ORIGINAL ARTICLE

# Apnoea and brain swelling in non-accidental head injury

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**Aims:** (1) To identify whether infants and young children admitted to hospital with subdural haematomas (SDH) secondary to non-accidental head injury (NAHI), suffer from apnoea leading to radiological evidence of hypoxic ischaemic brain damage, and whether this is related to a poor prognosis; and (2) to determine what degree of trauma is associated with NAHI.

**Methods:** Retrospective case series (1992–98) with case control analysis of 65 children under 2 years old, with an SDH secondary to NAHI. Outcome measures were presenting symptoms, associated injuries and apnoea at presentation, brain swelling or hypoxic ischaemic changes on neuroimaging, and clinical outcome (KOSCHI).

**Results:** Twenty two children had a history of apnoea at presentation to hospital. Apnoea was significantly associated with hypoxic ischaemic brain damage. Severe symptoms at presentation, apnoea, and diffuse brain swelling/hypoxic ischaemic damage were significantly associated with a poor prognosis. Eighty five per cent of cases had associated injuries consistent with a diagnosis of non-accidental injury.

**Conclusions:** Coma at presentation, apnoea, and diffuse brain swelling or hypoxic ischaemia all predict a poor outcome in an infant who has suffered from SDH after NAHI. There is evidence of associated violence in the majority of infants with NAHI. At this point in time we do not know the minimum forces necessary to cause NAHI. It is clear however that it is never acceptable to shake a baby.

Most subdural haemorrhages (SDH) in infancy result from non-accidental head injury (NAHI) when the infant has been shaken.[1] Many of the victims sustain associated extracranial injuries; significant injury to the brain and the morbidity and mortality are high.[1] There is an ongoing debate about the mechanisms and forces involved, what particular neuropathology is caused, whether diffuse axonal injury (DAI) or hypoxic-ischaemic injury is of greatest importance, and how these changes influence outcome.

SDH arises from rotational acceleration-deceleration forces that cause tearing of the bridging subdural veins.[2 3] Despite Duhaime's argument that a shaking injury with blunt impact is required to elicit forces great enough to cause an SDH together with the associated brain injuries,[4 5] it is now agreed that shaking alone can produce severe head injury in infants.[3 6–8] Expert witnesses in court are frequently asked to give opinions about how hard these babies are shaken. Evidence to answer this question is inconclusive and is derived from animal,[9] cadaver, biomechanical models,[5] and accidental head injury studies.[3 10] A recent publication suggests that "it may not be necessary to shake an infant very violently to produce shaking injury to the neuraxis" and that "it is possible that severe acceleration deceleration injury does not occur in shaken baby syndrome".[11] These statements have had a significant impact on the criminal justice system, as a recent decision of the Court of Appeal indicates.[12] A childminder had been convicted of the murder of a baby before the publication of the research by Geddes and colleagues.[11] During her appeal against the conviction, the Court was referred to the research and concluded that the less serious charge of manslaughter was the only safe verdict. The Court commented, "If the jury had had the additional benefit of hearing the fresh medical evidence we have heard, they might well have come to the same conclusion".

Geddes and colleagues[11 13] identify a high incidence of widespread microscopic neuronal hypoxic brain damage in their cohort of children with NAHI, with a low incidence of diffuse axonal injury. In a proportion of infants, they showed significant microscopic changes of focal axonal damage to the craniocervical junction or the cervical cord, a finding supported by Shannon and colleagues.[14 15] In infants who presented with a history of apnoea and hypoxic brain damage, they propose a mechanism of cervical hyperextension/flexion during shaking, leading to damage to the brain stem respiratory centres, with a consequent outcome of death from severe hypoxic brain damage. There is some support for this theory in earlier literature when Hadley *et al* described damage to the craniocervical junction in babies where there was no evidence of impact.[8] Johnson *et al* concluded that trauma induced apnoea causes cerebral hypoxia, which has a more detrimental influence on outcome than the primary mechanism of injury or the structural intracranial damage incurred.[16]

In our retrospective study of children under the age of 2 years who sustained an SDH from NAHI, we have addressed the following key questions: Does apnoea and generalised hypoxic brain injury predict a poor prognosis in infants who have sustained SDH from NAHI? What degree of trauma is associated with NAHI when an infant presents with SDH?

