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Inflicted head injury in infants

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Abstract

There is scant neuropathological information in the child abuse literature; even the best reviews include assumptions based on the findings of a few inadequate early studies. Our recent series of 53 fatal cases (Brain 124 (2001) 1290, 1299 [1,2]) demonstrated age-related patterns of brain injury and showed the substrate of severe encephalopathy in the infants to be hypoxic brain damage, not diffuse traumatic axonal injury ('DAI'), as had previously been thought. About one-third had craniocervical injuries, particularly in the brain stem, suggestive of stretch injury to the neuraxis. Our interpretation was that this finding implied a mechanism of injury—brain stem damage from stretch injury to the neck with resultant apnoea—that could account for the clinical scenario in many cases, and for which violence would not *necessarily* be required.

Since publishing this study we have turned our attention to the subgroup of infants who die without objective signs of injury, such as skull fracture or impact, whose carers are accused of abuse, usually, ''violent shaking'', on the pathologic findings alone. Given the striking discrepancy that there often is in such cases between the relatively trivial findings in the brain and the accusations of violence, we have been looking at the pathogenesis of the typical intracranial bleeding. A histologic study of dura from 50 paediatric autopsies, none of whom had suffered a head injury, has led us to propose that the subdural and retinal bleeding in such cases may well have a physiological aetiology, rather than being caused directly by trauma.

Keywords: Infant head injury; Non-accidental injury; Hypoxia; Craniocervical injury; Subdural haemorrhage; Retinal haemorrhages

1. Introduction

Most reviews of the so-called 'shaken baby syndrome' are quite clear about the neuropathological consequences of injuring young babies. Shaking, they say, causes retinal haemorrhages, subdural haemorrhage, brain swelling and tissue tears. In severe fatal cases, widespread brain injury also occurs, as a result of which the child loses consciousness. This brain injury, according to the literature, is primarily traumatic, of the type known as diffuse axonal injury (DAI), though hypoxic brain damage may also occur (and, indeed is invariably detected by neuroimaging [3]). The question of whether DAI is indeed an integral feature of inflicted infant head injury is not merely of academic significance: the conditions necessary to produce it are known to be extreme, and any head injury in which DAI has occurred is by definition severe traumatic injury.

A critical survey of the literature, however, reveals that there have been no systematic formal neuropathological studies of infant head injury, accidental or non-accidental, merely a few series looking at specific aspects, such as tissue ('contusional') tears [4], mechanisms of injury [5], or axonal damage [6]. It also reveals that the evidence base for DAI being a common finding in infant head injury [7–9] is poor, and that the belief originated from a single limited study by Vowles et al [10] performed before the diagnostic criteria for DAI were established [11–13], and before the advent of modern diagnostic techniques (see [2] for further discussion). The first formal study of microscopic damage in non-accidental injury (NAI) was published by Shannon and his colleagues, some 10 years after the Vowles paper, by which time the idea that DAI occurred was widely accepted in the

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literature. Shannon's findings, suggesting that axonal injury in such cases was virtually all ischaemic or vascular in origin, not traumatic [6], have been largely ignored or misunderstood. A subsequent claim by Gleckman et al [14] to have shown DAI in a small series of fatally injured infants is not fully documented, and appears to have misinterpreted ischaemic axonal injury for traumatic damage [2].

Our recent study of 53 cases of fatally injured children [1,2] included 37 infant cases, examined in detail. For microscopic brain damage, multiple blocks were taken, and immunohistochemistry for amyloid precursor protein performed on all [13]. The most frequent histological finding (in 84% of cases) was global hypoxic damage, apparently the result of an episode of significant apnoea occurring at presentation (documented in 70%). Diffuse axonal injury was present in only two children, both of whom had severe head injuries with multiple skull fractures. In 11 cases, however, there was macroscopic and/or microscopic evidence of injury to the craniocervical junction: in 8 of these axonal damage was localised to the corticospinal tracts in the caudal brain stem. This pattern of involvement of brain stem long tracts is identical to what has previously been described in adult cervical hyperflexion/hyperextension injury [13,15–17], and suggests that stretch injury to the neuraxis occurs in some of these infants. The resultant damage to the brain stem would account for the apnoea that is so frequently described, and this in turn would lead to the hypoxic brain swelling that is the immediate cause of death.

In summary, our study showed that in fatal non-accidental injury most infants have little in the way of traumatic brain damage, and demonstrated for the first time pathology which indicated how some of them might have been injured. This mechanism is one for which violence is not a prerequisite, despite the fact that there may be evidence of violence in many cases, in the form of extracranial injuries and/or skull fractures. And since a scenario of stretch to the neuraxis would not fit the biomechanical conditions for rupture of bridging veins and production of subdural haemorrhage [18], we have looked again critically at the possible aetiology of intracranial bleeding in these infants. In doing so, we have specifically asked the question: *is direct trauma to bridging veins the only possible explanation for subdural bleeding in these cases*? Further work leads us to suggest that the intracranial bleeding, both subdural and retinal, may in fact be a secondary phenomenon, not a direct consequence of trauma.

