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Review

Retinal haemorrhages in inflicted traumatic brain injury: the ophthalmologist in court

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Abstract

Child abuse is a significant social and public health problem in many societies. One particular form of physical abuse is inflicted traumatic brain injury (ITBI) – the so-called 'Shaken Baby Syndrome'. One of the key features associated with ITBI is characteristic ocular findings – only observed on fundal examination. It is crucial *not* to miss the diagnosis of ITBI as there may be implications for the safety of the child. The ophthalmologist is one key contributor to the process required to make the diagnosis of ITBI accurately and on the basis of all the relevant evidence. This article discusses some medicolegal implications of examining a child with retinal haemorrhages in the context of suspected ITBI, with practical advice for the ophthalmologist to be as objective and prepared as possible. The scientific validity of some alternative explanations advanced in court is discussed, with a strong emphasis on the literature.

Key words: child abuse, medicolegal, retinal haemorrhage, shaken baby syndrome, traumatic brain injury.

Introduction

Child abuse is a significant social and public health problem in many societies. One particular form of physical abuse is inflicted traumatic brain injury (ITBI) – the so-called 'Shaken Baby Syndrome' (SBS), which has an estimated annual incidence of 15–20 per 100 000 in New Zealand. Within the Maori population the incidence is even higher – between 33 and 39 per 100 000.[1] Several high profile cases in recent years have drawn public attention to this preventable cause of infant morbidity and mortality. One of the key features associated with ITBI is characteristic ocular findings – only observed on fundal examination. It is crucial *not* to miss the diagnosis of ITBI as there may be implications for the safety of the child. The ophthalmologist is one key contributor to the process required to make the diagnosis of ITBI accurately and on the basis of all the relevant evidence. Although less than half of these cases proceed to a criminal trial,[2] the observation of positive findings renders the ophthalmologist liable to appear as an expert witness in that trial, so clear documentation is essential.

The orthodox scientific consensus view on the significance of retinal haemorrhages (RH) in ITBI is often challenged in criminal courts in Australasia. The ophthalmologist may be put under great pressure to defend both their examination findings and their interpretation of the significance of those findings. Alternative explanations for the retinal findings are often proposed in court, and these suggestions are often similar from case to case, regardless of the specific circumstances of each case.

This article discusses some medicolegal implications of examining a child with RH in the context of suspected ITBI, with practical advice for the ophthalmologist to be as objective and prepared as possible. The scientific validity of some alternative explanations advanced in court is discussed, with a strong emphasis on the literature.

Although the randomized controlled trial is widely regarded as the gold standard in evidence-based medicine, it is clearly impossible to perform

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an experiment in which human infants are randomized to various forms of shaking and/or impact, followed by dilated fundoscopy. A fully adequate animal model of the relationship between RH and trauma has not yet been described. Although recent attempts at computer modelling (finite element analysis) have been undertaken to investigate the retinal forces produced,[3,4] to date the evidence from observational studies, carefully documenting corroborated causes for RH in infancy, becomes very important.

Terminology

'Shaken Baby Syndrome' is a term frequently used in the ophthalmic literature, but it may be suggested that this entity does not exist. That suggestion centres on two issues. First, in many cases of ITBI (particularly those who die), evidence of impact to the head may be found. Second, the biomechanical forces generated by impact are significantly greater than the forces predicted to result from shaking alone, and some have suggested that it is biomechanically improbable that shaking alone could cause the brain injuries seen in SBS.[5,6] This stands in contrast to the data from perpetrator confessions that suggest that shaking alone can indeed cause these injuries.[7,8] Although this data has been challenged,[9,10] clinical cases continue to be published where shaking is confessed and no evidence of impact can be found,[11] and the biomechanical science is complex and far from certain.[12,13] There is no need for the ophthalmologist to become embroiled in a theoretical discussion on the biomechanics of head injury. It is reasonable to acknowledge that the possibility of impact can never be excluded in a case of so-called SBS, and therefore preferable to use a term that does not limit the diagnosis of non-accidental injury to one particular mechanism. Preferred terminology includes ITBI, or non-accidental head injury (NAHI).[14]

Summary of ophthalmic findings observed in inflicted traumatic brain injury

The appearance of RH in ITBI in children is often characteristic and best observed through dilated fundal examination. The haemorrhages are typically too numerous to count, and present in multiple layers of the retina (preretinal, intraretinal, subretinal) therefore have a myriad of morphologies – dot, deep blot and flame (Fig. 1). They often extend to the periphery. These characteristic retinal findings, even if unilateral (in 20%)[15] are highly specific for ITBI. However the RH does not exist in isolation but in conjunction with other signs in a clinical context. Evidence of external injury is often absent.[16–18]

One highly characteristic form of RH is often observed, specifically the dome-shaped haemorrhage or retinoschisis, sometimes manifesting as a traumatic macular fold. Recent reports suggest this lesion may rarely result from crush and other severe injuries;[19–21] however, the common denominator is likely to be significant force impacting directly or indirectly on the infant retina.

