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FORENSIC FORUM

"Shaken baby syndrome" and forensic pathology: an uneasy interface

Roger W. Byard

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"Shaken baby syndrome," as it was once known, has become a very contentious and hotly debated area in the fields of forensic pathology and medicine of infants and young children [1, 2]. Described by Caffey in the 1970s to encompass situations where intracranial injury was present in the absence of external signs of head trauma, it was believed to result from violent shaking of an infant [3]. Infants and children under 2 years of age are thought to be the most vulnerable as their heads are disproportionately heavy relative to their bodies, the neck muscles are weak, the brain is soft as many axons are unmyelinated, and the base of the skull is flat [4, 5]. It has been asserted that the nature of the shaking is such that it should be recognized by the perpetrator as likely to be harmful [5], i.e., it is not something that could happen during normal playfully handling.

Classically infants may present with a hyperacute encephalopathy (cervicomedullary syndrome) with acute respiratory failure and severe cerebral edema due to brain stem injury, an acute encephalopathy with a depressed conscious state, fits, apnea, and hypotonia, with raised intracranial pressure, bilateral subdural hemorrhages and retinal hemorrhages, a subacute non-encephalopathic state with less severe brain injury and various combinations of subdural and retinal hemorrhages, rib fractures and bruising, or with a chronic state with isolated subdural hemorrhage, rapidly expanding head circumference, and signs of raised intracranial pressure [6].

R. W. Byard (✉)
Discipline of Anatomy and Pathology, Level 3 Medical School North Building, The University of Adelaide, Frome Road, Adelaide, SA 5005, Australia
e-mail: roger.byard@sa.gov.au

In milder cases an infant or child may present with nonspecific features such as vomiting, poor feeding, lethargy, and irritability. While some children will recover, survivors may have residual neurologic sequelae such as spasticity, seizures, cortical blindness, microencephaly, chronic subdural fluid collections, cerebral atrophy, and porencephalic cysts [5].

In a lethal case argument exists as to whether the infant may appear quite normal for a period of time (i.e., have a so-called "lucid interval"), or may instead be immediately symptomatic [7]. Given the severity of the injury (i.e., the event has resulted in death) and the rapidity with which intracranial pressure has been shown to rise in animal models following blunt trauma [8] the author considers it very unlikely that an infant would be able to engage in normal activities in an apparently unaffected manner after such an insult. Death is believed to occur in approximately 15–38 % of cases and the findings at autopsy, as noted, include subdural hemorrhage that is usually bilateral, subarachnoid hemorrhage, and retinal hemorrhage. The subdural hemorrhage tends to be maximal in the inter-hemispheric fissure, usually measures less than 5–10 ml on each side, and does not cause a mass effect [4, 5].

However, the validity of the diagnostic criteria and the evidence for underlying mechanisms have been questioned [9–11]. A "unified hypothesis" was proposed in 2003 which suggested that subdural and retinal hemorrhages in infants who had been shaken were due to hypoxia with brain swelling, rather than to blunt trauma [12]. This study has, however, been challenged [13] and it has been suggested that there were "serious flaws in the methodology" [14]. A retrospective multicenter study of infants and young children with proven hypoxic brain injury in the absence of trauma was certainly not able to demonstrate intracranial hemorrhage [15]. Similarly there has been little support for

the proposal that the pathological findings in typical cases could be caused by paroxysmal coughing [16] or immunization. Although the study on the lack of association of hypoxic encephalopathy and subdural hemorrhage has been criticized [17, 18], studies supporting this association have not controlled for cerebral trauma from delivery [19].

A Court of Appeal in the United Kingdom (UK) dealing with a series of cases of shaking and inflicted head injury in infants also questioned the validity of the hypothesis with the statement in paragraph 69 of the judgment that "the unified hypothesis can no longer be regarded as a credible or alternative cause of the triad of injuries" (i.e., encephalopathy, subdural hemorrhage, and retinal hemorrhage) [20]. A conclusion from a closed, invitation-only meeting of pathologists that was convened by the Royal College of Pathologists in the UK in 2009 however proposed that "…….the presence of "the triad," even in its "characteristic form," should not be regarded as absolute proof of traumatic head injury in the absence of any other corroborative evidence" [21].

The terminology in this area also presents difficulties, with terms such as "abusive head trauma," and "non-accidental head injury" being used. As these terms imply intent, the author is personally more comfortable with using more general terms such as "lethal craniocerebral trauma," which allow for a more objective discussion of possible scenarios that may have resulted in the injuries [22].

Biofidelic and animal models have been used to determine whether the forces required to cause such significant head trauma could be generated simply by shaking, or whether blunt force impact is also be required. A recent study involving anesthetized lambs did, however, clearly demonstrate that shaking alone was capable of causing death associated with widespread axonal injury in the absence of hypoxic–ischemic injury [23]. While this study has been criticized because of the paucity of subdural hemorrhages, it has shown that shaking on its own may be lethal in young anesthetized lambs. Whether this translates to awake human infants is, of course, another issue.

Certainly it is well recognized that misdiagnosis of shaking occurs, sometimes with devastating legal and other consequences [24]. However it is important not to throw out the baby with the bath water, i.e., difficulties in diagnosis should not be cited as evidence that a syndrome does not exist. These issues merely emphasize the complexities that continue to surround the contentious area of pediatric craniocerebral trauma.

Given the disagreements that are present in the literature, the high profile nature of many of these cases (sometimes over many years), and the difficult position of forensic pathologists when cases such as these come to court, it was considered appropriate to use the *Forensic Forum* section of the journal to give a range of contributors an opportunity to voice their opinions and to present their data. Contributors have been asked to focus on specific issues or questions that they consider particularly important.

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