

# Dural haemorrhage in non-traumatic infant deaths: does it explain the bleeding in 'shaken baby syndrome'?

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## Dural haemorrhage in non-traumatic infant deaths: does it explain the bleeding in 'shaken baby syndrome'?

A histological review of dura mater taken from a *post-mortem* series of 50 paediatric cases aged up to 5 months revealed fresh bleeding in the dura in 36/50, the bleeding ranging from small perivascular haemorrhages to extensive haemorrhage which had ruptured onto the surface of the dura. Severe hypoxia had been documented clinically in 27 of the 36 cases (75%). In a similar review of three infants presenting with classical 'shaken baby syndrome', intradural haemorrhage was also found, in addition to subdural bleeding, and we believe that our findings may have relevance to the pathogenesis of some infantile subdural haemorrhage. Recent work has shown that, in a proportion of infants with fatal head injury, there is little traumatic brain damage and that the significant finding is

craniocervical injury, which causes respiratory abnormalities, severe global hypoxia and brain swelling, with raised intracranial pressure. We propose that, in such infants, a combination of severe hypoxia, brain swelling and raised central venous pressure causes blood to leak from intracranial veins into the subdural space, and that the cause of the subdural bleeding in some cases of infant head injury is therefore not traumatic rupture of bridging veins, but a phenomenon of immaturity. Hypoxia with brain swelling would also account for retinal haemorrhages, and so provide a unified hypothesis for the clinical and neuropathological findings in cases of infant head injury, without impact or considerable force being necessary.

Keywords: infant head injury, non-accidental injury, subdural haemorrhage

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### Introduction

For many authors, subdural haemorrhage (SDH) in infancy has become synonymous with inflicted trauma, particularly when associated with retinal haemorrhages, despite the fact that accidental injury in this age group has also been documented to result in subdural and retinal bleeding [20,28,33]. The existence of isolated reports of

supratentorial SDH occurring *in utero* [2,17,21], or in neonates and infants from causes apart than trauma [8,26,30,31], is an indication that as in other areas of medicine, several mechanisms may lead to the same clinical picture [6]. Nevertheless, the term 'shaken baby syndrome' tends to be automatically applied to any infant with a swollen brain, subdural and retinal bleeding. This label, alleging as it does non-accidental injury, effectively precludes any further discussion of how these clinical features might have been caused, even though all of them, both singly and in combination, may be seen in conditions

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other than trauma. We have reviewed sections of dura taken from a *post-mortem* series of 50 cases who had died under 1 year of age, none of whom had sustained a head injury, and compared them with the findings in three cases of classical 'shaken baby syndrome'. Our findings lead us to propose an alternative, non-traumatic, aetiology for subdural and retinal bleeding in these infants.

## Subjects and methods

A review of samples of dura from paediatric autopsies was approved by the Tissue Subcommittee of the East London and City Health Authority Research Ethics Committee. These were a consecutive series of infant cases in which informed parental consent had been obtained for tissue to be taken at *post-mortem*; a formal review was initiated to investigate the significance of haemorrhage associated with the dura.

The series comprised 50 cases with age at birth of 18–41 weeks. There were 17 intrauterine deaths (IUD), three spontaneous abortions, 16 perinatal deaths (less than 7 days after birth), five neonatal deaths (occurring within the first postnatal month) and nine deaths in infancy (children under one year of age). Median survival of the 30 perinatal, neonatal and infant cases was 1 week. The two oldest children were aged 5 months, although one had been born prematurely at 23 weeks and survived 10 months (corrected developmental age 5 months). Cases in which there had been a therapeutic termination of pregnancy were excluded. None of the cases had suffered a head injury. A full *post-mortem* (with the exception of the eyes, which were not examined) had been performed by a paediatric pathologist, and the clinical course of all those children who had survived after delivery was available. Clinical details were used in addition to the *post-mortem* report to establish the mode of death in any case where the child had lived; otherwise, this was determined from the underlying condition, where known, and from the macroscopic and microscopic changes found at *post-mortem*.