In reported cases of confirmed child abuse where the perpetrator confessed[7,8,11] the retinal findings are consistent with the findings described in the preceding text. Characteristically there is *no* evidence of direct ocular trauma – no periorbital bruising or lid swelling or lacerations, the eyes are white and the anterior segment examination unremarkable.

The major non-ocular feature is intracranial injury. Subdural haemorrhage is the most common lesion seen. It is thought this bleeding comes from tearing of bridging veins as they cross from the brain to the dura, precipitated by very forceful acceleration and deceleration of the head.[22] In many cases, the subdural bleeding consists of a thin layer, often bilateral, which does not in itself have a mass effect on the underlying brain. It is recently suggested that, in young infants with non-traumatic subdural bleeding, the source may be the highly vascular dural plexus. It is unclear what, if any, relevance this has for the subdural bleeding seen in ITBI.[23]

A characteristic feature of ITBI is the associated brain injury. ITBI has a higher mortality, and a higher morbidity, than accidental head injuries associated with subdural bleeding.[24–30] The reason for this difference in outcome is debated, and it is likely that hypoxia plays a significant role.[31] Some papers suggest a correlation between the severity of acute neurologic injury and the severity of the RH.[32,33]

There may also be other characteristic features of child abuse such as old fractures detected on a skeletal survey, although often the injuries are restricted to the head alone. It is always important for the ophthalmologist to work in conjunction with the rest of the clinical team involved with the child. If possible, this should at least involve a paediatrician with expertise in child protection and in the differential diagnosis of inflicted head injury. Other important personnel might include a neurosurgeon and intensivist with expertise in paediatric head injuries, a paediatric radiologist and a social worker with child protection expertise.[34] In this fashion, the presence, type and distribution of the RH can be placed in the context of the other injuries present, and a collaborative decision made as to whether the nature of the described trauma is, or is not, consistent with the injuries. The RHs do not exist in isolation, but in a clinical context.

This article does not discuss rare genetic diseases (such as glutaric aciduria type 1), which may be

