FOR DEBATE

Shaken Baby Syndrome: fundamental questions

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More than 30 years have passed since the original publication by Caffey\(^1\) proposing the hypothesis of causing injury to infants by manual shaking, and the Shaken Baby Syndrome currently enjoys worldwide clinical attention. In the United States alone, large amounts of federal funding, passing through the individual states, provide support for comprehensive child protective services, including the evaluation of reported instances of injury by shaking, the establishment of cases of purported child abuse by shaking, and the prosecution and resolution of these cases. These cases achieve widespread national and sometimes international\(^2\) publicity, and well-intentioned health-care providers, such as physicians, nurses, paramedical personnel and social workers are often held up to close public scrutiny regarding their respective positions on this subject. While the legislatures, the media, and even some portions of the health-care professions have been quick to adopt a posture regarding the presence of child abuse as a general premise, and avoidance of injury to children by shaking specifically, a careful review of the facts surrounding the original hypothesis, plus subsequent recorded experimental data, raises questions which may have some significance here.

To begin with, Caffey in 1972 published the first definitive clinical article entitled ‘On the Theory and Practice of Shaking Infants’, referenced earlier. In reading this article, the only reference directly linking subdural haematomas, regarded as the hallmark injury in shaking, with violent to and fro movement of the neck, is an article published one year earlier by Guthkelch,\(^3\) in which the latter author references as a biomechanical basis for the concept of manual shaking causing subdural hematomas in children, earlier experimental work by Ommaya.\(^4\) Indeed, in reading the original papers by Guthkelch and Caffey, Ommaya’s work emerges as the only experimental verification for the hypothesis of causing subdural haematomas in infants by manual shaking, all else being retrospective clinical observation or anecdotal report. It would seem therefore that the original work by Ommaya should be examined closely.

In 1968, Ommaya attempted to establish experimentally whether intracranial injuries could be produced by rotational displacement of the head on the neck alone, without significant direct head impact. This series of experiments was performed in the light of previously published reports of cerebral concussion and other evidence for central nervous system involvement after whiplash injury in man. In this experiment, anaesthetized rhesus monkeys were secured in a contoured fibreglass chair mounted on a rigid carriage. This carriage was mounted on wheels placed on a track, and a piston was used to deliver an impulse to the carriage, propelling the animal forward, simulating a rear end motor vehicle collision, the actual point of interest in the experiment. The entire event was photographed with a high-speed camera, enabling a calculation of rotational acceleration of the head, this acceleration being measured in radians/s\(^2\), the radian being that portion of the circumference of a circle enclosed by arc of 57.29 degrees, or the radius of a circle measured along its circumference. Ommaya was able to produce clinical and pathologically demonstrated intracranial injury in the form of concussion, subdural haematoma, and parenchymal injury to brain tissue in 19 animals, 11 of which also had pathologically demonstrated neck injuries. He also demonstrated that rotational acceleration exceeding 40,000 radians/s\(^2\) was sufficient to produce experimental concussion, without impact to the head. Ommaya postulated from these data that the levels of angular acceleration required to produce cerebral concussion and brain injury in man without impact should be in the order of 6000–7000 radians/s\(^2\) a figure he revised downward later to 4000 radians/s\(^2\).\(^5\) This is based on a formula...
work, which established that an observation which has been borne out in later order to satisfy this law of basic Newtonian physics, an observation which has been borne out in later work, which established that \( F = ma \) is a biological constant for neural tissue inversely proportional to the mass of neural tissue raised to the 2/3 power. Following this reasoning and in accordance with the above referenced experimental data, the smaller mass of the infant head, with nonimpact injury, should require greater angular acceleration and not less, than the adult head. Moreover, no information existed at the time as to whether a human being could generate the amount of angular acceleration necessary to cause such injury by shaking an infant, although it was doubtful. At some point after the publication of the 1968 paper, Caffey contacted Ommaya and was so advised, but apparently misunderstood or disregarded the information (personal communication). In 1987, Duhaime, Thibault et al., studied precisely this question, and showed that human subjects, shaking dolls with hinge necks and equivalent massed heads, generated mean angular accelerations of 1138.54 radians/s\(^2\), or approximately \( \frac{1}{4} \) the predicted concussive injury thresholds, whereas impacts against either a hard metal or padded surface generated a mean of 52, 475.70 radians/s\(^2\), well within predicted thresholds.

Taken as a whole, the above research would seem to raise some questions about whether the intracranial injuries hitherto ascribed to shaking are really the result of such and this is also noted in the last cited study, hence the genesis of the expression ‘shaken-impact’. Obviously, this has far-reaching implications.

Another point which bears scrutiny is the most recognized intracranial manifestation of the described constellation of findings ascribed to shaking, the unexplained subdural haematoma. Some amplification and clarification is appropriate here. While the acute subdural haematoma most often is seen following obvious impact injury, it must clearly be differentiated from the chronic subdural haematoma, which is some key ways may be considered a different form of injury. While it is certainly correct that the chronic subdural haematoma started as an acute form, it must logically also follow that the chronic subdural haematoma is, by very definition, the acute subdural haematoma that was missed in its genesis. This need not imply that the injury was kept hidden, rather, that the injury was not severe enough to be considered serious at the time, or even to be brought to medical attention. Most obstetricians are aware that subdurals in infants can occur after apparently normal birth, and a true incidence (and prevalence) of birth related subdural bleeding has yet to be determined. Hence, it is by no means beyond the realm of possibility that a child sustaining subdural bleeding at the time of birth would present clinically weeks or even months later with a chronic subdural haematoma. By definition, this might well be interpreted as a chronic subdural haematoma without explanation, i.e. child abuse. It is also obvious that not every infant suffering an apparently minor fall, (with impact) is evaluated or scanned. One can easily see the genesis of the ‘unexplained’ injury under such circumstances. The mechanism of membrane formation as a means of resorption of chronic subdural hematoma in infants has never been demonstrated to be different from that in adults, and pathologic specimens obtained appear identical under the microscope. Rebleeding in subdural haematomas may occur, with minimal or no trauma, owing to the nature of the membranes and the process of resorption,\(^6,9\) explaining the slowly enlarging subdural which suddenly becomes symptomatic. Common sense would seem to indicate that not all the subdural haemorrhages in children are inflicted injuries and prior to 1972 the presence of retinal haemorrhages was a diagnostic aid in detecting the presence of chronic subdural haematoma in children, and has long been known among neurosurgeons to reflect an abrupt increase in retinal venous pressure, as a manifestation of an abrupt increase in intracranial pressure.\(^10,11\) Lastly, a simple point for consideration: When an adult presents with a chronic subdural haematoma, abuse is rarely a diagnostic consideration. Given the similar pathology of the subdural haematoma in adults and children, why, logically, should the opposite be true in a child?

No one would possibly disagree that the protection of innocent children is a good and desirable end. This protection, however, must be grounded in solid, logical, reproducible scientific concepts, if for no other reason than that once child abuse is suspected, a formidable apparatus of child protective agencies and the courts is engaged. Should the science be erroneous or ill founded, the paradigm shifts and, paradoxically, another category of innocents becomes imperiled, the wrongly accused. This is not justice.

Reference

2 Commonwealth of Massachusetts v Louise Woodward (1997).


