

The triad of retinal haemorrhage, subdural haemorrhage and encephalopathy in an infant unassociated with evidence of physical injury is not the result of shaking, but is most likely to have been caused by a natural disease

YES

In January of this year, the British Crown Prosecution Service dealt another blow to the 'shaken baby' hypothesis in their latest guidance¹ when they abandoned the term 'shaken baby syndrome' in favour of 'non-accidental head injury'. Although shaking remains the mechanistic lynchpin of their theory, the name change belatedly acknowledges that the shaking hypothesis has been seriously undermined by research of the past two decades. It is 23 years since Duhaime wrote: "It is our opinion based on the clinical data and the studies outlined, that the 'shaken baby syndrome' is a misnomer, implying a mechanism of injury which does not account mechanically for the radiographic or pathological findings".²

Background

At the heart of this problem is the diagnostic dilemma of young infants, usually less than six months of age, who present with the triad of retinal haemorrhage (RH), thin-film subdural haemorrhage (SDH) and encephalopathy.

Forty years ago, Guthkelch and others seized upon recently published biomechanical studies in adults to seek a traumatic explanation for this

triad.³ Since fractures, abrasions, bruises and other objective evidence of trauma were often lacking, they hypothesised that these infants must have been shaken and that the characteristic bilateral thin-film subdural bleeds were the result of bridging vein rupture from rotational forces induced by shaking.

Problems with the hypothesis

The first problem with the shaking hypothesis is empirical: in nearly 40 years, no one has ever witnessed shaking to cause the collapse of a well baby. The only three witnessed cases in the world literature were babies who had already collapsed.^{4,5}

The second problem is biomechanical. Once Duhaime demonstrated that even minor impacts generated forces considered sufficient to cause the triad while shaking did not, the term 'shaken impact syndrome' was born. However, there is no evidence that shaking must precede or accompany impact to cause brain injury; impact of itself is enough. Since then, multiple biomechanical studies have validated Duhaime's conclusion and endorsed the commonsense view that violent

Waney Squier

Consultant Paediatric Neuropathologist, Department of Neuropathology, Level 1, West Wing, John Radcliffe Hospital, Oxford, OX3 9DU, UK
Waney.Squier@clneuro.ox.ac.uk

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While evidence can help inform best practice, it needs to be placed in context. There may be no evidence available or applicable for a specific patient with his or her own set of conditions, capabilities, beliefs, expectations and social circumstances. There are areas of uncertainty, ethics and aspects of care for which there is no one right answer. General practice is an art as well as a science. Quality of care also lies with the nature of the clinical relationship, with communication and with truly informed decision-making. The **BACK TO BACK** section stimulates debate, with two professionals presenting their opposing views regarding a clinical, ethical or political issue.



Waney Squier



Lucy B Rorke-Adams

BACK TO BACK this issue:

shaking would cause neck injury, which is rarely identified.^{2,3} Biomechanical models are criticised as not fairly representing the biological structure of the human infant, but the same models, based on animal, mathematical and tissue experiments and injury reconstruction are used to design car seats, head restraints, airbags etc. We all depend on them in our daily lives. They are also consistent with experience. In one real-life example, a baby who suffered serious neck injury in a 70 mph crash had no SDH or RH,⁶ confirming the vulnerability of the infant neck⁷ and raising the question: if 70 mph whiplash does not produce the triad, how can the 'single firm shake' do so, as so frequently cited in Court?

A final problem is anatomical. While models and hypotheses may be criticised, there is no arguing with the anatomy. Anatomically, the hypothesis that shaking can cause thin-film subdural bleeding by bridging vein rupture is untenable. As these vessels are few in number and carry large volumes of blood, rupture would lead to large localised bleeds and would occur in the sub-arachnoid space.² The thin diffuse bleeds in triad babies are more likely to originate in the dura, reflecting the extensive vascularity characteristic of the infant dura.^{10,11} If sufficient, this intradural bleeding leaks onto the dural surface, creating a 'subdural' bleed. Since subdural and retinal bleeds are seen in about half of asymptomatic neonates, and bleeding into the dura is almost universal at neonatal autopsy whatever the cause of death, the hypothesis that these bleeds are caused by shaking and are immediately symptomatic cannot be supported.