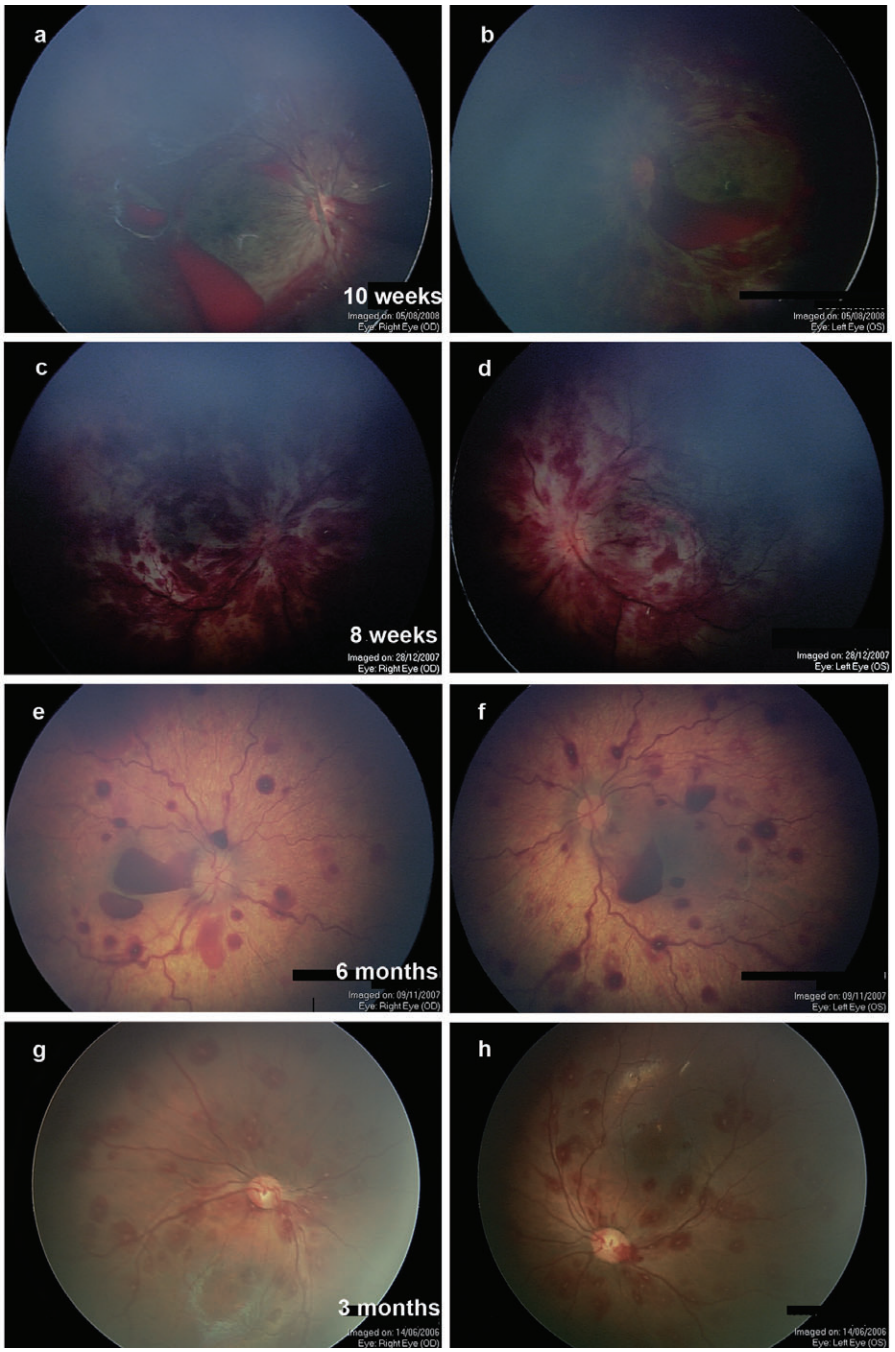

Figure 1. RetCam fundus photographs demonstrating a range of presentations of retinal haemorrhages in inflicted traumatic brain injury with the common features of multiple retinal haemorrhages affecting all layers of the retina (preretinal, intraretinal and subretinal), too numerous to count, and extending to the periphery. The images are from four infants: (a and b) infant A (right and left eyes, respectively) – a 10-week infant; (c and d) infant B (right and left eyes) – an 8-week-old infant; (e and f) infant C (right and left eyes) 6 months; (g and h) infant D (right and left eyes) from a 3-month-old infant. Infants A and B had pigmented fundi, which influences the quality of the RetCam photos. Injuries sustained by Infants B, C and D were ultimately fatal.

associated with RH,[35] or the multitude of medical disorders that may occasionally incorporate some type of RH. Many of these are reviewed elsewhere,[36,37] and most alternative diagnoses are easily distinguished from ITBI if the case is assessed by an experienced multidisciplinary team.

The examination

Use 'Universal precautions': when examining a child with suspected ITBI, treat every case as if you, as the examining ophthalmologist, will be summoned to court as an expert witness to discuss your

findings, and the interpretation of such findings. The importance of clear documentation cannot be overemphasized

1 Ideally, a detailed history as to the proposed mechanism of injury will already have been taken by the paediatric staff. You should not need to repeat this, but should summarize your understanding of the proposed mechanism of injury in the notes.
2 Examine the infant as soon as possible after admission to hospital. It may be suggested in court that the RH are not a direct effect of the head injury, but developed hours or days later. Although it may be difficult to arrange, it is best to examine the child yourself, as soon as you can.
3 If the child is awake/alert, document their apparent visual function – do they fix and follow, objection to occlusion? Is there a full range of eye movements?
4 Observe and document the presence or absence of any facial bruising, swelling, lacerations, including affecting the eyelids. Are the eyes white? Is there any subconjunctival haemorrhage?
5 Are there clear corneal reflexes? Intubated children may occasionally have an element of corneal exposure that can render fundal examination more difficult. Clear view of iris details and pupils usually excludes any anterior segment problem, such as a hyphaema.
6 The child will usually be dilated pharmacologically under the paediatrician's instructions. If the child is not adequately dilated, put more drops in the eye and wait. A good detailed examination is difficult to obtain with a small pupil. If you visualize a good red reflex and view of the posterior pole, then the ocular media are clear. If intracranial pressure monitoring is in place in intensive care, the intensivist and neurosurgeon will usually agree to pupil dilation. Occasionally, in a symptomatic infant on the ward, you may be asked not to dilate the pupils for a day or two.
7 Use an indirect ophthalmoscope, with a speculum and eye depressor if possible so as to rotate the globe. Topical anaesthetic drops should be instilled prior to insertion of the speculum. A 20-D lens is usually adequate but in a smaller infant a 28-D will give a better view.
8 Document as clearly and accurately the type, extent and distribution of haemorrhages. There is often pressure to do an expedient exam in the intensive care unit, surrounded by machines that are beeping and alarming. As well as nursing staff, there may be family members who sometimes can be hostile. Resist the urge to rush and perform a thorough documented exam. If multiple haemorrhages are present in each eye, but with distinctive morphologies and distributions between the two eyes, examine one eye, draw the retinal drawing, then move onto the other eye.

If the view was difficult or a complete view was not obtained, state so in the notes.

9 If haemorrhage is present, obtain retinal photographs. RetCam is the best modality in this situation.
10 If no photographic documentation can be obtained, consider review by a second ophthalmologist, particularly if this is outside your area of expertise. In a recent case a non-subspecialty trained ophthalmologist drew retinal drawings of a child examined in one hospital, that differed from RetCam images and retinal examination performed 2 days later at a tertiary referral centre. The defence used this point of difference to speculate that the haemorrhages must have occurred while the child was in the hospital. There is no evidence to suggest that retinal bleeding from this, or any cause will continue to expand after the inciting event (see following text), but this became a key issue for the defence.

