The ROYAL COLLEGE of OPHTHALMOLOGISTS

**Clinical Guideline**

# Abusive Head Trauma and the Eye

January 2024

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# Contents

# Abbreviations

| Abbreviation | Description | Abbreviation | Description |
|---|---|---|---|
| AEI | Accidental eye injury | NAHI | Non accidental head injury |
| AHT | Abusive head trauma | NAI | Non accidental Injury |
| ALTE | Apparent life threatening event | ND | Named Doctor |
| APP | Amyloid precusor protein | NFL | Nerve fibre layer |
| BRUE | Brief resolved unexplained event | NICE | National Institute of Clinical Excellence |
| CAF | Critical appraisal form | NN | Named Nurse |
| CML | Classic metaphyseal lesion | OCT | Optical coherence tomography |
| CMV | Cytomegalovirus | ONSH | Optic nerve sheath haemorrhage |
| CNS | Central nervous system | PP | Perplexing presentations |
| CPR | Cardiopulmonary resuscitation | PM | Post mortem |
| CRVO | Central retinal vein occlusion | RH/s | Retinal haemorrhage/Retinal haemorrhages |
| CSF | Cerebrospinal fluid | RCT | Randomised control trial |
| CT | Computerised tomography | RCO | Royal College of Ophthalmologists |
| ECMO | Extra corporeal membrane oxygenation | RCPCH | Royal College of Paediatrics and Child Health |
| ED | Emergency departments | ROP | Retinopathy of prematurity |
| FII | Fabricated or induced illness | RPE | Retinal pigment epithelium |
| GPP | Good practice points | SAH | Sub arachnoid haemorrhage |
| GWP | Guideline working party | SCBU | Special care baby unit |
| ICP | Intracranial pressure | SCH | Sub conjunctival haemorrhage |
| MRI | Magnetic resonance imaging | SBS | Shaken baby syndrome |
| MVA | Motor vehicle accident | SIGN | Scottish intercollegiate guidelines network |
| MVC | Motor vehicle crashes/collisions | SDH | Subdural haemorrhage |

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### Conflicts of interest

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# 1. Introduction

## 1.1. Background

Child maltreatment includes neglect and abuse. Child abuse may be physical, psychological, emotional, sexual and financial. Child abuse is when a child is intentionally harmed by an adult or another child – it can be over a period of time but can also be a one-off action. It can happen in person or online. It can also be a lack of love, care and attention – this is neglect[1]. Children have a right to be protected from abuse and neglect and it is the duty of all doctors to act on any concerns they have about the safety or welfare of a child or young person[2]. It is therefore incumbent on every ophthalmologist to be familiar with the clinical presentations, clinical signs of child maltreatment and be aware of child protection investigations and procedures. In a hospital setting children and young people being investigated for child maltreatment may be referred to an ophthalmologist as part of the clinical evaluation of the child. Referrals to the ophthalmologist are predominantly for evaluation of a child for signs of physical abuse associated with abusive head trauma (AHT). Occasionally the ophthalmologist may be the first doctor to evaluate a child whose history may not be consistent with the clinical presentation as in children with a fabricated or induced illness (FII) or a pre mobile well infant who may present with a subconjunctival haemorrhage which may be a sentinel sign of abusive injury[3].

Child protection is a difficult area of practice that can involve making decisions that are emotionally challenging, complicated by uncertainty and sometimes go against the wishes of parents. Evidence based reviews of literature have been published by members of the child abuse working party of the Royal College of Ophthalmologists (RCO) in 1999 and 2004 to support ophthalmologists in practice[4, 5]. In 2013 the RCO in collaboration with the Royal College of Paediatrics and Child Health (RCPCH) produced a clinical guideline bringing these previous publications by the child abuse working party of the RCO together with updated evidence. This document included clinical signs, differential diagnosis and confounding conditions that may have overlapping signs of AHT. It provided guidance and good practice points in evaluation documentation in child protection[6, 7]. These documents also support the ophthalmologist in dealing with legal proceedings that may ensue from child protection.

This document updates the previous guideline with evidence-based literature published since 2011. A review of newborn retinal haemorrhages, non-vitreoretinal ocular manifestations of physical injury and fabricated or induced injury (FII) are included. The document follows the same structure as the previous guideline with some new clinical questions and provides a narrative of updated evidence if this exists below each section of the previous document. Though this document is primarily aimed at ophthalmologists in practice it provides an evidence-based resource for any doctor dealing with children and those practicing in child protection.

**For ophthalmologists who are called upon to evaluate a child suspected of child abuse this document provides guidance on:**

1. The ophthalmic findings, differential diagnosis and confounding conditions that may have overlapping features of AHT.
2. How to evaluate a child with suspected AHT and the methods of documentation and imaging.
3. Advice to child protection teams on the interpretation of reported signs and further investigations that may be required.
4. How to find the local procedures to follow when dealing with potential child protection issues.
5. How to provide a factual report of clinical findings and a balanced interpretation of these findings based on current literature.

## 1.2. Terminology

Abusive head trauma (AHT) will be used synonymously with previously accepted terminology of non accidental injury (NAI), non-accidental head injury (NAHI) and shaken baby syndrome (SBS) used in previous RCO publications to describe the forms of physical abuse most relevant to the ophthalmologist. Apparent life threatening events (ALTE) used in the previous guideline will be used synonymously with the current terminology of brief resolved unexplained events (BRUE).

## 1.3 Aims of the guideline

1. To critically appraise the literature since the time of the last publication.
2. To update the previously published articles.
3. To identify good practice in the management of cases referred with suspected AHT and encourage evidence based standardised assessment of such children.
4. To identify new information concerning conditions which may simulate the ocular findings in abusive head trauma in children.

## 1.4 Scope of guideline

This guideline deals with the new literature in the field of AHT to supplement and update the previous guideline published by the RCO and the RCPCH. The previous guideline asked a series of questions based on the published literature and provided a synopsis of the literature for each question at the time. This document will include an update of the answers to questions asked in the previous publications and additional questions that a review of the current literature identifies.

The guidance will be presented as 4 chapters:

i. Aetiological factors and experimental models.

ii. Clinical features and pathology.

iii. Differential diagnosis and confounding conditions.

iv. Guidance for the ophthalmologist.

# 2. Methods

## 2.1. Clinical Questions

The guideline working party (GWP) consisted of paediatric ophthalmologists, a histopathologist, a neurosurgeon and a paediatrician who represented the RCPCH. This group of professionals are directly involved in the care of, and investigations of children with suspected abusive head trauma, are involved in current research in child protection or in the writing of medical reports and appearing as expert witnesses in Court in cases of abusive head trauma in children. Input from the College lay advisory group was sought.

For the purpose of literature searches and appraisal the following nine header clinical questions with sub questions were developed to accommodate the previous questions under nine areas. This included new questions on newborn retinal haemorrhages non-vitreoretinal ocular injury and FII and neglect.

1. **What differences are found between abusive head trauma retinal findings versus non-abusive head trauma retinal findings?**
   - (i) Are retinal haemorrhages more common in infants than older children?
   - (ii) What is the site and extent of retinal haemorrhages in child abuse?
   - (iii) Are any findings pathognomonic of abusive head trauma?
   - (iv) Are unilateral haemorrhages compatible with abusive head trauma?
   - (v) Are there any retinal findings without radiological evidence of intracranial injury?
   - (vi) Can accidental injury cause retinal haemorrhage?
   - (vii) Can short falls cause retinal haemorrhages?

2. **What are the differential diagnoses of retinal haemorrhages in children with clinical features associated with child abuse?**
   - (i) What other conditions in childhood may have retinal haemorrhages?
   - (ii) Can a bleeding diathesis or blood dyscrasia cause retinal haemorrhages similar to those seen in abusive head trauma?
   - (iii) Can seizures alone cause retinal haemorrhages as seen in abusive head trauma?
   - (iv) Can cardiopulmonary resuscitation cause retinal haemorrhages?
   - (v) Can prolonged vomiting or gagging cause retinal haemorrhages?
   - (vi) Is an apparent life-threatening event (ALTE) or brief resolved unexplained event (BRUE) associated with retinal haemorrhages?
   - (vii) Are vaccinations associated with retinal haemorrhages?
   - (viii) Do high cervical injuries cause retinal haemorrhages?
   - (ix) Do retinal haemorrhages occur with raised intracranial pressure?
   - (x) What are the ocular findings in ocular crush injury?

3. **What are the features of retinal haemorrhages in newborn infants?**
   (i) What are the retinal findings in newborn infants?
   (ii) What are the obstetric correlates to retinal haemorrhages in the newborn?
   (iii) What is the evolution of newborn retinal haemorrhages?
   (iv) Are new born retinal haemorrhages associated with intracranial bleeding? (abuse excluded)

4. **Which features or characteristics of non-vitreoretinal eye injury are present in child maltreatment, neglect and fabricated or induced illness?**
   (i) What are the non-vitreoretinal ocular and ocular adnexal injuries seen in (a) Abuse (b) Fabricated or induced injury and (c) Neglect?

5. **Can retinal haemorrhages be dated?**
   (i) Can intraocular haemorrhages increase after injury?
   (ii) Is it possible from examining the retina to estimate the time at which the injury occurred or whether they have occurred at more than one time?

6. **What are the postulated mechanisms of retinal haemorrhages in abusive head trauma?**
   (i) What forces are necessary to produce retinal haemorrhages?
   (ii) Can retinal haemorrhages and intracranial haemorrhages occur with vigorous play?
   (iii) Does hypoxia cause retinal haemorrhages?
   (iv) What are the animal, biomechanical and computer models of ocular findings in abusive head trauma?

7. **What is the ophthalmic/ocular pathology seen in abusive head trauma?**
   (i) What is the orbital and optic nerve pathology?
   (ii) What is the vitreous/ retinal pathology?

8. **Which methods of eye examinations and imaging of the retina are useful in abusive head trauma?**
   (i) Indirect ophthalmoscope?
   (ii) Retcam or other?
   (iii) Non mydriatic camera?
   (iv) Optos?
   (v) Optical coherence tomography?
   (vi) Neuro imaging?

9. **What are the methods used to document / record eye findings?**
   (i) What are the standardised methods of recording eye findings?

## 2.2. Gathering evidence

### 2.2.1. Search strategy

A search strategy was developed based on the clinical questions with the support of an information specialist. The search was aligned to the main clinical questions and the text words were chosen from the previous guideline search with additional text words selected for the new questions and those that did not appear in the previous guideline list. The search strategy depended on the database chosen and the strategy for Medline ALL is provided as an example with text words, the strategy for other databases can be provided by the chairman of the GWP on request. (Appendix 1)

### 2.2.2. Databases included

CINAHL (*Cumulative Index to Nursing and Allied Health Literature*), Cochrane Library, EMBASE, MEDLINE ALL; Scopus, Web of Science Core Collection: Citation Indexes, Science Citation Index Expanded (SCI-EXPANDED), Social Sciences Citation Index (SSCI), Conference Proceedings Citation Index- Science (CPCIS), Conference Proceedings Citation Index- Social Science & Humanities (CPCI-SSH).

### 2.2.3. Search results and data extraction

A total of 20,338 titles and abstracts were identified for the period 2011 and 2021 for questions included in the previous guideline and for the period 1970 to 2021 for the new questions (see flow diagram based on PRISMA appendix 2). After screening for relevancy 159 abstracts were selected for full paper review of which 44 were excluded. Critical appraisal of 115 studies using a critical appraisal form (appendix 3) (CAF) selected 70 studies to be included to inform the update of the guideline. Each paper was reviewed by two appraisers.

### 2.2.4. Levels of evidence and grades of recommendations

Though this work will be graded according to an adapted Scottish intercollegiate guidelines network (SIGN) (see Table 1) grades of recommendation will be used based on the table below. In addition a further grade 'E' was added for experimental studies. Expert opinion was excluded in the update given the polarized views that some experts express in this field. 'Good practice points' are used in the guidance for the ophthalmologist section.

Table 1: Levels of evidence and grade of recommendations[8]

| Levels of Evidence | |
|---|---|
| 1++ | High quality meta-analyses, systematic reviews of RCTs, or RCTs with a very low risk of bias |
| 1+ | Well-conducted meta-analyses, systematic reviews, or RCTs with a low risk of bias |
| 1- | Meta-analyses, systematic reviews, or RCTs with a high risk of bias |
| 2++ | High quality systematic reviews of case control or cohort or studies. High quality case control or cohort studies with a very low risk of confounding or bias and a high probability that the relationship is causal |
| 2+ | Well-conducted case control or cohort studies with a low risk of confounding or bias and a moderate probability that the relationship is causal |
| 2- | Case control or cohort studies with a high risk of confounding or bias and a significant risk that the relationship is not causal |
| 3 | Non-analytic studies, e.g. case reports, case series |
| 4 | Expert opinion |
| **Grade of Recommendation** | |
| A | At least one meta-analysis, systematic review, or RCT rated as 1++, and directly applicable to the target population; or A body of evidence consisting principally of studies rated as 1+, directly applicable to the target population, and demonstrating overall consistency of results |
| B | A body of evidence including studies rated as 2++, directly applicable to the target population, and demonstrating overall consistency of results; or Extrapolated evidence from studies rated as 1++ or 1+ |
| C | A body of evidence including studies rated as 2+, directly applicable to the target population and demonstrating overall consistency of results; or Extrapolated evidence from studies rated as 2++ |
| D | Evidence level 3 or 4; or Extrapolated evidence from studies rated as 2+ |
| E | Experimental studies |
| GPP | Good practice points recommended by the GWP |

### 2.2.5. External peer review

1. Members of Child maltreatment working party of the Royal College of Ophthalmologists.
2. Members of the Paediatric subcommittee of the Royal College of Ophthalmologists.
3. Members of the Royal College of Ophthalmologists Scientific committee.
4. Members of the Royal College of Ophthalmologists.
5. Members of the Royal College of Paediatrics and Child Health.
6. Lay advisory group of the Royal College of Ophthalmologists.

### 2.2.6. Date for Review of Document

This document will be reviewed for an update in 2028.

### 2.2.7. Facilitation and Funding

This document was commissioned by the RCO. The paediatric subcommittee of the RCO elected the chairman. The guideline working party (GWP) was recruited from ophthalmologists by an open invitation through on online forum of paediatric ophthalmologists, the histopathologist was invited by the chairman based on recent chapter written on AHT. The paediatric neurosurgeon was invited by the chairman from the regional area, The child protection paediatrician was recommended by the RCPCH. The work carried out by the information specialist was funded by the RCO. The RCO facilitated the online meetings of the GWP. The Chairman worked with the GWP, the reviewers and those undertaking the critical appraisals electronically. There was no external funding for the time, expertise and commitment of members of the GWP, the reviewers and appraisers who completed tasks designated to them by the chairman within stipulated timelines. The members of the GWP and the reviewers have no financial interest in the publication of this guideline.

# 3. Clinical Questions and Evidence

## 3.1. Aetiological factors and experimental models

### 3.1.1. Forces

**Clinical question: What forces are needed to produce retinal haemorrhages and other signs of intraocular trauma in infants without direct ocular injury?**

**Evidence from previous reviews 1999 and 2012**
Retinal injury can be produced by positive or negative acceleration without impact or direct ocular injury. It has been suggested that this retinal injury may be due to vitreous traction[9]. Evidence for retinal haemorrhages caused by angular acceleration without impact comes from adults exposed to emergency aircraft ejection[10], children involved in road traffic accidents without direct trauma to the eye[11], bungee jumping[12,13,14] and from perpetrator confessions[15]. Indirect evidence of the forces involved in the production of retinal injury comes from extrapolating data of cerebral and eye injury[16,17] from animal experiments[18], infant models[19] and the observation that not all abused infants with retinal haemorrhages have signs of impact[20,21]. Retinal injury from accidental trauma is very rare and is predominantly seen in severe injury[15,22]. Tangential injury causes more brain deformation, and shear may explain the propensity for retinal injury with a shaking injury[15]. Retinal haemorrhages are not reported in household falls even with skull fractures and are only seen in severe motor vehicle accidents[23]. Additional retinal findings like perimacular retinal folds, sub retinal, choroidal and vitreous haemorrhage may indicate application of greater severity of forces[9,17].There are no absolute values for the angular acceleration, forces required to produce retinal haemorrhages, but there is evidence that this must be considerable.

The forces involved in producing retinal haemorrhages in abusive head trauma are not known. Information on the forces involved is extrapolated from observational, experimental studies using computational and animal models. Please see sections 3.1.2, 3.1.3, 3.1.4 and 3.1.6.

**Evidence from review update 2022**
Computer eye modelling has shown higher pressure and strain in shaking simulations using dummies representative of a 6 week old baby than in fall simulations[24]. The authors reported that the softest shaking led to pressure in the retina that was 4 times higher than the most severe impact. The authors reported it was the rotational eye movement that was the key factor to induce injury. A further study by the same authors reported that their work supported the mechanism of vitreous traction on the retina but they noted that the thresholds that they used had been found after animal experiments to study retinal detachment so could not be directly compared to retinal haemorrhage[25].

A further computer modelling study has shown that in simulated shaking the greatest stress was along the blood vessels and their bifurcations, with maximum stress at the macula, the peripapillary area and at the ora serrata. The authors found that shaking the eye at a frequency as low as 2.2 cycles per second could produce stress levels of 7-10 kPa along the retinal vessels. Using work from adult vitreo adhesion studies and extrapolating the results the authors suggested that a pressure of 7kPa was required to produce vitreo retinal peeling. It was suggested that shaking even at a modest frequency could exert pressure forces on the retina causing splitting of the retinal layers or detachment of the vitreous which could result in retinal haemorrhage[26].

**Conclusions:** **D,E**

Studies using finite element models of simulated infants report on the suggested location in the retina, the amount of force and the type of rotational forces involved in AHT. The limitations of computer models in extrapolating the forces involved in producing retinal injury in infants subjected to abusive injury is acknowledged. While the actual forces involved in human subjects is not known, this update suggests that the forces are considerable, associated with rotation injury and located at the vitreoretinal interface along blood vessels and the macula.

### 3.1.2. Impact

**Clinical question: Is additional impact (in addition to shaking injury) necessary for the production of very severe ocular injury?**

**Evidence from previous review 1999 and 2012**
Direct impact (blunt trauma) is likely to be involved in the most severe cases of abusive retinal haemorrhages as experimental models suggest direct impact involves greater deceleration forces than does shaking[15,19]. An autopsy series of 10 cases where children had died from suspected inflicted injury described signs of head injury where signs of impact where sometimes occult[27]. Other studies have described severe eye injuries with and without evidence of direct impact[9,11,20,21,28].

There were no studies found which were designed explicitly to address the question of the nature of retinal injuries seen in cases of shaking alone as compared with cases of shaking plus direct impact. A biomechanical analysis of animal and neonatal cadaver models has suggested that the forces necessary for shaking to cause retinal haemorrhages would also lead to substantial cervical spine injuries. As neck injuries are uncommonly described in AHT, the authors suggest a re-evaluation of the relative significance of impact and shaking as mechanisms of injury[29].

Severe retinal haemorrhages are described after some, but not all crush injuries to the head. They were reported in 6 of 10 children aged < 3 years who died with crush injuries to the head[30]. In an autopsy study one of the 16 children who died after a television fell on their head had retinal haemorrhages (to mid-equator) and 1/ 169 with crush injuries to the head had haemorrhages extending to the ora; this child had been unrestrained in a car accident[31].

In five cases where a perpetrator had confessed to shaking and causing a direct impact, retinal haemorrhages were seen bilaterally with no description of severity in three infants who had retinal examination as compared with in 17/24 where the perpetrator did not confess to causing impact, although 2 of these cases did have evidence of impact nonetheless[32]. Severe haemorrhages were seen in 10/110 children referred for possible AHT and all were found to have definite evidence of inflicted injury, on the basis of evidence of impact to skull, or other bony fractures and no relevant history[33].

However severe haemorrhages are also reported where there is no evidence of impact. In 13/16 (81%) cases of admitted AHT severe haemorrhages were seen, whilst only 9/24 (38%) of these had evidence of impact[34], and similarly in another study severe haemorrhages were present in 25/45 (57%) cases of cases of admitted AHT, whilst only 10/45 (22%) of these had evidence of impact[35]. No correlation was found between the number of retinal haemorrhages and evidence of direct impact, in 75 children with confirmed abusive head injuries[36].

A systematic review concluded that whilst it is difficult to confirm the relative contributions of shaking and direct impact to the presentation of retinal haemorrhages, the full spectrum of ocular injury can occur without evidence of direct impact[37].

**Conclusions:** **B,C,D**

In addition to a shaking injury, impact may contribute to the severity of retinal damage but is not mandatory for its production. In experimental research there remain uncertainties regarding the likely mechanical events leading to particular patterns of injury. Severe retinal haemorrhages occur in some crush and head impact injuries but may be seen without any associated evidence of direct impact to the head.

**Evidence from review update 2022**
No additional studies were included in this update

### 3.1.3. Vigorous handling and play

**Clinical question: Can normal handling (such as vigorous play) cause retinal and intracranial haemorrhages?**

**Evidence from previous review 2004 and 2012**
In 2001, Geddes[38,39] reported the results of histopathological examination of the central nervous system (CNS) following fatal head injuries in 37 infants and 16 children. These studies have been interpreted by sections of the press[40] and some experts as suggesting that minor trauma, such as might occur during rough play, could cause the clinical picture commonly attributed to the Shaken Baby Syndrome (SBS). These studies[38,39] showed that, in infants with abusive head trauma, there was evidence of hypoxic/ ischaemic neuronal damage rather than the diffuse axonal changes associated with traumatic brain injury. This concept is supported by neuroimaging[41,42]. In 13 of 37 infants reported by Geddes[39] there was axonal damage at the cranio-cervical junction. It was suggested that this injury to the respiratory centres had a role in producing hypoxia and brain damage. These studies raise the possibility of alternative explanations for the observed pathology. While previously, it was assumed that the forces used must be sufficient to cause shearing injury, it is now apparent that shearing is not the sole pathogenesis of the changes seen in the brain (these reports specifically excluded discussion of the aetiology of retinal haemorrhages). Although the precise level of force required to cause retinal haemorrhages remains uncertain, the majority of these children were unequivocally the victims of severe trauma. Despite having access to full documentation in 52 cases, the authors did not cite a case in which they could link a less than violent event with fatal head injury. Nonetheless, in eight infants there was no bruising, skull fracture, or extra cranial injury to specifically indicate a violent event. It is now appreciated that evidence from mechanical models[19] may reveal an imperfect picture of the events occurring during injury[43]. It has been conceded that, 'whilst controversy still exists as to the exact mechanism, most authors now agree that the forces necessary to cause this type of injury are far from trivial and, in fact, are considerable' and 'that this sort of injury is unlikely to be inflicted 'accidentally' by well-meaning carers who do not know that their behaviour can be injurious'[44].

It is highly unlikely that the forces required to produce retinal haemorrhage in a child less than two years of age would be generated by a reasonable person during the course of (even rough) play or an attempt to arouse a sleeping or apparently unconscious child.

Some case studies are pertinent to this question but many do not give sufficient evidence of exclusion of abuse[45-50] or do not have ophthalmological input into all cases[51]. On the basis of a few case series[15, 52] and case studies[53], there is anecdotal evidence that retinal haemorrhages can occur in healthy children less than two years old during handling accidents and accidents during play. It is to be noted however that in all these studies where retinal haemorrhages have been reported in association with the accidental injury significant head injury coexists. These retinal haemorrhages are likely to be few, small and unilateral.

More severe haemorrhages in these circumstances should initiate concerns of there being an underlying medical condition or the possibility of prior non-accidental injury even in fully independently witnessed accidents.

**Conclusions:** **C,D,E**

There is no published evidence of retinal haemorrhages occurring as a result of normal handling or vigorous play with infants or children.

**Evidence from review update 2022**
No additional studies were included in this update

### 3.1.4. Mechanisms

**Clinical question: What are the postulated mechanisms of retinal haemorrhages in abusive and non-abusive head trauma?**

**Evidence from previous reviews 1999 and 2012**
Non-abusive or accidental head trauma is in general due to impact injury, rather than shaking injury. Retinal haemorrhages are not associated with witnessed accidental trauma unless the injury forces are very severe[11,15,22,23]. Terson's syndrome (retinal haemorrhage associated with intracranial haemorrhage) is rare following accidental head trauma (or neurosurgery) in infants and young children[54].

The mechanisms of retinal haemorrhage that occur in abusive head trauma (AHT) are thought to be:

1. Direct vitreous traction on the retina[9], leading to angular positive and negative acceleration forces.
2. Sudden rise in intracranial pressure.
3. Sudden rise in central venous pressure.

AHT may be due to impact injury, repetitive flexion / extension shaking injury, or a combination of both. It should be noted that within the shaking injury of repetitive flexion and extension the infant's head might impact their chest and or back at the extremes of movement. The deceleration produced by impact is much greater than that produced by shaking flexion / extension of the head[15,19]. Signs of impact head injury is usually[11,27], but not always[9,20,21,28] found when retinal haemorrhages and cerebral damage are present.

Evidence that angular acceleration without impact can produce retinal haemorrhages includes:

1. Adults who have sustained emergency ejection forces, and in deceleration experiments[10].
2. Road traffic accidents in children[11].
3. Bungee jumpers[12,14].
4. Confessed shaking injuries[11,15].
5. Not all infants with retinal haemorrhages associated with abuse have signs of impact injury[9, 11,20,21].

