

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

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. 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/

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or fractures. Pathogenesis of the triad has been ascribed to severe acceleration–deceleration forces consequent to shaking, plus or minus impact.

An enormous body of evidence based upon peer-reviewed studies has established the high frequency of association between the triad and shaken impact syndrome, with the caveat that this triad may not be pathognomonic for inflicted trauma.³ Specifically, one or more components may signal a naturally occurring disease, including among others, a variety of haematological/coagulopathic disorders, rare metabolic diseases, vascular malformations, etc.

Routine diagnostic evaluation of infants who present with one or more features of the triad therefore includes a search for one of the known diagnostic possibilities in the context of history and ancillary investigations.^{4,5}

Those who challenge the triad as a sentinel of possible nonaccidental trauma have advanced alternative disease states to explain its occurrence. Their list includes hypoxia-ischemia, birth injury, excessive coughing/vomiting, infections, vaccinations and venous thromboses.⁴ It is of note that these alternative suggestions purporting to account for the features of the triad have been extant for a relatively short time, first appearing in 2003.⁶

This was a publication by Geddes et al, who theorised that pathogenesis of SDH and retinal haemorrhage was hypoxia-ischemia and not trauma. The study upon which this extraordinary claim was based was severely flawed, including, for example, no clinical or pathological examination of the eyes; two years later it was retracted by Geddes, but by that time, the evil genie had escaped Pandora's box, repercussions of which have been far-ranging. A considerable literature has since accumulated with contributions both from Geddes's supporters (even after her retraction) and a host of challengers.⁷ Of primary importance is the fact that, to date, no reliable evidence base supporting a pathogenetic relationship between hypoxia-ischemia and subdural bleeding or retinal haemorrhages has been forthcoming.

Also lacking is evidence-based literature supporting the assertion that late consequences of 'birth

injury' may be mistaken for nonaccidental head trauma. Experienced paediatric pathologists have documented falcine and small SDH in perinates dying of problems unrelated to the CNS, e.g. congenital anomalies, infections etc., and recent radiological studies have confirmed these observations.⁸ The majority of the haemorrhages have resolved by one month of age, and if the infant comes to postmortem after a month or more, a delicate avascular membrane is sometimes found. The assertion that it is highly vascularised and may bleed spontaneously or consequent to minor trauma has no documented factual base.

It is also well established that retinal haemorrhages occur peripartum and these, too, disappear by four weeks of age.⁹

The claim that venous thromboses cause the triad is blatantly false. Although intracerebral haemorrhages are common, no standard texts of radiology or pathology document association of thromboses with SDH, although it is conceivable that small posterior pole retinal haemorrhages may result from increased intracranial pressure.⁹

Although subdural effusions and retinal haemorrhages are sometimes found in infants with bacterial meningitis, SDHs are exceptionally rare, even if the agent is haemolytic 'strep'. The retinal haemorrhages are basically caused by increased intracranial pressure and distinguishable by an experienced ophthalmologist from those consequent to trauma.⁹

Assertions that vaccinations or excessive coughing/vomiting cause subdural and retinal haemorrhages are clearly ludicrous. There is, in fact, strong evidence to the contrary concerning coughing/vomiting.¹⁰⁻¹² Surridge et al.¹⁰ studied 72 patients who required intensive care because of pertussis, 97% of whom were less than 12 months of age, and reported CNS complications to include seizures and encephalopathy; three patients died. They found neither SDH nor RH clinically or pathologically.

A companion study by Cherry¹¹ of children with severe croup with/without pneumonia (including some with diphtheria) made no mention of SDH/RH as a complication in severely affected

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patients. Similarly, Fitzpatrick et al., who studied a group of children with cyclical vomiting syndrome, found none with complicating SDH/RH.¹²

The scientific base for shaken impact syndrome has accumulated over a period of at least 150 years, although sporadic writings of physicians, anatomists and writers commenting about effects of CNS trauma, in particular concussion, appeared long before that time.

The concept that SDH was a consequence of shaking was advanced in 1930, and innumerable observations of traumatised infants by Caffey, Kempe, Gutkelch and countless others, laid the foundation for the objective base of shaken impact syndrome upon which contemporary investigators continue to build.

Contributions by paediatricians, neuroradiologists, neurosurgeons, clinical and forensic pathologists, physiologists, ophthalmologists, biomechanical engineers, social workers, and law enforcement agents have formed the evidence base that currently supports the diagnosis of shaken impact syndrome.

Although components of the syndrome include the triad, the diagnosis is actually based upon a complex constellation of clinical-pathological-investigative findings. These include:

1. investigative data
2. clinical history, examination and therapeutic requirements
3. laboratory studies to rule out natural disease, and
4. radiological, ophthalmological and pathological findings, all of which are evaluated against a knowledge base of clinical disease and features of accidental versus nonaccidental trauma.

The triad is an important component within this complex constellation, but does not stand alone.

Specialists involved in the tragic field of child abuse remain ever mindful of the wisdom of John Dewey who said: "Intelligence is not something possessed once and for all. It is in constant process of forming, and its retention requires constant alertness in observing consequences, an open-minded will to learn and courage in re-adjustment." Those who offer untested hypoth-

eses to defend individuals who have harmed infants do considerable disservice to science and to the victims.

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