In making an assessment of the findings in the notes, use an expansive phrase such as 'A distinctive pattern of multiple retinal haemorrhages affecting all layers of the retina, extending to the periphery (±retinoschisis) in the context of subdural haemorrhage (and other injuries if present) with no evidence of external ocular trauma, nor other contributing factors is highly suggestive of non-accidental injury.'

Use of the term 'consistent with' may be problematic. Some would suggest that the term should only be used if one also states the alternative diagnoses with which the findings may also be consistent. If, in your view of the evidence available, there is no other diagnosis that is likely to explain your findings, it is reasonable to state that clearly. One must always be ready to explain why, in your view, alternative diagnoses are not consistent with the findings you observed.

The code of conduct for expert witnesses

A key role of an expert witness is to be an impartial assistant to the court: in effect, to educate a lay audience as to the significance of the medical findings. A 'Code of Conduct For Expert Witnesses' was adopted by the High Court of New Zealand in 2002, and is identical to that adopted in many juris-

dictions internationally. According to this code, an expert witness has 'an overriding duty to assist the Court impartially on relevant matters within the expert's area of expertise' and 'an expert witness is not an advocate for the party who engages the witness'.

Although this code was developed originally for civil proceedings, it has been widely adopted, and a medical expert witness would be wise to be aware of its content, and to practice in accord with it.[38] A similar code of conduct for Australian colleagues varies between different jurisdictions. However useful guidelines can be found on the Royal Australian and New Zealand College of Radiologists website.[39]

Alternative explanations proposed in court

Retinal haemorrhages are not diagnostic of abuse and there are many different causes of such haemorrhages.

With respect to RH, it is important that the court understand that not all RH are the same, just as not all rashes are the same. From an ophthalmologist's perspective, a single dot haemorrhage at the posterior pole and multi-layer RH extending to the periphery are two different entities. If one ignores the obvious clinical distinctions, there are indeed many causes of RH. However, the role of the ophthalmologist is not to ignore the distinctions, but to explain to the court the likely explanations for this particular constellation of retinal findings in this particular patient.

It is helpful to explain to the court that RHs of different types are seen in a number of different medical conditions. However, if none of the alternative diagnoses apply in this case, and if the literature and the ophthalmologist's own clinical experience teach that it is extremely rare to see this type of RH outside the context of ITBI, it is the ophthalmologist's duty to inform the court of that fact.

It may be helpful to make a distinction between the evidence for the strong association between RH and ITBI (which is very well documented),[11,16,17,24–26,28,30,40–45] and the theories as to why that association exists. A common legal tactic is to try to generate a perception that there is confusion and controversy in the medical community, even when that controversy may not be directly relevant to the issue at hand.

There are two major theories that have evolved to explain the relationship between ITBI and RH. One theory relates to the anatomy of the infant, specifically the strong attachment of the vitreous to the retina in infancy. It is suggested that RH results from a direct mechanical effect at the vitreoretinal interface with resulting traction from (possibly repeated) acceleration–deceleration.

A second theory suggests that the RH result from an indirect effect of events elsewhere in the head or body, and may not therefore require that excessive force is applied to the retina. If this is so, it could be argued that the RH tell us nothing about the degree of trauma (and therefore the issue of child abuse), but are merely an epiphenomenon of whatever the primary event was. One version is that the RH is due to raised venous pressure in the retinal vasculature because of sudden increases in thoracic or head pressure – as suggested in Valsalva retinopathy, Terson syndrome (TS) or Purtscher retinopathy.

Retinoschisis seen in ITBI is seen in other clinical settings.

One particular distinctive feature of ITBI is the dome-shaped haemorrhage, also called haemorrhagic retinoschisis lesion and/or traumatic macular folds (Fig. 2). Other terms used to describe this lesion include haemorrhagic macular cyst, or sub-internal limiting membrane (ILM) haemorrhage, caused by separation of the ILM, or an even deeper retinoschisis.[46–48]

Some years ago, Massicotte *et al.* demonstrated persistent attachment of the vitreous to the ILM at the apices of the perimacular folds, and therefore suggested that this finding might be related to violent shaking.[49] This was supported recently by Sturm *et al.* who clearly demonstrated by OCT persistent attachment of the vitreous at the apices of the perimacular folds in macular retinoschisis, and confirmed this vitreoretinal traction to be present in schisis haemorrhages in ITBI.[50,51]

However, recently, retinoschisis has been demonstrated to occur in major crush injuries to the infant head. Three cases have been reported since 2004,[19–21,52] thereby generating an increased awareness in the ophthalmic literature. These three specific cases of crush injury involve

1. A television falling on a child's head,[19]
2. A 63 kg person falling on a 4-month-old infant[20] and
3. A mother holding a 10-week infant in a front-holding papoose, who tripped and fell forward crushing the infant's head between a wooden barrier and the mothers chest.[21] All three cases had massive head injuries with skull fractures. Two of the infants died.