## METHODS

We have a database of 90 children under the age of 2 years who were admitted to hospital in South Wales and South West England between 1992 and 1998 where a subdural haemorrhage or effusion was diagnosed by computed tomography (CT), magnetic resonance imaging (MRI), or at postmortem examination. Cases were notified from inpatient data, paediatric neurology and neurosurgery units, and the Welsh Paediatric Surveillance System (run along the same lines as the British Paediatric Surveillance System[17]). From these data we have used the inclusion criteria of Geddes *et al* to identify 65 children with SDH from NAHI[11 13]:

**Abbreviations:** CT, computed tomography; DAI, diffuse axonal injury; KOSCHI, King's Outcome Scale for Head Injury; MRI, magnetic resonance imaging; NAHI, non-accidental head injury; SDH, subdural haematoma

**Table 1** Relation between KOSCHI outcome score for 59 cases and signs and symptoms at presentation (Bender score): apnoea: hypoxic ischaemia and no apnoea/intracerebral radiological change

| | KOSCHI 1 Dead n=16 | KOSCHI 2/3 Severe disability n=12 | KOSCHI 4 Moderate disability n=6 | KOSCHI 5 Good outcome n=25 | Total n=59 |
|---|---|---|---|---|---|
| Bender score: symptoms at presentation to hospital | | | | | |
| 1 mild symptoms | 1 | 2 | 0 | 4 | 7 |
| 2 drowsy with neuro deficit | 0 | 7 | 4 | 17 | 28 |
| 3 stuporous | 2 | 2 | 1 | 2 | 7 |
| 4 comatose | 10 | 1 | 1 | 2 | 14 |
| dead | 3 | | | | 3 |
| Apnoea at presentation | | | | | |
| Apnoea | 10 | 5 | 1 | 5 | 21 |
| No apnoea | 6 | 7 | 5 | 20 | 38 |
| Diffuse brain swelling/hypoxic ischaemic damage on radiology (for 57 cases who had neuroimaging and outcome known) | | | | | |
| No radiology | 2 | | | | |
| Brain swelling/hypoxic ischaemic damage | 11 | 3 | 1 | 3 | 18 |
| No radiological intracerebral damage evident or clinical apnoea | 1 | 4 | 2 | 14 | 21 |

(1) Head injury where there was a confession by the perpetrator (n = 19).

(2) Cases where NAHI was established as a result of criminal conviction in the criminal court where there were unexplained extracranial injuries (n = 10).

(3) Cases where there were unexplained injuries elsewhere in the body, other than head injury, but no conviction (all diagnosed at case conference except two who died) (n = 15).

(4) Cases where the carer was convicted of injuring the child but in which there was no extra cranial injury (n = 0).

(5) Cases where there was major discrepancy between the explanation given by the carer and significant injury, such as a skull fracture, or if the history was developmentally incompatible (NAHI diagnosed at case conference in all except one who died) (n = 21).

Of those excluded, 16 had witnessed accidental or established medical causes and became a small comparison group. Nine did not meet the inclusion criteria.

The clinical research officer extracted clinical details from hospital notes, observation charts, and community case notes. We used the King's Outcome Scale for Head Injury (KOSCHI) to categorise outcome[18] (1 dead, 2 vegetative, 3 severe disability, 4 moderate disability, 5 good recovery). Child protection records, police files, and legal case records were also examined.