The sole remaining basis for the shaking hypothesis rests on confessions,⁸ which must be viewed with caution given the number of confessions which have been shown to be unreliable following DNA exonerations. The confession data on shaking has not been subject to critical review, but a recent study⁹ found little correlation between confessed accounts of shaking and objective brain scan observations.

Alternative explanations

Triad infants appear to be manifesting a response to disruption of intracranial homeostasis predi-

cated on the immaturity of the infant intracranial structures. Even the staunchest supporters of shaking agree that there is a multitude of causes of the triad, including trauma, birth defects, metabolic or genetic conditions, cardiorespiratory arrest, seizures, ruptured aneurysms, infection, stroke and sinovenous thrombosis.

Triad babies, whose deaths are presumed to be nonaccidental, have many features in common with cot death babies, whose deaths are presumed to be natural. There are distinguishing features; cot death babies are found dead and have no pathological findings, but we still don't know why they die. But the most obvious, and the most frequently overlooked, distinctive feature of many triad babies is an extended period of hypoxia prior to resuscitation and ventilation, frequently with a 'downtime' of over 30 minutes. This period of hypoxia damages vascular endothelium; subsequent reperfusion and the pressure surges of resuscitation and ventilation can be expected to produce the triad. The association of these factors with RH has already been demonstrated.¹²

In every case one must ask: what caused this baby to collapse? If there are fractures, bruises and abrasions, we may assume the triad was due to impact injury, inflicted or accidental. If there is neck injury, whiplash (and shaking) may be implied. We can all agree that it is never safe to shake a baby, since severe shaking could damage the vital centres of the brain stem and spinal cord, with disastrous consequences. But without objective evidence of trauma, the triad remains nonspecific. In a case of my personal experience, a mother found her baby comatose in hospital during an admission for suspected infection. A brain scan showed SDH and a swollen brain, with RH found at autopsy along with a small ruptured vein of Galen varix, hidden in the dural folds. How different this story would have been if the mother had discovered the collapsed baby at home! Once the triad was identified, the mother, as the sole carer, would almost automatically have been accused of shaking her baby. This is a salutary lesson; the triad may occur on an open hospital ward, just as natural diseases may present at home.

Failure to recognise abuse risks leaving a perpetrator at large and other children unpro-

tected. Failure to look beyond the simplistic and increasingly untenable shaking hypothesis risks incalculable damage by wrongfully removing children from loving parents or incarcerating innocent people. Further, by focusing on shaking or inflicted trauma to the exclusion of accidental and natural causes, we are almost certainly missing opportunities to save babies through prevention, early diagnosis and treatment.

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NO

It has been the practice of physicians to organise historical, physical and laboratory findings which occur with some frequency into syndromes or specific disease entities, and contributions by pathologists often provide a morphological base for the disorder. Thus, in the century and a half interval since Rudolf Virchow's studies earned him the sobriquet of 'Father of Pathology', innumerable diseases have been recognised, although unfamiliar constellations continue to challenge the diagnostic acumen of physicians, requiring ongoing clinical and pathological investigations to establish their place in the spectrum of disease.

Among this group are those that appear to be associated with child abuse. Although there is ample historical documentation of child abuse throughout the ages, a scientific approach to

define the nature and extent of such abuse is a relatively recent phenomenon.¹ Whereas abuse may take many forms, the majority do not cause death, e.g. psychological or sexual abuse, but infliction of injury to the central nervous system (CNS) is among the most lethal; about two-thirds of child abuse victims who die do so because of CNS trauma.²

Clinical and pathological studies have documented three features associated with CNS trauma that occur so frequently they are commonly referred to as 'the triad', specifically, subdural haemorrhage (SDH), retinal haemorrhage (RH), and encephalopathy.

This triad is found in infants who may/may not exhibit other injuries, such as bruising and/

Lucy B Rorke-Adams
MD, Senior
Neuropathologist, The
Children's Hospital of
Philadelphia; Consultant
Forensic Neuropathologist,
Office of the Medical
Examiner, City of
Philadelphia and Clinical
Professor of Pathology,
Neurology and Pediatrics,
University of Pennsylvania
School of Medicine, USA
rorke@email.chop.edu