To further investigate the significance and frequency of these findings, a subsequent retrospective

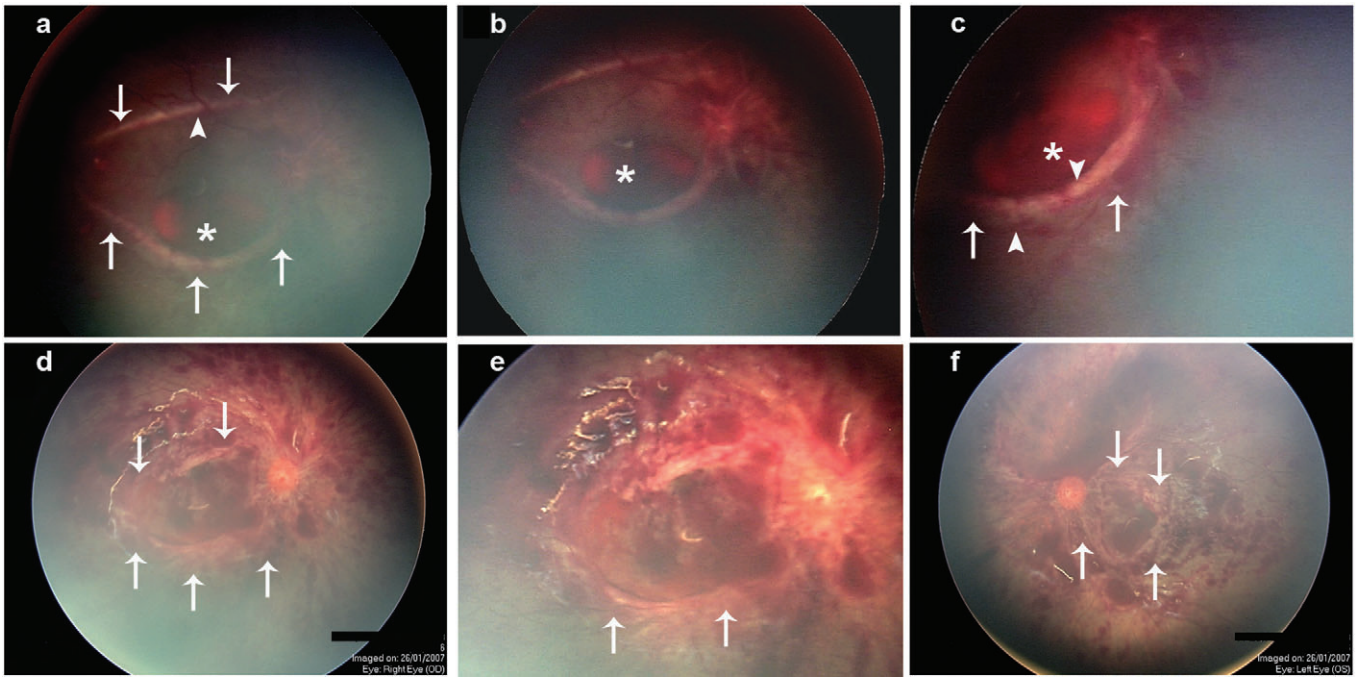

Figure 2. Traumatic retinal folds and retinoschisis haemorrhages in two infants demonstrated on RetCam fundal photographs. (a–c) Infant E, right eye; infant F, (d and e) right eye, (f) left eye. Arrows delineate the macular folds, with an arrowhead showing the undulation emphasized with a vessel lying over the fold. A dome-shaped haemorrhage (asterisk) has break through bleeding in to the preretinal space in (a–c). Haemorrhage in the macular areas in infant F also had dome-shaped configurations consistent with retinoschisis haemorrhage. The amount of haemorrhage in infant F would have rendered the child functionally blind. Both infants E and F had pigmented fundi, and the injuries sustained in infant F were fatal.

and pathological review of crush injuries in children was undertaken by Gnanaraj *et al.*[52] observing RH in crush injuries from falling televisions and nine autopsy findings with crush and skull fracture. They concluded that intraretinal and preretinal haemorrhages particularly in the posterior pole can occur in crush injury in the paediatric head. Haemorrhage under the ILM or extending to the ora serrata were only seen in situations where crush injury was part of a fatal trauma scenario related to motor vehicles. Retinal folds and the typical macular schisis associated with abusive head injury were not observed.

In sum, although retinoschisis haemorrhages have been observed in crush injuries, they are extremely rare. These cases illustrate the importance of placing the retinal findings in the context of the history given. In most cases of ITBI the mechanism proposed is that of a minor fall, often from a height of 1 m or less, without the massive force which existed in these three cases.

> The eye findings may be the result of a *Valsalva manoeuvre*, – that is, Valsalva retinopathy.