2. Dural bleeding in 50 paediatric cases without head injury

A high proportion of dural samples from routine autopsies carried out by a paediatric pathologist at the Royal London Hospital had been noted to contain florid intradural and subdural haemorrhage (Fig. 1), something that is not commonly seen in samples of dura from autopsies of older children and adults. We set out to study this bleeding

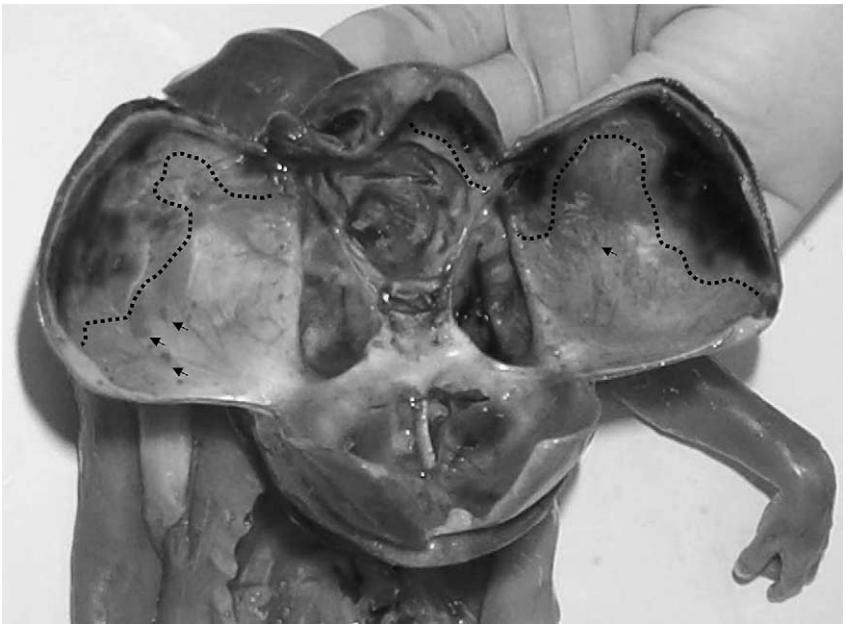

Fig. 1. A 26-week fetus that died rapidly in utero after placental abruption. The brain has been removed. The dotted lines surround very large confluent areas of intradural bleeding present under the calvaria, on both sides of the falx, as well as in the tentorium cerebelli. Some other smaller areas of haemorrhage are indicated by the arrows. While there was no obvious blood free in the subdural space at post mortem, histological sections revealed blood on the undersurface of the dura over both hemispheres, i.e. microscopic subdural bleeding.

systematically, and assembled a series of consecutive autopsies for which the appropriate parental consent had been obtained. The series, which is reported in detail elsewhere [19], reflects the practice of a paediatric pathologist working in a large teaching hospital, and comprises 50 cases, ages at birth from 18 weeks to 41 weeks. There were 17 intrauterine deaths, 3 spontaneous abortions, 16 perinatal, 5 neonatal and 9 infant deaths, the two oldest being 5 months of age. None of the children had suffered a head injury. A full autopsy had been performed on every case, the brain weighed accurately, and the cranial cavity inspected, before samples of dura were taken for histological examination. The medical notes were used in conjunction with the autopsy findings to establish the cause of death: in the majority of the subjects profound hypoxia, attributable to causes such as placental insufficiency, septicaemia, birth asphyxia, congenital cardiac or pulmonary disease, and bronchopneumonia, was a significant feature.

The principal pathology, in 36/50 cases, was bleeding inside the dura (intradural haemorrhage). In 11 of these, there was not only florid haemorrhage throughout the dura, but also on the surface, which in some cases was identifiably the subdural surface. The dural haemorrhage was fresh in all cases, and was a significant feature of the autopsy series, being found in deaths at all ages from the intrauterine deaths to infants of 5 months of age. The likelihood is that we will have underestimated the extent of bleeding in the cohort, because in many cases only one sample of dura was examined.