The brain weight had been recorded in every case. The pathologist had noted on removal of the brain that the dura in many cases was 'haemorrhagic' or 'congested', but in only one case had there been macroscopic evidence of subdural bleeding: a female born at 25 weeks who developed fulminating *Enterobacter* septicaemia secondary to severe chorioamnionitis, and died 1 week later, having been in intensive care since birth. A large unilateral sub-

dural haematoma was found at *post-mortem*, acting as a mass lesion.

Between one and three strips of dura from each case had been processed and embedded on edge, and a Perls stain and immunohistochemistry for CD68 (PG-M1) performed. The slides were reviewed by a single pathologist, who was blind to the clinical details.

## Infants with head injury

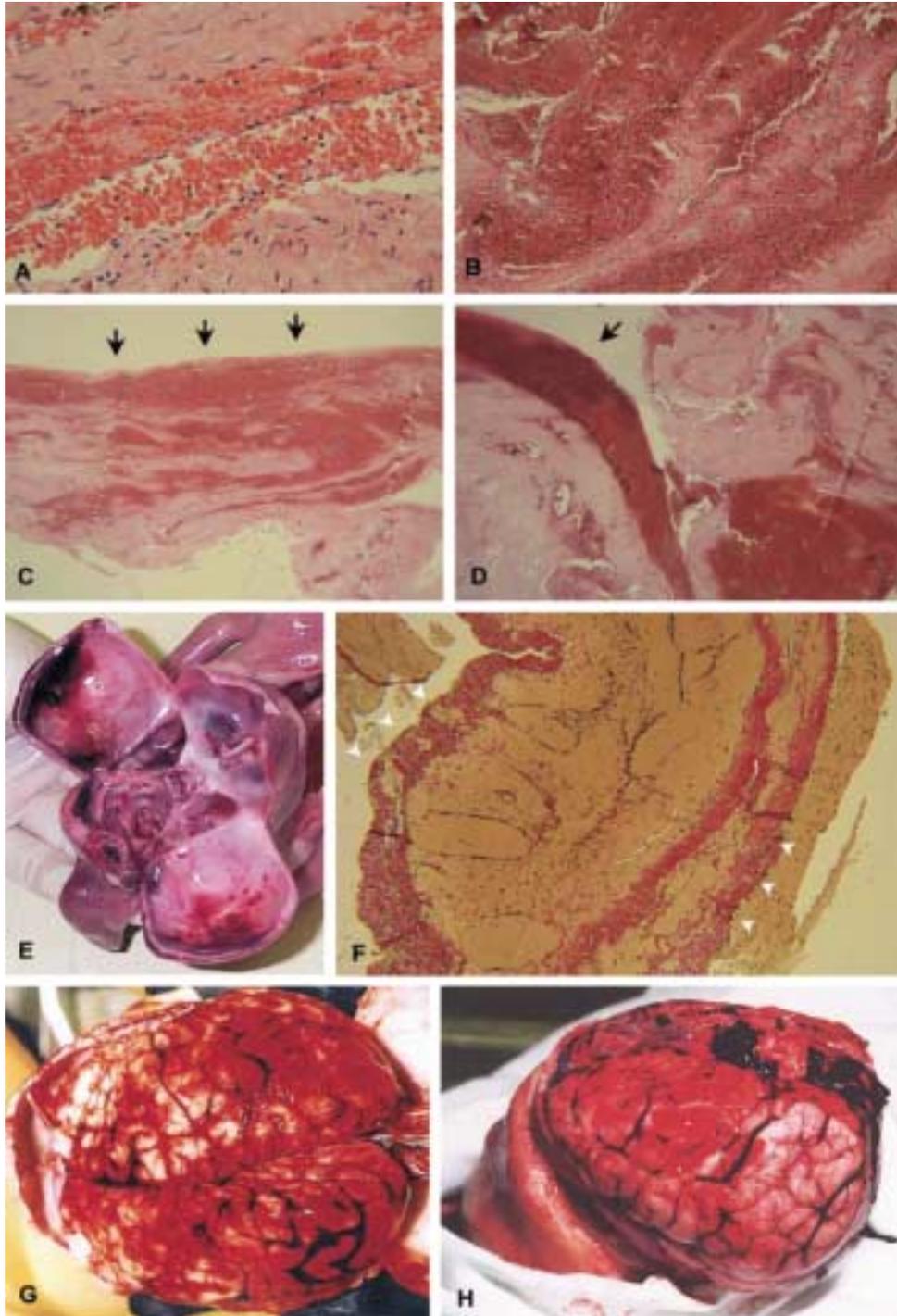
An additional series of dural samples was reviewed from three infants from an acute head injury, believed to have been inflicted. The children were aged 8 months, 7 weeks and 5 weeks at the time of injury, and the immediate cause of death in all had been raised intracranial pressure (ICP). They all had documented retinal haemorrhage. Bilateral subdural bleeding had been seen on *pre-mortem* scans and was confirmed at *post-mortem*. None had either a skull fracture or subscalp bruising, and all had marked brain swelling, manifest as significantly increased brain weight, with severe hypoxic brain damage on histology. One of the three had extracranial injury in the form of old rib fractures. The three had previously been included in a large series of non-accidental head injury [12,13], and were the only infant cases from that series in which sections of dura were still available.