To investigate the association of retinopathy in children associated with a *Valsalva manoeuvre* Herr *et al.*[53] studied 100 children with hypertrophic pyloric stenosis, who suffered excessive vomiting with facial petechiae, subconjunctival haemorrhage and even respiratory arrest associated with vomiting. None of these children were found to have RH. Papers quoted to support Valsalva retinopathy are perforce derived from the adult literature. Valsalva is also highly unlikely to be a causative factor in many of the reported scenarios

> The intracranial bleeding and pressure changes may be the reason for the eye findings, a condition known as Terson syndrome.

The term TS was first suggested in 1900 describing a vitreous haemorrhage that occurred as a direct consequence of a subarachnoid haemorrhage (SAH). This term has evolved to include the presentation of any type of intraocular haemorrhage after spontaneous or trauma-induced intracranial haemorrhage, but usually SAH. Taking the definition as it now stands in adults, all infants with ITBI associated with RH have TS, and the term becomes meaningless. The term is only useful in infants if it provides some insight into the mechanism of RH. In adults with massive SAH (for example after a ruptured aneurysm or AV malformation), TS is observed in 27–40%,[54] and one proposed mechanism is the presumed

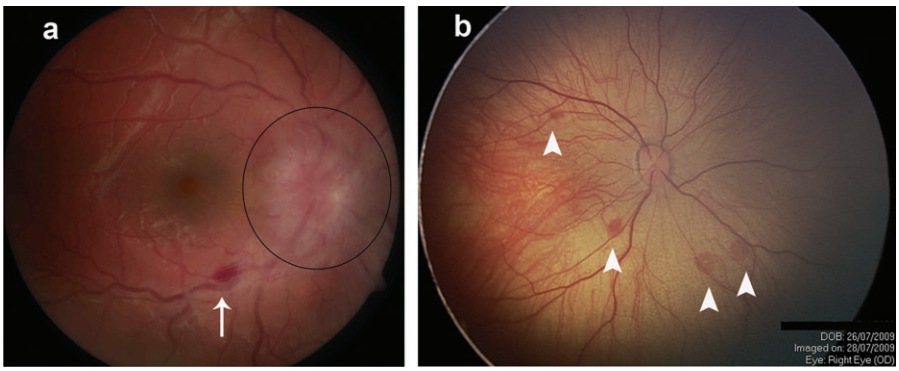

Figure 3. RetCam fundus photographs of retinal haemorrhages in cases where inflicted traumatic brain injury did not occur. (a) demonstrates the clinical picture in papilloedema with swelling of the optic nerve head and nerve fibre layer adjacent (within circle). Despite marked swelling that presumably has a compressive effect on vascular outflow, there is only one visible haemorrhage (arrow), in the nerve fibre layer, with a 'flame'-shaped configuration, and limited to the posterior pole. There are also dilated venous vessels. (b) demonstrates the appearance of an incidental finding of retinal haemorrhages in a 2-day-old infant in whom an ophthalmology consult was requested for systemic investigation of other congenital malformations. These retinal haemorrhages (arrowheads) are intraretinal and homogenous in appearance, sparse ($n = 4$, i.e. can be counted), and isolated to the posterior pole (photographs [a and b] courtesy of Dr Shuan Dai).

sudden rise in intracranial pressure, but there are a number of other suggested mechanisms. In adults, macular schisis cavities have been reported.

However, TS (in the sense of major RH of the type seen in ITBI, but caused indirectly by SAH) has not yet been reported in the paediatric literature. One prospective study described 57 consecutive children with known intracranial haemorrhage from non-abuse causes, who underwent dilated eye examination.[55] Fifty-five patients (96%) had no evidence of intraretinal or vitreous haemorrhage. Only two patients had abnormal eye examinations. One patient had a single dot haemorrhage associated with presumed infectious white retinal lesions. The second had three flame and two deeper dot intraretinal haemorrhages after a motor vehicle accident. Retinoschisis was not observed. If TS does occur in children (viz RH resulting from an indirect effect of intracranial bleeding), it would appear to be rare compared with adults. The only context in which severe multi-layer RH are routinely seen in infancy is ITBI.

> Raised intracranial pressure is the cause of the retinal haemorrhage.

Retinal haemorrhages can sometimes be seen in association with optic nerve swelling or papilloedema (Fig. 3a). However, the type and extent of RH observed with optic nerve swelling are well documented as being distinct and very different from that observed in ITBI.[56] Evidence from the literature supports this. A large series[57] demonstrated that papilloedema after acute head injury is not common, being observed in 15 of 426 patients (3.5%). The youngest patient in the series with papilloedema was 5 years old, the next youngest 9 years old. This data are therefore probably not directly applicable to children, as discussed with Valsalva retinopathy and TS.[53,55]

This paper also states that 'Papilledema was of a low grade in all patients' and that 'Retinal nerve fibre layer haemorrhages were not common and exudates were not seen'.

