaken baby syndrome debate by Greenwald [17], before the uses of optical coherence tomography and finite element models, this idea was not investigated as much as the hypothetic increase in intracranial pressure or obstruction of retinal vasculature theories, although these theories were ruled out by clinical evidence. This has unfortunately led to meaningless discussion on the existence of shaken baby syndrome. Numerical simulations have helped to understand what is happening within the eye when a shaking event occurs and the vitreo-retinal traction hypothesis appears now to be the most likely cause of retinal hemorrhages in shaken baby syndrome.

Subdural hematoma

Subdural hematomas are the most common injury seen in shaken baby syndrome even though they are not pathognomonic of shaking. Noted by Tardieu [26] in 1860 in some battered children, Guthkelch explained their appearance in shaken baby syndrome as a consequence of the "rupture of one or more of the delicate bridging veins which run from the cerebral cortex to the venous sinuse" and with Caffey, they connected it to the act of shaking [4–6]. They pointed out the unmyelinated baby brain, the suppler skull with larger fontanels and pliable sutures and the weak neck muscles for worsening the outcome of shaking.

Caffey cited also Ommaya et al. [6] for having found that "inertial effect of the easily deformable brain moving with a time lag after rotating displacement of its much less deformable mature container, the skull" is an important factor in whiplash injuries.

Skeptical that shaking alone induced injuries, Duhaime et al. [7] tried to quantify the angular acceleration of the head of a 1-month-old baby model during both shaking and impact. The results were compared to primate brain injury thresholds for concussion, subdural hematomas and diffuse axonal injury; those thresholds were taken from Thibault et al. [27]. From this comparison, they claimed that values during shaking are

well below the impact-induced injury ones. As in their clinical cases, deaths were associated with increased intracranial pressure and brain swelling with signs of blunt impact to the head, and based on their experiments, the authors concluded that shaking alone could not be responsible for injuries seen in shaken baby syndrome.

Cory et al. [28] highlighted two questionable points of this study: the biofidelity of the baby model and the tolerance limits used in the study. Indeed, after replicating the baby model, the authors found that parameter changes increased the head angular acceleration which "exceeded the original Duhaime results and spanned two scaled tolerance limits for concussion." They also stated that the thresholds used for head injuries were for primates, and thus may not be reliable for human, especially for babies.

Furthermore, the thresholds have been obtained from experiments concerning lateral impacts on primates but not on shaking experiments. The difference between them is huge: An impact lasts a few milliseconds, whereas a back and forth head sequence lasts hundreds of milliseconds as it is estimated to be around 4 Hz [23, 25, 29].

As quoted above from Guthkelch, the first reason for subdural hematomas to occur seems to be bridging veins rupture. This idea was supported by an experience from Ommaya [4]:

> *An ordinary round-bottomed litre glass flask is filled with liquid paraffin in which have been suspended a few spoonfuls of desiccated coconut. When this mixture has been agitated the flakes will remain stationary in a state of even dispersion for periods of several minutes at a time. The flask is completely filled, closed with a rubber bung, and held firmly by the neck. It will be found that the flakes can be more readily set in motion, and will continue to swirl about for longer, after shaking for a few seconds than after the hardest blow that can be delivered without breaking the flask.*

In the above described experiment, the flakes model the brain and the liquid models the cerebrospinal fluid; the swirling effect of the flakes in the liquid, which appears both in the shaking and the impact (blow) cases, shows the desynchronized motions of the brain and the skull. The bridging veins, which are tensioned even in the "resting state" of the head, are put under high stress during the differential movement of the skull and the brain and when they exceed their tolerance limit, they become teared, causing subdural bleeding inside the head.

This was also corroborated by Caffey [6], who cited a correspondence with Ommaya about the movement of the brain with a time lag with the skull [6]: Indeed, due to the presence of the cerebrospinal fluid (CSF), the skull and the brain are not moving in a synchronized during the rotational movement of the head.

But in the early 2000s, Geddes made a complete work about inflicted head injury in children, and gave a conclusion, among others, that subdural hematomas in shaken baby syndrome was due to hypoxia and brain swelling rather than to bridging veins ruptures [9, 30, 31]. Logically, one should find brain swelling instead of ruptured bridging veins in babies who died from shaken baby syndrome. Matschke [32] attempted to verify this conclusion but found no correlation between brain swelling and subdural hematomas in his series. In our series, whenever whiplash-shaking has led to rapid death by cardiorespiratory arrest due to cervical spine injury, subdural hematomas from bridging veins rupture was always seen as a thin layer. If death occurs rapidly, there is no major brain swelling; if not, brain swelling seems to be the consequence of a subdural hematoma associated with hypoxia either because of a cervical trauma with prolonged apnea or resuscitation from cardiopulmonary arrest, or because of a prolonged seizure.

