Shaken baby syndrome: the quest for evidence

Review

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Shaken baby syndrome (SBS), characterized by the triad of subdural haemorrhage, retinal haemorrhage, and encephalopathy, was initially based on the hypothesis that shaking causes tearing of bridging veins and bilateral subdural bleeding. It remains controversial. New evidence since SBS was first defined three decades ago needs to be reviewed. Neuropathology shows that most cases do not have traumatic axonal injury, but hypoxic–ischaemic injury and brain swelling. This may allow a lucid interval, which traumatic axonal injury will not. Further, the thin subdural haemorrhages in SBS are unlike the thick unilateral space-occupying clots of trauma. They may not originate from traumatic rupture of bridging veins but from vessels injured by hypoxia and haemodynamic disturbances, as originally proposed by Cushing in 1905. Biomechanical studies have repeatedly failed to show that shaking alone can generate the triad in the absence of significant neck injury. Impact is needed and, indeed, seems to be the cause of the majority of cases of so-called SBS. Birth-related subdural bleeds are much more frequent than previously thought and their potential to cause chronic subdural collections and mimic SBS remains to be established.

See end of paper for list of abbreviations.

The diagnosis of shaken baby syndrome (SBS) is characterized by a triad of clinical signs: subdural haemorrhage (SDH), retinal haemorrhage (RH), and encephalopathy. The syndrome remains controversial; this review examines the current evidence for and against the two main hypotheses put forward to explain it. The topic has a bearing on the practice of all paediatricians caring for infants with suspected abuse, as well as those who are asked to give expert opinion.

The 'accepted' hypothesis

The term non-accidental head injury (NAHI), which bears no implication for the mechanism of injury, is preferred to the term 'shaken baby syndrome'. In addition to the triad of signs described above, two further factors of circumstantial evidence are often adduced: (1) that the injuries are invariably inflicted, unwitnessed, by a sole carer; and (2) that the given history is incompatible with the severity of the injuries. The term 'incompatible' is highly subjective and open to circular logic. In other words, the history will be regarded as inadequate if it does not correspond to the paediatrician's notion of the history required to produce the injuries observed. Even the term 'sole carer' may be applied when other family members are present in adjacent rooms. Notably, there is no requirement for there to be any objective evidence of trauma.

The association of SDH with child abuse has been recognized since the mid-nineteenth century.[1] In 1962, Kempe et al. described 'battered child syndrome', a combination of SDH, multiple skeletal injuries, and bruises.[2] In the 1970s Caffey described the association of long bone fractures and SDH with child abuse.[3,4] While he admitted that the cerebral lesions of whiplash-shaking had not been studied systematically, and that much of his evidence was circumstantial, he promoted a nationwide education campaign which led to widespread acceptance of SBS as 'a serious and clearly definable form of child abuse'.[5]

It is, however, Guthkelch who is credited with recognizing the importance of whiplash during shaking in causing intracranial injuries.[6] Basing his hypothesis on a study by Ommaya et al.,[7] Guthkelch suggested that the rotational forces of shaking would cause tearing of bridging veins and bilateral subdural bleeding. Ommaya subjected Rhesus

monkeys to whiplash forces that induced concussion, SDH, and white matter shearing injury (diffuse axonal injury) in 18 out of 50 adult animals, 11 of whom also had neck injury. By applying Newton's second law of motion (Force = mass × acceleration) mass scaling could be applied. In other words, smaller brains required larger rotational accelerations to cause injury than larger brains, and in this way it was thought possible to predict the force required to cause intracerebral damage in infants.

Extrapolation of the findings of these early studies on adult primates to human infants requires careful consideration of the differences in structure and tissue properties of the infant head and neck, including the very specific response of the infant brain and unfused skull to head injury. When considering the implications of his experimental results on the proposed injury mechanism associated with SBS, Ommaya himself noted that: 'It is improbable that the high speed and severity of the single whiplash produced in our animal model could be achieved by a single manual shake or even a short series of manual shaking of an infant in one episode.'[7]

Nonetheless, the acceptance of shaking as a cause of severe intracranial injury has led to it being considered capable of generating force of enormous magnitude, often equated to a high-speed motor vehicle accident or a fall from a second-storey window.