Papilledema has been reported in two series as occurring in approximately 5% of cases of ITBI, so it is unlikely to account for the RH seen in 83–85% of those cases.[32,47]

It may still be suggested that the RH are caused by raised intracranial pressure, even if the ophthalmologist can see no evidence of papilloedema on ocular examination. One study has tried to examine the relationship of intracranial pressure and RH and could demonstrate no relationship.[32] Given that cerebral oedema is a common pathway for an injured brain to follow, it is difficult to explain why, if raised intracranial pressure is the causative factor, severe RH is almost never seen after serious or fatal accidental trauma.[45] This issue was discussed in a legal review of cases of 'shaken baby syndrome' in the High Court in the UK in 2005. At that time, the hypothesis under discussion was the so-called 'Geddes hypothesis', which (among other things) implicated raised intracranial pressure as a possible cause for RH in ITBI.[58,59] In the High Court, it was pointed out to Dr Geddes that one of the cases had no brain swelling at all on brain scan, and still had both subdural and retinal bleeding. She conceded that she had no explanation for this, and that her hypothesis was flawed. 'As an example of why the hypothesis is not correct Dr Jaspan, giving evidence in the appeal

of Rock, demonstrated that CT scans taken of Heidi's brain showed that there was little or no brain swelling at a time when subdural haemorrhages and retinal haemorrhages were shown to be present'.[60]

> Anaemia and retinal haemorrhages often occur together.

Typical retinal findings observed in anaemia consist of occasional small dot or flame haemorrhage, but these are infrequent, and uncommon. Carraro *et al.* demonstrated in mild and moderate anaemia only 5% of patients (adults) showed RH.[61] In severe anaemia RHs were observed in 83%, and were flame-shaped in 41%, *discrete* in 64%, white centred in 3% with a subhyaloid haemorrhage observed in one patient only. Three representative photos in that paper are shown that each have less than four haemorrhages. There is very little paediatric literature on the subject.

> Convulsions caused the retinal haemorrhages.

Three studies have specifically observed the retina of children admitted to hospital following convulsions, including a total of 247 infants and children.[62–64] Two infants had RH, and both were eventually diagnosed with abuse.[62]

> Cardiopulmonary resuscitation (CPR) caused the retinal haemorrhages.

Gilliland and Luckenbach looked at post-mortem eyes of 169 children who had undergone CPR. No case was found in that study to support the hypothesis that RH are caused by resuscitation attempts.[65]

> The haemorrhages weren't seen by/were not as marked when first seen by Dr X- a paediatrician, or emergency medicine specialist, therefore the bleeding must have occurred in hospital.

A study by Morad *et al.*[66] showed that non-ophthalmologists did not attempt to (36%) or were 'unable to' (19%) examine the fundus in 72 children with SBS. When the retina was examined, non-ophthalmologists were accurate in recognizing the absence or presence of RH in 87%. However, false-negative examinations occurred in 13%. Similarly Kivlin *et al.* reported that non-ophthalmologists missed the haemorrhages in 29% of affected patients.[47]

The most likely explanation for RH not being seen on admission, but being described later by an ophthalmologist, is the experience and technique of the original examiner. It is difficult to ensure that all cases of suspected ITBI are examined within minutes or hours of admission, and no published studies have described repeated ocular examination in the first few days in hospital. All that can be definitively stated is that there is no good evidence that significant evolution of RH over time occurs. Gardner[67] published one case report suggesting this occurred, but issues with that case report make it difficult to draw a conclusion (see following text).

Theoretically there is potential that RH might extend, for example if the child had a severe coagulopathy. An extensive literature search failed to identify any reports of such an event. In anticoagulated patients on warfarin, RH was observed in 3%, none of which was visually significant.[68] Similarly although RH can occur in coagulation disorders, this is an infrequent occurrence[69] with no report of extension of an existing RH found. Extension is theoretically unlikely given the tamponade from retinal tissues and intraocular pressure, the retinal milieu contains unique clotting abilities[69] as well as the absence of abnormal vasculature or fragile neovascularization, particularly in ITBI.

> The haemorrhages are unilateral, therefore cannot be due to abuse.

Many of the large series demonstrate an incidence of unilateral RH ranging from 0% to 42%.[15,47] In a series of 123 children with non accidental injury prospectively examined, 13% were unilateral.[47]

> Retinal Haemorrhage is a consequence of head trauma in general, and is not specific to non-accidental injury.

Retinal haemorrhages are exceedingly rare in accidental head trauma, and (with a few exceptions) are usually only observed in severe life-threatening situations associated with extensive evidence of external injury, such as in a serious motor vehicle accident. There are many papers assessing RH in accidental trauma, compared with ITBI.[11,16,17,24–26,28–30,40–42,44,45,70]

It may be argued that these papers are contaminated by circular reasoning, if the authors used RH as a criterion for the diagnosis of ITBI. That is, if a study begins with a fixed view on the significance of RH, there is a risk that alternative explanations will be, a priori, excluded. It is certainly the case that in a number of retrospective papers, the retina was not examined in all cases of accidental head trauma assessed.