We analysed the first cranial CT/MRI scans and follow up neuroimaging undertaken within the first two weeks of diagnosis for evidence of intracerebral damage. The Bender grading scheme was used to classify symptoms of severity on presentation[19]: 1 mildly symptomatic, 2 drowsy variable neurological deficit, 3 stuporous, 4 comatose (GCS 3–5).

We identified 31 cases from the Geddes *et al* studies[11 13] of infants who died from NAHI and had SDH, and 28 infants and young children with shaken baby syndrome from the study of Johnson and colleagues,[16] and compared them to our case series.

In our analysis we used standard $\chi^2$ with appropriate degrees of freedom, and Fisher's exact probability test for 2×2 contingency tables when sample sizes are small. The study was approved for epidemiological analysis by MREC S Wales.

## RESULTS

Of the 65 cases, the age at head injury ranged from 19 days to 23 months (mean 5.2 months). There were 57 infants, 48 of whom were 6 months or under. Three children were dead on arrival at hospital, 15 were comatose, eight were stuporous with fluctuating levels of consciousness, 32 were moderately drowsy with fits or neurological deficit, and seven had mild symptoms or general malaise.

Table 1 details the Bender score for presenting symptoms with respect to the KOSCHI outcome score for the 59 children where the outcome is known. There is a significant correlation between the severity of symptoms at presentation to hospital and poor outcome ($\chi^2 = 29.07$ on 9 df, $p < 0.001$) (calculations exclude the three children who were dead on arrival).

Macroscopic findings of extensive bleeding around and damage to the cervical spinal cord or brain stem were evident on four of the 14 postmortem examinations. No micropathology findings were available.

Twenty two (34%) children had documented apnoea (table 1), nine had a history of apnoea before admission (one was dead on arrival and three had impaired levels of consciousness), and 13 were recorded as apnoeic at admission (five had documented respiratory arrest). Fourteen of these children required ventilation. Apnoea at presentation was strongly associated with death or severe disability ($\chi^2 = 13.3$ on 3 df, $p < 0.005$).

Overall 85% of children had further injuries. These were characteristically multiple. Thirty one children had more than 83 fractures between them (10 had a single fracture, 21 had fractures at multiple sites); 16 children had rib, nine skull, eight long bone, and eight metaphyseal fractures. Eight infants were reported as having recent fractures (four had concurrent old fractures). In 14 the fractures were old (in four reports there was no comment about the timing of the fractures; five cases only had skull fractures that cannot be aged). Forty children were recorded to have bruises; in 28 these were multiple (average four bruises per child, one child had 21 recorded). Two children had adult bites and three burns.

Forming 2×2 contingency tables, the data of table 2 showed that there was no significant difference between the degree of associated injury in children with apnoea and those without. Although the sample size is small, the data indicate a significantly higher number of children with skull fractures in children who presented with apnoea.

SDH was diagnosed on an initial CT scan in 61 cases, on MRI in two, and at postmortem examination only in two cases; 28 children had follow up MRI scans (18 in week 1, seven in week 2, three long term follow up). In 53 cases the investigations were undertaken or reported by a neuroradiologist with a specific interest in paediatric neuroimaging in the tertiary centre. Of the 28 children who were ventilated all scans were undertaken on the day of admission. Table 2 details the intracerebral damage evident on neuroimaging. Of 21 apnoeic children, 10 had cerebral swelling or hypoxic

**Table 2** Associated injuries and intracerebral neur radiology findings in the group of children with apnoea and those without apnoea