To highlight bridging veins ruptures, Maxeiner [32] provided X-ray scans of the brain in suspected shaken baby syndrome case: The right hemisphere blood supply was normal, the left one had a diffuse bleeding. Further, the author stated although subdural hematomas alone could be a controversial diagnose for child abuse as for accidental injury, "in cases of SDH (mostly of insignificant volume), combined with several bridging veins ruptures and an acute life-threatening condition of the patient, neither a minor fall nor other trivial injury can be accepted, rather, a trauma of an extraordinary degree has to be assumed as the cause."

Contrary to impact experiments, where the acceleration can be measured and thresholds can be drawn for brain injuries, the inertial movement of the brain during shaking needs another approach: Finite element modeling, as for retinal hemorrhages. Several finite element models were created, particularly for head impact simulations, but they all lacked the modeling of the CSF. Morison [29] in 2002 developed the first 3-D infant head model taking into account the CSF and after simulations and concluded that shaking alone could produce bridging veins stretching close to tolerance for failure and thus subdural hematomas.

Using another finite element model, Roth and coauthors [33] simulated a shaking event and the same anteroposterior shaking followed by an impact at the back of the head with angular velocities given by Prange et al. [34], and concluded that shaking could induce bridging veins rupture at the same level as a shaking-impact to the back of the head.

Spine injuries

As seen before, spinal injuries are an emerging evidence for shaken baby syndrome cases. Geddes et al. [9] have pointed out the particular frequency of cervical trauma in young

infants due to their vulnerability to hyperextension/flexion. In a supporting way, Porzionato et al. [35] presented a case of a 30-month-old child presenting no external signs of trauma, but cervical dissection showed "hemorrhagic infiltration of the clavicular head of the sternocleidomastoid muscle, carotid region, posterior musculature of the pharynx and esophagus, and retro-pharyngeal/esophageal spaces." Although the victim seemed to have had several types of abuses, authors claimed shaking as having been harmful to the spinal region. Geraut and associates [36, 37] described three cases of child abuse with associated spine injuries. Saternus et al. [38] and Ghatan et al. [39] also gave findings about spine injuries in shaken baby syndrome cases.

Geddes et al. [9, 30, 31] interestingly stated that epidural cervical hemorrhage and focal axonal damage to the brain stem and spinal nerve roots are the consequence of cervical hyperextension/flexion. Even if it was found especially in very young infants, it does not rule out a violent shaking event. Raul and Ludes [15], who also found injuries in the spine in their shaken baby syndrome series, went further by giving an explanation for the brain swelling cases from Geddes et al. [9, 30, 31] associated with a small number of subdural hematomas and spine injuries. Infants, usually younger than 6 months old, are not able to hold their heads; consequently, when shaken, their spines are more likely to be injured due to the pointed cervical weakness and this may lead to cardiopulmonary arrest or to apnea resulting in hypoxia and brain swelling. Moreover, those infants often present a thin layer of subdural hematoma. When infants grow older, their cervical musculature becomes able to protect the cervical spine from whiplash-induced injuries.

Conclusion

From the early 1970s, theories and research emerged and debates focused on the possibility of shaking alone to create the injuries seen in shaken baby syndrome. Recent works have shown that whiplash-shaking alone is enough to generate vitreo-retinal traction leading to retinal hemorrhages and to cause the rupture of bridging veins, which leads to subdural hematomas. To a certain extent, the eye-vitreous system has the same inertial behavior as the skull-brain system during shaking.

The association of retinal hemorrhages and subdural hematomas with spine injuries and/or bone fractures where the child can be held is of major importance for diagnosing shaken baby syndrome as the major mechanism of injury. The use of finite element models as well as the systematic examination of the spine have helped to understand the mechanisms of injury related to shaken baby syndrome.

As shaken baby syndrome leads to different clinical presentations (seizure, neurological impairment, apnea, coma, cardiopulmonary arrest), it is important to have a global view of the injuries sustained by the infant to correctly discuss the biomechanical aspects of this syndrome. Thus, global head and neck finite element models and simulations seem to be a promising field that can help to advance understanding of the mechanisms of injury not only within the skull but also at the cervical level. Given the differences between adult and infant skull and neck, both in terms of mechanical properties and behavior, acute tissue properties and more realistic models are needed to obtain thresholds of injury and to use these models in the wide field of abusive head trauma.

Conflicts of interest None

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