Experimental information about forces necessary to produce brain injury is available. As the severity of brain injury and eye injury correlate, it has been argued that brain injury experiments may be extrapolated to eye injury. These include:

1. Whiplash injury experiments in monkeys[18].
2. Experiments in infant models[11,19].
3. Tangential acceleration is associated with much more brain deformation and shear than an equivalent linear acceleration. This may account for the relationship between retinal haemorrhages and shaking injury in infants[15].
4. Severe eye injuries are likely to be associated with the severe level of forces that cause severe brain injuries[9,17].

Speculation that hypoxia might cause retinal haemorrhages in the context of cervical injury-related apnoea, is not supported by evidence[55].

An understanding of the mechanisms of retinal haemorrhages (RHs) in AHT provides clinical insights into the condition, and enables the critical evaluation of alternative explanations of injury volunteered by those present at the time of the injury. As direct experimentation is not possible, much of the evidence for the mechanisms of RHs in AHT is both indirect, and incomplete.

RHs occur because of loss of integrity of blood vessel walls. In trauma, mechanical rupture of blood vessel walls may occur because of external traction, compression, and shearing forces transmitted by the vitreous, or by internal forces of increased intravascular pressure, which may occur due to transient acceleration effects or be secondary to transient or sustained raised intracranial pressure. The mechanical strength of retinal blood vessel walls may be low in infants, however the mechanical properties of human infant ocular tissues are not known.

While both internal and external forces on the blood vessel walls probably contribute to the occurrence of RHs in AHT, there are pointers towards the dominant factor in particular situations. For instance, the widespread, severe RHs occasionally seen in severe crush injuries of the head[31,56] are likely to be due to transient very high intravascular pressure effects. In contrast, sustained periods of raised intracranial pressure and intrathoracic pressure have not been associated with the severity of RHs[36]. In most circumstances it is not possible to differentiate the effects of transient external forces on retinal blood vessel walls and transient elevation of intravascular pressure.

Case series of AHT in which the perpetrator has confessed and described the abuse have shown that violent shaking, with or without head impact, has occurred[32,57,58]. The onset of neurological signs is immediate, or occurs soon after the incident[32,57,58]. RH have been found in almost all cases, and in most cases have been numerous and bilateral[32,35,57,59].

A post mortem case series of 10 cases of severe road traffic accidents showed severe RHs in most cases[30]. Retinoschisis and retinal folds were described in association with numerous retinal haemorrhages in a case of accidental severe crush injury of the head[56]. However, in a series of 27 cases of fatal and non- fatal head crush injury, while retinal haemorrhages were found in some cases, there were no cases of retinoschisis or retinal folds[31]. In a case series of 75 cases of abusive head trauma the severity of retinal haemorrhages correlated with the severity of head trauma[36]. However, the severity of RHs did not correlate with periods of raised intracranial or intrathoracic pressure, head impact trauma, or the side of brain / retina injury[36]. Conversely, in a series of 154 cases assessed following a fall, only three cases were found to have RHs. All 3 cases had unilateral RHs, and epidural haemorrhage was present in all 3 cases[52]. In general, it would appear to be the case that severe RHs may occur in association with severe head injury, either due to inflicted shaking with or without impact, or due to severe (often fatal) accidental high velocity or crush injuries.

Attempts have been made to measure the forces that occur in various types of injury. Animal studies have added little information to clinical accounts. Intraocular haemorrhages, including peripheral RHs, have been induced by rapid head rotation in neonatal pigs[60]. However, the pattern of haemorrhages was different from that seen in human abusive head trauma cases. Retinal haemorrhages have also been produced in mice shaken in brain injury studies, but no description of the pattern of retinal haemorrhages was given[61]. Retinal haemorrhages have not been produced in other animal studies of shaking induced brain injury[62,63].

Mechanical experiments and mathematical models have also added only limited additional understanding to date. A case of the violent use of a baby rocker chair was found in a re-creation engineering experiment to produce greater forces than those in shaking injury[64], however a similar experiment did not confirm this[46]. A number of theoretical models of the mechanics of ocular trauma in shaking injury have been developed[65-67]. However, as the mechanical properties of the tissues of human infant eyes is not known, it is difficult to be clear as to how well these models relate to clinical situations[65-67]. The studies may provide some insights into the patterns of injury seen. The possible significance of the tethering effect of the optic nerve in shaking injury has been considered[66]. Modelling of stresses at the retinal surface has indicated that maximum stress may occur at the posterior pole, and at the retinal periphery[67]. Engineering calculations of shaking and impact head injury based on available mechanical and animal models have been developed[43]. It has been pointed out that forces required to produce brain injury is inversely proportional to its mass – brain injury will occur with lower levels of force than eye injury[43].

Models of the mechanics of head trauma in adults have also been developed[68], but once again it is difficult to relate these to clinical situations[69]. Test dummy experiments showed that shaking with impact produces much greater acceleration /deceleration forces than either impact from a short fall, or shaking alone[68]. Test dummy experiments of short falls demonstrated a low risk of significant head (or limb) injury[70]. However, the use of head injury models using test dummies to assess infant shaking / impact injuries have been criticised as not being biofidelic by engineers, who propose using theoretical modelling[71], and alternative models using "Head Injury Criterion" have been proposed[71]. Similarly, engineering calculations of the forces experienced in the neck of shaken infants found that severe cervical spine injury would be expected to occur in the context of the magnitude of forces assumed to occur with head shaking in an infant[29]. There is therefore a discrepancy between the mechanical assumptions and calculations derived from these, and clinical experience. Engineering modelling of floor surfaces have shown that wide variation in the degree of injury will occur, dependent on the mechanical properties of the surface that the head impacts[72].

Possible mechanisms for RHs in abusive head trauma include: external forces on blood vessel walls (shaking or impact); internal forces of raised intravascular pressure (pulsed or maintained); biomechanical properties of blood vessel walls though this is not known and abnormalities of the blood (in various pathologies).

**Conclusions:** **C,D,E**

Evidence is indirect, and the mechanisms may well be mixed in most cases. There are some pointers towards the likely predominant mechanism in certain circumstances: external forces; confessed shaking injury, with / without impact is very often associated with numerous, bilateral RHs; Shaking may indicate potential vitreous traction with external forces, and additional internal forces. In accidental impact trauma RHs are only associated with severe, high velocity injuries. Head crush injury (abrupt severe elevation intravascular pressure) can produce severe RHs, however the severity of RHs does not correlate with periods of raised intracranial or intrathoracic pressure.

**Evidence from review update 2022**
No additional studies were included in this update;

### 3.1.5. Hypoxia

**Clinical question: Does hypoxia give rise to the clinical picture of AHT?**

**Evidence from previous reviews 2004 & 2012**
The suggested consequences of axonal injury at the cranio-cervical junction are apnoea followed by hypoxia. However, in the absence of circulatory collapse or the vascular changes associated with high altitude[73,] acute hypoxia alone is insufficient to cause subdural and retinal bleeding. Similarly, acute hypoxia similar to that which occurs in the lungs, heart, and occasionally other viscera, has not been shown to cause retinal haemorrhages[55].

It has been suggested that intradural and juxtadural bleeding in children dying from non-traumatic, hypoxic conditions provides an explanation for the subdural bleeding in SBS[74]. In all, 72% of cases had intradural and juxtadural bleeding histologically identical to that found in three cases of SBS. However, these children suffered 'profound' hypoxia before birth for various reasons including bronchopneumonia and placental insufficiency. There is no indication in this study that apnoea from localised axonal damage can produce a similar picture. The authors hypothesise a sequence of events where severe hypoxia leads to brain swelling, raised intracranial pressure, subsequent subdural, and retinal haemorrhage this study however did not examine eyes.

Acute hypoxia resulting from transient apnoea has not been shown to result in the SBS picture. Hypoxia coupled with circulatory collapse may produce potentially fatal brain swelling.

In a postmortem study of premature babies and neonates intradural and subdural haemorrhages have been demonstrated in the setting of hypoxic ischaemic encephalopathy in the neonatal and perinatal period, but examination of the eyes was excluded and hence it is not known if retinal haemorrhages were present or that these postmortem findings could have any relevance to babies who survive with hypoxic ischaemic encephalopathy[75]."Dysphagic choking, apparent life threatening event (ALTE)" has been proposed as a mimic of non- accidental injury in a 4 month old infant where intracranial and bilateral retinal haemorrhages associated with disseminated intravascular coagulation were noted. The brain on autopsy demonstrated hypoxic changes however abusive injury could not be ruled out[76].

There are no clinical or experimental studies which directly address the question of hypoxia as a cause of retinal haemorrhage, but clinical studies in cases of ALTE reveal no retinal haemorrhage in 292 cases[77-79] and post-mortem ocular endoscopy has revealed that in a single case of sudden infant death syndrome (SIDS) there was no evidence of any retinal pathology[80]. One hundred cases where severe coughing led to hospitalisation revealed no instance of retinal haemorrhage[81] and 0/100 cases of intractable vomiting due to pyloric stenosis showed retinal haemorrhage, including one case in which there was respiratory arrest[82].

Local hypoxic damage of the retina has been associated with preretinal and vitreous haemorrhage where peripheral retinal non-perfusion in eyes of infants at variable time intervals following abusive head trauma have been reported[83,84] and may result in the later development of preretinal fibrovascular proliferation and tractional retinal detachment[83,85].

**Conclusions:** **D**

Hypoxia resulting from apnoea or associated with choking or respiratory arrest during vomiting has not been shown to cause the clinical picture of AHT. Retinal hypoperfusion ischemia as a result of non-accidental injury may lead to the later development of preretinal fibro vascular proliferation and tractional retinal detachment.

**Evidence from review update 2022**
No additional studies were included in this update.

### 3.1.6. Computational and biomechanical models of ocular injury in AHT

**Clinical Question: What are the current computer and biomechanical models of AHT?**

**Evidence from previous reviews 1999& 2012**
Biomechanical models do not provide a satisfactory explanation for the events during ocular injury[19,43].

There is no single biofidelic 3D model available, which takes into account all the biological features of an infant head, especially with regards to the presence of a fontanelle, nature of CSF, biological characteristics of all the tissue components and neck properties[68,86]. In addition there are no biofidelic infant eye models mimicking characteristics of vitreous or sclera[67]. A range of values of physical parameters such as angular acceleration, angular velocity, peak 'g' force have been derived from mathematical models, accident reconstruction, adult volunteer and cadaveric studies. Tolerance limits derived from adult or subhuman primate studies and scaled down for assessing injury in AHT may not be valid in human paediatric population[46]. To date, there is no biomechanical model which can be applied specifically to quantitatively assess head injury tolerance levels in the paediatric population[46]. Small parametric variations (type of neck, head-neck insertion and chest and back padding) in Duhaime's original infant model[19] allowed for significant increase in the value of head angular acceleration. This makes Duhaime's original suggestion that shaking alone may not cause fatal injury doubtful[86]. Anthropomorphic and rigid body models (not biofidelic) suggest that shaking alone cannot produce the head accelerations necessary to cause the brain injuries associated with SBS[87].

Finite element model studies suggest that shear stress forces between the eye and the intra orbital fat and the orbital bone oscillation and the forces produced at the posterior pole of the retina may be the mechanism for injury[66,67,88,89]. In cases with no evidence of impact, end point impact, of the chin to chest and the occiput to the back during shaking can exceed the injury tolerance limits[72].

A perfect biofidelic model of an infant does not exist and hence forces that are required to produce ocular injury calculated on current models will be imprecise. Data from recent computational models using finite element analysis suggest that shear forces are exerted maximally at the posterior retina. The precise force required to produce the ocular features of abusive head trauma is not known.

**Evidence from update 2022**
Computer modelling has been used to review the effects on the eye of both shaking and falls. One finite element eye model system found that falls produced a higher linear acceleration and a lower rotational acceleration compared to shaking cases where the rotational acceleration was high[24,25]. Another study found that the same stress was experienced by the preretinal, intraretinal and subretinal layers. The authors reported that if the force generated during shaking was greater than the force needed to maintain vitreoretinal adhesion it could lead to retinal and vitreous separation and the formation of retinal haemorrhages. Their model predicted the stress patterns consistent with diffuse retinal haemorrhages typically found in the posterior pole and around the peripheral retina in abusive head trauma[26].

**Conclusions:** **E**

Computer modelling has been used to examine the effects of shaking and falls on the eye. These are helpful in understanding the mechanisms producing retinal bleeding.

### 3.1.7. Animal models

**Clinical Question: Are there any suitable animal models of AHT?**

**Evidence from previous review 2012**

There are relatively few experimental studies that inform our understanding of inflicted injury and the formation of retinal and optic nerve sheath haemorrhages in man. Intraocular haemorrhages, including peripheral retinal haemorrhages, have been induced by rapid head rotation in neonatal pigs[60]. However, the pattern of haemorrhages was different from that seen in human abusive head trauma cases. Retinal haemorrhages have also been produced in mice shaken in brain injury studies, but no description of the pattern of retinal haemorrhages was given[61]. Retinal haemorrhages have not been produced in other animal studies of shaking induced brain injury[62,63]. In six piglets, 50 minutes of conventional, closed chest cardiopulmonary resuscitation gave rise to no retinal haemorrhages[90]. Animal studies give various potential insights into the mechanism of the formation of retinal haemorrhages. It is well – known that retinal haemorrhages can form in cases of meningitis and this does not appear to be entirely the result of altered coagulation. One study has shown that experimental meningitis is associated with loss of autoregulation of retinal blood flow[91] raising the possibility of this being a contributory factor in the genesis of retinal haemorrhages. When fluid is rapidly infused into the cisterna magna, optic nerve sheath and retinal haemorrhages have been induced in a non-human primate but not in the dog[92]. It has been suggested that the wood-pecker's resistance to retinal haemorrhages despite substantial rotational forces being applied to the head, is a function of lack of vitreous attachment, combined with rigidity of the sclera and limited capacity for ocular movement within the orbit[93]. There is an experimental study of some value to histopathologists examining eyes post-mortem (PM). In a study of 20 eyes of rhesus macaques with experimental central retinal vein occlusion it was concluded that it takes about two days for haemosiderin to be detectable and that in some but not all eyes, iron deposition can be found many months later[94].

**Conclusions:** **E**

There are no satisfactory primate or non-primate models reported that replicate retinal injury seen in children with abusive head trauma.

**Evidence from review update 2022**

No additional studies were included in this update.

## 3.2. Clinical features and pathology

### 3.2.1. Age of child at presentation

**Clinical question: Are retinal haemorrhages in child abuse more common in infants than in older children?**

**Evidence from previous reviews 1999 & 2012**
Retinal haemorrhages due to shaking baby syndrome or shaking infant syndrome are most commonly described in infants less than 24 months of age and they become less frequent and less severe in older children[28,95,96]. Retinal haemorrhages in older children are more commonly reported in accidental head injury[15,97]. Reports of retinal haemorrhages occurring in older children and adults are anecdotal and rare[98-101].

Much of the existing published literature involves a selected series of children below the age of 36 months reflecting the difficulty that perpetrators might have in inflicting a shaking injury in older, heavier children, with stronger neck muscles and the smaller head to body size ratio compared to infants. In addition, many reported series have an upper age limit, which may exclude analysis RH in older children. One notable series of 123 children[102], all under the age of three years, diagnosed with AHT showed an increased incidence of retinal haemorrhages with increasing age of the child. Twenty-two out of 24 children (91.6%) greater than 12 months of age and diagnosed with AHT were noted to have any type of RH versus 69 out of 99 infants (69.6%) less than 12 months of age having any type of RH. However, statistical analysis was not possible due to far fewer older children being present in the analysis. A few cases of children as old as eight years presenting with retinal haemorrhages in the context of abuse have also been reported[103,104]. There is no upper age limit to the presence of retinal haemorrhages, but the incidence is likely to be low with increasing age of the child.

**Evidence from review update 2022**
The evidence continues to strongly support the findings of previous reviews that ocular findings secondary to AHT occur almost exclusively in younger children. Clark 2020, in a large series of accidental and non-accidental eye injuries in children from Western Australia, found no children >24 months of age with NAI related retinal haemorrhages[105]. Carrim 2012 (UK), also found that retinal haemorrhages associated with non-accidental injury occurred in younger infants with a mean age of 15 weeks (range 2-47 weeks)[106]. Similarly, a series from Taiwan reported a mean age of 7 months (range 1-36 months)[107].

**Conclusions:** **D**

Retinal injury in AHT is commonly reported in children under three years of age. The highest prevalence is seen in infancy although case studies of older children have been reported.

### 3.2.2. Characteristics of retinal findings

#### 3.2.2.1. Site of retinal haemorrhages

**Clinical question: What is the usual site and extent of retinal haemorrhages in AHT?**

**Evidence from previous reviews 1999 and 2012**
Retinal haemorrhages (RHs) are predominantly seen at the posterior pole but retinal periphery may be difficult to examine clinically[108]. Typically all layers of the retina are involved, but haemorrhages may be confined to a single layer[9,16,96,109-112].

Severe retinal haemorrhages and perimacular folds are more likely seen in NAI but are not pathognomonic[27,113,114], RH can be unilateral or highly asymmetrical[17,115,116].

A systematic review on the diagnostic accuracy of ocular signs has reported a sensitivity of 75% and a specificity of 94% of intraocular haemorrhage in AHT[37]. Unilateral retinal haemorrhages are commonly reported[117]. However, RHs are often too numerous to count and extend from posterior pole to ora serrata[34,36,118-120].

**Evidence from review update 2022**
The majority of studies continue to describe retinal haemorrhages attributed to AHT to be numerous, multi-layered and extending from the posterior pole to the retinal periphery[105,107,121-124]. One study reported that in children with retinal haemorrhages secondary to AHT, none extended to the periphery (zone 3) and, in a significant number of eyes, the haemorrhages were limited to the posterior pole (zone 1)[125].

**Conclusions:** **B,C,D**

RHs may be unilateral and asymmetrical but they are often reported as severe, multi-layered, and bilateral.

### 3.2.2.2. Ocular fundus findings

**Clinical question: Are any ocular fundus findings pathognomonic of child abuse?**

**Evidence from previous reviews 1999 & 2012**
Severe retinal haemorrhages (preretinal, intraretinal and subretinal) and perimacular retinal folds are frequently seen in child abuse[27,108,113] but they cannot be said to be pathognomonic[126]. They have also been reported in severe accidental trauma and Terson's syndrome[127] although they are more common in AHT. The presence of perimacular folds may suggest that there have been cycles of acceleration and deceleration[108,114]. Haemorrhagic retinal cysts and retinoschisis are probably much more common in child abuse[27,128,129] than accidental trauma.

There are no clinical ocular findings that are pathognomonic for abusive head trauma. Combined data from prospective studies indicate that intra ocular haemorrhages have a high specificity (94%) and sensitivity (75%) for child abuse[34,37]. The specificity is further increased when there is bilateral involvement, peripheral involvement, intraretinal, preretinal, premacular haemorrhages and moderate to severe intraocular haemorrhages[130].

Perimacular retinal folds and traumatic retinoschisis have a low sensitivity and high specificity for AHT. These findings are however not pathognomonic and have been reported in infants after fatal motor vehicle accidents[30] and crush head injuries[56,131]. Optic nerve sheath haemorrhages are significantly more common in abuse than in other conditions in autopsy studies[132].

There have also been case reports of RPE tear[133], epiretinal membrane[134], giant retinal tear[135] macular hole[136], associated with abusive head trauma.

**Evidence from review update 2022**
There are no clinical ocular findings that are pathognomonic for abusive head trauma. Maguire et al analysed individual patient data from 6 comparative studies of children < 3 years with intracranial injury to determine the association between AHT and combinations of apnoea; retinal haemorrhage; rib, skull, and long-bone fractures; seizures; and head and/or neck bruising. When rib fracture or retinal haemorrhage was present with any 1 of the other features, the OR for AHT is >100 (PPV >85%)[137].

The combination of subdural brain haemorrhage, retinal haemorrhage and encephalopathy is not pathognomic for abusive head trauma[138]. Traumatic retinoschisis in children is highly associated with subdural haemorrhage, neurologic symptoms, and poor outcomes and is associated with likely abuse in most cases[139]. Additionally, there is a strong association between traumatic retinoschisis and significant neurological morbidity or, fatal outcome.

**Conclusions:** **B,C,D**

There are no fundus findings pathognomonic of child abuse though multi-layered retinal haemorrhages that are bilateral involving the posterior pole and periphery, retinal folds and retinoschisis may be highly suggestive of it. The ocular fundus findings need to be evaluated with other systemic findings present.

#### 3.2.2.3. Unilateral retinal haemorrhages

**Clinical question: Are unilateral retinal haemorrhages compatible with child abuse?**

**Evidence from previous reviews 1999 & 2012**
Unilateral retinal haemorrhages are not uncommon with indirect child abuse injuries. This is based on the following evidence:

1. A report of three consecutive cases[140].
2. In 13 autopsy cases, 3 of the 9 in a group with direct head trauma had unilateral retinal haemorrhages[16].
3. Of 14 consecutive cases of presumed shaken baby, three had unilateral retinal haemorrhages[17].
4. Of 20 cases of shaken infants, four had unilateral retinal haemorrhages[115].
5. In one case out of 7 babies with "physical child abuse" there were haemorrhages on histopathology in only one eye[141].

Unilateral or highly asymmetrical retinal haemorrhages may also occur in pathological disease states and in accidental trauma[127,141-149].

Unilateral retinal haemorrhages have been described in abusive head trauma[37,117,130,150] and accidental head trauma[30,37,52,130,151]. Bilateral haemorrhages are frequently seen in abusive head trauma[37,130], and are less prevalent in accidental head trauma[52,130]. When retinal haemorrhages are unilateral, they may be ipsilateral to the side of brain trauma[117,152,153], although one study failed to demonstrate this association[36].

**Evidence from review update 2022**
In the majority of cases, retinal haemorrhages secondary to AHT are bilateral. Nonetheless, unilateral haemorrhages are consistently reported in many studies[106,123-125,154].

**Conclusions:** **D**

While retinal haemorrhages are more commonly bilateral in cases of abusive head trauma, cases with unilateral haemorrhages do occur. They also occur in accidental head trauma, and in other pathologies. When they occur in association with unilateral intracranial haemorrhage, they are often but not exclusively ipsilateral.

### 3.2.2.4. Frequency of intraocular haemorrhage

**Clinical question: Can intraocular haemorrhage increase after injury?**

**Evidence from previous reviews 1999 & 2012**
*Retinal haemorrhages (RHs) rapidly increase in extent and severity while the trauma and immediate effects are in progress. However, many of the intracranial events that are associated with RHs do progress gradually. Therefore the finding of increasingly extensive intraocular haemorrhages could reflect observer error, further trauma, further haemorrhages as a result of continuation of the effects of the original trauma, or a spread of the haemorrhage from one layer or area of the retina to another, for example from the subhyaloid space into the vitreous. No references are provided to support this. (* See new published evidence in the update below which reports that retinal haemorrhages do not increase after injury).

Studies specifically dealing with whether or not RHs can increase after injury were not found in the literature reviewed. Most studies include single eye examinations with only a few including repeated or serial eye examinations. In a study of 123 children with subdural haematomas secondary to abuse[102] retinal haemorrhages were present in 83%; eye exams were approximately weekly. Authors document the rate of resolution of RHs of different appearances and layers of the retina[102]. A further study of 241 consecutive infants (<3 years) with subdural haematomas[34] where ophthalmic examinations took place at regular intervals until retinal haemorrhages resolved. Neither of these studies reported any increases in retinal haemorrhages though this was not specifically studied. There is a single study which reports that new retinal haemorrhages may appear in premature babies being screened for retinopathy of prematurity (ROP), even in the absence of ROP[155]. These haemorrhages are small in number and may resolve very slowly. There was however no control group and no detail regarding the eye screening exams, in particular no reference as to whether RetCam screening was employed. There is also little or no information on the infants underlying medical status. Progressive increasing severity of RH in AHT has not been reported.

**Evidence from review update 2022**
A prospective retinal imaging study of children with accidental head injury, abusive head trauma and non traumatic encephalopathies found that 60% of all retinal haemorrhages progressively declined before resolving but in 35% the initial area of haemorrhage increased before reducing and resolving. The mean increase in area was 35% for intraretinal haemorrhages and 99% for pre-retinal haemorrhages. It was suggested that this pattern could be due to spreading of the haemorrhage or from fibrinolysis with water being drawn into the haemorrhage. No increase in the number of retinal haemorrhages was found[156].

**Conclusions:** **C,D**

The area of retinal haemorrhage can increase in about one third of cases, but the number of haemorrhages do not increase. This has implications for timing of injury based on area of retinal haemorrhage/s.

### 3.2.2.5. Timing of haemorrhage injury

**Clinical question: Is it possible to determine from an examination of the retina, the time at which an injury occurred or whether there have been haemorrhages at more than one time?**

**Evidence from previous reviews 1999 & 2012**
Data on the age of bruising of the skin by its change in colour is controversial[157] (Current scientific evidence concludes that is not possible to age bruises from inspection[158]) and a parallel can be drawn with retinal haemorrhages. It can be extrapolated from retinal haemorrhages (RH) related to birth trauma that superficial flame shaped RH resolve in 24 hours[159,160] moderately severe haemorrhages take a few weeks to clear[161] and severe retinal haemorrhages with vitreous haemorrhage may take months to clear[162,163].