| Associated injuries | Apnoea n=22 | No apnoea n=43 | All n=65 | p value |
|---|---|---|---|---|
| Extracranial injury | 19 | 35 | 54 | 0.421 |
| Bone fractures | 10 | 17 | 27 | 0.583 |
| Evidence of impact injury to head (skull fracture/bruising to head) | 11 | 18 | 29 | 0.364 |
| Skull fracture | 6 | 3 | 9 | 0.034 |
| **Neuroradiological findings (n=63)** | **Apnoea n=21** | **No apnoea n=42** | | |
| Cerebral swelling | 9 | 7 | 16 | |
| Hypoxic/ischaemic change | 1 | 1 | 2 | |
| Infarction | 1 | 2 | 3 | |
| Contusion | 1 | 2 | 3 | |
| Diffuse axonal injury | 1 | 0 | 1 | |
| Intraparenchymal haematoma | 4 | 3 | 7 | |
| White matter change | 0 | 2 | 2 | |
| Mixed picture | 3 | 3 | 6 | |
| Cerebral atrophy | 0 | 3 | 3 | |
| Total cases with intracerebral damage | 13 | 18 | 31 | |
| No intracerebral damage | 8 | 24 | 32 | |

**Table 3** Comparison of our data with other relevant studies

| | Inclusion criteria | Case series | Apnoea | Skeletal fractures | Skull fracture | Extracranial injury | Hypoxic ischaemia |
|---|---|---|---|---|---|---|---|
| Geddes *et al* | Fatal NAHI in infants: SDH cases extracted | 31 | 24 (77%) | 16 (52%) | 12 (39%) | 17 (55%) 22 (71%) if skull fractures included | 27 (87%) (pathology diagnosed) |
| Kemp | Fatal NAHI: SDH children under 2 years | 16 | 10 (63%) | 9 (56%) | 3 (19%) | 16 (100%) | 11 (85%) of 13 who had CT/MRI |
| Johnson *et al* | Shaken baby syndrome: infants and very young children | 28 | 16 (57%) | 18 (64%) | 8 (29%) | | 20 (72%) (radiology diffuse brain swelling) |

ischaemia on the initial scan compared with eight of 42 without apnoea. Fisher's test indicated a significant difference between the two groups with $p = 0.02$.

Table 1 shows a highly significant association between diffuse brain swelling/hypoxic ischaemic damage seen on neuroimaging and poor clinical outcome ($\chi^2 = 19.56$ on 3 df, $p < 0.0003$). In a group of 21 cases who had no reported apnoea or intracerebral damage of any kind, only one child died (massive SDH with disseminated intravascular coagulation). There was a significant correlation with a good outcome ($\chi^2 = 9.34$ on 3 df, $p = 0.025$).

Of the 16 cases that sustained SDH or effusion from a recognised medical or witnessed accident, only one had apnoea at presentation. Two of the children with severe trauma had diffuse brain swelling. This group had an older age profile range, 85 days to 21 months (mean 8 months). Three children died (two road accident victims, one major trauma). Four had a KOSCHI score of 3, six had a KOSCHI score of 4, and three had a KOSCHI score of 5). The poor outcome was likely to be related to the underlying medical condition in nine cases (meningitis, haemorrhagic disease of the newborn, post-neurosurgery, complex perinatal problems relate to prematurity).

We compared our data with that of other studies (table 3). Although inclusion criteria and definitions vary between studies, the associations between apnoea, other associated injuries, and hypoxic ischaemia are remarkably similar.

## DISCUSSION

We have confirmed that SDH from NAHI is particularly prevalent in very young babies who present to hospital with a wide range of symptoms. A significant proportion of these children had apnoeic episodes during the acute phase of their illness. Apnoea is associated with radiological evidence of generalised brain swelling, an early indicator of hypoxic brain damage.[20 21] Coma at presentation, apnoea, and diffuse brain swelling/hypoxic ischaemic damage are associated with a poor prognosis.

