

“Shaken baby syndrome” and forensic pathology

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In the paper by Byard [1] it is noted that shaken baby syndrome (SBS) has become a very contentious and hotly debated issue in the forensic pathology community as well as in the rest of medicine that concerns itself with young children. I certainly agree with that opinion. Byard then goes on to point out a number of specific points of contention, for example lucid interval and whether the pathological findings of subdural and retinal hemorrhage are caused by trauma or by hypoxia. There is also mention of some of the biomechanical studies which have tried to model child head injuries. The author invites commentary on this controversy from individuals who have been involved in some of this ongoing discussion.

My practice has been as a forensic pathologist who is also a neuropathologist for the past 38 years in a large metropolitan area serving a population of approximately 2.5 million, with two large tertiary care children’s hospitals. I have had the opportunity to autopsy several hundred children with head injuries, as well as to perform the neuropathological examinations on those cases done by all the other forensic pathologists who work in this system. In doing these examinations, I was also aware of all other autopsy findings, medical histories, and investigational information on the cases, and I assisted the individual forensic pathologists in coming to conclusions about the cause and manner of death in these cases. My opinions regarding the “controversy” over SBS are very much based

upon these experiences. This extensive exposure to hundreds of children who have died from head injuries from all causes has led me to the conclusion that many of these children sustained their injuries by inflicted trauma. Inflicted head injuries are common injuries and the pathological features are fairly uniformly the presence of subdural blood in small or greater amounts over the cerebral convexities, small amounts of subarachnoid hemorrhage in patches along the parasagittal convexities, brain swelling, and retinal hemorrhages in many cases which tend to be very numerous, in multiple retinal layers, and extending far into the periphery of the retina. On neuropathological examination by β -amyloid precursor protein staining, many of these young children demonstrated patterns of traumatic axonal injury. Clinically, these children were neurologically devastated rather abruptly and some were dead at the scene, while others survived in deeply comatose states for varying periods. All of these cases occurred in areas, often homes, where there were no unbiased witnesses to the events that preceded the collapse. These cases varied in that some of the children demonstrated extensive additional injuries such as bruises, fractures, and/or burns, while others had less external evidence of trauma, and a small number had no external evidence of trauma, even to the head. In all these cases, very extensive investigation of all aspects of the family’s and child’s medical history, birth records, genetic screening, and the circumstances surrounding the collapse of the child, was carried out. After consideration of all these aspects in these several hundred children, I have come to the conclusion that these children have diffuse brain injury of a traumatic nature and that the explanations offered in many of the cases were not consistent with the severity of these injuries, when that explanation was of a short fall, or no explanation was given at all.

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I have also encountered a small number of children who have sustained lethal head injuries in accidental circumstances in and around the home. These head injuries are much less common than those that I felt to be due to inflicted trauma, and they appeared to be distinctly different from the diffuse brain injuries of the inflicted head injury group. The accidental head injuries were generally the result of either focal contact injury or crushing head injury. The focal contact injuries were skull fractures with epidural hemorrhage or focal (at the site of the fracture) subdural hemorrhage and these caused death by creating increasing intracranial pressure which occurred over a period of time, several hours to over a day, with the child becoming more and more lethargic, with persistent nausea and vomiting, before eventually becoming unconscious and dying. These children tended not to have retinal hemorrhages. Their mechanism of injury involved a short fall. The crushing head injuries occurred when the stationary head was compressed by a heavy weight such as a television or heavy object falling onto the child's head. The resulting injury consisted of comminuted skull fractures, lacerations, and fracture contusions of the brain, and these children sometimes have very extensive retinal hemorrhages. Neither of these head injuries, focal contact or crushing injury, resembles the pathological features of the inflicted diffuse brain injury.

I do not find it unreasonable to diagnose an inflicted diffuse head injury which does not have evidence of an impact simply because I cannot scientifically prove shaking is harmful. I think the more important decision is whether an injury is a diffuse injury versus a contact injury or crushing head injury. The “controversy” suggests that because science cannot prove that someone can shake a baby and cause significant damage, then we must not diagnose any inflicted head trauma, even when there is very obvious impact trauma and/or additional evidence of inflicted injury. The “controversy” is used to distract from the central issue that someone has injured a child [2–4].

References

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