The presence of haemosiderin noted in a retinal haemorrhage on pathology suggests it had been present for at least three days[164], hence the presence and absence of haemosiderin in different retinal haemorrhages indicates haemorrhages of different ages[27,164].

A study reporting on the timing of RHs as shown by clearance of retinal bleeding in AHT gives an wide time frame of 1-11 months[165]. Indirect evidence on timing can be extrapolated from two sources where the time of 'injury' is known: birth (in humans) and experimental induction of central retinal venous occlusion in adult monkeys. The human birth studies suggest that whilst extensive intra retinal bleeding would be expected to clear by four weeks, with approximately 86% of them clearing within two weeks, discrete intra retinal haemorrhages can be seen up to 58 days after birth[166,167]. An animal study reported resolution of intra retinal bleeding ranging from two days to one month. Histopathologically haemosiderin has demonstrated in the retina which occurs as early as two days after CRVO and may last up to 16.8 months[94].

Direct evidence from cases of AHT and indirect evidence from birth studies; give similar timings for clearance of confluent intraretinal bleeding noted clinically. The majority of birth related retinal haemorrhages clear within two weeks though some intraretinal haemorrhages may persist up to 58 days (see section 3.3.3). Histopathologically the presence of haemosiderin in the retina may represent a haemorrhage, which has occurred between two days and 16.8 months prior to examination.

**Evidence from Update 2022**
In infants with retinal haemorrhages associated with hypoxic ischaemic encephalopathy those RHs involving the macula have been reported to take longer to resolve (mean 38.57 days) than haemorrhages not involving the macula (mean 24.27 days)[168].

A systematic review of new born retinal haemorrhages identified 13 studies. 83% of retinal haemorrhages resolved within 10 days with isolated cases persisting to 58 days. The haemorrhages were mainly bilateral, intraretinal and posterior in situation[169].

A retrospective review of retinal haemorrhages in children under two years of age with abusive or accidental head trauma found that intraretinal haemorrhages cleared rapidly, within 1-2 weeks with one haemorrhage persisting for 32 days. Pre-retinal haemorrhage persisted for 5-111 days. The presence of "too numerous to count" intraretinal haemorrhages indicated that trauma had occurred within a few days prior to the examination but the presence of preretinal haemorrhage with no or few intraretinal haemorrhage suggested days to weeks since the trauma[170].

In a prospective retinal imaging study of children with accidental head injury, abusive head trauma and non traumatic encephalopathies it was found that the duration of intraretinal haemorrhage was consistent with other reports with a mean resolution of intraretinal haemorrhage of 12 days (range 1-57 days) and a mean resolution of pre-retinal haemorrhage of 29 days (range of 10-72 days)[156].

**Conclusions:** **B,C,D**

The majority of intraretinal haemorrhages will clear within two weeks but there is a range reported of up to 58 days for isolated intraretinal haemorrhage to resolve. Pre-retinal haemorrhage takes longer to clear. The presence of multiple intraretinal haemorrhages with preretinal haemorrhage suggests recent trauma within a few days of the retinal examination, but an isolated preretinal haemorrhage with few or no intraretinal haemorrhages may suggest a longer interval from the trauma.

### 3.2.2.6. Intracranial findings or encephalopathy

**Clinical question: Are there any retinal findings without intracranial findings or encephalopathy?**

**Evidence from previous reviews 1999 & 2012**

There is a correlation of the severity of retinal haemorrhages and intracranial Injuries[9,17,171] though it can be difficult to estimate the severity of, or diagnose intracranial injury on an initial CT scan[172]. AHT patients with subdural haemorrhages and other intracranial injuries typically exhibit retinal haemorrhages, but the association is not invariable[9]; between 11%[173] and 39%[96] of abused children do not have retinal haemorrhages. Conversely there is little reference to retinal haemorrhages occurring in the absence of intracranial findings although it is suggested that violent chest compression in child abuse may cause retinal haemorrhages[174-176].

There are case series/case reports suggesting that retinal haemorrhages may be identified in the presence of normal neuro-imaging[150,177-179]. In the majority of these studies the imaging performed was a CT scan. The CT scan appears to be relatively insensitive to picking up very thin film subdural bleeding. Morad et al[178] identified eight children with cerebral oedema on CT scanning. Seven of these had very severe cerebral oedema and five died. All had extensive retinal haemorrhages but the CT scan failed to detect any signs of intracranial bleeding. The authors hypothesised that rapidly developing cerebral oedema causes increased intracranial pressure and subsequent tamponade of any accumulating intracranial blood. They also indicated that the prognosis in such cases was very poor. The same group[177] identified nine children diagnosed with AHT with normal CT scan results but retinal or vitreous haemorrhage. All had a normal CT scan at admission to hospital but three were found to have evidence of subdural bleeding on subsequent MRI scanning. The authors recommended that an MRI scan be performed 5-7 days after the presumed injury and postulated that earlier scans may fail to detect acute subdural haemorrhage (because an acute haematoma's density is similar to CSF or brain tissue). They note that MRI scans are also superior to CT in detecting small accumulations of blood, non-haemorrhagic intra-axial injuries such as cortical contusion or shearing and in dating blood collections. They conclude that the diagnosis of AHT can still be made in children with normal CT imaging but emphasised that careful assessment of the combination of clinical signs at presentation is important in children suspected of suffering an inflicted injury. Healey and Schrading[150] reported a single case of a child with a history of apnoea, abnormal neurological signs and unilateral retinal haemorrhages. The CT and MRI scans were normal but the mother admitted shaking the child.

**Conclusions:** **C,D**

RHs are relatively rare in the absence of encephalopathy or intracranial bleeding in AHT. CT imaging is relatively insensitive in picking up small subdural haemorrhages and very thin film subdural bleeding. MRI is thus used as definitive method to CT scan to increase diagnostic yield. However, it appears that in some severely injured children, early CT and MR imaging may indicate an absence of intracranial haemorrhage but reveal extensive cerebral oedema. Marked cerebral oedema may prevent intracranial blood accumulation and may in fact be a marker of more severe head injury.

**Evidence from review update 2022** **C,D**
While there were no papers that were included in this update on the presence of retinal findings without intracranial injury or encephalopathy; it was reported that children with an intracranial injury (ICI) alone have a probability of AHT of 4%, this rises to 58% if ICI and RHs are present (Maguire 2011). This paper also highlights that though the presence of apnoea is often not recorded, it also correlates significantly with AHT[137].

### 3.2.2.7. Intracranial bleeding

**Clinical question: Are retinal haemorrhages secondary to intracranial bleeding?**

**Evidence from previous review 1999, 2004 & 2012**
There is indirect evidence linking brain and eye injury though there is no clear indication that RHs are secondary to intracranial haemorrhage. Reports suggest similar causation for RHs and intracranial haemorrhage[9,11,13-17,22,23].

AHT with subdural haemorrhages are often associated with retinal haemorrhages[9,17,171]. However infants with other intracranial injuries may have retinal haemorrhages[9,96,173].

Terson's syndrome[127], described as an association of subarachnoid haemorrhage (either from rupture of intracranial arterial aneurysms or arteriovenous malformations) and retinal haemorrhage in adults[149,180].

Further reports of Terson's syndrome describe a wider association of any intracranial bleeding with intraocular bleeding[54,181-183]. A prospective observational study revealed very little evidence for Terson's syndrome in children[54] reporting isolated haemorrhages in children with a mean age of 10.3 years. Intracranial bleeding with associated intraocular haemorrhage was seen in older children in this group.

Terson's syndrome is rare in children and haemorrhages if they occur tend to be concentrated around the optic disc.

A report[152] of a seven month old infant who was diagnosed with a ruptured complex fusiform aneurysm of the right middle cerebral artery resulting in a large acute subarachnoid haemorrhage was found to have extensive right-sided pre and intraretinal haemorrhages.

A further report[184] of a ruptured middle cerebral aneurysm resulting in a subarachnoid haemorrhage in a seven month old infant revealed bilateral extensive intra and preretinal haemorrhages extending to the ora serrata with haemorrhagic optic nerve sheaths on post-mortem examination. It was hypothesized that the ocular and optic nerve sheath haemorrhage was due to a rapid rise in intracranial pressure.

Bilateral retinal haemorrhages, retinal detachment, subdural haemorrhage, subarachnoid haemorrhage, cerebral and cervical cord swelling with multiple rib fractures in a 4.5 month old infant who presented with choking and received cardiopulmonary resuscitation has been reported[76]. Though abusive injury could not be excluded the retinal bleeding was attributed to disseminated intravascular coagulopathy, raised intracranial pressure and the attempted CPR.

An infant born to a mother with primary anti-phospholipid syndrome had an extensive occipital infarction with bilateral vitreous haemorrhage and hyphaema[185]. It has been postulated that the retinal haemorrhage may have been due to maternally transferred antibodies causing a hypercoaguable state in the neonate resulting in retinal vascular occlusion, or massive cerebral hypoxia and subsequent raised pressure leading to a Terson's like syndrome.

A case of a 38 week gestation infant who died with severe brain oedema, had bilateral extensive peripapillary haemorrhage noted on postmortem examination which was postulated to be caused by a Terson-like phenomenon[155].

Accidental low impact head injury associated with epidural haemorrhage and retinal haemorrhages have been reported[186]. In this study the retinal haemorrhages were seen in five cases and were unilateral in four. The retinal haemorrhages were mild concentrated in the posterior pole. It is hypothesized that the mechanism of retinal haemorrhage was secondary to a sudden rise in intracranial pressure due to the epidural haemorrhage. However surgical evacuation of the epidural haemorrhages and the initial trauma may play a role as causal factors of the retinal haemorrhages.

In a prospective study[52], 154 children who sustained vertical falls from a height, 16 infants had epidural haemorrhages and three had mild unilateral retinal haemorrhages. This suggests that RHs with epidural haematomas are rare in children who sustain vertical falls unless there is a midline shift.

**Conclusions:** **C,D**

Current evidence demonstrates that intra ocular bleeding secondary to intra cranial haemorrhage from intracranial pathology or accidental trauma is rare in young children. Retinal findings however may closely resemble those seen in AHT. Cases where intra ocular and intra cranial haemorrhage are seen together include those with clear evidence of a ruptured aneurysm, severe accidental head injury, epidural haemorrhage and neurosurgical intervention. The severity of the ocular findings being directly related to the severity of the intracranial event with an acute, sharp rise in intracranial cranial pressure playing a role in the mechanism of retinal haemorrhages.

**Evidence from review update 2022**
No additional studies were included in this update.

### 3.2.2.8. Intracranial damage and retinal haemorrhage

**Clinical question: Is intracranial damage always accompanied by retinal haemorrhages in AHT?**

**Evidence from previous review 1999 & 2012**
There is a close correlation between the severity of retinal haemorrhages and intracranial injuries[9,17,171] although it is sometimes difficult to estimate the severity of or even to diagnose intracranial injury, on the initial CT scan[172].

It is usual for AHT patients with subdural haemorrhage and other intracranial injuries to have retinal haemorrhages, but the association is not invariable as between 11% and 39% of abused children do not have retinal haemorrhages[96,173].

There is conflicting information about the incidence of retinal haemorrhage in abusive head trauma in the literature.

A systematic review of the diagnostic accuracy of ocular signs in abusive head trauma found that intra-ocular haemorrhage had a 75% sensitivity for abusive head trauma[37]. Retinal haemorrhages are reported in 53-89% of cases of AHT[34,36,102,120,130,187,188]. As intracranial haemorrhage is used as inclusion criterion for the diagnosis of abusive injury in many of these studies this introduces circular logic potentially confounding efforts to make an accurate assessment of incidence. These studies suggest that though retinal haemorrhages are frequently seen in the presence of intracranial injury they are not always present.

A report of prosecuted perpetrators of fatal AHT revealed bilateral RH in all 6 cases[132].Though this report provides evidence that retinal haemorrhages are usual in AHT, limitations of the study prevent firm conclusions as to whether retinal haemorrhages are always associated with brain injury in AHT as the diagnosis was made on the basis of the findings of subdural haematoma of the brain and retinal haemorrhages. Hence it is possible that children with brain injury from abusive head trauma without retinal haemorrhages could have been excluded from the study. Further perpetrator confessed studies of AHT report RH in 46/54 cases[189] and unilateral retinal haemorrhages in 3/17 cases[59].

**Conclusions:** **B,C,D**

There is clear evidence that retinal haemorrhage is found in approximately two-thirds of intracranial injury caused by abusive head trauma.

**Evidence from review update 2022**
No additional studies were included in this update.

### 3.2.3. Pathology

#### 3.2.3.1. Optic nerve and orbital pathology

**Clinical question: What is the optic nerve and orbital pathology seen in AHT and non AHT ?**

**Evidence from previous review 1999 & 2012**
Optic nerve sheath haemorrhage associated with the rupture of a subarachnoid vascular malformation was reported in 2 children who were initially suspected of having AHT[149,180].

The first description of optic nerve sheath haemorrhage (ONSH) in AHT was reported in 1986[190]. Since then, there have been 17 studies in which optic nerves have been examined pathologically in cases of deaths due to AHT, accidental trauma, and non-traumatic causes[9,16,27,31,95,96,108,109,112,114,132,141,153,191-194]. Four involve the sequential inclusion of autopsy case material between 1984 to 1999[96,109,112,194], therefore the last study of this series was included for analysis[194]. Two studies contain details of orbital pathology in addition to the optic nerve[27,193]. Six studies are controlled, with inclusion of non-AHT case material[9,16,109,141,193,194]. One study focuses on accidental crush head injury[31]. Overall, in the studies included, ONSH was found in 229 of 486 (47%) of cases of AHT and retinal haemorrhage was found in 250 cases (51%). There was variation from series which included all forms of child abuse[191,194,] where 50% were found to have ONSH, to series which included RH as an inclusion criterion[108,132,192] where 86% had ONSH. Where this feature was documented, subdural ONSH was more prevalent than subarachnoid haemorrhage. Most cases in which there was ONSH also had RHs; a small number of cases occurred in which ONSH or RHs were found in isolation.

In non-AHT cases, ONSH was found in 28 of 545 cases (5%) and RH in 34 (6%). In two controlled studies, severe accidental head injury was associated with optic nerve sheath haemorrhage in 7/21 cases (33%)[141,193]. A post-mortem study on orbital pathology found that subarachnoid haemorrhage in the optic nerve sheath was only seen in cases of non-accidental injury. They also found that orbital pathology was more common in cases of AHT. Extraocular muscle haemorrhage was only seen in cases of AHT (28%), and bleeding into orbital fat was more common in AHT (50%) compared with accidental injury (22%). Bleeding in the posterior orbit was also more common. This study focussed on the examination of the optic nerves and orbital contents[193]. A study of severe accidental crush head injury found ONSH in 9 cases (33%)[31]. In controlled studies where death was due to non-traumatic causes (e.g. Sudden Infant Death Syndrome, asphyxia, etc.) ONSH was found in 1 of 18 cases[16,112,141], and in that case ONSH was demonstrable only on microscopic examination[16].

Bleeding at the optic nerve-scleral junction in the region of the vascular circles of Zinn and Haller (peripapillary intrascleral haemorrhage) is noted in 97 of 208 cases (47%) of AHT, where this feature was sought[27,31,95,132,190]. In 89 control cases a study which included cases of accidental head injury, there was a case of peripapillary intrascleral haemorrhage (1%)[95]. Some authors have hypothesised that bleeding at this location was consistent with the action of shearing/torsional forces at the junction of the eye and the optic nerve and, thus was suggestive of, shaking[195]. Over 50% of the cases in their study showed no evidence of impact, all had perimacular folds, and 98% had optic nerve haemorrhage. In a study of Terson's syndrome in adults, the author[195] proposed an alternative explanation for the peripapillary intrascleral bleeding, invoking rapid and severely raised intracranial pressure transmitted into the optic nerve sheath, impeding retinal venous outflow from the optic nerve and blood flow via the retinochoroidal anastomoses at the optic nerve-scleral interface. This model also provides an explanation of the optic nerve sheath, premacular and retinal bleeding seen in Terson's syndrome.

Optic nerve sheath haemorrhages are common in AHT, however they may also be seen in cases of accidental head injury. The only study which specifically examined orbital tissues in detail found that bleeding into orbital fat and especially the extraocular muscles and posterior orbit may be of significance in distinguishing AHT from accidental injury, but more work is required to confirm this. Scleral haemorrhage at the junction between the optic nerve and sclera is common in AHT, and is seen in association with ONSH and RH, but the number of studies documenting this finding is small.

**Evidence from review update 2022**

ß-Amyloid Precursor Protein (ß-APP) immunohistochemistry was performed on the eyes and distal optic nerves of 5 infants diagnosed by medical examiners with shaken baby syndrome (SBS), 3 diagnosed as combined shaking-impact syndrome, one with blunt head trauma and 4 non-traumatic controls[196]. 3/5 SBS cases and all 3 combined shaking-impact cases showed optic nerve axonal injury, with strongly ßv-APP positive beaded or swollen axonal segments, in most cases concentrated to a band in the anterior optic nerve just caudal to the optic nerve head and lamina cribrosa, with scattered ß-APP positive axonal segments more posteriorly. This pattern was not seen in the blunt head trauma case or non-traumatic controls. The SBS and combined shaking-impact cases also showed focal ß-APP positive axonal swellings within the retinal nerve fibre layer, almost always adjacent to areas of retinal haemorrhage. ß-APP positive beaded or swollen axonal segments were not seen in the retina of non-traumatic cases. The axonal injury highlighted by ß-APP was not visible on haematoxylin and eosin or neurofilament protein stains. The authors highlighted that the small size of the series, with only one case of blunt trauma, was inadequate to draw conclusions about the specificity of optic nerve axonal injury for shaking or shaking-impact trauma[196].

A case report described a 2 year old child with a fatal head crush injury from a toppled 27" cathode ray tube television set[197]. There were multiple contusions to the head, a 17cm widely displaced skull fracture extended from the right parietal bone across the vertex to the left parietal bone with an associated dural tear, and a further basilar fracture involved the floor of both middle cranial fossae. The brain was swollen with bilateral acute subdural haemorrhage, patchy subarachnoid haemorrhage, bilateral mild hippocampal uncal herniation, haemorrhagic contusion-laceration with disruption of the inferior surface of the frontal lobes and petechial and streak-like haemorrhages of the anterior corpus callosum. Acute bilateral optic nerve sheath haemorrhages were seen macroscopically (microscopy not detailed)[197].

An 8 week old baby boy, born at term, was found unconscious lying in bed with his father[198]. He received 35 minutes of CPR with chest compressions. Laboratory tests on admission revealed evidence of disseminated intravascular coagulation (DIC) in addition to anaemia with haemoglobin 6.4. Three hours after admission, a further 18 minutes of chest compressions were carried out. CT scan revealed hypoxic-ischaemic brain injury but no subdural or subarachnoid haemorrhage. Funduscopic examination revealed extensive bilateral intraretinal haemorrhages (see section 3.2.3.2). Skeletal survey revealed no fractures. The baby had anoxic encephalopathy and died after

4 days. Autopsy revealed no external signs of trauma or scalp bruising and a swollen brain with no subdural or subarachnoid haemorrhage. Microscopy revealed bilateral subdural optic nerve sheath haemorrhages along the nerves and focal right intradural optic nerve sheath haemorrhage. Gross examination of orbital soft tissue revealed multiple petechial haemorrhages. Pathological retinal findings are described below in 3.2.3.2.Cardiac examination revealed anomalous origin of the right coronary artery within the left sinus of Valsalva with an associated subacute infarct adjacent to the atrioventricular node. Although the Child Protection Team were suspicious of abuse due to the extent of the retinal haemorrhages, the medical examiner gave a cause of death as cardiac arrest due to myocardial infarction (MI) in the region supplied by the anomalous coronary artery, with contributing anaemia. DIC on admission was thought likely secondary to cardiogenic or hypotensive shock. The authors commented that in such a case, it may be difficult to determine whether abuse led to MI and subsequent coagulopathy or whether the infant suffered an MI and then developed signs of cerebral hypoxia and retinal haemorrhages as a result of the complications of the MI and its treatment[198]. The potential contribution of CPR in this case is discussed in section 3.3.5.2.

**Conclusions:** **D**

ß-APP immunohistochemistry is a useful adjunct to routine histopathological staining to allow detection of axonal injury in the optic nerve. In a small case series, optic nerve axonal injury was demonstrated in cases diagnosed as shaking or shaking-impact head trauma[196]. Further work is required to determine the specificity of these findings for abusive head trauma and mechanism of injury.

Head crush injury can be associated with bilateral optic nerve sheath haemorrhages, as described in the previous review[197].

Optic nerve sheath haemorrhages occurring in the context of DIC, anaemia and infantile myocardial infarction with an anomalous origin of the right coronary artery may be difficult to distinguish from abusive head trauma[198].

### 3.2.3.2 Vitreoretinal pathology

**Clinical question: What is the vitreous/retinal pathology in AHT and non AHT ?**

The three studies described above in section 3.2.3.1 also reported on retinal findings.

In the study of ß-Amyloid Precursor Protein (ß-APP) immunohistochemistry, the SBS and combined shaking-impact cases showed focal ß-APP positive axonal swellings within the retinal nerve fibre layer. However, due to the presence of adjacent retinal haemorrhage and detachment in most cases, the authors cautioned against the appearance in the retina being a specific marker for a shaking type of injury[196]. ß-APP positive beaded or swollen axonal segments were not seen in the retina of non-traumatic cases.

In the case of a 2 year old child with a fatal head crush injury from a toppled television set, histological examination of the eyes revealed bilateral acute retinal haemorrhages, predominantly in the nerve fibre layer (further information regarding extent and location were not provided)[197].

The 8 week old baby with an anomalous right coronary artery and MI underwent funduscopic examination 10 hours after admission[198]. This revealed extensive bilateral intraretinal haemorrhages extending from the posterior pole to the ora serrata, preretinal haemorrhages in the right eye and a left subhyaloid haemorrhage. Histological examination of the eyes revealed bilateral marked nerve fibre retinal haemorrhage extending to the ora serrata. As discussed above and in section 3.2.3.1, the primary event in such a complex case may be difficult to identify.

A case series described the histopathological findings in eyes of 5 patients consecutively received that demonstrated perimacular folds and/or detachment of the internal limiting membrane (ILM) and/or superficial retinal layers ("retinoschisis")[199]. Cases had a diagnosis of abusive head trauma as determined by the Medical Examiner's Office in agreement with the multidisciplinary child abuse team. All cases had other systemic manifestations of abusive trauma and intracranial injury.
The 5 cases all featured bilateral retinal haemorrhages extending to the ora serrata. 4 were symmetric and 1 asymmetric, all included intraretinal haemorrhage with 4/5 being multilayered, also featuring preretinal and/or subretinal haemorrhage. 2 cases showed vitreous haemorrhage. All cases showed bilateral subarachnoid and subdural optic nerve sheath haemorrhage (4 symmetric, 1 asymmetric), 3/5 with additional intradural haemorrhage. 2/5 cases showed peripapillary intrascleral haemorrhage and 4/5 showed orbital fat haemorrhage. One case also featured extraocular muscle sheath and cranial nerve sheath haemorrhage. All cases demonstrated histologically, in at least one eye, condensed vitreous attached to the apices of perimacular folds and/or the dome of the intervening ILM. The elevated ILM remained attached to the apices of the folds and extended across the intervening "crater". These histological findings support vitreoretinal traction due to acceleration-deceleration forces as the mechanism underlying retinoschisis and perimacular fold formation. The authors discussed other scenarios in which appearances similar to posterior traumatic retinoschisis can occur, and postulated alternative pathophysiological mechanisms in such cases. For example, in fatal head crush injury, high level falls, fatal motor vehicle collisions and aneurysmal rupture, there is a sudden hyperacute rise in intracranial pressure that differs from the slower rise seen in AHT. This may cause occlusion of the central retinal vein with insufficient time for arterial pressure to down-regulate, leading to acute retinal vascular rupture of sufficient pressure to elevate the ILM, which in turn might raise retinal folds. Obstruction of retinochoroidal anastomoses at the scleradural junction could be contributory[199].

**Conclusions:** **D**

ß-APP immunohistochemistry is a useful adjunct to routine histopathological staining to allow detection of axonal injury in the retina, as in the optic nerve. In a small case series, retinal axonal injury was demonstrated in cases diagnosed as shaking or shaking-impact head trauma[196]. Further work is required to determine the specificity of these findings for trauma.

Head crush injury can be associated with bilateral retinal haemorrhages, as described in the previous reviews[197].

Retinal haemorrhages occurring in the context of DIC, anaemia and infantile myocardial infarction with an anomalous coronary artery may be difficult to distinguish from abusive head trauma[198].

Condensed vitreous attached to the apices of perimacular folds has been demonstrated histologically in 5 cases of abusive head trauma, supportive of vitreoretinal traction as a pathogenic mechanism for macular retinoschisis and perimacular fold formation[199].

## 3.3. Differential diagnosis and confounding conditions

### 3.3.1. Accidental injury and retinal haemorrhage

**Clinical question: Can accidental injury cause retinal haemorrhages?**

**Evidence from previous review 1999 & 2012**
Retinal haemorrhages (RHs) are rare after accidental injury. They may be seen after severe head injury complicated by fractures and diffuse or focal intracranial damage. Children who sustain fatal injuries after reported short falls are more likely to have been subjected to non-accidental trauma[15,22,23,95,97,112,141,173,200-202].