We did not see a high incidence of radiological DAI (white matter shearing injury). We acknowledge that we may underestimate DAI on CT scan. It can be more confidently excluded on MRI,[22 23] but is ultimately a neuropathological diagnosis. Previous neuropathology literature reported DAI in NAHI in infants[24–26]; this lead to the theory that shaking impact forces frequently cause shearing injury within the brain with consequent poor long term outcome. In the light of current knowledge and more up to date histopathological techniques, Geddes and colleagues[11 13] dispute the validity and proposed pathogenesis of these findings. Recent studies argue that DAI is less commonly seen than previously thought.[11 13 16 27] Our radiological findings support the conclusions of Geddes *et al* and Johnson and colleagues[16] who advocate that hypoxic-ischaemic injury is of greater importance in terms of both symptoms and signs at presentation and of long term outcome. Diffuse brain swelling, evident on neuroimaging, is known to be associated with a high mortality rate in children with severe head injury,[21 28] more specifically in NAHI.[29]

Our findings are based on observations recorded in notes and radiological reports drawn from many centres. Our case series includes survivors of NAHI. Nevertheless comparison with a similar case series of Johnson and colleagues[16] and Geddes and colleagues' subgroup of fatal cases[11] are remarkably consistent. Our findings of four children who died and

had evidence of macroscopic damage to the cervical spinal cord lend some support to the suggestion that hyperextension/flexion injury to the craniocervical junction may contribute to apnoea and secondary hypoxic ischaemia and diffuse brain swelling. In light of the growing number of reports[8 30] of cervical cord damage in these cases, we would recommend that neuroimaging is extended to include the spinal column.

Most infants and young children with NAHI have associated, serious injuries that would in themselves be sufficient to make a clinical diagnosis of physical child abuse. There is evidence that these infants have been subject to a considerable degree of violence at or around the time of NAHI. In the absence of a high incidence of DAI, Geddes *et al* conclude that "it may not be necessary to shake an infant very violently to produce stretch injury to its neuraxis" and that "it is possible that the severe acceleration-deceleration injury does not occur in shaken baby syndrome". These are biomechanical hypotheses proposed by the authors, who, themselves identify 71% of infants with SDH who have significant extracranial injury or skull fracture[11] and 85% of children with impact head injury at postmortem examination.[13] This is indicative of a significant level of force in the majority of their case series. We are concerned that these statements have been widely interpreted in the media and used by defence lawyers[12] to suggest that minimal shaking forces alone may cause "shaken baby syndrome", when there is no evidence currently to support this.

The majority of SDH seen in NAHI are very shallow and often do not exert significant mass effect on the underlying brain. This may be why the true significance of these collections is sometimes not appreciated when the initial neuroimaging investigations are performed. The SDH itself is often the diagnostic marker of a mechanism of injury and a collection of symptoms and signs that are typically seen in "shaken baby syndrome". The SDH is rarely the prime cause of the presenting symptoms or responsible for the severe clinical outcome which are more likely to be related to the degree of associated hypoxic-ischaemic damage to the brain.

The factors associated with shaking are multiple and the forces elicited will vary according to the mechanism of injury, be it shaking or shaking impact, the strength and intent of the perpetrator, the size and muscle tone of the baby, and where the baby is held. Small babies have relatively poorly developed respiratory centres and are susceptible to apnoeic episodes. Damage to the cervical cord or brain stem,[11] squeezing of the chest, and concussion,[31] will all impair respiration. Extreme shaking forces may well cause shearing and axonal injury within the brain.[4] Associated impact injury will contribute to focal brain haemorrhage, contusion, and skull fractures. Together with secondary cerebral hypoperfusion,[32] hypoxic ischaemia, and refractory seizures the brain will sustain a combination of diffuse and focal damage.[33]

Decisions on forces and mechanisms of injury in this field are clearly complex. The current evidence base is insufficient to make any accurate comment about the degree of force that would be necessary to cause intracranial damage. It is important that multiagency collaborative research to pool biomechanical, clinical, neuropathology, radiology, and sociolegal findings continues to build up this resource. This issue has recently been highlighted by the President of the Family Division, Dame Elizabeth Butler-Sloss, who has called for "further research on the mechanism of subdural haematomas and the degree of force required to cause them in young children and babies, emphasising that this would be 'highly desirable' and very helpful for the medical profession faced with the results of injury in hospital, for the child protection teams and the judges and magistrates who try such cases".[34]

Although here is evidence of significant associated violence in the majority of infants who suffer from NAHI, at this point in time we cannot quantify the minimum forces required to cause brain damage when a baby is shaken. However, this condition does not arise from normal childcare or play activities. Implicit in this paper and the research literature is that it is never acceptable to shake a baby or child. Where shaking is clinically evident and the care given to the child is deemed "not being what it would reasonable to expect a parent to give",[35] then the child is in need of protection from further harm.