Aware of this concern, several large series of infants with TBI have either deliberately excluded RH as a diagnostic criterion for ITBI,[16,25,71] examined the retina in all cases regardless of cause[41,42,45] or sought to address the issue of circularity by assessing only corroborated or confessed mechanisms of injury.[11] All these papers have confirmed that RH is

rare in infants and children with accidental TBI, yet common in ITBI. A recent and very cautious meta-analysis of the literature confirmed that RH were strongly associated with ITBI, with a positive predictive value of 71% (97.5% CI 0.483 to 0.868), and an odds ratio of 3.504 (97.5% CI 1.088 to 11.280, $P = 0.03$).[44]

Overall, these papers demonstrate that based on history, most household falls are neurologically benign, that in accidental trauma, RH is not common and usually associated with major force or external injury, and that one should consider the diagnosis of child abuse when RH are present in the absence of a documented history of major trauma.

The severity of RH observed in accidental trauma is also less than in ITBI. Vinchon *et al.* classified the RH as 'Grade 1' (dot, blot or flame at the posterior pole only), present in the few accidental trauma patients with RH. Overall, RH had a 93% specificity for NAI, 66% if grade 2 (small dome- or pearl-shaped), 100% if grade 3 (large dome-shaped, to periphery and possible preretinal).[45] In his 2009 paper, Vinchon *et al.* found that severe RH had a specificity of 0.974 for child abuse and concluded that 'severe RH in the absence of facial trauma are specific of inflicted head injury'.[11]

> Retinal haemorrhages are associated with short distance falls.

This is one of the most common arguments encountered. An American Forensic Pathologist, Dr John Plunkett,[70] searched the US Consumer Product Safety Commission database retrospectively for fatal injuries related to playground equipment over a period of 11.5 years. The denominator was 75 000 head and neck injuries, with 18 deaths resulting from falls, of which 8 were under the age of 3 years. Many of the falls were complex and involved swings or jungle gyms, and several were unwitnessed. The series included the case of a toddler videotaped falling from a plastic play gym, an event resulting in her death with subdural and retinal bleeding. Three cases (including this toddler) had RH, although the RHs are poorly described. However given the number of infants who fall in daily life, RH from low falls must be an exceedingly rare occurrence.[72]

A further case attributes an 11-month infant's injuries to a fall backward from the infant's own height, reported by a 5-year-old child.[67] The possibility of benign external hydrocephalus is also raised as an explanation for the infant's subdural bleeding, although insufficient information is provided to assess that possibility. No retinal photographs are presented, only drawings of the retinal lesions. The infant was examined twice by the same examiner, and the retinal findings (as reported by Dr Gardner) appear to have changed significantly over time. It is clear however from these drawings and the accompanying description that NO retinoschisis cavity was present.

This case report[67] is annotated by the editor of *Paediatric Neurosurgery*, referring the reader to two other articles in the same issue. One article[34] warns that Dr Gardner's conclusions are speculative at best. 'The author assumes that a backward fall of an 11-month-old is in fact the mechanism that caused the injury because it is witnessed. There is no doubt that a 5-year-old could have witnessed the 11-month-old fall backwards, but the reliability of the witness must be considered before accepting that this is in fact the mechanism of the injury. . . . There are also gross inconsistencies in this case in the descriptions of the eye findings . . . The incomplete presentation of the eye findings challenges the credibility of this report.'

Another relevant paper describes a series of three children.[73] Each sustained an accidental head injury with acute subdural or SAH, and RH in the posterior pole of one eye. Each recovered fully. None had retinoschisis. This paper merely reminds us again that every RH must be put in the context of the clinical history.

> Retinal haemorrhages are commonly seen as a consequence of normal deliveries.

This is of course true, as demonstrated in Figure 3b. RH may be seen in up to 50% of births in studies published to date, usually intraretinal and restricted to the posterior pole. They are usually gone by 4 weeks of age, although Hughes described one infant where some form of RH persisted to 58 days.[74–76] The age of the infant in most cases of ITBI, mean that birth injury is not a credible explanation for the RH.

Summary

An Ophthalmological assessment of the cause of RH in an infant should not be based on the presence of haemorrhage alone, but on the entire clinical picture. Specifically it is the *combination* of the *type*, *extent* and *distribution* of the RHs, in the *context* of the history and other findings in the case, which enables a diagnosis of ITBI to be made. The ophthalmologist functions as part of a team, which reaches a collaborative diagnosis based on the best evidence available.

As the examining ophthalmologist, take care to document the nature and extent of ocular injury carefully and accurately, including retinal photographs if possible. Acknowledge the difficulty of the examination if this is the case, and acquaint yourself in detail with the literature so when called to be an

expert witness, you are as objective and as informed as possible. In court, it is your role to be fair and balanced in your discussion of the scientific evidence, and to provide a reasoned explanation for your opinion, which can be understood by a lay audience. Legitimate flaws in the scientific evidence must be acknowledged, but you are not obliged to accept alternative explanations which, in your view, are consistent neither with the literature or your own experience. Ultimately, it is the role of the court to determine the guilt or innocence of the accused. Your role is purely to form a reasonable opinion as to the aetiology of the RH, and to explain that opinion to the court.

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