Data from falls in 3357 premobile infants reported fractures in less than 1% with less than 16 infants requiring hospitalization[203]. Short falls in infants only rarely show evidence of neurological or ocular damage[204,205]. In studies where a history of short falls are elicited the injuries were ultimately found to be inflicted[23,206]. Retinal haemorrhages in children who sustained accidental household trauma were reported in three cases which were 'mild' unilateral, in posterior pole and with no extension to ora serrata[53]. A further study of short falls which were estimated to range from 0.6 to 2.4 metres in witnessed playgrounds incidents reported three children with bilateral retinal haemorrhages[48].

Subdural haemorrhage (SDH) with RHs is rarely associated with high velocity accidental injuries in children. Child abuse needs to be considered when SDH and RH exist without an adequate explanation or a medical cause.

An accidental injury is deemed to have occurred when there is a consistent history, a witnessed accident, an accident in a public place, which was witnessed or where the clinicians and investigating authorities concluded an accidental injury occurred[207] Retinal haemorrhages (RHs) are rare in accidental injury and occur in 0% to 15% of children less than 3 years[33,35,52,130,208,209]. RHs due to accidental trauma involve falls[33,35,45,52,54,130,186,207,208,210,211] motor vehicle accidents (MVA)[30,35,54,130,207,209,212] crush injury[31,56,131,212,213] to the head between unyielding surfaces and accidents during play[49].

Retinal haemorrhages are predominantly unilateral, mild and located in the posterior pole in case series reporting falls which range from less than 4 feet to over 10 feet[35,52,130]. RH confined to the posterior pole, few in number and either intra or pre retinal are reported from short falls (a couch, bed, tables, caretakers arms, baby walkers and trolleys)[35,52,186]. Falling backwards from a sitting position on a carpeted floor in a 11month old infant was associated with bilateral retinal haemorrhages in all four quadrants of the retina[45] and a 21 month child who fell from a standing position on a chair onto a tiled floor sustained bilateral retinal haemorrhages with retinal folds[210]. An 11metre fall of a 24 month old girl who fell onto a concrete surface was associated with bilateral preretinal, intraretinal and subretinal haemorrhages with bilateral macular retinoschisis and a possible retinal fold[211].

Retinal haemorrhages that occur in children who survive MVA's have been reported as mild pre and intra retinal and located in the posterior pole[35,130,207,209,212]. However in children who die from their injuries bilateral, multilayered, numerous RH in the posterior pole and periphery associated with retinal folds are described in autopsy series[30].

In crush injuries to the head the severity of RH varies from mild mainly in the posterior pole, pre and intra retinal[31] to extensive multilayered bilateral RH associated with retinal folds and retinoschisis[56,131,212,213].

RHs are rare in accidental injury. Though the majority of reports suggest that these RHs are mainly mild in severity and predominantly unilateral, few in number either pre or intra retinal and located to the posterior pole, a pattern of bilateral extensive multi-layered RHs extending from the posterior pole to the periphery, associated with retinoschsis and retinal folds have been the subject of a few recent case reports and autopsy series with predominantly severe crush head injury.

**Evidence from review update 2022**

Accidental crush injuries continue to be an uncommon but recurring cause of retinal haemorrhages. Raj et al presented a case of an infant with confirmed accidental trauma sustained from an adult-worn baby carrier fall with superimposed head crush injury resulting in significant intracranial, and retinal findings[214]. On examination, the 2-month old infant was awake and irritable with a full anterior fontanelle and scalp swelling, with no other external signs of injury. CT of the head revealed biparietal skull fractures with subjacent soft tissue swelling and bilateral subdural hematomas but no orbital fracture. Subsequent MRI revealed bilateral subdural hematomas and scattered foci of parenchymal injury. Retinal examination by a paediatric ophthalmologist with indirect ophthalmoscopy revealed numerous intra- and preretinal haemorrhages bilaterally, located primarily in the posterior pole, with fewer more anteriorly in the retinal periphery. There was no retinoschisis or retinal fold. A skeletal survey did not detect any additional injuries.

Adams et al described a 14-month old infant who had subhyaloid retinal haemorrhages with schisis cavities and perimacular folds. This ultimately was a fatal traumatic brain injury from a witnessed fall while being carried and, which involved falling down 10 steps with multiple impacts. The child also had a subarachnoid and a subdural brain haemorrhage[215].

**Conclusions:** **C,D**

Further evidence indicates that crush injury could be associated with severe and bilateral RHs. Accidental fatal head injury can also be associated with RHs, retinal folds and retinoschisis.

### 3.3.2. Other childhood conditions and retinal haemorrhage

**Clinical question: What other conditions of childhood may have retinal haemorrhages?**

**Evidence from previous review 1999 & 2012**

- Leukaemia: Reports suggest that RH seen in leukaemia may or may not be associated with other blood abnormalities like thrombocytopenia[216,217].
- Haemorrhagic disease of the newborn: A study states that there is no association between thrombotest results and birth related RH[159].
- Sickle Cell retinopathy: The retinal findings have distinguishing features which suggest a diagnosis of sickle cell retinopathy[218].
- ECMO treatment: The intraretinal haemorrhages reported may not be due to the ECMO itself but due to other comorbidity[143,148,219,220].
- Retinopathy of prematurity (ROP): The retinal features suggest a diagnosis of ROP.
- Galactosaemia: Vitreous haemorrhages described[221].
- Henoch-Schonlein purpura: Transient punctate RH is reported[222].
- Thrombocytopaenic purpura: RHs have been reported[223].
- Maternal ingestion of Cocaine: RHs are reported that last longer than typical newborn RHs[224].
- Meningitis: RHs associated with meningococcal meningitis is reported[144] .
- Intracranial vascular malformation: Subhyaloid and RH is reported in an infant with intracranial arterial aneurysm, and optic sheath haemorrhage in child with ruptured Subarachnoid malformation[149,180].
- Optic disc drusen, tuberous sclerosis, X linked retinoschisis: Vitreous haemorrhage is reported in these conditions[225,226].

- Intracranial pathology: Chronic, severe papilloedema may cause peripapillary haemorrhage, and acute, severely raised ICP may cause widespread haemorrhage.
- Intraocular surgery: Multiple RHs are reported[227,228].
- Severe hypertension: RHs are often present.
- Galactosaemia: Further evidence noting vitreous haemorrhage is reported[229].
- Homocystinuria: RHs with vitreous haemorrhages and SDH is reported in a five week old baby[230].
- Glutaric aciduria: Numerous studies describe the association between glutaric aciduria and RHs as well as other retinal features, in young infants many of who may have co-existent SDH. However, neuroradiologically this condition has characteristic findings[231-233].
- Osteogenesis Imperfecta: Three cases are reported with RHs, and one case with vitreous haemorrhages, only in the posterior pole. One case had multiple, large shaped, preretinal, flame dot and blot haemorrhages but no other associated retinal findings[234].
- Osteoporosis- pseudoglioma syndrome: One study noted the presence of RH in the context of exudative vitreoretinopathy, although unlikely to pose a clinical dilemma[235].
- Incontinentia Pigmenti: One newborn infant noted to have RHs and vitreous body haemorrhage, progressing to retinal detachment, in the context of a family history and characteristic skin findings[236].
- X-linked retinoschisis: A further 2 cases are recorded with bilateral vitreous haemorrhage by three months of age[237].
- Central retinal vein occlusion: A newborn with macular haemorrhages and associated hydrops with intracerebral haemorrhages, diagnosed as CRVO[238].
- Prematurity proposed as a predisposition to RH from hyperflexion/extension of head during normal handling: This is an unsubstantiated hypothesis relating to preterm infants undergoing ROP screening, of whom 11 had RH or vitreous haemorrhages. Abuse was not explicitly excluded, as it was implied that since the infants were in a special care baby unit (SCBU) it had not occurred. They detail retinal bleeding, and propose that retinal vascular immaturity may play a role in bleeding in these cases[155].
- Infections: Newborn CMV noted to cause retinitis, including RHs[239], a child with HSV 6 noted to have a retinal vein occlusion[240], acute retinal necrosis caused by HSV 1 in a newborn[241] numerous reports detail RHs in Malaria with associated characteristic changes of the disease, often using it as an indication of cerebral malaria[242-244], meningoccocal septicaemia causing multiple posterior RHs in 5/12 children with severe septicaemia and disseminated intravascular coagulation[245] streptococcus pyogenes meningitis in a neonate with glycosylation disorder noted to have RHs[246] and also in a neonate with Strep. pneumoniae meningitis had bilateral 'too numerous to count' RHs found extending to ora serrata in association with CRVO in one case[247], congenital toxoplasmosis infants noted to have RH in association with extensive ocular features of the disease[248].
- Fibromuscular dysplasia: a nonatheromatous, non-inflammatory,multifocal segmental angiopathy of uncertain aetiology. A single case has been recorded of an 11 month old infant with intracerebral and intraventricular haemorrhage in association with extensive subhyaloid and intraretinal haemorrhages[249].
- Terson's syndrome: Although this association is well recognised in the adult literature, this is the first study including children. It included 16 deceased individuals, whose age ranged from 3 months to 90 years. Unfortunately details were only given on one 7 year old child, involved in a MVC, and no specific details of this case were given, nor details as to how Terson's was confirmed as the aetiology of the RH[250].

- Asphyxia: Two cases of asphyxia causing RH are described, one reports an asphyxiated 12 year old 'playing a scarf game' with others who developed a dense preretinal haemorrhage in his right eye[251], while the other study reported an asphyxiated neonate who had had the umbilical cord tightly wrapped around his neck, with oedematous and ecchymotic eyelids. On day 13 an ophthalmological examination noted a preretinal haemorrhage in right eye, and diffuse reddish discolouration thought to be severe RH; normal left eye. He died and post mortem (PM) examination revealed multiple RHs in the right eye[151].
- Preterm neonates undergoing RetCam recordings: A 33/40 infant being screened for ROP was examined including a RetCam image, showed no RHs and two areas of vitreous haemorrhage. He was re-examined 10 minutes later by a senior ophthalmologist and noted to have moderate intraretinal dot and blot haemorrhages and a few flame haemorrhages in right posterior pole, predominantly peripapillary[252]. Unilateral RH was reported in an 25/40 preterm infant following RetCam screening for retinopathy of prematurity examination[253]. However in response to the above study another group examined 50 eyes in 25 children 60 minutes after a RetCam examination for ROP, and none had developed RHs[254].

**Evidence from review update 2022**

- Critical illness: Retinal haemorrhages were reported in 24 of 159 (15%) critically ill children admitted to an intensive care unit who were prospectively examined with the RetCam II or the indirect ophthalmoscope. The retinal haemorrhages were reported to be mild or moderate in 75% of children and severe in 25%. There were 24 children (50% were 24 months or younger) between the ages of 6 weeks and 16 years with retinal haemorrhages. Children with AHT or suspected AHT were excluded. All cases had a history of accidental trauma or an underlying medical diagnosis. These included acute leukaemia, late onset haemorrhagic disease of the newborn with a short fall, sepsis, bronchiolitis, pulmonary hypertension, acute respiratory distress, pneumonia, post bone marrow transplant, cerebral aneurysm, AVM and severe combined immunodeficiency[215]. The reason for admission to intensive care in these children was predominantly sepsis, respiratory failure, traumatic and non-traumatic encephalopathy. An underlying coagulopathy was present in 45% of the children with retinal findings. Both sepsis and coagulopathy were significant risk factors for retinal haemorrhages[255]. A further prospective observational study in critically ill intubated children less than 4 years of age which did not exclude children with AHT reported retinal haemorrhages in 6 of 85 children, 4 of whom had a diagnosis of AHT, one had CPR and one traumatic brain injury. A more recent prospective study in children less than 4 years of aged admitted to intensive care and intubated reported a prevalence of retinal haemorrhages in 6 of 85 (7%) of children. Four children were diagnosed with AHT, one with accidental blunt head trauma in a motor vehicle accident and one with hypoxic ischaemic encephalopathy and sudden infant death syndrome. Excluding AHT in the latter study reduced the prevalence of retinal haemorrhages to 2.4%[122].
- Accidental trauma. Further to the previous guideline (refer section 3.3.1) evidence from a study on DW- MRI (diffusion weighted magnetic resonance imaging) in children compared infants with abuse (29 infants) with those with witnessed accidental trauma (16 infants). Hypoxic ischaemic brain injury (HII) was found only in the abuse group with retinal haemorrhages present in 12% of those with accidental head trauma and 38% of abuse cases. In the accidental injury cases the retinal haemorrhages were all graded as mild whereas in the abuse victims 38% were severe[256]. No RH were found in 4 cases of accidental trauma in a recent series of cases evaluated for suspected AHT[121]. A case study of a 2 month old girl who sustained a witnessed crush injury from fall when carried in a front holding baby carrier reported bilateral intra and pre retinal haemorrhages mainly in the posterior pole and fewer in the periphery. This child also sustained multiple skull fractures and bilateral subdural haemorrhages[214] (see section 3.3.9). Shuman and Hutchins[138] reported on a 14 month toddler who died after a witnessed short fall while standing on a moving toy train in a mall. The autopsy revealed an occipital fracture and extensive intracranial,

spinal cord and optic nerve sheath haemorrhages. His eyes showed bilateral retinal haemorrhages with a retinal fold in the right eye. A fundal clinical exam was not carried out while alive.

- Cerebral sinovenous thrombosis (CSVT). Retinal haemorrhages were found in 17% (5 of 29) of children aged between 7 weeks to 17 years with a confirmed diagnosis of CSVT. These RH were recorded within 10 days of a diagnosis if CSVT. In 4 of the 5 children with RH were few, superficial, peripapillary and intraretinal associated with swollen optic discs. One child with meningitis, sepsis and multiple cerebral infarctions had bilateral 30-35 RH in the posterior pole which were intraretinal. There were no children less than 3 years with CSVT and RH. RH seen in CSVT is associated with papilloedema or a medical cause and AHT is not identified as a risk factor for CSVT[257].
- Hyponatremia. A single case of RH in a child with hyponatremia with cerebral oedema was reported from a series of cases evaluated for AHT however no details on the demographics or the laterality, number or distribution was provided[121].
- Birth haemorrhages (see section 3.3.3.): RH were reported in 6 cases with birth trauma in a case series evaluated for suspected abuse with infants examined within the first week of life to 40 days postnatally. The RH were intraretinal and some preretinal though laterality was not mentioned[121]. A study on optical coherence tomography (OCT) on birth haemorrhages showed an intraretinal nasal and foveal haemorrhage noticed on routine eye examination at postnatal age 38 days. **C,D** The authors suggest that if seen earlier the haemorrhages could have been more severe and bilateral. The message from this report was that the foveal haemorrhage imaged with OCT lasted for 12 weeks but left no permanent sequelae like macular hole and epiretinal membranes as has been reported with foveal haemorrhages that resolve in AHT[258]. Li et al reported 769 (21.5%) of 3573 term newborn babies, examined within a week of birth, with RH of which 27% were graded as severe and 8% with macula haemorrhages. The laterality and distribution was not provided in detail[259]. Pu et al reported a prevalence of 18% (550 of 3123 infants) of RH in infants from high risk pregnancies or neonatal asphyxia. Most of these resolved within 28 days however 6 children with vitreous haemorrhages took 4 months to resolve and 1 required a vitrectomy at 8 months[260].

  Rossin et al report progressive retinal haemorrhage in a premature infant born at 31 weeks. The fundus revealed bilateral equatorial large (2 disc diameters) pre retinal haemorrhages 3600 equatorially these haemorrhages increased when subsequently examined a week later. Incidentally CMV viraemia was found but no evidence of vasculitis on fluorescein angiogram nor was there any evidence of neovascular retinopathy of prematurity. This infant also had small subarachnoid haemorrhages in the superior frontal sulcus. The authors suggest this may labelled as delivery associated retinopathy or Terson's syndrome though no explanation could account for the new retinal haemorrhage noted a week later[261]. Simkin et al[262] using the RetCam for newborn screening with dilated pupils reported an incidence of 14.5% (50 of 346 infants) with RH. Babies were screened in hospital or the community. Those screened in the community had a higher incidence of RH (25%) compared to those screened in hospital (8%), this was thought to be due to the screening in the community occurring earlier than those screened in hospital. The haemorrhages were bilateral in 68%, they ranged from mild to severe and were more common in normal and instrumental delivery. When examined within 6 weeks later 94% had resolved. There were 2 babies born with instrumental deliveries that had RH persisting till 3 months.(see 3.3.3).
- Acute disseminated encephalomyelitis (ADEM) with bilateral optic neuritis: A case report of a 2 year-old child who was previously well, presented with drowsiness and bilateral visual loss. The child had absent pupillary reflexes with bilateral multi-layered RHs, in the posterior pole and periphery and bilateral optic nerve swelling. A diagnosis of ADEM and bilateral optic neuritis was made after numerous investigations ruled out AHT and an infective cause. Blood tests for oligoclonal, aquaporin 4 and myelin oligodendrocyte glycoprotein antibodies were negative. There were no signs of raised intracranial pressure (ICP) to account for the swollen discs. RH with

optic neuritis is rare and is mainly around the peripapillary area. The authors suggest that the retinal findings were secondary to ADEM and optic neuritis due to a possible vaso-occlusive event like central retinal vein occlusion[263].

- Arteriovenous malformation (AVM) – intracranial: Shuman and Hutchins[138] report on a 3-month-old boy with subdural haemorrhage with large preretinal haemorrhage in the right eye and schisis cavities and a possible vitreous haemorrhage in the left eye when examined alive. The child died and a clinical diagnosis of abusive head trauma was made. On autopsy an arteriovenous malformation of the falx cerebri was reported as the cause of death. **D**
- Aneurysm – intracranial: Silva et al[264] reported a 4 week old infant born vaginally at term presenting with left focal seizures leading to status epilepticus. A large left expanding subdual haemorrhage was found on CT neuroimaging. Retinal examination revealed bilateral superficial peripapillary retinal haemorrhages. A diagnosis of AHT was made and the child protection team, the social services and police were involved. A subsequent MRI scan revealed a ruptured saccular aneurysm of the left middle cerebral artery. Embolization of the aneurysm and evacuation of the subdural haemorrhage led to a rapid recovery of the infant and child safeguarding was stopped.
- Bleeding disorders: Thau et al[265] (see 3.3.4).

### 3.3.3. Newborn retinal haemorrhage

**Clinical question: For how long can birth-related retinal haemorrhages persist?**

**Evidence from previous review 1999 & 2012**

RHs are common at birth, occurring in between 2.6% and 59% of newborn infants[159,160,266-268]. The RHs are present after birth and disappear rapidly with very few exceptions. The finest nerve fibre layer (NFL) haemorrhages can disappear in 24 hours, whilst even extensive NFL haemorrhages are usually gone within a few days. Half of all haemorrhages disappear within 48 hours[266] and 2.6% persisted at 72-120 hours after birth[160]. Larger subhyaloid and larger intraretinal haemorrhages seem to persist for the longest time[159]. Most neonatal retinal haemorrhages have no visual sequelae[267,269], although occasional cases of long-term impairment have been reported in association with macular haemorrhages[270].

Emerson et al examined 149 newborns within 30 hours of birth using binocular indirect ophthalmoscopy[166]. Intraretinal haemorrhage was present in 34% of newborns and varied from a single dot haemorrhage in one eye to bilateral widespread haemorrhages, occasionally with white centres. The incidence of haemorrhage was higher for vacuum-assisted (75%) than normal vaginal delivery (33%) and was least if born by caesarean section (7%). The mean maternal age was greater for infants with retinal haemorrhage. Within two weeks after birth, retinal haemorrhage resolved in 86% of eyes, and at four weeks no intraretinal haemorrhage was detected, although a single subretinal haemorrhage persisted until six weeks after birth[166]. Hughes et al reported similar findings[167]. RHs occurred in 34% of births (77% if vacuum; 30% if forceps or normal vaginal delivery (NVD); 8% if caesarean section). All haemorrhages were intraretinal and all resolved by 16 days of age except two infants (post vacuum birth) where a small number of discrete foveal haemorrhages persisted for up to two months after birth. RHs are common in newborn babies; they are more prevalent in assisted births and least common after elective caesarean sections. They may range from few to widespread bilateral RHs. They largely resolve within two weeks though dense foveal intraretinal RHs may persist for months.

**Evidence from update 2022**

In addition to determination of how long can birth related haemorrhages persist for other clinical questions included: What are the retinal findings in neonates? What are the obstetric corelates? Are neonatal haemorrhages associated with intracranial bleeding seen in newborn (when abuse is excluded)?

Watts et al[169] in a systematic review of 13 studies (that met inclusion criteria) found that birth related RHs occurred in 25% of 1777 infants born by spontaneous vaginal delivery (SVD). The incidence of RHs was increased after vacuum assisted delivery (42.6%) or double instrumental (forceps and vacuum) delivery (52%). Forceps alone was not associated with an increased incidence. RHs were most commonly bilateral (59%) and varied in severity but were predominantly intra-retinal and located at the posterior pole. 83% of RHs had resorbed within 10 days. A few persisted for up to 58 days.

Naderian et al[271] also described extensive RHs after delivery by vacuum extraction. The RHs in this case report persisted for 5 weeks.

Zhao et al (2015) in a large prospective study of 1199 healthy full-term infants reported birth related RHs in 24.5%[272]. The authors found that SVD and cephalhaematoma correlated positively with RHs while caesarean delivery was negatively correlated.

Yanli et al[273] carried out a prospective study of 3054 infants designed to identify correlations between obstetric risk factors and severity of RHs. RHs were observed in 39.4%. The study concluded that SVD, a prolonged second stage of labour, advanced maternal age and neonatal intracranial haemorrhage were associated with an increased risk of RHs.

Pu et al (2017) performed a prospective study of 3123 infants delivered after high-risk pregnancy or identified neonatal asphyxia. The overall incidence of RHs was 18%. Delivery mode (SVD or instrumental) and neonatal asphyxia were found to be risk factors for RHs in higher risk infants[260].

Kara et al (2018) reported a case in which the utility of Optical Coherence Tomographic (OCT) imaging for documenting foveal RH resolution was demonstrated[258].

Kim et al (2018) performed a retrospective comparative study of RetCam images of infants with RHs following AHT and NVD. They found that RHs in both groups shared similar characteristics but that RHs associated with AHT were significantly larger and more likely to involve all 3 layers of the retina[154].

Simkin et al (2019) in a prospective neonatal study of 346 infants found RHs in 50 (14.5%) and also noted RHs were more common after SVD in comparison to caesarean delivery[262].

Ji (2019) in a prospective cohort study of 234 new born infants found RHs in 17.5% and sub-conjunctival haemorrhages (SCHs) in 9%. They noted no correlation between the presence of RHs and SCHs. SCHs were associated with high birth weight but not instrumental delivery[274]. RHs were more common after instrumental delivery. They postulated different pathophysiological aetiologies for RHs and SCHs with chest compression of larger infants in the birth canal, with concomitant increased venous pressure, suggested as a factor underlying SCH production.

Eris et al (2019) retrospectively studied RetCam images of 148 eyes of 74 infants with hypoxic ischaemic encephalopathy (HIE) treated with therapeutic hypothermia. They found an overall incidence of 24.3%. Resorption of RHs was noted by 38 days. RHs were more frequent in those infants with severe HIE. It was suggested that hypoxia may be a factor affecting severity of retinal bleeding[168].

Rossin et al (2020) described an infant born at 31/40 by SVD with moderate RHs and a subarachnoid haemorrhage (SAH) thought to relate to birth. The RHs were noted to enlarge and evolve. The SAH and its effect on ICP was suggested to have influenced RH genesis and evolution[261].

Mao et al (2020) demonstrated that neonatal RHs can be graded objectively and accurately via deep convolutional neural network model[275].

**Conclusions:** **B,D**

Recent papers confirm that RHs are common in neonates affecting up to 40% of infants born by SVD. Most resolve rapidly within 2 weeks. They are more prevalent after assisted births, particularly those involving vacuum extraction, but are less common after elective caesarean sections. Subconjunctival haemorrhages in seen in newborns in 9%.

Obstetric and perinatal risk factors for RHs include older maternal age, neonatal intracranial bleeding, and hypoxic ischaemic encephalopathy.

The use of modern technologies including wide-field imaging, OCT and deep neural network analysis of images can assist objective assessment.

### 3.3.4. Bleeding diathesis or blood dyscrasia

**Clinical question: Can bleeding diathesis or blood dyscrasia cause retinal haemorrhages similar to those seen in child abuse?**

**Evidence from previous review 1999 & 2012**
A report suggests that a bleeding diathesis should be excluded in all suspected abuse cases[276]. RHs in leukaemia can be difficult to distinguish from those in AHT, but the abnormal white cell count would help to distinguish leukaemia from AHT[216,277]. Extensive RHs have been described in Von Willebrand's disease[278], vitreous haemorrhages in protein C deficiency[279,280]. Bleeding disorders may co-exist with Abusive Head Trauma[281,282], and a haematological opinion should be sought.