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## COMMENTARY

Ideas are changing in the field of non-accidental injury (NAI). The biomechanical evidence that shaking can produce subdural and retinal haemorrhage (SDH, RH) has recently been shown to be dubious,[1 2] and the assumption that fatally abused infants suffer severe traumatic brain damage has been disputed.[3 4] Low level accidental falls in childhood *may* occasionally cause death, producing SDH and RH.[5] This paper by Dr Kemp and her colleagues provides further food for thought. Having shown that hypoxic brain damage appears to be the significant feature of fatal cases, rather than diffuse axonal injury, the authors tackle the vexed question of how much force is needed to inflict a head injury on an infant. Even though there were serious associated injuries in 85% of their cases, they conclude honestly that "the current evidence base is insufficient to make any accurate comment about the ... force ... necessary to cause intracranial damage". Nevetheless, it is almost universally accepted that violence is a prerequisite of "shaken baby syndrome", partly because of the frequent finding of skull fractures or other injuries, and partly because of a widespread conviction that shaking subjects an infant brain to severe traumatic ("shearing") forces.

A recent study showed that traumatic brain damage is in fact rare in NAI, and produced evidence for a mechanism of injury (stretch to the neuraxis) that does not intrinsically require much force[3]—in apparent contradiction with the fact that, according to current dogma, only severe brain movement will cause SDH. Such a striking discrepancy must be addressed. Is trauma the cause of the bleeding? There is no way of proving that the typical infantile subdural, so different from the unilateral mass lesion seen in older children and adults, is the result of tearing of bridging veins during injury. Or, for that matter, of proving that the retinal bleeding is traumatic. Should we perhaps not focus on a neck injury causing reflex apnoea, with SDH and RH resulting directly from leakage from hypoxic vessels, in the setting of raised central venous pressure due to brain swelling? Haemorrhage is known to occur in many organs, including both brain and retina, as a result of asphyxial blood vessel damage and/or severe venous congestion, particularly in the fetus and infant.[6] There is some evidence to support just such an aetiology for SDH and RH in NAI, which would provide a physiological explanation for all the events and findings of fatal cases,[6] whether or not force had been used.

There is no doubt, as Dr Kemp and many others have shown, that most abused children have had violence inflicted on them. Nevertheless, it is important to think carefully about the actual mechanism that causes these babies to die, because in a number of fatal cases the neuropathology, even in the brain stem, is trivial; what has led to death is the *response* of the child's brain to the insult—that is, the brain swelling. From adult trauma work we now know that such a response is genetically determined, and that genotype may profoundly influence the clinical course after head injury.[7]

So, if there is even a possibility that the bleeding in these infants is a secondary event, and not caused directly by trauma, it must make us worry about those who have no objective evidence of injury in the form of bruising, skull fractures, or extracranial injuries, who present apnoeic and are found to have hypoxic brain swelling, with intracranial bleeding. Is it possible that some of them may have had neither a head injury nor a neck injury? Is it possible that (say) gastro-oesophageal reflux could have triggered an apnoeic attack that produced hypoxic brain swelling and intracranial bleeding? Could the explanation offered by the carer in fact be true?

How can we be sure? The only honest answer is that in this particular group of children, we can't. It is for such cases, those in which there are no grounds apart from medical opinion for allegations of abuse, that we need to keep trying to elucidate the actual mechanisms of injury, to ensure that any allegations we may make are supported by scientifically robust data.

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