## Results

### Findings in 50 cases without head injury

The immediate causes of death were infection ( $n=6$ ), hypoxia ( $n=26$ ), infection with documented severe hypoxia ( $n=8$ ) and Sudden Infant Death Syndrome (SIDS) (4). Six deaths, five of them intrauterine, remained unexplained after *post-mortem*.

The only significant pathology, present in 36/50 cases, was bleeding inside the strips of dura (intradural haemorrhage, IDH) as illustrated in Figure 1. All the intradural bleeding was fresh: there was no haemosiderin, Perls' positivity or evidence of a macrophage reaction in any of the 36 cases, suggesting that the bleeding found at *post-mortem* was likely to have occurred less than 2–3 days before death [3]. In at least 24/36 (66%), delivery could be excluded as an aetiological factor in the bleeding, because IDH was present in 11 of the 17 cases that had died *in utero* and in 13 of the 18 infants who lived for 5 days or more (median 23 days). Fifteen children had not died *in utero*



**Figure 1.** (a–d) Intradural and juxtadural bleeding in four non-traumatic infant autopsies. (a) Showing minimal extravasation of red blood cells; (b) More extensive bleeding confined to the dura. In (c) and (d), arrows indicate where blood has ruptured through the dura and lies on the surface (all haematoxylin and eosin). (e) Florid intradural bleeding with some subdural blood adherent to the dura of a 26-week-old-fetus, who died *in utero* after placental abruption. (f) Dura from a case of non-accidental head injury, stained with haematoxylin van Gieson; the two edges of the dura are indicated by white arrowheads. Blood (yellow) has dissected between layers of collagen (red), and ruptured through on to the subdural surface, on the right. (g,h) Two cases of non-accidental infant head injury at *post-mortem*, showing the typical appearance of the subdural bleeding in such cases. The bleeding lies over both hemispheres. In (h), there is a small collection in the interhemispheric fissure. Note that virtually all the surface blood is subdural; a small amount of subarachnoid haemorrhage is present in the sulci.

but survived less than 5 days. Eight of these had a spontaneous vaginal delivery, of whom five had IDH. Of the five delivered by Caesarean section, four had IDH, three of them severe (++). Two others, both with severe IDH, had a ventouse extraction and a failed ventouse plus Caesarean section, respectively.

The extent of the haemorrhage in the samples was graded as '0' (meaning none, or only minimal extravasation of small groups of red cells), '+' (small haemorrhages round several vessels) and '++' (florid IDH; widespread foci of haemorrhage, sometimes occupying the full thickness of the dura). Table 1 summarizes the data according to degree of IDH and type of death. In 11 of 50 cases, blood had ruptured right through the collagen, on to its outer surface (Figure 1c,d, Table 2). We have used the term 'juxtadural bleeding' to describe these cases, where we were

unable to tell from sections whether the blood was lying in the extradural or subdural space.