Support for the 'accepted' hypothesis

Support for the 'accepted' hypothesis is based on a number of sources, particularly the literature, well documented episodes, witnessed shaking events, and confessions and convictions.[8]

'The literature' is fraught with problems,[9] including poor case ascertainment and circular logic. These problems were well expressed by Hobbs et al. who said of their study of infant SDH: 'As the study was retrospective it was inappropriate to apply specific and accepted criteria with which to define non-accidental and accidental injury, the most contentious area of diagnosis. Also there is no absolute or gold standard by which to define NAHI.'[10]

Leestma reviewed the literature from 1969 to 2001 and found 324 cases with detailed individual case information.[11] Of 54 cases in which shaking was admitted, only 11 were without evidence of impact and could, therefore, be considered 'pure' shakes. Independently witnessed shakings are even more uncommon; only three cases appear to be recorded in the world literature.[11,12]

Confessions and convictions are regarded as important in supporting SBS, but confessions are unreliable unless the circumstances in which they are made are known. Confessions may be made by one parent to prevent the remaining children from being taken from the other parent and put into care. Such confessions can result from intense police interrogation of distraught or frightened parents, or they may be part of plea-bargaining. Convictions were demonstrated to be unreliable by the UK Court of Appeal[13] in 2005 where two of the four cases before it had their convictions overturned and one was reduced from murder to manslaughter. One appeal was dismissed.

Evidence which undermines the 'accepted' hypothesis

BIOMECHANICS

In 1987, Duhaime et al. used dummies (similar to the crash test dummies used in road-safety research) modelled on a typical 6-month-old human infant to test the accepted hypothesis. Using a variety of neck models Duhaime predicted injury thresholds using Ommaya's data.[14] College students shaking the dummies generated mean acceleration of 9.2G, considered well below the threshold necessary for intracranial damage, while impacting the dummies on to a metal bar generated 428G. Duhaime wrote: 'It is our conclusion that the shaken baby syndrome, at least in its most severe acute form, is not usually caused by shaking alone. Although shaking may, in fact, be a part of the process, it is more likely that such infants suffer blunt impact. The most common scenario may be a child who is shaken then thrown into or against a crib or other surface, striking the back of the head and thus undergoing a large, brief deceleration. This child then has both types of injury-impact with its resulting focal damage, and severe acceleration/deceleration effects associated with impact causing shearing forces on the vessels and parenchyma.' In 1979, the term 'shaken-impact syndrome' was introduced, despite the finding that impact itself is a sufficient cause of brain damage without any need for prior shaking.[15]

Duhaime's experiments were reproduced by Cory and Jones.[16] These authors created forces which exceeded the injury threshold for concussion, but noted that there were chin and occipital contacts at the extremes of the shaking motion in their dummies. Their shaker volunteers fatigued after 10 seconds and the authors expressed their concerns regarding the difficulties in extrapolating to human infants the findings in both dummy and animal models. They concluded: 'It cannot be categorically stated, from a biomechanical perspective, that pure shaking cannot cause fatal head injuries in an infant.' In the same year (2003), Prange et al. published the results of studies using a dummy modelled on a 6-week-old infant.[17] In these studies, shaking and inflicted impact onto foam were insufficient to cause accelerations considered necessary for SDH or axonal injury, while impact onto a firm surface exceeded the acceleration of a 1.5m fall onto concrete or carpet. Falls onto foam caused much less rotational force to the head as the foam cradled head and neck together. This is an important observation as it is frequently not recognized that falls and impact to the head produce significant rotational forces due to impact forces which are not aligned through the centre of gravity of the head, and the effect of the hinging of the head on the neck. These authors also commented on the problems of modelling the infant skull and neck and extrapolating results from dummy and cadaver studies to human infants.

Neck injuries may be under-reported in infants who die after severe abuse.[18] In Ommaya's study, 11 of 19 primates had neck injuries; these were adult animals with mature neck structure and musculature. It is likely that the forces required to cause intracranial injury would also damage the weak infant neck.[19] This makes logical sense to all those of us who drive cars fitted with head restraints: they are designed to prevent whiplash, not SDH or RH. In road traffic accidents infants who suffer single severe hyperextension forces have cervical fractures, dislocations, spinal cord injury, and torn nerve roots, not SDH.[20,21]