A case of minor head injury in an 11 month old who fell backwards from standing height onto a wooden floor, sustaining extensive SDH and bilateral, extensive, multilayered RHs. Original investigations for coagulopathy were normal, abuse was diagnosed, but six months later a re-evaluation of the bloods suggested mild von Willebrand's disease which was then accepted as a cause of the bleeding[283].
A 14 year old boy with acute myeloid leukaemia is described as having bilateral, extensive multilayered RHs, including perimacular folds, in the absence of trauma[284]. Other bleeding disorders with RHs described include Hemansky- Pudlak syndrome[285], where a seven week old infant presented with SDH and RHs in the posterior pole, involving subhyaloid, intraretinal, and subretinal on one side, and a single macular haemorrhage in the other eye; a case of haemophagocytic lymphohistiocytosis in an 11 day old, who presented collapsed with SDH and peripheral perivascular RHs[286]; a 36 day old pre-term infant being examined for possible ROP who was found to have bilateral retinal and vitreous haemorrhage in the absence of ROP with low fibrinogen levels[287]; an eight year old boy who presented four months after bone marrow transplant for aplastic anemia, who was noted to have atypical ischaemic maculopathy, including scattered flame shaped haemorrhages bilaterally[288].

**Evidence from update 2022**
Bleeding disorders: Thau et al[265] conducted a review of reported retinal haemorrhages in bleeding disorders and found that RH have been reported in Vitamin K deficiency, disseminated intravascular coagulation, thrombocytopenia, platelet function disorders, factor 1, Factor V, factor VIII, factor IX and factor XI deficiency. These haemorrhages may be unilateral or bilateral and be distributed in the posterior pole or periphery of the retina. They have been described as sub hyaloid, superficial, intraretinal, subretinal or multi-layered. The authors argue that coagulopathy may be seen frequently in head trauma and are not the cause of RH. However, in the presence of intracranial haemorrhage and RH they cite the report on standardised tests that may be performed to evaluate a bleeding disorder where there is a suspicion of abusive injury.

**Conclusion:** **D**

Bleeding diathesis should be excluded in all suspected cases of AHT with RHs.

### 3.3.5. Confounding conditions

#### 3.3.5.1. Seizures

**Clinical question: Can seizures alone cause retinal haemorrhages as seen in cases of child abuse?**

**Evidence from previous review 1999 & 2012**
There were no retinal haemorrhages in 560 cases of adult seizures[289]. There were no retinal haemorrhages seen in 65 children after seizures[116,147]. There was one case of extensive retinal haemorrhages in an adult after status epilepticus[290]. It was considered that convulsions alone in children rarely, if ever, cause retinal haemorrhages.

A number of studies have looked at children presenting with recent onset seizures. Known cases of trauma were excluded and an ophthalmologist performed a fundus examination with an indirect ophthalmoscope within 48 or 72 hours of admission. In a study of 153 children aged less than two years, only one retinal haemorrhage in an eight month old child was detected[291]. A study of 31 children reported two children each with only one retinal haemorrhage[292]. A further study examined 182 children under two years and found retinal haemorrhages in two cases that also had subdural haemorrhages (SDH) and were considered to be due to inflicted head injury[293]. The reported cases of RH attributed to convulsions seem to be confined to very few haemorrhages located close to the optic discs.

**Conclusions:** **D**

Retinal haemorrhages are rarely caused by seizures alone. If RHs are found in a child with convulsions, this finding should prompt a search for another cause.

**Evidence from review update 2022**
No additional studies were included in this update.

#### 3.3.5.2. Cardio-pulmonary resuscitation (CPR)

**Clinical question: Can cardio-pulmonary resuscitation cause retinal haemorrhages?**

**Evidence from previous review 1999 & 2012**
Closed chest massage, even by trained doctors, can cause a marked rise in intracranial pressure[294]. Violent chest compression in child abuse may cause retinal haemorrhages[174,175]. Attempted CPR by untrained individuals has been advanced as an innocent cause of retinal haemorrhages in a single case report[295] although AHT was not convincingly excluded.

There have been other reports of retinal haemorrhages occurring after CPR without other explanation for the haemorrhages[145,296]. One neonate with meconium aspiration who had CPR during the first day of life before being put onto ECMO suffered seizures with vitreous haemorrhages being noted on the 11th day. A further study found no case of retinal haemorrhage in their autopsy study of 169 cases, 131 of which had prolonged resuscitation[297].

Retinal haemorrhages were found on non-mydiatric ophthalmoscopy in only 1 of 45 children who had not had prior trauma and were successfully or unsuccessfully resuscitated: the child with RHs had severe arterial hypertension and seizures. Of 9 trauma victims, 5 had retinal haemorrhages; 4 of these were correctly suspected as being due to child abuse and the other had head and chest injuries in an

automobile accident. A further 5 children could not be examined who had corneal clouding or miosis. The conclusion was that 'when retinal haemorrhage is detected in the paediatric patient after CPR, prior trauma should be assumed and retinal haemorrhage should not be attributed to the mechanical effects of CPR[175]. Experimental evidence from studies on piglets showed no retinal haemorrhages despite high, monitored intra-cranial and intra-thoracic pressure[90].

A study reports a case of a preterm infant with retinopathy of prematurity (ROP) that had bilateral retinal haemorrhages following CPR. The infant was a black male infant born at 24 weeks with bilateral stage 3 ROP. His left eye had 7 clock hours of stage 3 plus and some intra-retinal haemorrhage with the right eye having no retinal haemorrhages. During intravenous sedation in preparation for laser treatment, the child suffered severe apnoea and needed 15 minutes of chest compression. Fundoscopy 90 minutes later showed multiple flame-shaped intraretinal haemorrhages, some with white centres, diffusely distributed throughout entire vascularised retina (including the posterior pole) in both eyes. The child's immature retina may have been a factor in the development of these haemorrhages[298].

CPR is very unlikely to cause retinal haemorrhages, even if carried out by unskilled individuals. A caveat could be added to exclude very premature children with acute ROP on the basis of the case report.

**Evidence from review 2022 update**

Further evidence from a prospective study of 38 children aged 1 week to 17 years who were administered CPR reported RHs in 3.2% (1 of the 31 cases without a co-existing medical condition to explain the RHs) that could be associated with CPR. This case had an out of hospital cardiac arrest from drowning and had a single unilateral RH which was superficial and peripapillary. The age of the child was not reported. The average duration of CPR in this study was 10 minutes, and a fundus examination was completed within 48 hours of cardiac arrest. Though a total of 7 children were reported to have RH, 4 were diagnosed as AHT from non ocular features such as rib fractures, SDH, SAH and abdominal injury (RH numerous diffuse, multi-layered, posterior pole and periphery, 3 with bilateral and one unilateral findings). One was associated with a ruptured intracranial AVM (RH were 4-8, peripapillary, intraretinal, preretinal and bilateral) and 1 child had septic shock (RH were 1-2 intraretinal posterior pole bilateral). One child had an unwitnessed cardiac arrest due to drowning and had a single unilateral peripapillary intraretinal haemorrhage. The authors describe the patterns of RH in these cases as consistent with the diagnosis[299]. Levison et al[198] report an 8 week old child who had 35 minutes of CPR who was found to have bilateral multi-layered RHs in the posterior pole and periphery. This baby had laboratory evidence of disseminated intravascular coagulation (DIC). The autopsy revealed a cardiac anomaly causing a myocardial infarction with hypoxic brain injury and no subdural haemorrhage. In this case, the CPR, DIC and hypoxic brain injury could all have contributed to the development of RHs.

**Conclusions:** **D**

CPR alone continues to be unlikely in isolation to cause retinal haemorrhages, and when associated the pattern of RHs reported are mild. The additional presence of DIC and hypoxic brain injury may be a contributing factor.

### 3.3.5.3. Choking, gagging and vomiting

**Clinical question: Can prolonged vomiting, or gagging cause retinal haemorrhages?**

**Evidence from previous review 1999 & 2012**
Various causes of Valsalva manoeuvre may be associated with retinal haemorrhage in adults[300].

The medical literature in this area remains limited. Geddes and Talbert[301] published a hypothesis paper in 2006 suggesting a computer modelling approach to investigate feeding accidents as a trigger for intracranial and retinal bleeding. A dynamic circulatory model of a three-month-old infant was induced to "cough" and the response to changes in physiological variables monitored. The authors proposed that paroxysmal coughing could cause intracranial and intraluminal pressures to rise exponentially to approach a level which could be sufficient to damage veins. Barnes et al[76] reported a four and half month old male infant with no previous health problems in whom dysphagic choking was suggested as a component in the production of retinal haemorrhages and subdural haemorrhages. However, the authors indicated that the carer's account was inconsistent with the clinical and imaging features and that AHT could not be ruled out. Herr et al[82] examined 100 infants with hypertrophic pyloric stenosis. No retinal haemorrhages were identified in any of the children although one had developed facial petechiae and two had subconjunctival haemorrhages. However, although a dilated funduscopic examination was performed in all, an ophthalmologist did not necessarily carry this out.

**Evidence from review update 2022**
No additional studies were included in this update. However, a study of dysphagic choking and an ALTE (apparent life threatening event) as a cause of the features seen in AHT including RHs, concluded that ALTE's cannot be supported as a mechanism for the findings in AHT[1]. For more details see following section on ALTE/BRUE[302] (3.3.5.4).

**Conclusions:** **D,E**

RHs appear to be very rare (if they occur at all) as a result of Valsalva manoeuvre in young children.

### 3.3.5.4. Apparent life-threatening event (ALTE) or brief resolved unexplained event (BRUE)

**Clinical question: Is an apparent life-threatening event associated with retinal haemorrhages?**

**Evidence from review update 2012**
An apparent life-threatening event in an infant is defined as a condition characterized by some combination of apnoea, colour change, marked change in muscular tone, choking or gagging and is frightening to the observer[79].

In a study of 128 infants[79] who presented to the emergency department with an ALTE where the mean age of the infants was 2.1months (range 0.07-16 months) 71 infants had a funduscopic examination by a paediatric ophthalmologist. There was one child in this group who had a diagnosis of abuse with bilateral RHs, rib fractures and a confession of vigorous shaking by the father. In a series reporting on the ophthalmic findings 120 children (less than 12months of age) who presented with an ALTE only the children diagnosed with AHT had retinal haemorrhages[77]. There were no retinal findings in 114 children with non AHT[77,78]. In a recent prospective study of 108 infants with a median age of 1.5 months (range 0.5-13.8 months) who presented with an ALTE a detailed fundoscopic examination by an ophthalmologist using indirect ophthalmoscopy and dilated pupils revealed no retinal haemorrhages.

Combining these studies reporting on 292 children less than 24 months who presented clinically with an ALTE, not due to AHT, none of the children had any retinal haemorrhages. ALTE has not been reported to be associated with RHs.

**Evidence from review update 2022**
In a study evaluating whether dysphagic choking/ALTE now called BRUE (brief resolved unexplained event) can be the causative mechanism of the findings seen in AHT, the authors studied a cohort of infants with subdural haemorrhages (SDH) with or without BRUE. BRUE/dysphagic choking has been hypothesised to caused hypoxic ischaemic encephalopathy and RH. The authors hypothesise that if this were true the extracranial findings of retinoschisis, abdominal injury and fractures reported in AHT should be absent in BRUE. They studied 170 cases of which 106 were those with SDH without BRUE and 64 cases with BRUE. They found that subjects with SDH and BRUE were more than 5 times likely than the SDH group without BRUE to manifest extracranial features (OR 4.8- CI:1.9-12.1). When looking at retinoschisis alone 16 in the BRUE group had retinoschisis versus 8 in the group without BRUE (OR 4.1; CI: 1.6-10.2). Hence the study concludes that BRUE/ALTEs or dysphagic choking are not supported as a causative mechanism for the findings in AHT which includes RHs[302].

**Conclusions:** **D**

The literature does not support BRUE (formerly known as ALTE) as a causative mechanism of retinal findings.

#### 3.3.5.5. Vaccinations and retinal haemorrhages

**Clinical question: Are vaccinations associated with retinal haemorrhages?**

**Evidence from review update 2012**
A number of authors have hypothesized a link between the presence of multiple bone fractures, subdural haemorrhages and retinal haemorrhages and routine vaccination due to elevated histamine and reduction of vitamin C[303,304]. However, this remains unsubstantiated by clinical evidence and there are no reported cases in the literature of retinal haemorrhages occurring following childhood immunizations although cases of retinal vein occlusion have been associated with hepatitis B vaccination in adults[181,305]. The association of RH and vaccinations has not been reported in children.

**Evidence from review update 2022**
A study was undertaken to test the hypothesis whether vaccinations in children cause retinal haemorrhages. The authors retrospectively studied the 7675 fundus examinations of 5197 children less than 23 months. Complete immunisation records were available in a subset of 2210 children who had 3425 funduscopic examinations. There was prevalence of 0.18% of RH (4 of 2210) with all these children having AHT diagnosed from non-ocular clinical findings. There were no RH observed within 7 days of immunisation, 1 child had RH within 14 days and no additional child had RH within 21 days. There was no association of RH with vaccinations in children[306].

**Conclusions:** **D**

The literature does not support vaccinations or immunisations as a cause retinal haemorrhages.

### 3.3.6. Other causes of retinal haemorrhage

#### 3.3.6.1. Cervical (neck) injury and retinal haemorrhages

**Clinical question: Do high cervical injuries from any other source give rise to retinal haemorrhages?**

**Evidence from previous review 2004 & 2012**
Cervical injuries alone did not give rise to retinal haemorrhages but could give rise to apnoea. Inflicted cervical spinal injury coupled with circulatory collapse had the potential to produce hypoxic ischaemic encephalopathy[307,308].

Two studies reported the results of biomechanical experiments on whether forceful rocking or bouncing in baby seats could account for subdural and retinal bleeding and may involve high cervical injury in the process. The experiments were conducted to assess a carer's account about how infants under their care suffered injury. Both infants were eight weeks of age and had suffered bilateral subdural and bilateral retinal bleeding. The retinal bleeding was not described by an ophthalmologist in either case.

The outcomes in the two experiments differ with Jones[46] reporting that violent rocking was insufficient to cause the threshold values for subdural bleeding, whilst the study by de San Lazaro[64] reported that violent rocking in a baby chair (which may have included head impact on the floor) could give rise to a sufficient acceleration/deceleration to cause head injury. This study therefore gives the mechanism as acceleration /deceleration in the baby chair similar to shaking.

A case report of a 21 month old child who died after a short fall was found at autopsy to have a fatal cervico medullary cord lesion and a cranial impact site[210]. The child suffered bilateral subdural and subarachnoid haemorrhage with bilateral retinal bleeding. The child was reported to have had bilateral retinal haemorrhages and perimacular folds whilst on intensive care, but at autopsy the retinal bleeding was identified in the ganglion cell layer, more anteriorly than posteriorly, with bilateral optic nerve sheath haemorrhage. The child was confirmed as having raised intracranial pressure on intracranial monitoring.

**Conclusions:** **D,E**

These studies describe a mechanism of injury, which may involve the cervical cord but do not prove that high cervical injury alone can give rise to retinal bleeding.

**Evidence from review update 2022**
No additional studies were included in this update. Though there is recent evidence of a higher prevalence of radiological injury to the high cervical spine in AHT compared to accidental injury and non-traumatic cases where the spine was imaged the retinal findings were not reported[309].

### 3.3.6.2. Short distance falls

**Clinical question: Can short distance falls cause retinal haemorrhages?**

**Evidence from previous reviews 2004 & 2012**
Tangential acceleration associated with shearing forces is well documented as causing retinal haemorrhages but the situation is less clear with short distance falls. Short distance household falls were found to be neurologically benign with no associated retinal haemorrhages even in the presence of skull fractures[15,22]. Accidental trauma, or rough play would be unlikely to cause retinal haemorrhages in children less than two years of age[38,39].

A few reports have described unilateral or bilateral haemorrhages in children following short falls[48,53]. However, it seems clear that minor falls can, only exceptionally, give rise to subdural and retinal bleeding (see section 3.1.3).

A group studied the association of haemorrhages and epidural haematomas associated with accidental head trauma following short distance falls. Of the nine children included in the study, five were found to have retinal haemorrhages. These were all superficial, few in number, confined to the posterior pole and no deep or sub retinal haemorrhages were noted. The authors pointed out that all the examinations were done after surgical evacuation of the haematoma that may have had an impact on the development of the haemorrhages[186].

Minor head trauma, in the absence of underlying medical conditions such as osteogenesis imperfecta type I, is only very rarely associated with severe intracranial injury or retinal haemorrhage, particularly the extensive multi- layered haemorrhages extending to the ora serrata, as seen in two thirds of the victims with SBS[310]. In the absence of a known major accidental injury involving severe intracranial trauma, such as a crush injury or high force MVC, extensive retinal haemorrhages are highly indicative of occult, severe intentional injury[30]. Although the rate of retinal haemorrhage after severe accidents such as motor vehicle accidents is higher, the medical literature continues to confirm the rarity (<3%) of haemorrhagic retinopathy after other forms of minor accidental trauma. When it does occur, the haemorrhages are usually few or at most modest in number and rarely extend beyond the posterior pole[311].

A prospective study that looked at the incidence of RH in 154 children younger than two years that had sustained a vertical fall. The prevalence of RH was found to be 1.95% (three patients). Of these, all were unilateral and associated with epidural haematomas and a midline shift. They concluded that accidental short distance falls could result in RH but these were unilateral, small and isolated as opposed to the diffuse, deep and bilateral RH seen in AHT[52].

A retrospective review of 287 children found that for falls less than four feet, none of the children in the accidental group had RH and 25% of those in the abused category did. They concluded, "RHs are virtually never seen in short falls" [120]. A prospective study that looked at 45 cases of confessed inflicted head injury and 39 cases of accidental trauma found RH in 56.8% and 15% in the two groups respectively. In the accidental trauma group, five of the six cases had mild, flame shaped haemorrhages while one case that sustained facial trauma and direct impact to the globe had a severe RH[35].

A case of an 11-month-old Asian infant who sustained RH and SDH following a witnessed fall backwards from a sitting position has been reported. However, the ophthalmic examination was carried out following surgical evaluation of the intracranial hematoma that may have affected the RH[45].

An update on SBS reported that extensive retinal haemorrhages are not caused by short falls[312].

A prospective study looked at 87 children hospitalised with head trauma over a two-year period and divided them into accidental and AHT groups. Retinal haemorrhages seen in children with accidental head trauma were most often unilateral, involved a single retinal layer and in 42% of cases were a single haemorrhage. In contrast, abusive head trauma is associated with multiple RHs, usually bilateral, involving the preretinal and intraretinal layers, covering the macula and extending to the periphery of the retina. Thus it is not the presence of RH but the location and number that is most helpful in distinguishing accidental from inflicted head injury[130].

**Evidence from review update 2022**

Raj et al present the case of an infant with confirmed accidental trauma sustained from an adult-worn baby carrier fall with superimposed head crush injury, resulting in significant intracranial, and retinal findings. On examination, the 2-month old infant was awake and irritable with a full anterior fontanelle and scalp swelling, with no other external signs of injury. CT of the head revealed biparietal skull fractures with subjacent soft tissue swelling and bilateral subdural hematomas but no orbital fracture. Subsequent MRI revealed bilateral subdural hematomas and scattered foci of parenchymal injury. Retinal examination by a paediatric ophthalmologist with indirect ophthalmoscopy revealed numerous intra- and preretinal haemorrhages bilaterally, located primarily in the posterior pole, with fewer more anteriorly in the retinal periphery. There was no retinoschisis or retinal fold. A skeletal survey did not detect any additional injuries. This case likely represents a combination of short distance fall plus superimposed crush injury[214].

Minns et al, reported children admitted to PICU from a variety of causes including AHT and accidental causes. While RHs were found in some children who fell from significant height, no RHs were found in 6 children (12 eyes) who had falls of <1m[124].

**Conclusions:** **C,D**

RHs associated with short falls are rare. Short distance falls are unlikely to cause retina haemorrhages if the injury is not severe or associated with crush injury. In rare cases accidental falls, especially those associated with SDH may be associated with RHs, but these tend to be unilateral, localised and superficial.

#### 3.3.6.3 Raised intracranial pressure

**Clinical question: Do retinal haemorrhages similar to AHT occur with raised intracranial pressure?**

**Evidence from review update 2012**
Animal studies have demonstrated that retinal haemorrhages can be induced by a rapid marked rise in intracranial pressure (ICP) to the point where consciousness is impaired[313].

In adults humans, retinal and optic nerve sheath haemorrhage occur when there is very marked increase in intracranial pressure[314], with emphasis on rapidity of onset[195,315,316].

A number of case reports provide additional evidence that retinal haemorrhages occur with acute extreme ICP rise.

In two adults studies inadvertent iatrogenic experiments of this phenomenon have been reported. A patient suffered brief acute ICP rise secondary to compression of a meningocoele during a routine medical investigation, resulting in bilateral retinal haemorrhages[317]. Another patient developed bilateral retinal haemorrhages when subject to accidental severe elevation in preoperative intraventricular pressure[318].

There have been two case reports demonstrating that infants develop extensive retinal and optic nerves heath haemorrhages with severe ICP rise[152,184]. The retinal haemorrhages in the two infant cases were different to those reported in adults, with a wider distribution throughout the retina, and presence in different layers[152,184]. Both infants who were 7months old had ruptured intracranial aneursyms with subarachnoid haemorrhages. In one infant[152] the ICP that was measured was high and this infant had extensive unilateral, multilayered retinal haemorrhages extending from the posterior pole to the periphery of the retina. In the second infant[184] who died, acute raised ICP was diagnosed on the postmortem findings of diffuse cerebral oedema and a subarachnoid haemorrhage. This child had bilateral multiple multilayered retinal haemorrhages which were distributed in the posterior pole and periphery.

The intracranial bleeding present in these cases does not seem to be the direct cause of retinal haemorrhages: only one child, aged 7 years, developed retinal haemorrhages in a series of 57 children with intracranial haemorrhage, and, whilst not explicitly stated in the article, this child had marked intracranial midline shift and/or large volume parenchymal cerebral haemorrhage that may have reflected marked intracranial pressure elevation[54].

As part of a wide-ranging study a series of children with apparent abusive head trauma, evaluation was made to see if there was a correlation between presumed signs that might suggest raised intracranial pressure and eye findings[36]. No relationship was identified however intracranial pressure was not measured. Instead, a correlation was found between severity of cranial injury and eye findings.

Experimental and clinical data demonstrate that an acute dramatic rise in intracranial pressure (ICP) may produce unilateral or bilateral retinal haemorrhages in infants that resemble those reportedly caused by abusive head trauma.

**Evidence from review update 2022**
A prospective study of children with atraumatic elevated ICP confirmed by lumbar puncture evaluated dilated fundus examinations for retinal haemorrhages. The mean ICP was 35 cms of H2O (range 20-56 cms of H2O). Of the 100 children studied 74 had papilloedema of whom 16 had RH (21.6%). All 16 had a very high ICP with a mean of 42 cms of H2O. The pattern of RH was peripapillary, superficial, and intraretinal haemorrhages adjacent to the swollen optic disc in 8 cases and splinter haemorrhages on the disc in further 8 cases. All cases were examined within 48 hours of the lumbar puncture. Four children had associated cotton wool spots and lipid exudates. No children had RH beyond the peripapillary area, in the macula, along the vascular arcades or in the periphery. There were no children with retinal folds or retinoschisis. This study should be interpreted with caution as the children included ranged from 3-17 years and the exact duration of elevated ICP was not known, as symptoms ranged from days to months[319]. It is possible that ICP elevation was not acute.

A more recent prospective multicentre study of nontraumatic causes of raised ICP in 56 children younger than 4 years reported no retinal haemorrhages[320]. Only one child in this group had papilloedema. The causes of increased ICP included hydrocephalus, intraventricular haemorrhage, congenital malformations, malfunctioning shunts. The presence of intracranial space-occupying lesions and raised ICP was diagnosed by opening pressures, clinical criteria and other clinical signs of raised ICP. It was estimated that the ICP was raised from 4 hours to 27 weeks.

**Conclusions:** **D**

RH are rare in raised ICP in nontraumatic causes. RH are not seen without papilloedema. When present they are small, do not extend beyond the peripapillary area and are associated with papilloedema.

### 3.3.6.4. Crush injury

**Clinical question: What are the ocular findings in crush injury?**

**Evidence from previous review 2012**
A crush head injury occurs when the head is trapped between two objects or hard surfaces resulting in the application of bilateral, static, compressive forces on the head. The forces are applied more slowly and over a larger area compared to impact injury and result in deformation of the skull with cranial fractures[210].

There have been a small number of case reports and a retrospective review of the ocular findings in crush injury to the head in children. In the case reports all the children had sustained skull fractures.

Lantz et al[131] described a 14-month-old child who died after head trauma sustained by a TV set weighing 19.5 kg falling on top of him, which was determined as an accident after a forensic recreation of the scene. The child was found to have extensive bilateral retinal haemorrhages (dot and blot intra retinal and preretinal), which extended out to the ora with perimacular retinal folds. At autopsy optic nerve sheath haemorrhages were identified.

Lueder[213] reported a witnessed crush head injury in a four-month-old child sustained when a 12-year-old weighing 63 kg fell onto the infant's head. Ophthalmological examination revealed vitreous haemorrhage on the right side, and in the other eye retinoschisis with perimacular folds and diffuse, extensive, multi-layered retinal bleeding. Forensic examination determined that the injury was accidental.