The cause of the large unilateral SDH in the 25-week-old-fetus described above was not found, but assumed to have resulted from severe sepsis and disseminated intravascular coagulation [26].

Brain weights were compared to standard paediatric tables. Where they fell outside the normal range for the age, values were compared with the actual size and weight of the body. The weight of the brain was found to be normal in 32 cases, reduced in five and raised in one. In 12 further cases, the clinical history and/or the degree of maceration suggested that the child had been dead *in utero* for more than 24 h, so the brain weight was disregarded for the purposes of this review because it possibly did not reliably reflect the state of the brain at the time of death. The one case in which the brain weight was increased to a level that might have caused raised ICP was a 1-month-old infant whose cause of death remained unascertained after *post-mortem*. Intradural bleeding was present in that case.

There was an association between the occurrence of severe IDH (graded '++') and the type of case: florid bleeding was seen in 55% (11/20) intrauterine deaths and spontaneous abortions; 69% (11/16) perinatal deaths and only 14% (2/14) neonatal and infant deaths ( $P < 0.001$ ) (Table 2). The results for IUD and spontaneous abortion were similar, and so these two groups were combined as cases that had no postnatal survival; for the same reasons, the results for neonatal and infant deaths were also combined. Table 3 documents the association between IDH, infection and hypoxia in 40 cases for which the cause of death was known: there was no evidence of a relationship between IDH and infection ( $P = 0.72$ ) and only a slight indication of one with hypoxia ( $P = 0.15$ ); the percentage of cases that were hypoxic and had an IDH was 79% compared to only 50% in the cases that were not hypoxic. Although this could be considered to be an important difference, chance cannot be ruled out. However, because the  $P$  value was not far from 0.05, the lack of statistical significance is probably due to the small number of cases, in particular those without hypoxia, of which there were only six in total.

**Table 1.** The number of cases according to type of death and degree of IDH

Type of death	IDH grading*			All
	0	+	++	
Spontaneous abortion	2	1	0	3
Intrauterine death	5	0	10	15
Stillbirth	0	1	1	2
Perinatal death	2	3	11	16
Neonatal death	2	3	1	6
Infant death	3	4	1	8
All	14	12	24	50

0, No haemorrhage, or only minimal extravasation of small groups of red cells; +, small haemorrhages round several vessels; ++, florid IDH; widespread foci of haemorrhage, sometimes occupying the full thickness of the dura.

**Table 2.** Relationship between severe intradural bleeding (graded '++') and type of case

Type of case:	Severe IDH present?	
	Yes (++)*	No (0, +)*
IUD, spontaneous abortion	11 (55%), 7 had JDH	9
Perinatal death	11 (69%), 3 had JDH	5
Neonatal and infant death	2 (14%), 1 had JDH	12
Total	24 (48%)	26

JDH, Juxtadural haemorrhage; bleeding that had ruptured the outer layer of the collagen, with clot lying on the outer surface of the dura (Figure 1c,d). \*0, no haemorrhage, or only minimal extravasation of small groups of red cells; +, small haemorrhages round several vessels; ++, florid IDH; widespread foci of haemorrhage, sometimes occupying the full thickness of the dura.

## Cases of infant head injury

The samples of dura from three cases of presumed head injury all showed recent IDH, which had not been noted in the original histological report, as well as SDH. In these

**Table 3.** Relationship between the presence of intradural bleeding, infection and hypoxia in 40 cases (after exclusion of six unexplained deaths and four SIDS)

Cause of death:	IDH present? Yes (+, ++)*	No (0)*
Infection?		
Yes	10 (71%)	4
No	20 (77%)	6
Hypoxia?		
Yes	27 (79%)	7
No	3 (50%)	3
Mixture		
Infection only	3 (50%)	3
Hypoxia only	20 (77%)	6
Infection & hypoxia	7 (88%)	1

0, No haemorrhage, or only minimal extravasation of small groups of red cells; +, small haemorrhages round several vessels; ++, florid IDH; widespread foci of haemorrhage, sometimes occupying the full thickness of the dura.

cases, the two surfaces of the dura were identifiable and, in two of the three, the blood appeared to have dissected from the dura into the subdural space (Figure 1f).