BRAIN PATHOLOGY

Encephalopathy associated with NAHI was thought to depend on white matter tearing or diffuse axonal injury. However, in 2001 Geddes et al. published a detailed microscopical study of a large series of infants thought to have suffered inflicted brain

damage.[22,23] The study overturned previous ideas by showing that most of these infants were not suffering from diffuse axonal injury, as seen in adult trauma, instead most had hypoxic–ischaemic injury. Only a minority had axonal injury and this was restricted to specific areas of the brainstem. Her conclusion was supported by subsequent neuroradiological observations.[24]

This has important clinical implications. Whereas axonal tearing results in immediate loss of function and a lucid interval is unlikely, brain swelling takes place with a speed and severity of huge individual variation, which may allow a lucid interval or a period in which cerebral function is gradually impaired.[25,26] This is particularly likely to occur in infants whose skulls still have unfused sutures, and are, therefore, distensible.[27]

The alternative hypothesis

Geddes had observed that infants with NAHI commonly have a thin bilateral film of subdural blood, sometimes so small that it is missed on scans.[22,23] Neuroradiological observations have confirmed that these bleeds may be as small as 2 to 3ml and easily missed at autopsy.[28] Geddes proposed that these very thin subdural haemorrhages may not be the result of traumatic rupture of bridging veins, which causes thick unilateral space-occupying clots, but may occur when intracranial vessels are damaged by hypoxia in the presence of abnormal haemodynamic forces such as venous hypertension, systemic arterial hypertension, or episodic surges in blood pressure.[29] The paper sparked considerable debate,[30,31] perhaps somewhat surprisingly as over 100 years ago virtually the same hypothesis was proposed by Cushing: 'The intracranial hemorrhage is usually of venous origin and follows the rupture of some of the delicate and poorly supported venous radicles of the cerebral cortex. Such an injury may be the direct result of undue traumatism during labor or may occur when too great strain has been put upon the vessels by the profound venous stasis of postpartum asphyxiation; just as in later months they may rupture under the passive congestion brought about by a paroxysm of whooping cough or a severe convulsion.'[32]

Support for the Geddes hypothesis

Can infant SDH arise from sources other than traumatic rupture of bridging veins?

Perhaps the best information regarding infant SDH has been derived from the study of neonates. The preceding clinical circumstances are known and the older pathological literature contains detailed descriptions of large numbers of autopsy cases. Bridging vein rupture is uncommon: Craig described 120 neonatal autopsies with 62 showing SDH but only three with torn bridging veins, all with overriding sutures.[33] Larroche described 700 autopsies, 18% with SDH.[34] She noted a common association with hypoxic–ischaemic injury. She also described venous congestion, particularly with asphyxia and anaemia, and after exchange transfusion causing raised atrial pressure. She did not identify torn veins but referred to the work of Cushing, who both operated on his cases and subsequently did the autopsies. Even he had difficulties: 'In two of the cases I have examined I have satisfied myself that such ruptures were present. A positive statement, however, cannot be given even for these cases, since the dissection and exposure, difficult enough under any circumstances owing to the delicacy of the vessels, is the more so when they are obscured by extravasated blood.'[32] The difficulty of autopsy verification of traumatic rupture of bridging veins has been addressed more recently and contrast injection of the veins attempted.[35]

Are there alternative, non-traumatic, sources of subdural bleeding? The dura, falx, and healing chronic subdural membranes are all potential sources of widespread, thin-film subdural bleeding. Volpe considered that SDH is by no means always traumatic and arises from tributaries of the venous sinuses within the layers of the dura.[36] Intradural bleeding is frequently seen in asphyxia,[37] and brain scans of infants with severe hypoxic–ischaemic injury frequently show high signal consistent with congestion or haemorrhage in the falx where extensive venous sinuses are to be found.[38,39]

The natural healing process of SDH is by formation of a membrane containing numerous fragile, thin-walled capillaries which rebleed spontaneously.[40] Small subdural bleeds are frequent after birth, described in up to 26% of vaginal deliveries,[41,42] this may confer vulnerability to rebleeding on these infants which may be exacerbated by hypoxia and hypertension, for example, after coughing and choking[43]

Evidence that undermines the Geddes hypothesis

The most commonly cited argument against the Geddes hypothesis is that SDH is not seen in cases of hypoxia, asphyxia, drowning etc., but these cases are rare and very few will be scanned in any one department. Further, as noted above, these bleeds may be small and readily missed on a scan or at autopsy. There has been no detailed prospective study to support this argument. A single retrospective case study gives insufficient clinical detail to assess the cases of the appropriate age group.[44]

The 2003 Geddes hypothesis paper[29] was criticized because only one of the 50 infants who presented with dural bleeding