Watts and Obi[56] reported the eye findings in a witnessed crush injury in 10 week-old infant and compared this with the ophthalmic examination in a 14 week- old infant with abusive head trauma and noted that similar ophthalmic findings could be seen in the two conditions. The ocular findings in the infant with crush injury were bilateral asymmetric retinal haemorrhages with a small number of posterior polar haemorrhages in one eye and in the other eye multi-layered, extensive retinal bleeding extending from the posterior pole to the ora, with retinoschisis and retinal folds. The child with abusive head trauma had one retinal haemorrhage in the right eye and in the left there were multi-layered, extensive retinal haemorrhages extending to the ora with a macular retinoschisis and retinal fold. Retinal photographs of the two cases show an almost identical fundal appearance.

Gnanaraj and colleagues[31] undertook a retrospective clinical and pathological review of paediatric crush head injuries. They reviewed two groups of patients, 16 children admitted to the hospital over a ten-year period 1992-2002 with head injuries caused by falling televisions (TV) and nine children who died from crush head injury between 1982-1989. In this time period 400 children had died and 169 were prospectively studied, and the nine children with crush head injury were identified from within this prospectively studied group. In the falling TV group 11/16 children had fundus examinations and one child had multiple preretinal and blot retinal haemorrhages extending from the posterior poles to the equator (it is not clearly stated whether this was unilateral or bilateral); this child had skull fractures. Four of the nine children dying of head injury had retinal haemorrhages, confined to the posterior pole in three patients and out to the ora in the other patient (who had been an unrestrained passenger involved in a road traffic accident). It is variably stated that all or 8/9 of these children had multiple skull fractures. The child with haemorrhages out to the ora also had optic nerve sheath haemorrhage. No child in either group had retinoschisis or retinal folds.

These cases indicate that extensive, multi-layered, bilateral retinal bleeding, or bilateral asymmetric bleeding, retinoschisis and perimacular folds can occur in isolated cases of accidental crush head trauma. Few to widespread RHs have been reported in crush injuries to the infant head.

**Evidence from review update 2022**
Raj et al report a 2-month-old female who sustained a crush injury when her grandmother who was carrying her in a front holding baby carrier fell crushing the baby's head between her and a hard surface. The accident was witnessed by three independent witnesses. The baby was noted to have a soft tissue haematoma, multiple skull fractures and bilateral subdural haematomas. A paediatric ophthalmologist who examined the baby's eyes with an indirect ophthalmoscope reported bilateral multi-layered (intra retinal and pre retinal) haemorrhages mainly in the posterior pole with fewer in the periphery. Because of the intracranial and eye findings the child abuse team were involved and abuse was ruled out[214]. (see sections 3.3.1 & 3.3.2)

**Conclusions:** **D**

This report provides further evidence of RH in cases of accidental crush injury to the infant's head.

### 3.4. Imaging of the retina in AHT

**Clinical question: Which methods are useful in imaging the retina in AHT?**

**Evidence from previous review 2012**
Photography of retinal findings in abusive injury supplement detailed clinical descriptions while providing a permanent patient record. Hand held non-contact cameras have been used to capture high quality details of the posterior pole but do not lend themselves to imaging the retinal periphery. The RetCam (Clarity Medical Systems, Pleasanton, California) a contact digital fundus camera providing a 130 degree of view of the retina allows documentation of findings in the posterior pole and periphery thus supporting the clinical notes on the patterns of retinal features in abusive head injury. In a pilot study the RetCam has been shown to provide high quality images at the bedside of retinal findings in abusive head trauma[321]. When used as a telemedicine tool in abusive head trauma the RetCam 120 degree had a 100% sensitivity and 85.7% specificity when compared to indirect ophthalmoscopy in detecting the presence or absence of retinal haemorrhages[119]. Caution has been suggested when interpreting retinal haemorrhages seen after a previous examination with an indirect ophthalmoscope and scleral depression or imaging with the RetCam where the examination may induce retinal haemorrhages[252,253]. A prospective audit on pre and post RetCam imaging in 50 children screened for ROP failed to show any retinal haemorrhages[254].

The Retcam images of abusive trauma have been used to study inter and intra observer variability classifying the type[322], development of a new zonal classification[323] development of a retinal tool[324] and development of standardized reporting system[325] for describing retinal haemorrhages which may be used for clinical documentation and as evidence in legal proceedings.

Optical coherence tomography (OCT) has been applied to examination of infants subjected to abusive injury to define the relationship of the vitreoretinal interface and the level of retinal pathology[85]. The development of the hand held spectral domain OCT (Bioptigen Inc, North Carolina) allows imaging of a supine infant and has been successfully used to define both acute[326] and chronic[327] vitreoretinal findings in infants with abusive head trauma.

Imaging of the retina in abusive head trauma supports detailed documentation of retinal findings and provides a permanent record of retinal findings. The use of the OCT helps in elucidating the vitreoretinal relationships in AHT.

**Evidence from review update 2022**
The capture of retinal haemorrhages through digital imaging lends itself to the application of deep learning tools and artificial intelligence to objectively locate and quantify retinal haemorrhages. A study by Mao et al found that the application of a new scoring system based on a deep convoluted neural network which automatically segments out the optic disc and retinal haemorrhages and locates the macular region and calculates the ratio of retinal haemorrhage area to optic disc size can more accurately grade retinal haemorrhages than older grading systems[275].

In a study by Donaldson et al [121] where 57 children were investigated with the input of a multidisciplinary child protection team, 29 (50.9%) were determined to have had abusive head trauma as determined by the multidisciplinary child protection team. Sixteen were documented to have retinal haemorrhages of which 14 were imaged with a widefield digital retinal camera (RetCam®). They found retinal haemorrhages had a specificity of 55% and sensitivity of 68% for abusive head trauma. Retinal haemorrhages too numerous to count, multi-layered haemorrhages, and extension to the peripheral retina were more likely to be associated with abusive head trauma than other aetiologies. All cases of retinal haemorrhage were associated with intracranial haemorrhage and the finding of retinoschisis was only seen in abusive head trauma cases.

Bhardwaj et al[328] performed both indirect ophthalmoscopy and widefield retinal imaging in a total of 118 infants admitted with head injuries between the ages of 1 and 36 months. There were 21 cases of abusive head trauma, as determined by a child protection paediatrician and a multidisciplinary team, in which retinal haemorrhages were seen in 14 (66%) of cases and retinal folds or macular schisis were seen in 8 cases (38%). They noted that in 86 cases of accidental injury RH was present in only 2 cases (2%). Their imaging study confirmed that RH in infants with head injury had a high positive likelihood ratio for AHT. Severe haemorrhagic retinopathy, particularly if they were associated with perimacular folds or macular retinoschisis had the highest positive predictive value for AHT. However, they also documented cases of extensive multi-layered RH extending to the retinal periphery and peripheral haemorrhagic schisis in 36% of cases of indeterminate cause of head injury.

The distribution of densest RHs in their study was at the posterior pole, followed in decreasing order of density by the superior retina, nasal retina, and temporal peripheral retina showing the least dense distribution of haemorrhages.

A study by Minns et al [124] compared the number, location and layer of retinal haemorrhages found on RetCam image analysis in children with AHT as determined by multiagency child protection team, with those seen in accidental traumatic brain injury and children with non-traumatic encephalopathies. Significantly more RHs in all retinal zones with far greater numbers of dot-blot haemorrhages were seen in AHT. Moreover, the distribution of RHs were more peripheral in AHT than other aetiologies. The likelihood of AHT was far greater in those < 3 years and with greater than 25 dot-blot haemorrhages.

Morphological and temporal patterns of resolution of RHs can be defined by imaging which is important for medicolegal reasons. A study by Jones et al[156] showed that there were two main forms of resolution with one form, "pattern A" seen in 60% of cases of RH's, where there was a gradual and progressive decrease in the size of individual RH's and a second, asymmetrical form of decay, "pattern B" where an initial increase in the area of an individual RH was followed by a gradual decrease. An infrequently seen form of resolution, "pattern C", was characterised by a fluctuating increase and decrease in size over time. Follow-up imaging gave an indication of time to clearance of haemorrhages, which was found to be around 13.7 mean days (range 1-57 days) in "pattern A" RHs and was seen to be quicker for intraretinal haemorrhages (mean 12.0 days; range 1-57 days) compared to pre-retinal haemorrhages (mean 29.0 days; range 10-57 days). For "pattern B RHs" the mean (range) number of days from the day of first imaging to the day of maximum haemorrhage area was 2.7 (1-35) days. For intraretinal RHs the mean (range) number of days from the day of first imaging to the day of maximum haemorrhage area was 2.2 (1-21) days. For preretinal RHs the mean (range) number of days from the day of first imaging to the day of maximum haemorrhage area was 9.8 (1-35) days. Subsequent clearance of "pattern B" haemorrhages from their maxima followed a similar time course to pattern A with intraretinal RHs clearing at a mean of 8.2 (1-52 days) and for pre-retinal RHs 28.3 days (21-42 days). Of note in their study, whilst the area of individual haemorrhages could increase during follow up, there was no increase in the total number of haemorrhages seen. The authors commented that no inferences could be made by interpreting the area, or change in area, of individual retinal haemorrhages in relation to the timing of the inciting event.

The area of retina covered by retina can be analysed in photographs. It has been shown that the haemorrhage covered fraction of the retina (a 40-degree field centred on the central retina) is statistically greater in definite abuse cases compared with indeterminate cases and also in cases associated with axial skeletal fractures or severe intracranial injury[329]. Furthermore, imaging studies clearly distinguishes the characteristics of retinal haemorrhages caused by raised intracranial pressure (ICP) and those caused by trauma. In 100 children (mean age 12 years (range 2-17 yrs)) with raised ICP, 93 were imaged within 48 hours of undergoing a lumbar puncture. Optic disc swelling was present in 74 % overall, but in those with opening pressures of > 20 mmH2O or > 28 mm H2O, 18 (56%) had disc swelling. 16 children had retinal haemorrhages which were described as splinter haemorrhages on the optic disc (8 children) or superficial intraretinal haemorrhages in the peripapillary retina (8 children).

Mean opening pressure was > 40 cmH2O in these cases. No children had RH in the central or temporal macular area, along the retinal vascular arcades distal to the peripapillary region or in the retinal periphery. No cases of pre-retinal or subretinal haemorrhages, retinoschisis or macular folds were observed. They concluded that a small number of children with elevated intracranial pressure with florid papilloedema had haemorrhages which were restricted to optic disc or peripapillary area[319].

Whilst contact wide-angle digital retinal cameras have dominated the published literature on retinal imaging in AHT, a small series of 10 eyes from 5 infants aged from 1 month to 15 months were successfully imaged using the ultra-widefield Optomap non-contact digital retinal camera (Optos P200MA scanning laser ophthalmoscope). Four out of 5 infants were imaged without the requirement of sedation, using a carefully devised imaging protocol to optimise imaging and monitoring of infants. Only 1 infant required sedation. The described advantages of the Optomap imaging included better quality imaging due to confocal optics, requirement for just a single image to capture a 200-degree horizontal field facilitating a speedier process of image capture and avoidance of applying pressure on the globe as would be the case with traditional contact imaging devices. A significant barrier to the use of the Optos P220MA device is the lack of portability and inability to use in a supine infant such as typically encountered in paediatric intensive care facilities[330].

Use of the hand-held OCT to demonstrate longer-term changes in retinal structures in severe cases of AHT associated with dense bilateral multi-layered retinal haemorrhages has been reported. Focal posterior vitreous separation and disruption of the retinal layers at the macula was demonstrated along with correlative abnormalities in electroretinography[331].

Non-ophthalmoscopic imaging techniques have also been described that may detect features retinal features of AHT. These include ultrasound scans of the globe which may reveal the presence of traumatic retinoschisis up to 60 hours before dilated retinal examinations can be performed. The advantages include earlier detection following admission, where use of pharmacological pupil dilatation may be prohibited due to the requirement for initial neuro-observations. The disadvantages pointed out with this technique is the lack of evidence that milder forms retinal injury following abusive head trauma, not associated with retinoschisis, may be detected[332].

Retinal haemorrhages may also be detected on MR scans. A form of gradient-echo imaging, called susceptibility weighted imaging (SWI), which is a volumetric three-dimensional sequence tailored to be particularly susceptible to signal heterogeneities within the magnetic field induced by paramagnetic and magnetic materials, such as haemosiderin and calcium, demonstrated RH's in 62% of infants with RHs detected by dilated fundus examinations using a standard protocol. However, through implementing an orbital protocol the detection rate increased to 80%. The MRI standard brain protocol had a sensitivity of 75% which increased to 83% for the orbital SWI protocol with a specificity of 100% for both protocols. The orbital imaging technique was less able to detect retinal haemorrhages when there were less than 5 haemorrhages and if these were intraretinal or subretinal, whilst pre-retinal haemorrhages were more easily detected[333].

Another study using routine brain imaging protocol demonstrated a 61% sensitivity and 100% specificity in detecting retinal haemorrhages. Of the various MR imaging protocols used, gradient recalled echo showed the best ability to detect retinal haemorrhages, and specifically, high-grade retinal haemorrhages that are particularly associated with AHT. The authors stated that using a 3-T magnet was more effective at detecting haemorrhages than the 1.5-T magnet. The adoption of an axial thin-slice dedicated orbital gradient sequence was advocated[334].

The presence of optic nerve sheath subdural, intradural and subarachnoid haemorrhages detected by orbital 3-D SWI imaging has also been demonstrated in a separate preliminary study[335].

These MR studies demonstrate the potential for early detection of ophthalmic features of abusive head trauma prior to detection using conventional retinal examination and ophthalmoscopic imaging but require further refinement to establish definitive protocols.

**Conclusions:** **D**

Retinal imaging continues to support detailed documentation and provides a permanent record of retinal findings. Imaging studies demonstrate both qualitative and quantitative differences in the number and distribution of retinal haemorrhages associated with AHT compared with other aetiologies. Imaging also lends itself to future use of automated image analysis. The resolution of retinal haemorrhages associated with AHT undergoes two main patterns, with an increase in area prior to gradual resolution seen in some cases. No increase in number of haemorrhages is encountered.

Use of non-contact ultra-widefield scanning laser ophthalmoscope (Optos P200MA) provides high quality imaging but its utility remains limited at present. The opportunity to detect evidence of retinal haemorrhages through ocular ultrasound and MR scanning early in the evaluation of AHT may be considered as part of the child protection protocol. OCT imaging in AHT helps in elucidating the vitreoretinal relationship in AHT, and in the long term may provide structural and functional correlates.

## 3.5 Non vitreoretinal injury, fabricated or induced illness and neglect

**Clinical question: Which features or characteristics of non-vitreoretinal eye injury are present in child maltreatment, neglect and fabricated or induced illness?**

This subject has not been considered in previous reviews. The review identified 15 relevant papers. This included a systematic review and two large retrospective case series.

### 3.5.1. Non vitreoretinal injury from physical abuse

A published systematic review identified 5 relevant papers, three case series and two case reports (describing 26 children in total)[3]. Papers by DeRidder 2013[336], Spitzer 2005[337], Baskin 2003[1,338], Calzada 2003[339] are also included in this review in their own right. Skarbeck-Borowska 2011 was not included in our review.

This review highlighted a relative paucity of good quality data available on abusive eye injuries. It was notable that whilst there was a wide range of reported eye injuries due to abuse, all of the children had subconjunctival haemorrhage, either unilateral or bilateral, suggesting that this sign has value as a potential sentinel injury that should prompt further enquiry as to the possibility of abuse. The authors note alternative possible explanations for SCH such as infectious, haematological and neoplastic disease, Valsalva manoeuvre, vomiting, and accidental trauma. They consider that in the absence of these aetiologies non-accidental injury must be considered as a cause of eye injury. The authors also observed that 22% of the children had been seen in the preceding weeks with an ocular complaint and discharged, without maltreatment being recognised. This suggests that some child abuse could be prevented by increased awareness of eye injuries and early detection. Abusive ocular injuries were seen in children ranging in age from one month to fourteen years (in the older children this was described as severe corporal punishment but in current UK parlance this would be considered child physical abuse). The spectrum of non-vitreoretinal injuries described in child abuse included periorbital oedema, chemosis, subconjunctival haemorrhage, corneal epithelial loss, hyphema, cataract, and globe rupture. In the described cases, a high proportion of all children with abusive eye injury also had other injuries, such as bruising, fractures (including occult fractures detected on skeletal survey in infants), soft tissue injury, abdominal or intracranial injury.

A large retrospective study of children admitted to a tertiary children's hospital in Australia with any type of eye or adnexal injury, identified 747 children admitted in a 14-year period[105]. 39 (5.2%) of cases were indicative or highly suspicious for NAI, the remainder being classified as Accidental Eye Injury (AEI). Children with NAI were younger (mean age 5.8 months vs. 7 years for accidentally injured children). NAI accounted for 74.2% (26/35), 19.2% (5/26), 5% (2/40) and 6.8% (3/44) of eye injuries in children aged <6 months, 7 to 12 months, 13 to 18 months and 19 to 24 months, respectively. No eye injuries due to NAI were found in children >24 months. Males were more likely than females to suffer both AEI and NAI (AEI 68.2%; NAI 57.8%). The most frequent presentations of children with eye injuries due to NAI were cutaneous bruising/hematoma (20/38; 51.2%), seizures (16/38), unresponsiveness/pallor (16/38) (42.1% each) and head injury (15/38; 39.4%). There was no history of trauma in 84.3%, with a history of fall in the remainder. Nearly half of AEIs (254/516, 49.6%) occurred outside the family home. Blunt trauma (385/704; 54.6%) was the most common mechanism of injury followed by injuries from sharp objects (112/704; 15.9%). All cases of AEI provided a history consistent with the injury. All NAI-related eye findings involved retinal haemorrhages in all retinal layers including optic disc haemorrhages. Haemorrhagic macular schisis was seen in 20% (5/25) of NAI cases where wide field fundus photos were acquired (25/39, 64.1%). There were no cases of anterior segment or adnexal trauma. The most common eye finding in AEI was a closed globe injury (306/694; 44%), followed by adnexal injuries (297/694; 42.7%) and open-globe injuries (91/694; 13.1%). Retinal involvement was unusual in AEI, with retinal injury noted in 2.1% (15/704) including retinal haemorrhages in 1.2% (9/704). All of these also had multiple associated globe injuries including corneal laceration, hyphema, lens subluxation, vitreous haemorrhage, choroidal rupture, macular hole and commotio retinae. None had clinically apparent non-ocular injuries. Non-ocular injuries described in the NAI cases included subdural haemorrhage (SDH), skull fractures, long bone and rib fractures and hypoxic ischemic brain injury. Three children died.

**Subconjunctival haemorrhages (SCH)**

A retrospective case note review of 1466 children seen as an inpatient and who had been referred to a child protection team in a tertiary children's hospital, identified 14 children with SCH who were subsequently diagnosed with physical abuse[336]. 5 (36%) cases were bilateral. Median age was 6.5 months (range 1-68 months, mean 16.5 months). 10 (71%) had initially presented because of eye/face findings. None had RH or conjunctivitis. None had history of chest compressions, cough, vomiting or constipation. 11 (79%) also had bruising. Affected body areas included the face, ears, scalp, neck, back, abdomen, and extremities. 2 (14%) had intracranial injury, 6 had fractures, one had liver laceration, 1 had a subgaleal haematoma, 1 had dried blood in the nares but no other physical or radiological findings (circumstantial evidence suggested smothering), one had severe HIE but no other apparent injury, with SCH presumed to be secondary to raised ICP. Injury of lips/mouth was mentioned in 3 cases. There was no case where the SCH was the only cause for concern after full investigation.

A further 3 cases (aged 5w, 4mo, 5mo) are described where an infant presented initially because of bilateral SCH and was subsequently found to have features of abusive trauma[337]. One of these infants had facial petechiae, a bruise to abdomen and rib fractures. There were previous safeguarding concerns. Another had bruising to the back and rib fractures. The third had perioral petechiae. This infant later presented with femur and tibia fractures and bruising to buttocks, and it was considered that the prior eye findings were a missed sentinel sign of physical abuse.

**Case Reports:**

Various case reports described eye findings that were identified in children diagnosed with physical abuse. The prevalence of other non-ocular findings suggestive of abuse was very high which is reassuring in that it makes circularity of argument very unlikely (i.e. the eye findings were not the sole reason for the diagnosis of child abuse).

A case report of a 4-month infant presenting with corneal haze, initially treated with antibiotics and steroids, who re-presented at 8 months of age with signs of abuse including SDH, RH, facial bruising, rib fractures. At follow up at 16 months the infant had amblyopia, eccentric fixation, esotropia, reduced visual acuity. Breaks of Descemet's membrane were noted. It is postulated that the original presentation was the result of abuse[340].

Two infants where new-onset eye deviation (esotropia or exotropia) was the presenting feature of child maltreatment, including abusive head trauma. One 6-month infant had bilateral abduction deficits that were thought to be due to bilateral sixth nerve palsy. It was also considered possible that the deficits were secondary to increased intracranial pressure or a traumatic brain injury. Findings included optic neuropathy, vitreous haemorrhage, retinal haemorrhage. This infant had no external features of trauma but was identified as having bilateral subdural hematomas of mixed ages, healing fractures of the bilateral proximal tibias and fibulas, as well as a healing right distal radial buckle fracture. The second case involved a 4½ month infant with progressively worsening exotropia for over a month. Retinal haemorrhage was present. Head circumference had increased over that time. The strabismus was attributed to his obscured visual axis due to a large pre-retinal haemorrhage, and resolved at follow-up. Again, there were no external features of trauma, but the infant was found to have bilateral, chronic SDH, features of chronic increased intracranial pressure and multiple long bone fractures (CML's)[341].

A 13-week infant presented with clinical features of severe AHT, that at follow up age 17 months was noted to have superior oblique underaction (CN IV palsy) bilaterally and RH. No squint had previously been noted. The authors hypothesise that this was more likely to have been secondary to trauma than a congenital palsy. At presentation the infant had a history of fall. Non-ocular findings included SDH, SAH, healing CML[342].

A case series of 7 children who had traumatic hyphaemas secondary to inflicted belt injuries. Other features in these children included corneal blood staining (2 cases), vitreous haemorrhage, traumatic cataract, face and back ecchymoses, commotio retinae, angle recession 320°, traumatic mydriasis. A range of outcomes was observed ranging from small hyphaemas with normal intraocular pressure and no vision loss, that resolved, to injuries with severe elevations of intraocular pressure and permanent, significant loss of vision. 3 of 7 cases had poor vision outcome, one underwent vitrectomy. The study highlights that severe consequences can arise from injuries inflicted using a belt[339].

A single case of a 2-month infant with Stickler Syndrome (COL2A1 mutation) that suffered AHT and subsequently had periorbital ecchymoses, vitreous haemorrhages, hyphaemas bilaterally, bilateral rhegmatogenous retinal detachments from giant retinal tears. There were no RH. There was a history of shaking by a family member. The infant also had rib fractures with different stages of healing, chronic cortical contusions and SDH[343].

A single case report of a 3-month ex-premature infant presenting at routine examination with impaired vision, found to have flare/cells in anterior and posterior chambers, subluxation and opacification of lenses and complete retinal detachments. The infant also had an unexplained healing 4cm cut below the right auricle. Abuse was apparently admitted by the caregiver[344].

A single case report of a 4-month infant who presented after an alleged fall, with findings of dense bilateral cataracts, lens dislocation, retinal detachment, vitreous haemorrhage. The infant also was found to have bruising to the torso, extremities and face, clavicle and rib fractures of various ages, resolving intracranial haemorrhages on MRI[345].

Two cases involving unilateral retinal detachment with other features suggestive of child abuse. Other eye findings included raised intraocular pressure, hazy cornea, retinal detachment and giant tear, hyphaema, swollen eyelid with injected conjunctiva. Multidisciplinary assessment led to the infants being safeguarded, with circumstantial evidence suggesting increased risk of maltreatment. Neither had external evidence of trauma. MRI showed SAH around the left temporal lobe in one case[346].

A case report of a 9-week infant presenting with hazy, enlarged corneas and raised intraocular pressure. Hyphaema, lens subluxation, cataract, vitreous haemorrhage were noted. This infant also had unexplained patchy and linear scars of the face, neck and chest[347].

A single case of an 8-week old infant presenting with a dilated and fixed pupil and facial bites. There was eyelid swelling and proptosis, hyphaema, iris sphincter irregularity, bilateral retinal detachment with giant tear. Abuse was apparently admitted. Non-ocular findings included the facial bites, faltering growth, and a previous admission with subdural haematoma and suspected neonatal sepsis that with hindsight was considered to be probable missed child abuse[348].

**Recommendations – Eye injury in physical abuse:** **B,D**

The range of eye injuries seen in abusive trauma is broad, and where an appropriate history is not provided, abusive trauma has to be carefully considered. There was no type of injury that stood out as being highly specific for abusive trauma, but the fact that all described cases in the Betts et al 2017 series[3] had subconjunctival haemorrhage suggests that this could be regarded as a 'sentinel injury' that should prompt further consideration of abuse. As is common in child maltreatment, the possibility of abuse may not be recognised on the child's first presentation due to the lack of history, and early detection requires vigilance and care.

Children presenting to hospital with abusive eye injuries in the Clark 2020 series[105] had certain characteristics that assist in discriminating abusive eye injury from accidental eye injury:

1. Young age <24 months (and particularly under 6 months when infants are usually non-mobile).
2. The presence of other associated non-ocular injuries.
3. The absence of a history of trauma at presentation, or history of simple fall that does not adequately explain the injuries.
4. Retinal involvement without evidence of other ocular or adnexal trauma.