## Discussion

The principal finding in this series of 50 non-traumatic paediatric autopsies, from deaths *in utero* up to infants aged 5 months, is that varying degrees of intradural and juxtadural bleeding were present in 72% of cases (36/50). In two-thirds of these, delivery-related trauma could definitely be excluded as a cause. This finding is particularly important because we have observed identical bleeding in three infants presenting as classical 'shaken baby syndrome'.

IDH (meaning haemorrhage within the dura as opposed to the more usual meaning in clinical practice of haemorrhage beneath the dura) is often seen in biopsies from adults or older children as an artefact of surgery, but not in dura taken at *post-mortem* from these age groups. However, it was very common in the paediatric autopsies we reviewed, and Figure 1 demonstrates that, in such cases, much of the dura of the calvaria may contain haemorrhage (e). Our study may well have underestimated the incidence of bleeding in this cohort, since, in most cases, only one sample of dura was taken.

The series we have studied reflects the practice of a teaching hospital paediatric pathologist, and most of the subjects had been profoundly hypoxic before death, as a

result of conditions such as bronchopneumonia, congenital heart or lung disease, placental insufficiency (both acute and chronic), septicaemia and birth asphyxia. Petechial haemorrhages in many internal organs, including in the dura, are well recognized by paediatric pathologists to be a characteristic finding in cases of birth asphyxia [11,35], while an early study documented intradural bleeding as a 'constant finding' in premature infants [5]. Similarly, venous hypertension and asphyxial cardiovascular damage have been implicated in the aetiology of acute convexity subdural and primary subarachnoid haemorrhage in newborn infants, and in intracerebral bleeding [11,15,34]. Mechanical compression of the head and rises in central venous pressure during delivery may contribute to neonatal intracranial haemorrhage, but they were not the cause of the IDH in at least 66% of our 36 cases, in whom the primary mechanism was likely to have been hypoxia-induced alterations in the permeability of immature vessels, causing blood to leak into the extravascular compartment. The collagen layers of the infant dura are generally much more cellular and less compact than in older subjects, and so do not tend to impede the extravasation of blood [5].

## Intracranial pathology in non-accidental injury

A recent study by Geddes *et al.* [14] described the neuropathology of 53 cases of inflicted head injury in detail. Perhaps the most unexpected finding was that few of the 37 infants in that series showed anything in the way of traumatic brain damage, apart from craniocervical damage in approximately one-third of the cases, the microscopy being compatible with stretch injury to the neuraxis. The authors proposed that in those cases hyperflexion-hyperextension of the neck had caused primary brainstem damage, which in turn had provoked apnoea, resulting in global hypoxia and catastrophic secondary brain swelling. Hypoxia, known from neuroradiological studies to be a cardinal feature of infant head injury, was an important finding in that series: 78% of infants presented with apnoea or respiratory difficulties, while 84% had microscopic evidence of global hypoxia. In fact, in a significant proportion, hypoxic damage was the only pathology found in the brain. Secondary hypoxic brain swelling was the most frequent cause of death, with subdural and retinal bleeding present in 84% and 70% of infants, respectively [12,13].

These findings suggested a mechanism which would explain most of the clinical picture of 'shaken baby syn-

drome', but which left the SDH unexplained. Because stretch injury to the craniocervical junction need not involve either violence or impact, it has been calculated that the maximal forces involved in shaking a baby, derived from experimental work [7,23], do not reach the threshold necessary to rupture cortical bridging veins, if there is no impact to the head. A number of fatal cases show no evidence of impact, even after careful examination, a fact that is usually explained away by suggesting that the head must have been decelerated (or 'slammed') against a soft surface, such as a mattress. However, what is usually ignored is that the typical subdural haemorrhage in infants is quite different from that seen in older children or adults. Characteristically spread over both hemispheres, often no more than a thin film, but sometimes with small collections of blood in the interhemispheric fissure, it does not require neurosurgical intervention; SDHs acting as mass lesions tend to be seen in older age groups [12,13]. The question arises: if the assumed circumstances of the incident (severe shaking, with or without impact, the force equivalent to a fall from a two storey window or involvement in a high speed road traffic accident) do not fit the observed neuropathology in many of the infants, could there be another source (and, possibly, mechanism) of bleeding for this unique form of SDH?