Intradural haemorrhage (meaning haemorrhage into the layers of the dura, not underneath it), was seen in a majority of the series, even though it is not a common autopsy finding in dura taken from older children or adults. We believe that, as with the haemorrhages familiar to paediatric pathologists in internal organs in cases of birth asphyxia or prematurity, the intradural and subdural bleeding in our series was a manifestation of severe hypoxia. Hypoxia is known to be an important aetiological factor in infant intracranial haemorrhage, including acute convexity subdural and primary subarachnoid bleeding in neonates and intracerebral bleeding in premature and low birth weight children [20–22]. Indeed, one early study we found in the literature actually describes intradural bleeding as a ‘constant finding’ in premature infants [23]. Despite the fact that in our series 79% of hypoxic cases had intradural bleeding as against 50% of those that were not documented to be hypoxic, the results lacked statistical significance ($P = 0.15$), but the P-value is probably explained by the very small number of cases that were not hypoxic (only six in total).

Having documented subdural as well as intradural bleeding in these children, we reviewed further sections of dura from three infants dying of an acute head injury, believed to have been inflicted. These children, who formed part of the group described in our earlier study, were 5 weeks, 7 weeks and 8 months old at the time of their injury, and their immediate cause of death was raised intracranial pressure. Retinal and subdural bleeding were present with marked brain swelling, and there was severe hypoxic damage on histology. Examination of sections of dura in all three cases revealed recent intradural and subdural haemorrhage: in two the blood from the dura appeared to have dissected out into the subdural space.

3. Hypothesis: relevance of hypoxic intra- and subdural bleeding to the pathology of ‘shaken baby syndrome’

The classic picture of fatal infant head injury is that of a moribund child, presenting after an episode of witnessed or presumed respiratory collapse. Brain swelling and subdural and retinal haemorrhages are detected in hospital, and for many doctors this pathologic triad is sufficient for an accusation of abuse. In a proportion of such cases, however, there are no objective signs of violence at autopsy, in the form of either subscalp bruising or skull fracture, and apart from cerebral swelling, there is remarkably little intracranial pathology. The blood in the subdural space is thinly distributed over the surface of the brain, insufficient to produce significant mass effect on its own. Subarachnoid haemorrhage, often overestimated at post mortem, is usually found to be negligible once the brain is fixed. There is rarely any intracerebral bleeding: contusions are not a feature, and parenchymal haemorrhages or tissue tears are not common [1]. On microscopy, traumatic axonal damage of any significance tends to be located in the brain stem long tracts and cervical nerve roots, the white matter of the hemispheres remaining remarkably undamaged [2].

Because the combination of unexplained subdural haemorrhage, retinal haemorrhage and brain swelling is so often associated with NAI, it is inevitable that an accusation of NAI may on occasion be based solely on these pathological findings, in the absence of other features suggestive of inflicted trauma. In such a case, the accusation originates from the doctors involved, who may persist with their allegations in the face of an alternative explanation from the carer, however, feasible. Phrases such as ‘violent shaking’, ‘fall from a two storey window’, ‘force comparable to that of a high speed road traffic accident’ are commonly employed by paediatricians, ophthalmologists, radiologists and pathologists. Such confident assertions of violence derive in part from studies of fatal accidental paediatric trauma, where extreme circumstances are known to produce subdural and retinal bleeding, and in part from the idea that DAI is common in these children (discussed above). There are, however, a number of objections to this. First, non-accidental injury is not the only cause of a swollen brain with subdural and retinal bleeding, and for this reason alone, extreme caution should be exercised in cases where there are no additional stigmata of injury. Second, while extreme force can undoubtedly produce the picture of ‘shaken baby syndrome’, we do not know the *minimum* force required to

rupture bridging veins and cause subdural bleeding. It is one thing to demonstrate that violence has been used to inflict a head injury in the majority of cases, and quite another to conclude from this that violence has always to occur. A third consideration is that we do not know for certain whether bridging veins actually do rupture in these cases, because the characteristic subdural bleed seldom if ever comes to neurosurgery. The few autopsy studies that have addressed this question [24–26] have merely shown venous contrast leakage in the subdural space, without being able to distinguish between traumatic rupture and abnormal vascular permeability.

In our series, having found intradural and subdural bleeding identical to that seen in hypoxic infants in three NAI cases, we propose that the aetiology of the subdural haemorrhage results from abnormal vascular permeability, caused by a combination of hypoxia-related damage to immature intracranial veins, combined with grossly raised central venous pressure. Our findings in the present series suggest that, in the immature brain, hypoxia is sufficient to cause extravasation of significant amounts of venous blood both in and under the dura. If intracranial—specifically, central venous—pressure is elevated, as it is in the majority of cases of infant NAI, vascular fragility and bleeding would be greatly enhanced.