**Recommendation: Eye findings in child abuse:**

The following non-vitreoretinal eye findings have been described in abused children in the papers identified in this review. Physical abuse should be considered in the differential diagnosis for these conditions. It is not possible to conclude how sensitive or specific these findings are for physical abuse on the basis of case reports:

- New onset eye deviation
- Optic neuropathy

- Hyphaema **B,D**
- Raised intraocular pressure
- Vision loss
- Flare or cells in anterior and posterior chambers
- Subluxation or dislocation of lens
- Cataracts
- Hazy cornea
- Corneal epithelial loss
- Swelling of eyelids
- Periorbital oedema
- Injected conjunctiva
- Chemosis
- Pupillary abnormalities
- Amblyopia
- Abnormal fixation
- Rupture of Descemet's membrane
- Proptosis
- Subconjunctival haemorrhage (see above)
- Globe rupture

Eye injury in an infant with a known propensity for eye complications such as a connective tissue disorder may still be associated with child abuse and physical abuse should be considered in the differential diagnosis.

**Recommendations – SCH and abusive eye injury:**
The review findings support the view that SCH without a clear causal history, that remain unexplained after the medical differential diagnosis of SCH has been considered, may be a sentinel sign for physical abuse and should prompt a thorough consideration of abusive trauma.

### 3.5.2. Fabricated or Induced Illness in Children

There were very few published accounts of eye injury in cases of FII. Only one relevant paper was identified by the review process.

A single case report of an infant with severe recurrent keratoconjunctivitis thought to be due to deliberate administration of caustic substances by the caregiver in the context of what would now be described in the UK as FII. There was spontaneous improvement in hospital with rapid recurrence of lesions after discharge from hospital. pH of tear fluid was alkaline which supported a hypothesis of administration of an alkaline caustic substance. Biopsy was useful in differentiating possible causes. In addition to the eye findings the infant had unexplained oral/lip lesions, also lesions of the face and oropharynx, linear cutaneous lesions of face and ear. Oesophageal and gastric erosions were identified on upper GI endoscopy that were considered to be characteristic of caustic ingestion. The parent later apparently admitted causing the injuries but the nature of the caustic agent was not confirmed[338].

**Recommendations – FII and eye presentations:** **D**
A high level of awareness/suspicion is needed to detect these cases at an early stage. It is important to gather and store medical samples for possible use as evidence where possible (fluids, gastric aspirates, biopsies, images). In the UK it would be considered important to follow RCPCH PP (perplexing presentations)/FII procedures, involve the Named Doctor/Nurse, use local multiagency safeguarding children procedures to help to explore the concerns and come to a diagnosis.

### 3.5.3. Neglect

Although neglect is mentioned in several of the above case reports as an associated feature where there has also been physical abuse involving eye trauma, this review did not identify any publications where neglect was the primary cause of presentations with eye problems

## 3.6 Guidance for the Ophthalmologist

### 3.6.1. Procedures for the ophthalmologist and documentation when a safeguarding concern arises.

In the United Kingdom all NHS Trusts (England)/Health Boards (Wales & Scotland) and Health and Social Care Trusts (Northern Ireland) that deal with children will have a safeguarding team with lead professionals. The arrangements vary between the fours UK nations but are broadly similar. In England and Wales there is a Named Doctor (ND) and Named Nurse (NN) for child protection who can support other professionals with safeguarding concens. The named professionals have a broad range of responsibilities but principally support all activities necessary to ensure that the organisation meets its responsibilities to safeguard/protect children and young people. They ensure that advice is available to the full range of specialties within the organisation on the day-to-day management of children and families where there are safeguarding/child protection concerns and can provide advice (direct and indirect) to colleagues on the assessment, treatment and clinical services for all forms of child maltreatment. In Scotland a similar function is carried out by named professional leads for child protection and in Northern Ireland by a named paediatrician and nurse.

The ND is usually a consultant paediatrician but in an eye hospital they may be an ophthalmologist who will need to have links with the Named Doctor in a neighbouring hospital. Rarely, there may be a need to escalate concerns to a Designated Doctor (England & Wales). Designated professionals have a strategic professional lead role on all aspects of the health service contribution to safeguarding children across the area but are not usually directly involved in clinical decision making.

Local safeguarding guidelines and procedures should be readily available to all health staff working with children, families or vulnerable adults. They will usually be found on the NHS Trust/Health Board/ Health and Social Care Trust's intranet pages. They identify key safeguarding processes and personnel together with relevant contact arrangements including those of the local Children's Social Care and the Police safeguarding teams. These teams are often combined in a Multiagency Safeguarding Hub (MASH).

Referral and assessment arrangements vary between local authorities. All Local Safeguarding Children Boards (LSCBs) in England, Regional Safeguarding Children Boards (RSCBs) in Wales, Child Protections Committees in Scotland and Safeguarding Board for Northern Ireland have websites with details of referral pathways, referral forms, thresholds and eligibility criteria, and children's services provision within their area.

How should I manage a child/young person where maltreatment is suspected or considered?

(Adapted from NICE Clinical Knowledge Summaries– Child maltreatment – recognition and management: 2019 revision)

All clinicians who are involved in the care of children should be familiar with the NICE guidance. Underpinning this is the Government guidance: 'Working Together to Safeguard Children: A guide to inter-agency working to safeguard and promote the welfare of children (July 2018)', which provides a framework that interprets the legislation around safeguarding children, predominantly the Children Acts of 1989 and 2004.

Many forms of child maltreatment may involve the eye. The ophthalmologist mainly encounters physical abuse (direct or indirect eye trauma, shaking, smothering) and occasionally fabricated or induced illness (FII, previously known as Munchausen Syndrome by proxy), sexual abuse, neglect and emotional abuse. If you are not sure whether a child or young person is at risk, or how best to act on your concerns, seek advice from senior colleagues, local safeguarding leads or if necessary, the Named or Designated colleagues.

If you have concerns about maltreatment you should:

- Take a detailed medical history to try to better understand the cause of the concern but do not over-step into a more detailed forensic enquiry as this is not the role of the ophthalmologist.
- Record any concerns carefully in the child's records including verbatim comments by the child or their carer, and any examination or investigation findings, electronic data or images and your working diagnosis/opinion.
- Manage individual injuries as appropriate.
- Arrange hospital admission where clinically indicated. Ensure that the receiving paediatrician is aware of your concerns.
- Make a referral if your concerns are not easily resolved.
- Sometimes hospital admission is arranged purely to ensure that the child is in a safe place and that safeguarding procedures can be followed. This should be discussed as part of making a referral. Admission to hospital is not an alternative to making a referral.

**Making a Referral**
The threshold for making a referral is a concern that a child may be at risk of significant harm. You do not have to be certain and should not delay referral as this may increase the risk to the child. Significant harm may adversely affect the child's health or development compared with what might be reasonably expected of a similar child.

Discuss your concerns with the child (if they are of sufficient age and understanding) and/or their caregivers and obtain consent before sharing confidential information unless there is reason to believe that this will increase the risk of harm to the child/young person. However, you should not let obtaining consent delay disclosure of important information regarding children/young people at risk of significant harm. The refusal of consent can be over-ridden in certain circumstances and in safeguarding situations the child's best interests take priority. Some parts of the UK have Mandatory Reporting which creates a legal duty to report safeguarding concerns. This applies in Wales but not in England or Scotland. In Northern Ireland it is an offence to fail to report a 'relevant offence' to the police.

If a child is thought to be in immediate danger, refer immediately to Children's Social Care and/or the police. Consider the safety of other children living with or in contact with the suspected perpetrator.

Otherwise, contact children's social care to discuss the need for a referral to them, using local multi-agency safeguarding procedures. Initial contact may usually be by phone or email depending on the degree of urgency, followed up with a referral form. This may trigger a child protection investigation, a family assessment to determine whether supportive services need to be offered, or other appropriate responses may be identified, e.g. if an alternative explanation for an injury is identified as a result of the investigation. Give your contact details so that you can be contacted, if necessary, in order that your concerns can be clarified or feedback given after the investigation. Professionals cannot make anonymous referrals.

**Responsibilities of doctors in training**
If a doctor in training suspects maltreatment while conducting an ophthalmic examination they should discuss with a senior colleague, consultant ophthalmologist or the senior nurse of the ward or department. This should not delay making a referral and any professional should make the referral using standard local procedures and forms if they are concerned. Nobody has a veto over a safeguarding referral and if the concerns remain, a referral should be made even if there is disagreement within a clinical team.

**Ongoing responsibility for safeguarding**
Anybody who raises a safeguarding concern has a responsibility to ensure that their concern is heard and acted upon appropriately. For children admitted to hospital, the admitting paediatrician will assume responsibility for ongoing investigation and general medical management of the child, including the ongoing safeguarding process. In hospitals where there is no identified safeguarding team and the assessment of a paediatrician is not immediately available, the consultant ophthalmologist will decide the lines of responsibility and discuss the case with colleagues in a neighbouring unit or with the Named or Designated Doctor, to ensure that the child has access to appropriate medical assessment and follow up.

In situations where eye hospitals are isolated from hospitals with a paediatric department and there are grave injuries or serious concerns about immediate risk to the child an escorted transfer of the case should be made to a neighbouring hospital where a paediatrician is available to examine the child. Statutory services (Children's Social Care or the police) must be involved in these cases.

The over-arching responsibility for investigating suspected child abuse lies with the Local Authority Children's Social Care department and the Police Child Protection team. However if an ophthalmologist has any concerns about safeguarding in children there should be no delay in making the appropriate referrals. The safety of the child is of paramount importance and if there is any doubt of potential safegaurding it is best to err on the side of caution and make a referral to the local child protection team.

**Child maltreatment – recognition and management: Summary**

(Adapted from NICE Clinical Knowledge Summaries – Child maltreatment – recognition and management: 2019 revision)

- Child maltreatment includes any type of abuse or neglect of a child/young person caused by inflicting harm or by failing to act to prevent harm. It can be classified as physical, sexual, or emotional abuse, neglect, and fabricated or induced illness.
  - Physical abuse involves causing physical harm to a child such as shaking, hitting, throwing, burning, or suffocating.
  - Sexual abuse involves forcing or tempting a child to take part in sexual activities.
  - Emotional abuse includes conveying to children/young people that they are worthless, unloved, or a burden.
  - Neglect includes the persistent failure to meet the child's basic physical and/or psychological needs.

- Fabricated or induced illness involves the misrepresentation of the child as ill by the caregiver by fabricating or inducing symptoms.

- Healthcare professionals should be alert to the possibility of maltreatment if the following features are present in the absence of a suitable explanation:
  - Frequent attendance or unusual pattern of presentation to healthcare services most commonly due to injuries, either inflicted or due to inadequate supervision. Less commonly, this may occur due to fabricated or induced illness.
  - Unusual or marked change in the child's behaviour or emotional state, different from what is expected for their age and developmental stage, and not explained by a medical condition, neurodevelopment disorder or stressful situation (outwith the maltreatment).
  - Injury or injuries with features suggestive of physical or sexual abuse, or in some instances neglect.
  - Any evidence of sexual activity in a child/young person, especially when they are underage.
  - Harmful interaction between parent/carer and child/young person.
  - The child/young person appears neglected.
  - Failure to ensure access to appropriate medical care or treatment, such as failure to attend hospital appointments or give prescribed medications.
- In general, it is recommended that:
  - If there is any uncertainty about when to consider or suspect maltreatment, or about the immediate risk of harm to the child, advice should be sought from a named professional for child safeguarding or a senior colleague.
  - Consent should be obtained before sharing confidential information unless this will increase the risk of harm to the child/young person.
- If there is suspicion that child maltreatment is occurring, children's social care should be contacted to discuss the need for a referral. If the child is thought to be in immediate danger, the police should be informed.
- If hospital admission is needed, the admitting paediatrician should be made aware of any safeguarding concerns.
- If maltreatment is considered one possible explanation for a report or clinical feature:
  - Other alerting features of child maltreatment should be sought.
  - Information should be obtained from other agencies and colleagues.
  - If these investigations lead to a suspicion of maltreatment, children's social care should be contacted to discuss the need for a referral.
  - If it is thought that referral is not justified, the child should be reviewed regularly.
  - A written record needs to be made of the outcome in cases where maltreatment has been considered.

**What should alert me to child maltreatment?**

(Adapted from NICE Clinical Knowledge Summaries - Child maltreatment - recognition and management: 2019 revision)

- When assessing the possibility of child maltreatment, be aware of risk factors that have been linked to abuse and neglect and the barriers to recognition of child maltreatment.

- Features that should be noted include the child/young person's appearance, behaviour or demeanour, and interaction with the parent or carer. Also assess for any alerting features in the child/young person's history, including frequency of attendance at primary and secondary care, any previous contact with social services, and obtain relevant information on other adults and children in the home.
- Be open-minded when considering the possible cause of an injury or other sign. In order to consider non-abusive causes for any presenting features, ask questions about:
  - Perinatal history – birth-related trauma, history of prematurity.
  - Medication and other possible iatrogenic causes.
  - Past medical history of fractures or bleeding disorders.
  - Family history of clotting disorders, metabolic disease, fractures, blue sclera, and deafness to exclude osteogenesis imperfecta).
- Signs that may alert a clinician to the possibility of child maltreatment can include:
  - Frequent attendance or unusual patterns of presentation to healthcare services, often due to injuries/features that may suggest physical or sexual abuse, neglect, or less commonly, fabricated or induced illness.
  - Inappropriately explained poor school attendance.
  - Refusal by the parent or carer to allow a child or young person to speak to a healthcare professional on their own when it is necessary for the assessment of the child or young person.
  - Unusual or marked changes in the child's behaviour or emotional state, that are unexpected for their age and developmental stage, and not explained by a medical condition, neurodevelopment disorder, or stressful situation (outwith the possible maltreatment).
- Other situations where maltreatment may be suspected can include:
  - Evidence of sexual activity in a child/young person.
  - Persistent harmful behaviour towards the child/young person from the parent/carer suggestive of emotional abuse.
  - Evidence of neglect.

*Suspect* physical abuse if there is/are any of the following, and an explanation is absent or unsuitable:

- Bruising:
  - In the shape of a hand, ligature, stick, teeth mark, grip, or implement, or, in the absence of an underlying medical condition (for example, a coagulation disorder):
    - On a child who is not independently mobile.
    - Multiple or clustered and/or of a similar shape and size.
    - On the ankles and wrists resembling ligature marks.
    - On the neck resembling attempted strangulation.
    - On any non-bony part of the body or face including the eyes, ears and buttocks.
- Bites: if a human bite mark is unlikely to have been caused by a young child

- Lacerations, abrasions, or scars:
  - On a child who is not independently mobile.
  - The injuries are multiple, especially with a symmetrical distribution.
  - Affected areas are usually protected by clothing (for example, back, chest, abdomen, axilla, genital area).
  - The eyes, ears and sides of face are affected.
  - Injuries have occurred on the neck, ankles, and wrists that look like ligature marks.
- Burn or scald injuries:
  - On a child who is not independently mobile.
  - Injury is observed on any soft tissue area that would not be expected to come into contact with a hot object in an accident (for example, the backs of hands, soles of feet, buttocks, back).
  - The burn is in the shape of an implement (for example, cigarette, iron).
  - Forced immersion is suggested by scalds:
    - To buttocks, perineum and lower limbs.
    - To limbs in a glove or stocking distribution.
    - To limbs with symmetrical distribution.
    - With sharply delineated borders.
- Fractures: Where one or more fractures occur/have occurred in the absence of a medical condition that predisposes to fragile bones (for example, osteogenesis imperfecta, osteopenia of prematurity).
- Intracranial injury: Occurring in the absence of major confirmed accidental trauma, or known medical cause, especially in a child aged under 3 years, and/or where there are other associated inflicted injuries.
- Eye trauma: Where there are retinal haemorrhages or injury to the eye in the absence of major confirmed accidental trauma or a known medical explanation, including birth-related causes.
- Spinal injuries: If there is injury to vertebrae or within the spinal canal in the absence of major confirmed accidental trauma.
- Visceral injuries: If there is an intra-abdominal or intrathoracic injury in the absence of major confirmed accidental trauma and without a suitable explanation, and/or there is a delay in presentation. There may be no external bruising or other injury.
- Poisoning – if:
  - There is a report of deliberate administration of inappropriate substances, including prescribed and non-prescribed drugs.
  - There are unexpected blood levels of drugs not prescribed for the child.
  - There is reported or biochemical evidence of ingestions of one or more toxic substances.
  - The child was unable to access the substance independently.
  - There have been repeated presentations of ingestions in the child or other children in the household.
    - Note: consider child maltreatment in cases of hypernatraemia without a medical explanation.

- Repeated apparent life-threatening events: Where the onset is witnessed only by one parent or carer and a medical explanation has not been identified. Consider maltreatment if there is an apparent event with bleeding from the nose or mouth and a medical explanation has not been identified.
- Submersion injury: If a child has a non-fatal submersion incident (near-drowning) and the explanation is absent or unsuitable, or if the child's presentation is inconsistent with the account.

Additionally, consider physical abuse/maltreatment if there is/are any of the following:

- Cold injuries: For example, swollen, red hands or feet with no obvious medical explanation, and/or if a child presents with hypothermia and the explanation is unsuitable.
- Oral injuries: if the explanation is absent or unsuitable.
- General injuries: if the explanation for a serious or unusual injury is absent or unsuitable.
- The child's behaviour towards their parent or carer shows any of the following, particularly if they are not observed in the child's other interactions:
- Dislike or lack of cooperation.
- Lack of interest or low responsiveness.
- High levels of anger or annoyance.
- Passivity or withdrawal.

**Recognising Neglect**

- *Suspect neglect if:*
  - A child is persistently dirty and smelly.
  - There are repeated observations/reports of a poor standard of home hygiene that affects a child's health, inadequate provision of food, and/or a living environment that is unsafe for the child's developmental stage.
  - Parents or carers fail to seek medical advice for their child to the extent that the child's health and wellbeing are compromised, including if the child is in ongoing pain.
- *Consider neglect if:*
  - A child has severe and persistent infestations, such as scabies or head lice.
  - A child's clothing or footwear is consistently inappropriate (for example, for the weather or the child's size).
  - A child displays faltering growth because of lack of provision of an adequate or appropriate diet.
  - Parents or carers persistently fail to anticipate dangers and to take precautions to protect their child from harm.
  - The explanation for an injury or incident (for example, a burn, sunburn, ingestion of a harmful substance, animal bite, or a non-fatal submersion incident) suggests a lack of appropriate supervision.
  - A child or young person is not being cared for by a person who is able to provide adequate care.
  - Parents or carers fail to administer essential prescribed treatment for their child, repeatedly fail to bring their child to follow-up appointments that are essential for their child's health and wellbeing, and/or persistently fail to engage with relevant child health promotion programmes, which include immunisation, health and development reviews, and screening.

- Parents or carers have access to, but persistently fail to obtain treatment for their child's dental caries.
- A child's behaviour towards their parent or carer shows any of the following, particularly if they are not observed in the child's other interactions:
- Dislike or lack of cooperation.
- Lack of interest or low responsiveness.
- High levels of anger or annoyance.
- Passivity or withdrawal.

**Further information sources in relation to eye injuries and child abuse**
In addition to this RCOphth Working Party report, the Royal College of Paediatrics and Child Health has produced a Child Protection Companion that gives evidence-based guidance for professionals facing concerns about child abuse or neglect. This includes a section on abusive head trauma and retinal findings. RCPCH has also published a systematic review on retinal findings in child abuse that is of direct relevance to Ophthalmologists.

**Eye examination after a sudden infant death** **GPP**
The RCPCH, RCPath and RCOphth issued a joint statement in January 2022 recommending immediate eye examination in infants brought in dead: rcpch.ac.uk/news-events/news/joint-statement-eye-examination-sudden-unexpected-death-children. Where local procedures have been developed they should be adhered to, but there is lack of concensus on the involvement of ophthalmologists in these examinations. The view of the working party was that it is not recommended. It is usual for paediatricians to examine the eyes including ophthalmoscopy (if not prevented by corneal clouding). They may lack the specialist experience to interpret the findings and abnormal findings may be difficult to interpret. There is not an evidence base that can be used to define ophthalmoscopic abnormalities in a postmortem retina. However evidence of retinal bleeding should prompt careful assessment for other features of child abuse, and should be discussed with the coroner.

**Suggested Reading**
https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/942454/Working_together_to_safeguard_children_inter_agency_guidance.pdf (accessed September 2023)

https://www.rcpch.ac.uk/sites/default/files/2019-08/named_doctor_for_child_protection_-_model_job_description_and_competencies_2019_0.pdf (accessed September 2023)

https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/338875/MASH.pdf (accessed September 2023)

https://www.nice.org.uk/guidance/ng76/resources/child-abuse-and-neglect-pdf-1837637587141 (accessed September 2023)

https://cks.nice.org.uk/topics/child-maltreatment-recognition-management/ (accessed September 2023)

### 3.6.2 Standards of Ophthalmology Assessment [85,321,322,324,326,327,349-351]

The ophthalmology assessment must be carried out by an ophthalmologist[351]. External eye examination: It is rarely possible to record the visual acuity in a very sick child and the reason should be recorded (eg: sedated/intubated). The periocular area, eyelids, conjunctiva, anterior segments, pupillary reactions and ocular movements (or dolls head manoeuvre if necessary) of both eyes should be examined and the findings recorded.

Posterior segment examination: With agreement of the managing team the pupils should be dilated with short acting mydriatics (e.g. phenylephrine 2.5%, tropicamide 0.5%) and the fundus should be examined with an indirect ophthalmoscope and a condensing lens (20D ,28D, 30D or a 2.2 panfundoscopic lens).

The retinal findings should record whether retinal haemorrhages are present unilaterally or bilaterally and details of the layer of retinal involvement (preretinal, intraretinal and subretinal) the location in the retina (posterior pole /periphery), the severity of the retinal haemorrhages in terms of number (few, many and too numerous to count) and size should be described. The presence or absence of additional features such as retinal folds, haemorrhagic retinoschisis and the presence of large macular or vitreous haemorrhages should be recorded. A standardised clinical proforma designed for documentation (appendix 4) should be used.

The use a hand held camera, the RetCam and a hand held OCT machine (Optical coherence tomography), Widefield photography (Optos) help to record findings if available.

Any report should include the following details written legibly (appendix 4)

1. Name and signature and status (e.g. Consultant, Specialist trainee).
2. Date, time and location of examination.
3. Reason for referral and referral physician.
4. Level of consciousness of the child at the time of examination.
5. What drops where used to dilate the pupils and the time of instillation. **GPP**
6. Methods of examination.
7. Photography details documented if done.
8. Clear description of ocular findings with an annotated diagram .
9. Follow up suggested.

### 3.6.3 Timing of Ophthalmology Assessment

As ophthalmology findings on external ocular examination, anterior segment examination and retinal examination may change within a few days (see section 3.2.2.5) it is recommended that ophthalmology assessment are carried out as soon as is practically possible. While there was no evidence from this review of the timing of ophthalmology assessment this should ideally undertaken within 24-48 hours of the initial paediatric assessment. This assessment should be undertaken by the on-call ophthalmology team. If any findings need to be reviewed by a paediatric ophthalmologist this should be undertaken preferably within 72 hours of the initial paediatric referral. **GPP**

# Appendix 1

**Search strategy:**

The search strategies used were separated into the clinical questions 1-9. This was to ensure the correct search terms were used for each question and the time period searched. For those questions 1-5 included in the previous review the search strategy was between 2011 and 2021. The search strategy for the new questions 6-9 added was between 1970-2021.

The search strategy for the database MEDLINE ALL for the questions is provided below. The search strategy for all other databases can be provided on request from the chairman.