### Relevance of intradural bleeding: a hypothesis

The three cases of inflicted head injury reviewed here also had intradural bleeding, and in two of the three blood was present through all the layers of collagen, in continuity with the blood in the subdural space. We suggest that hypoxia-related leakage of blood from veins both inside the dura and in the subdural space was the source of the subdural haemorrhage found in each case, rather than traumatic rupture of bridging veins.

What would be the mechanism of IDH in non-accidental injury? Most infants with inflicted head injury have severe hypoxic brain damage and rapidly develop grossly raised ICP from secondary brain swelling, which is documented on a scan taken on arrival at hospital, and confirmed at *post-mortem* by markedly increased brain weight [12,13]. Our observations in the present series indicate that, in the immature brain, hypoxia both alone and in combination with infection is sufficient to activate the pathophysiological cascade which culminates in altered vascular permeability and extravasation of blood within and under the dura. In the presence of brain swelling and raised intracra-

nial pressure, vascular fragility and bleeding would be exacerbated by additional haemodynamic forces, such as venous hypertension, and the effects of both sustained systemic arterial hypertension and episodic surges in blood pressure.

The interactions between factors such as venous pressure, blood pressure and brain swelling are potentially complex. Cerebral venous hypertension occurs when there is an obstruction to flow, which is the situation where there is cerebral swelling. The dural venous drainage, in the main, is via the meningeal veins which originate in plexiform venous channels within the dura, and drain eventually into the superior sagittal and other intracranial sinuses; only the posterosuperior group of vessels drain externally, via the jugular foramen [36]. We hypothesize that the presence of severe brain swelling with venous congestion would produce widespread 'oozing' from leaky hypoxic dural veins, possibly with a contribution from similarly leaky bridging veins, and that this is responsible for the typical thin film or patchy collections of subdural blood. A significant additional contributor would be the systemic arterial hypertension, both sustained and episodic, that is commonly documented in children with raised intracranial pressure. This may occur as part of Cushing's triad (bradycardia, hypertension and raised intracranial pressure), or in blood pressure surges, characteristically seen for 1–2 h around the time of brainstem herniation or tentorial pressure 'coning'. Alternatively, blood pressure lability may be neurogenic in origin, resulting from lesions of the nucleus of the solitary tract [4,10], which could be particularly relevant in an infant with craniocervical injury. Whatever the cause, a rise in arterial blood pressure in a patient with cerebral oedema will increase external carotid flow to the arterial branches in the dura, which in turn would be transmitted to the already congested venous side, and so compound the effects of raised venous pressure. Finally, brain oedema is exacerbated both by superimposed hypercapnia resulting from respiratory abnormalities, and by arterial hypertension [18,24,25,27]. These three factors, cerebral venous hypertension and congestion, arterial hypertension and brain swelling, coupled with immaturity and hypoxia-related vascular fragility, provide an alternative physiological scenario for the characteristic subdural bleed of the 'shaken baby syndrome'.

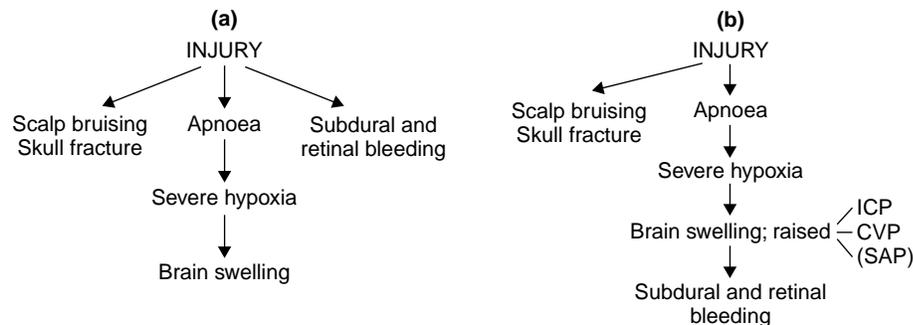
Similarly, retinal haemorrhages can be explained by rises in intracranial and central venous pressure, with and without hypoxia [19,22]; they are also seen in a propor-