There are a number of other physiological factors operating in these children that would promote bleeding. Very often systemic arterial pressure is raised, either as part of Cushing's triad, or in blood pressure surges, which occur in children as intracranial pressure rises. Additionally, blood pressure control by centres in the medulla [27,28] could well be deranged by an injury to the craniocervical junction. Whatever the mechanism, a rise in arterial pressure would increase external carotid blood flow to the dura, in a situation in which the venous drainage via the dural sinuses is already intensely congested because of raised venous pressure. Hypoxia also directly damages vascular integrity: it causes intracranial vessels to leak, via upregulation of vascular endothelial growth factor [29], and depletes glycogen in vascular smooth muscle, impeding its metabolism and normal function. In children with severe hypoxic brain swelling, all these factors are going to increase the likelihood of prolonged venous leakage, in veins in the dura and the subdural space.

Is it possible that retinal haemorrhages are also the result of hypoxic vascular damage with raised central venous pressure? The answer is: entirely possible. Retinal haemorrhages are known to be caused by rises in intracranial and central venous pressure, with and without hypoxia [30,31]; they are also a normal occurrence in some full term and premature births [32,33]. In the so-called 'shaken baby syndrome' it has never been shown that the retinal bleeding is the result of direct trauma to retinal vessels; rather, it has been widely assumed to be so [34], despite the fact that an authoritative recent review of the biomechanics of paediatric head injury has described the hypothesis as 'biomechanically improbable', and suggests there is compelling evidence that rapidly rising intracranial pressure is responsible [35].

Finally, if subdural and retinal bleeding have a 'physiological' rather than a 'biomechanical' aetiology, a number of important facts follow:

- The physiological scenario outlined above would account for all the features of some cases of infant head injury, without significant force being necessary.
- There would be no need to postulate impact in cases in which there appears to have been none.

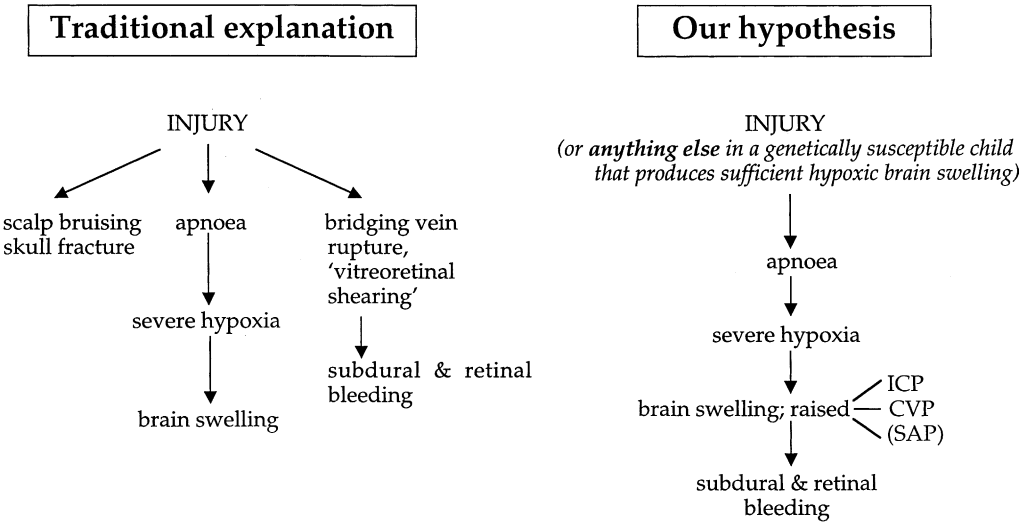

Fig. 2. The physiological cascade that may be involved in the production of intracranial bleeding in many infants. (See text for full explanation.)

- The proposed sequence of events would also account for the occasional case in which retinal and subdural haemorrhage occur, in the absence of trauma.
- It is possible that some cases of alleged 'shaking' may not in fact have had a head injury at all.

In relation to this final point, it is known that genetic factors play a critical role in determining the response of the individual brain to a given insult [36], and it is possible that in a genetically susceptible child *any* factor that triggers an episode of apnoea sufficient to cause severe hypoxic brain damage may precipitate the cascade illustrated in Fig. 2, resulting in subdural and retinal bleeding.

4. Conclusion

Our neuropathological studies of fatal cases of infant head injury and of dura from non-traumatic paediatric autopsies have led us to propose that the intracranial bleeding in NAI may be a secondary phenomenon resulting from deranged infant physiology, rather than a direct result of trauma. Such a possibility highlights the difficulty of being certain that abuse has occurred in cases where there is no objective evidence of either trauma or violence.

Note added in proof

After this paper was submitted an article by Dr. Reichard and his colleagues, ''Beta-amyloid precursor protein staining of nonaccidental central nervous system injury in pediatric autopsies'' was published in J. Neurotrauma 20 (2003) 347–355. This neuropathological study of 28 victims of abuse, including 10 children under 1 year of age, found diffuse traumatic axonal injury in only 1 infant, confirming our finding that DAI is a rarity in the very young.

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