Forensic Sci Med Pathol (2014) 10:248–250
DOI 10.1007/s12024-014-9533-z

COMMENTARY

"Shaken baby syndrome" and forensic pathology

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Accepted: 8 January 2014 / Published online: 28 January 2014

I welcome the creation of a forum to air opposing views in this "very contentious and hotly debated area" [1]. The critical issue is why, after more than 40 years, shaken baby syndrome/abusive head trauma (SBS/AHT) remains controversial.

Contrary to Byard's suggestion, the SBS/AHT controversy is not about whether infants can be damaged or killed by violent shaking or abuse; of course they can. The real controversy is over whether shaking or abuse may reliably be inferred from specific findings, classically, subdural and retinal hemorrhage with encephalopathy (the triad).

Although SBS/AHT is a neuropathological and biomechanical hypothesis, we have learned in recent decades that it does not comport with the neuropathology of the infant brain or the biomechanics of head injury. The SBS/AHT hypothesis assumed that the triad was caused by the physical rupture of bridging veins, retinal vessels, and axons within the brain, requiring forces often described as equivalent to a multi-story fall or major motor vehicle accident, causing immediate symptoms. In the absence of impact, it was assumed that the findings were caused by violent shaking.

We now know that these assumptions are wrong: the force of shaking is unlikely to cause these findings [2], the brain damage is hypoxic-ischemic rather than traumatic [3, 4], the subdural hemorrhages are too thin to reflect ruptured bridging veins and are also seen without trauma [3–5], and that there is a wide range of alternative causes, including short falls and natural disease [6]. Failure to consider these treatable differential diagnoses places infants at further risk.

For four decades, the medical profession and the courts have largely accepted the SBS/AHT hypothesis as fact. Today, we know that the hypothesis lacks a reliable evidentiary basis and Byard highlights its fragile and speculative nature: "it was *believed* to result from violent shaking," "infants are … *thought to be* most vulnerable," and "it has been *asserted* that." A recent meta-analysis acknowledges that the research supporting the SBS/AHT hypothesis is "fraught with circularity" and limited by selection, informational, confounding, and recall bias [7]. Some of the strongest supporters of the hypothesis acknowledge that: (1) shaking is supported solely by confessions [8], (2) the triad is a "myth" [9], (3) circularity is a serious consideration [10], and (4) all diagnoses in this area consist of "informed speculation" [11]. In other areas of medicine and law, these limitations alone would preclude diagnosis, let alone criminal convictions.

In addressing these problems, pathologists must first be objective. Byard dismisses terminology implying intent, preferring the term "lethal craniocerebral trauma." This term is also inappropriate since it assumes trauma. By objective analysis of the pathological evidence we can consider causation with an open mind, untainted by assumptions inherent in a name [12]. Guthkelch, whose speculations on the pathogenesis of subdural hemorrhage were largely responsible for the original concept of SBS [13], has recently proposed the term "retinodural hemorrhage in infancy" (RDH) [14].

Second, as in other areas of medicine, the carer's account is the cornerstone of diagnosis [15]. If there is a history or evidence of impact, accidental trauma must be distinguished from inflicted trauma. Biofidelic models may not accurately predict tissue injury, but they do allow a comparison of

This comment refers to the article available at doi:10.1007/s12024-013-9514-7.

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relative forces. If there is no evidence of trauma, the appropriate default diagnosis may be natural or unknown causes. The presence of RDH in almost half of asymptomatic neonates [16, 17] suggests vulnerability to bleeding due to age-related anatomy and physiology [18, 19]. Reflux into the dura may be a protective device that prevents backflow into the infant brain; indeed, this is the most likely explanation for the extensive intradural bleeding seen in neonates without evidence of trauma [20]. It would also explain why birth-related bleeds and the hemorrhages attributed to SBS/AHT are most often located in the highly vascular dural folds of the falx and tentorium.

Third, timing must be addressed. In the classic hypothesis, babies collapse immediately after shaking due to mechanical damage to nerve fibers; however, the brain pathology is almost invariably hypoxic-ischemic injury associated with brain swelling. Byard's opinion that a lucid interval is unlikely due to the speed of brain swelling relies on animal studies which, as he indicates, may not translate to human infants. The rate of brain swelling in humans is highly variable; cytotoxic edema takes 48–72 h to reach its maximum [21], reflecting a complex cascade of events. Not only does human pathophysiology allow for the possibility of lucid intervals, but clinicians also recognize that they can occur with "SBS" [22].

Fourth, simplistic explanations must be avoided and new explanations assessed in the light of our understanding of the developing brain. Few subjects have been more misunderstood, misinterpreted and over-simplified than the "unified hypothesis" [23]. This hypothesis, which built on Geddes' earlier work, did not suggest a simple association of hypoxia and SDH but described a complex pathophysiological cascade that has been largely validated for retinal hemorrhages, which are significantly associated with life support and cerebral edema rather than trauma [24]. The lamb studies, cited by Byard, indicate that while shaking may damage the brain, it is an unlikely cause for the triad, even after repeated, vigorous shaking. Interestingly, only the least mature lambs died and then after an interval of at least 2 h. None showed the classic triad [25].

It is irresponsible to treat a hypothesis that, after over forty years, remains unproven and whose key components are inconsistent with the pathophysiology of the developing brain, as established. Instead, we can best fulfill our commitment to child protection by engaging in the objective assessment of facts, not assumptions.

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