tion of normal infants at birth [9], as well as in premature babies [1]. The incidence of retinal haemorrhage in neonates reported in the literature varies, possibly due to different patient demographics, difference in time after birth to examination and different examination techniques. In healthy newborns, it has been documented that delivery by Caesarean section reduces the risk of haemorrhage [9], although this is not always the case [29]; in premature infants, the mode of delivery does not appear to be associated with a different incidence of retinal haemorrhage [1]. In a prospective study of a consecutive series of premature infants, the occurrence of retinal bleeding was approximately one-half of that seen in term infants, and is believed to be partly explained by the effects of ventilation in reducing hypoxia, and thus abnormal vascular permeability and blood vessel fragility [1]. In the setting of inflicted infant head injury, it has never been proved that retinal bleeding is directly caused by shaking; rather, it is widely assumed that it results from the shearing forces of the injury, which simultaneously cause retinal and subdural bleeding and diffuse brain damage [16]. However, with the knowledge that most infant victims of non-accidental injury show very little if any traumatic pathology in the brain [13], it is appropriate to re-evaluate this assumption. We suggest that in many cases of non-accidental head injury retinal haemorrhages occur for essentially the same physiological reasons as subdural bleeding, as outlined above.

This constellation of events, severe hypoxic damage to immature blood vessels, exacerbated by raised ICP, central venous and systemic arterial hypertension, is not proposed to be the cause of all infantile subdural haemorrhages: for example, traumatic rupture of one or more

bridging veins would be a more likely explanation of significant unilateral bleeds. Nor is it necessarily the sole mechanism of retinal haemorrhages. However, if retinal and subdural bleeding are essentially secondary phenomena, and not directly the result of trauma (Figure 2), the sequence provides an explanation for all the findings in many cases of 'shaken baby syndrome', without impact or violence being necessary. Conversely, in cases in which there has obviously been significant impact (scalp bruising and/or skull fracture with brain swelling), it would be unnecessary to postulate shaking as well, in order to account for retinal and subdural haemorrhages. Finally, a physiological rather than a traumatic mechanism for the bleeding would provoke further speculation: is it possible that occasional instances of 'shaken baby syndrome' may not be cases of 'shaking' or, indeed, of head injury at all? In a susceptible infant (for it is likely that, as in the adult, genetic factors play an important part in the individual response to a given cerebral insult [32]), subdural and retinal bleeding might result from any event that initiated apnoea or significant hypoxia, with brain swelling. Such a possibility highlights the difficulties posed by isolated cases in which there is nothing apart from subdural and retinal bleeding to substantiate an allegation of inflicted trauma; it is clear that extreme caution should be exercised by experts involved in such cases. In such circumstances, we suggest it would be better to use a descriptive term such as 'infantile encephalopathy with subdural and retinal bleeding', which has no aetiological implications, rather than 'shaken baby syndrome'.

Finally, the frequency with which IDH has been found in our *post-mortem* series certainly makes it likely that intradural bleeding may also occur in children who do not die.



**Figure 2.** (a) Illustrates the traditional view of events in infant head injury, according to which retinal and subdural bleeding are a direct consequence of trauma. Our findings suggest that the sequence outlined in (b) may be more likely, with the bleeding being a secondary event. A full description is provided within the text. ICP, intracranial pressure; CVP, central venous pressure; SAP, systemic arterial pressure.

For that reason, when examining sections of dura for medico-legal purposes, particularly in the context of 'timing' of injuries, histopathologists should not automatically ascribe the presence of any haemosiderin, macrophages (or even possibly organizing haemorrhage) to a previous head injury.

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