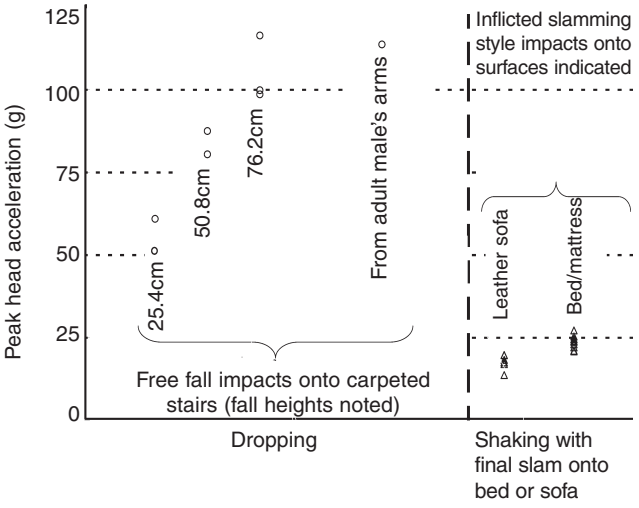

Figure 1: *A comparison of maximum head accelerations for head impacts resulting from free falls of various heights onto a carpeted surface with inflicted impacts onto relatively soft household surfaces. Free falls from only 24.5cm result in maximum head accelerations that are approximately twice as great as inflicted shaking style head impacts onto either a sofa cushion or a mattress. (C Van Ee, personal communication 2007).*

had macroscopic SDH.[30] My own experience is that if the dura is adequately sampled, dural and subdural membrane bleeding is frequent in association with macroscopic SDH. Again, careful prospective studies are needed.

Objective assessment of infant head injury

Biomechanics has an increasingly important role in our understanding of the forces involved in infant head injury. It has grown from studies of transport safety and essentially examines the effects of physical forces upon biological systems. With suitable scientific rigour it can determine applied forces in an objective and reproducible way, but the determination of resulting injury is far more complex. Motor vehicle testing laboratories have established injury criteria that are used in the development of car safety devices by detailed reconstruction of accidents.[45]

It has been shown that head impacts onto carpeted floors and steps from heights in the 1 to 3 feet range result in far greater head impact forces and accelerations than shaking and slamming onto either a sofa or a bed (C Van Ee, personal communication 2007; Fig. 1), reproducing the findings from Duhaime and Prange noted above.[18,19]

Biomechanical studies have shown that both falls and impacts generate rotational forces due to off-axis impact forces and the hinging of the head on the neck. The assumption that rotational forces are indicative only of shaking is clearly unfounded and biomechancially incorrect.

The clinical history

Clinical history is the cornerstone of good diagnostic practice.

I am very struck by the numbers of cases of infants with SDH, RH, and encephalopathy with a history of vomiting and choking, given consistently and in detail. Thousands of infants regurgitate and choke every day but they do not develop the triad. Is there any justification for thinking that just sometimes, in a few cases, they might? Inhalation of feed or vomit may play a part in sudden infant death[46] and awake apnoea associated with gastro-esophageal reflux is well recognized.[47] The physiological response to aspiration may be dramatic; foreign material on the larynx causes laryngospasm which is associated with startle, cessation of respiration, hypoxaemia, bradycardia, and a doubling of blood flow to the brain.[48] These circumstances with, or even without, vigorous resuscitation, may cause a pre-existing healing subdural membrane to bleed or cause the dura itself to become haemorrhagic and ooze blood into the subdural space. Awake apnoea may be precipitated by nappy changing or a change in posture; perhaps rough handling or shaking can do the same, the mechanism of brain injury being asphyxiation rather than traumatic venous tearing?

Expert evidence

Giving evidence is in itself daunting and many clinicians are reluctant to become involved. But clear, thoughtful, and balanced input is essential if we are to assist in the promotion of justice. Our responsibility is to examine all the details in every case of unexplained infant collapse and to adopt a rigorous and questioning approach. If we are intellectually honest we will sometimes be forced to admit that we simply do not know the cause. Children must be protected from harm, and parents and families must be protected from hasty and wrongful accusation, which itself can wreak dreadful and lasting damage.

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List of abbreviations

NAHI	Non-accidental head injury
RH	Retinal haemorrhage
SBS	Shaken baby syndrome
SDH	Subdural haemorrhage