**Search strategy for MEDLINE ALL:Questions 1, 2 and 5: Search period 2011-2021**

1 Child/ (1722716)
2 Child, Preschool/ (935197)
3 Infant/ (805489)
4 Infant, Newborn/ (615234)
5 (baby or babies or child* or neonate* or toddler*).mp. (2624963)
6 or/1-5 (3011099)
7 (Accidental* and injur*).mp. (17989)
8 Accidental Injuries/ (104)
9 "Wounds and Injuries"/ (78317)
10 Accidental trauma.mp. (496)
11 ((Battered or shaken) adj (baby or child* or infant*)).mp. (1837)
12 Battered Child Syndrome.mp. (808)
13 Abusive head trauma.mp. (577)
14 ((non-accidental or nonaccidental) adj3 (trauma or injur*)).mp. (1261)
15 Non?accidental head trauma.mp. (27)
16 (child adj (abuse or maltreatment or protection)).mp. (37163)
17 Shaken impact syndrome.mp. (18)
18 Shaken Baby Syndrome.mp. (886)
19 Soft tissue injur*.mp. (9213)
20 Neurosurgical injur*.mp. (35)
21 Short falls.mp. (99)
22 Physical abuse.mp. (6126)
23 (Intentional injur* or Intentional trauma).mp. (624)
24 or/7-23 (141474)
25 Bilateral retinal h?emorrhage.mp. (16)
26 Blot retinal h?emorrhage.mp. (1)
27 (Blunt ocular trauma or Blunt trauma).mp. (9783)
28 Bruise eyelid.mp. (0)
29 Conjunctival h?emorrhage.mp. (133)
30 Subconjunctival h?emorrhage.mp. (491)
31 Corneal laceration*.mp. (217)
32 Disc oedema.mp. (173)
33 Dot retinal h?emorrhage.mp. (1)
34 (ECMO or Extracorporeal Membrane Oxygenation).mp. (16976)
35 Extramacular dot h?emorrhage.mp. (0)
36 ecchymosis eyelid.mp. (3)
37 Eye h?emorrhage.mp. (999)
38 eye injur*.mp. (15880)
39 Flame hemorrhage.mp. (4)

40 Flame shaped h?emorrhage.mp. (11)
41 Foveal h?emorrhage.mp. (20)
42 Fundal h?emorrhage.mp. (9)
43 h?emorrhage.mp. (311244)
44 h?emorrhagic retinopathy.mp. (98)
45 H?emorrhagic retinoschisis.mp. (5)
46 Intracranial arterial aneurysm.mp. (72)
47 Intraocular h?emorrhage.mp. (308)
48 Intraretinal h?emorrhage.mp. (121)
49 Lacerated eyelid.mp. (1)
50 Lid injur*.mp. (13)
51 (LRP5-b or Low-density-lipoprotein-receptor-related protein).mp. (4762)
52 Ehlers danlos syndrome.mp. (4169)
53 (Vitamin C or Vitamin D).mp. (99531)
54 Protein s deficiency.mp. (1781)
55 Protein C deficiency.mp. (2064)
56 Coagulopathy.mp. (14459)
57 Fibrinogen levels.mp. (3828)
58 New born h?emorrhage.mp. (0)
59 Birth h?emorrhage.mp. (8)
60 (H?emorrhagic diseas* adj3 new born).mp. (8)
61 Hermansky Pudlak.mp. (714)
62 (Osteogenesis imperfecta or Glutaric aciduria).mp. (6540)
63 (Fibromuscular dysplasia or Methylmalonic aciduria or Homocyst*).mp. (31019)
64 (Vaccination* or Immunisation*).mp. (184707)
65 Multiple small punctate h?emorrhage.mp. (0)
66 Multiple punctate cerebral h?emorrhage*.mp. (1)
67 Nerve palsy.mp. (11929)
68 (Ocular adj (finding* or trauma)).mp. (4527)
69 (Ophthalmological finding* or extraocular finding*).mp. (483)
70 (Ophthalmology or Ophthalmoplegia or Opthalmoplegia).mp. (52068)
71 Orbital injur*.mp. (363)
72 Pale-centered h?emorrhage*.mp. (1)
73 (Palpebral fissure* or Papilledema or Papilloedema).mp. (8732)
74 Preretinal h?emorrhage*.mp. (187)
75 (Posterior pole or Purtscher retinopathy).mp. (3452)
76 Residual foveal h?emorrhage*.mp. (0)
77 (Retina or Retinal fold or Retinal injur*).mp. (119123)
78 Retinal Artery Occlusion.mp. (3097)
79 Retinal capillary network.mp. (42)
80 Retinal detachment.mp. (26921)
81 Retinal exudates.mp. (94)
82 Retinal Hemorrhage/ (5307)
83 Retinal h?emorrhage*.mp. (6638)
84 (Roth Spots or Ruptured retinal capillary).mp. (74)
85 Spinal cord arteriovenous malformation.mp. (59)
86 (Splinter h?emorrhage or Subconjunctival h?emorrhage or Subhyaloid haemorrhage).mp. (566)
87 (Subhyaloid h?emorrahage* or Subhyaloid macular h?emorrhage*).mp. (5)
88 (Subretinal h?emorrhage* or Terson Syndrome).mp. (967)
89 (Unilateral retinal h?emorrhage* or Vitreous h?emorrhage).mp. (4238)
90 (Valsalva maneuver or Valsalva retinopathy).mp. (5588)
91 Von Willebrand syndrome.mp. (476)

92 Whiplash injur*.mp. (3572)
93 Cerebral venous sinus thrombosis.mp. (1092)
94 Benign external hydrocephalus.mp. (37)
95 or/25-94 (908086)
96 6 and 24 and 95 (2525)
97 limit 96 to (english language and yr="2011 - 2021") (920)

**Search strategy for MEDLINE ALL:Question 4: Search period 2011-2021**

1 exp Infant, Newborn/ (617901)
2 exp Infant/ (1156731)
3 (infant* or newborn* or baby or babies or neonate*).mp. (1473048)
4 or/1-3 (1473048)
5 Bilateral retinal h?emorrhage*.mp. (82)
6 Blot retinal h?emorrhage*.mp. (10)
7 (Blunt ocular trauma or Blunt trauma).mp. (9767)
8 Bruise eyelid.mp. (0)
9 Conjunctival h?emorrhage*.mp. (183)
10 Subconjunctival h?emorrhage*.mp. (586)
11 Corneal laceration*.mp. (217)
12 Disc oedema.mp. (172)
13 Dot retinal h?emorrhage*.mp. (2)
14 (ECMO or Extracorporeal Membrane Oxygenation).mp. (16877)
15 Extramacular dot h?emorrhage*.mp. (0)
16 ecchymosis eyelid.mp. (3)
17 Eye h?emorrhage*.mp. (1000)
18 eye injur*.mp. (15852)
19 Flame hemorrhage*.mp. (15)
20 Flame shaped h?emorrhage*.mp. (57)
21 Foveal h?emorrhage*.mp. (23)
22 Fundal h?emorrhage*.mp. (18)
23 h?emorrhage.mp. (310807)
24 h?emorrhagic retinopathy.mp. (98)
25 H?emorrhagic retinoschisis.mp. (5)
26 Intracranial arterial aneurysm.mp. (72)
27 Intraocular h?emorrhage*.mp. (395)
28 Intraretinal h?emorrhage*.mp. (354)
29 Lacerated eyelid.mp. (1)
30 Lid injur*.mp. (13)
31 (LRP5-b or Low-density-lipoprotein-receptor-related protein).mp. (4682)
32 Ehlers danlos syndrome.mp. (4167)
33 (Vitamin C or Vitamin D).mp. (99390)
34 Protein s deficiency.mp. (1780)
35 Protein C deficiency.mp. (2062)
36 Coagulopathy.mp. (14421)
37 Fibrinogen levels.mp. (3828)
38 New born h?emorrhage*.mp. (0)
39 Birth h?emorrhage*.mp. (9)
40 (H?emorrhagic diseas* adj3 new born).mp. (8)
41 Hermansky Pudlak.mp. (711)
42 (Osteogenesis imperfecta or Glutaric aciduria).mp. (6550)
43 (Fibromuscular dysplasia or Methylmalonic aciduria or Homocyst*).mp. (31020)
44 (Vaccination* or Immunisation*).mp. (184819)

45 Multiple small punctate h?emorrhage.mp. (0)
46 Multiple punctate cerebral h?emorrhage*.mp. (1)
47 Nerve palsy.mp. (11907)
48 (Ocular adj (finding* or trauma or manifestation)).mp. (5148)
49 (Ophthalmological finding* or extraocular finding*).mp. (481)
50 (Ophthalmology or Ophthalmoplegia or Opthalmoplegia).mp. (51971)
51 Orbital injur*.mp. (361)
52 Pale-centered h?emorrhage*.mp. (1)
53 (Palpebral fissure* or Papilledema or Papilloedema).mp. (8731)
54 Preretinal h?emorrhage*.mp. (187)
55 (Posterior pole or Purtscher retinopathy).mp. (3462)
56 Residual foveal h?emorrhage*.mp. (0)
57 (Retina or Retinal fold or Retinal injur*).mp. (119146)
58 Retinal Artery Occlusion.mp. (3086)
59 Retinal capillary network.mp. (43)
60 Retinal detachment.mp. (26894)
61 Retinal exudates.mp. (94)
62 Retinal Hemorrhage/ (5299)
63 Retinal h?emorrhage*.mp. (6625)
64 (Roth Spots or Ruptured retinal capillary).mp. (74)
65 Spinal cord arteriovenous malformation.mp. (59)
66 (Splinter h?emorrhage or Subconjunctival h?emorrhage or Subhyaloid haemorrhage).mp. (565)
67 (Subhyaloid h?emorrahage* or Subhyaloid macular h?emorrhage*).mp. (5)
68 (Subretinal h?emorrhage* or Terson Syndrome).mp. (969)
69 (Unilateral retinal h?emorrhage* or Vitreous h?emorrhage).mp. (4234)
70 (Valsalva maneuver or Valsalva retinopathy).mp. (5583)
71 Von Willebrand syndrome.mp. (474)
72 Whiplash injur*.mp. (3573)
73 Cerebral venous sinus thrombosis.mp. (1085)
74 Benign external hydrocephalus.mp. (36)
75 exp Choroid Hemorrhage/ (444)
76 exp Vitreous Hemorrhage/ (1923)
77 exp Retinal Hemorrhage/ (5299)
78 (h?emorrhage* adj3 retin*).ti,ab. (3966)
79 ((retina* or eye) adj3 detachment).mp. (27608)
80 multilayer retinal h?emorrhage.mp. (1)
81 (retina* adj3 (trauma or injur*)).mp. (2549)
82 ("choroid h?emorrhage*" or "vitreous h?emorrhage*" or "macular h?emorrhage*").mp. (5055)
83 ("disc diameters" or "disc oedema" or "posterior pole").mp. (3862)
84 ((Intraocular or Intraretinal or intracranial or macular) adj (trauma* or injur*)).mp. (1380)
85 or/5-84 (909711)
86 exp Delivery, Obstetric/ (81824)
87 exp Vacuum Extraction, Obstetrical/ (1337)
88 exp Extraction, Obstetrical/ (3497)
89 exp Obstetrical Forceps/ (1673)
90 exp Obstetric Labor Complications/ (69991)
91 exp Parturition/ (17866)
92 exp Cesarean Section/ (46402)
93 exp Labor, Induced/ (9534)
94 Breech Presentation/ (3169)
95 exp Extraction, Obstetrical/ (3497)
96 exp Vaginal Birth after Cesarean/ (1686)

97 exp Labor Presentation/ (6837)
98 "spontaneous vaginal deliver*".mp. (1410)
99 "operative vaginal deliver*".mp. (922)
100 "assisted vaginal deliver*".mp. (464)
101 "mechanical vaginal deliver*".mp. (1)
102 "spontaneous vertex deliver*".mp. (70)
103 ((induction or extraction or suction or ventouse or prolonged or forcep* or cesarean) adj3 (deliver* or birth)).mp. (24784)
104 "normal vaginal delivery".mp. (893)
105 ("midcavity forceps" or "obstetric forceps" or "wrigleys forceps" or "outlet forceps").mp. (188)
106 ("low cavity forceps" or "neville barnes forceps" or "kielland forceps" or "ferguson forceps").mp. (42)
107 (prolonged labo?r or second stage of labo?r).mp. (3106)
108 ("delayed delivery" or "delayed second stage" or "failed forceps").mp. (271)
109 failed ventouse.mp. (6)
110 "Kiwi Omnicup".mp. (22)
111 "polyethylene vacuum cup".mp. (2)
112 "after coming head".mp. (21)
113 ((vacuum or vaginal) adj3 (birth or deliver*)).mp. (20221)
114 (instrumental deliver* or instrumental birth).mp. (1435)
115 ((breech or instrumental) adj3 (birth or deliver*)).mp. (3808)
116 "secondary to birth".ti,ab. (61)
117 exp Birth Injuries/ (5617)
118 (complicat* adj3 (birth or deliver* or labo?r)).mp. (23822)
119 birth related injur*.mp. (38)
120 ((trauma or injur*) adj3 (deliver* or labo?r or birth)).mp. (8870)
121 Caesarean delivery.mp. (2585)
122 (C-section or Caesarian section).ti,ab. (2319)
123 or/86-122 (175084)
124 exp Retinopathy of prematurity/ (6081)
125 Retinopathy of prematurity.ti,ab. (6713)
126 (rat: or mouse or mice or hamster: or animal: or dog: or cat: or rabbit: or bovine or sheep).mp. (12408848)
127 animal stud*.mp. (37704)
128 or/124-127 (12414308)
129 4 and 85 and 123 (6359)
130 129 not 128 (3702)
131 limit 130 to (english language and yr="2011 - 2021") (1134)

**Search strategy for MEDLINE ALL:Question 6-9: Search period 1970-2021**

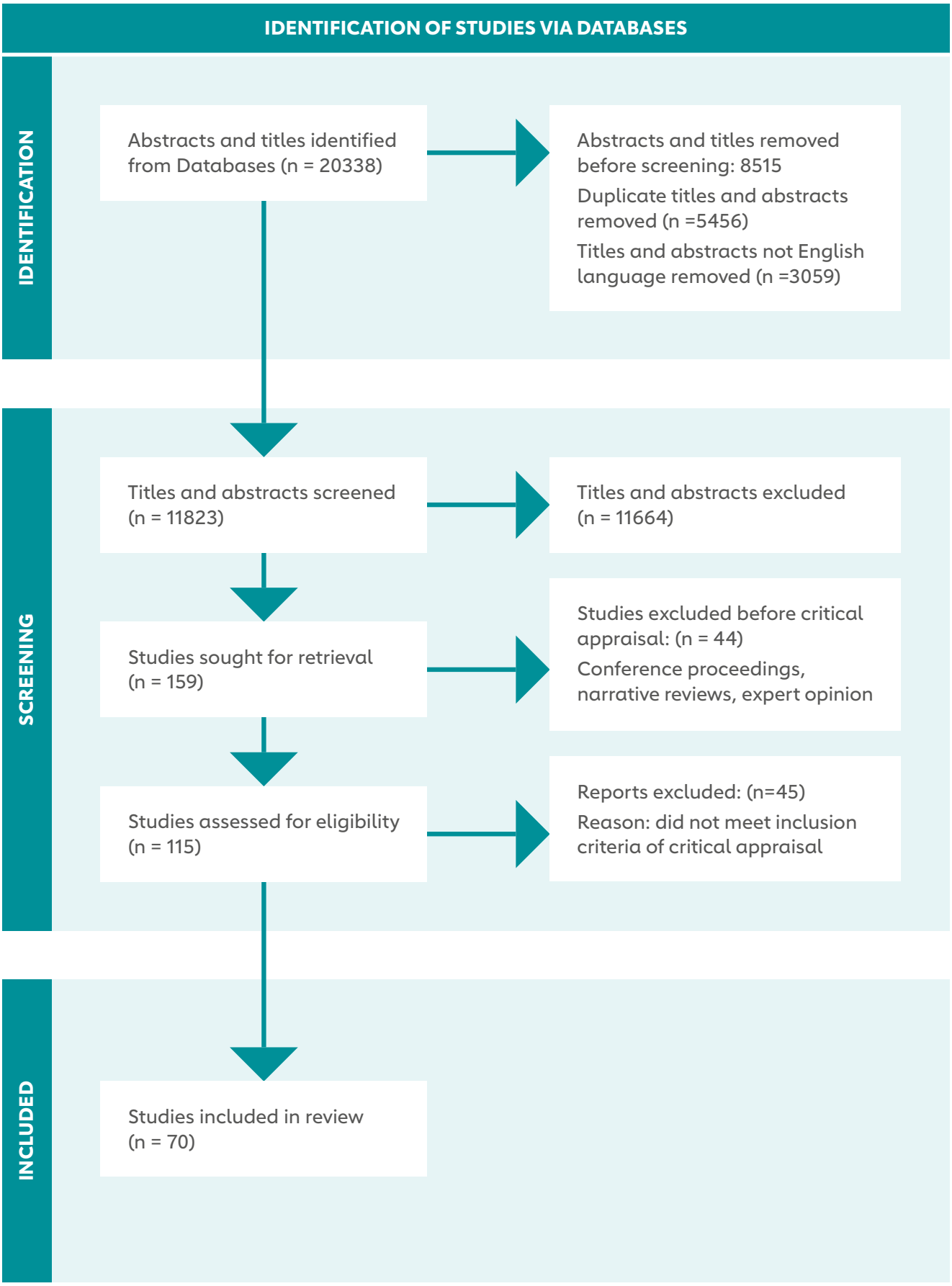
1 Child/ (1722124)
2 Child, Preschool/ (934919)
3 Infant/ (805290)
4 Infant, Newborn/ (615054)
5 (baby or babies or child* or neonate* or toddler*).mp. (2623448)
6 or/1-5 (3009507)
7 (Accidental* and injur*).mp. (17974)
8 Accidental Injuries/ (104)
9 "Wounds and Injuries"/ (78298)
10 Accidental trauma.mp. (495)
11 ((Battered or shaken) adj (baby or child* or infant*)).mp. (1835)
12 Battered Child Syndrome.mp. (808)
13 Abusive head trauma.mp. (576)

14 ((non-accidental or nonaccidental) adj3 (trauma or injur*)).mp. (1260)
15 Non?accidental head trauma.mp. (27)
16 (child adj (abuse or maltreatment or protection)).mp. (37136)
17 Shaken impact syndrome.mp. (18)
18 Shaken Baby Syndrome.mp. (884)
19 Soft tissue injur*.mp. (9211)
20 Neurosurgical injur*.mp. (35)
21 Short falls.mp. (99)
22 Physical abuse.mp. (6119)
23 (Intentional injur* or Intentional trauma).mp. (624)
24 or/7-23 (141409)
25 Bilateral retinal h?emorrhage.mp. (16)
26 Blot retinal h?emorrhage.mp. (1)
27 (Blunt ocular trauma or Blunt trauma).mp. (9778)
28 Bruise eyelid.mp. (0)
29 Conjunctival h?emorrhage.mp. (133)
30 Subconjunctival h?emorrhage.mp. (490)
31 Corneal laceration*.mp. (217)
32 Disc oedema.mp. (173)
33 Dot retinal h?emorrhage.mp. (1)
34 (ECMO or Extracorporeal Membrane Oxygenation).mp. (16930)
35 Extramacular dot h?emorrhage.mp. (0)
36 ecchymosis eyelid.mp. (3)
37 Eye h?emorrhage.mp. (999)
38 eye injur*.mp. (15876)
39 Flame hemorrhage.mp. (4)
40 Flame shaped h?emorrhage.mp. (11)
41 Foveal h?emorrhage.mp. (20)
42 Fundal h?emorrhage.mp. (9)
43 h?emorrhage.mp. (311052)
44 h?emorrhagic retinopathy.mp. (98)
45 H?emorrhagic retinoschisis.mp. (5)
46 Intracranial arterial aneurysm.mp. (72)
47 Intraocular h?emorrhage.mp. (308)
48 Intraretinal h?emorrhage.mp. (121)
49 Lacerated eyelid.mp. (1)
50 Lid injur*.mp. (13)
51 (LRP5-b or Low-density-lipoprotein-receptor-related protein).mp. (4760)
52 Ehlers danlos syndrome.mp. (4164)
53 (Vitamin C or Vitamin D).mp. (99447)
54 Protein s deficiency.mp. (1782)
55 Protein C deficiency.mp. (2063)
56 Coagulopathy.mp. (14439)
57 Fibrinogen levels.mp. (3826)
58 New born h?emorrhage.mp. (0)
59 Birth h?emorrhage.mp. (8)
60 (H?emorrhagic diseas* adj3 new born).mp. (8)
61 Hermansky Pudlak.mp. (713)
62 (Osteogenesis imperfecta or Glutaric aciduria).mp. (6535)
63 (Fibromuscular dysplasia or Methylmalonic aciduria or Homocyst*).mp. (31008)
64 (Vaccination* or Immunisation*).mp. (184475)
65 Multiple small punctate h?emorrhage.mp. (0)

66 Multiple punctate cerebral h?emorrhage*.mp. (1)
67 Nerve palsy.mp. (11919)
68 (Ocular adj (finding* or trauma)).mp. (4526)
69 (Ophthalmological finding* or extraocular finding*).mp. (482)
70 (Ophthalmology or Ophthalmoplegia or Opthalmoplegia).mp. (52024)
71 Orbital injur*.mp. (363)
72 Pale-centered h?emorrhage*.mp. (1)
73 (Palpebral fissure* or Papilledema or Papilloedema).mp. (8729)
74 Preretinal h?emorrhage*.mp. (187)
75 (Posterior pole or Purtscher retinopathy).mp. (3448)
76 Residual foveal h?emorrhage*.mp. (0)
77 (Retina or Retinal fold or Retinal injur*).mp. (119053)
78 Retinal Artery Occlusion.mp. (3094)
79 Retinal capillary network.mp. (42)
80 Retinal detachment.mp. (26910)
81 Retinal exudates.mp. (94)
82 Retinal Hemorrhage/ (5306)
83 Retinal h?emorrhage*.mp. (6635)
84 (Roth Spots or Ruptured retinal capillary).mp. (74)
85 Spinal cord arteriovenous malformation.mp. (59)
86 (Splinter h?emorrhage or Subconjunctival h?emorrhage or Subhyaloid haemorrhage).mp. (565)
87 (Subhyaloid h?emorrahage* or Subhyaloid macular h?emorrhage*).mp. (5)
88 (Subretinal h?emorrhage* or Terson Syndrome).mp. (967)
89 (Unilateral retinal h?emorrhage* or Vitreous h?emorrhage).mp. (4235)
90 (Valsalva maneuver or Valsalva retinopathy).mp. (5584)
91 Von Willebrand syndrome.mp. (476)
92 Whiplash injur*.mp. (3571)
93 Cerebral venous sinus thrombosis.mp. (1089)
94 Benign external hydrocephalus.mp. (37)
95 or/25-94 (907354)
96 6 and 24 and 95 (2522)
97 limit 96 to (english language and yr="1970 - 2021") (2218)

# Appendix 2

**Flow diagram based on PRISMA: Preferred Reporting Items for Systematic Reviews and Meta-Analysis**



# Appendix 3

| | |
|---|---|
| **Reviewer's name:** | |
| **Date:** | |

| | |
|---|---|
| **Study's first author, surname and year:** | |
| **Title:** | |
| **What is/are the research question(s) posed by the study?** | |

**SECTION A.**
**Key Questions:**

The main and sub questions being updated in this guideline are listed below. Please tick which question this study addresses – more than one question may apply.

We have listed the subject TITLE for each area to help simplify things due to large numbers of questions and sub questions for this review.

If none of the questions apply (option 10), the study should be EXCLUDED and further details are required in Section C.

| SECTION A.<br>Clinical Question | Yes | Comment (if possible mention sub question this addresses) |
|---|---|---|
| **1. Abusive head trauma and non:** What differences are found between abusive head trauma retinal findings versus non-abusive head trauma retinal findings?<br>(i) Are retinal haemorrhages more common in infants that older children<br>(ii) What is the site and extent of retinal haemorrhages in child abuse<br>(iii) Are any findings pathognomonic of abusive head trauma<br>(iv) Are unilateral haemorrhages compatible with abusive head trauma<br>(v) Are there any retinal findings without radiological evidence of intracranial injury<br>(vi) Can accidental injury cause retinal haemorrhage<br>(vii) Can short falls cause retinal haemorrhages | | |
| **2. Differential diagnoses:** What are the differential diagnoses of retinal haemorrhages in children with clinical features associated with child abuse?<br>(i) What other conditions in childhood may have retinal haemorrhages<br>(ii) Can a bleeding diathesis or blood dyscrasia cause retinal haemorrhages similar to those seen in abusive head trauma<br>(iii) Can seizures alone cause retinal haemorrhages as seen in abusive head trauma<br>(iv) Can cardio pulmonary resuscitation cause retinal haemorrhages<br>(v) Can prolonged vomiting gagging cause retinal haemorrhages<br>(vi) Is an apparent life threatening event (ALTE) or brief resolved unexplained event (BRUE) associated with retinal haemorrhges<br>(vii) Are vaccinations associated with retinal haemorrhages<br>(viii) Do high cervical injuries cause retinal haemorrhages<br>(ix) Do retinal haemorrhages occur with raised intracranial pressure<br>(x) What are the ocular findings in ocular crush injury | | |
| **3. Newborns:** Retinal haemorrhages in newborn infants:<br>(i) What are the retinal findings in newborn infants?<br>(ii) What are the obstetric correlates to retinal haemorrhages in the newborn?<br>(iii) What is the evolution of newborn retinal haemorrhages?<br>(iv) Are new born retinal haemorrhages associated with intracranial bleeding seen in newborns (abuse excluded) | | |

| SECTION A.<br>Clinical Question | Yes | Comment (if possible mention sub question this addresses) |
|---|---|---|
| **4. Non-vitreoretinal injury fabricated and neglect:** Which features or characteristics of Non Vitreo retinal eye injury are present in child maltreatment, neglect and fabricated or induced illness?<br>(i) What are the non vitreoretinal ocular and ocular adnexal injuries seen in (i) Abuse (ii) Fabricated injury and (iii) Neglect? (eg subconjunctival haemorrhages, burns nerve palsies etc) | | |
| **5. Dating:** Can retinal haemorrhages be dated?<br>(i) can intraocular haemorrhages increase after injury<br>(ii) is it possible from examining the retina the time at which the injury occurred or whether they have occurred at more than one time | | |
| **6. Mechanisms:** What are the postulated mechanisms of retinal haemorrhages in abusive head trauma?<br>a. Forces necessary to produce retinal haemorrhages<br>b. Can retinal haemorrhages and intracranial haemorrhages occur with vigorous play<br>c. Does hypoxia cause retinal haemorrhages<br>d. Animal, biomechanical and computer models of ocular findings in abusive head trauma | | |
| **7. Pathology:** What is the ophthalmic/ocular pathology seen in abusive head trauma?<br>(i) What is the orbital and optic nerve pathology<br>(ii) What is the vitreous/retinal pathology | | |
| **8. Imaging:** Which methods of eye examinations and imaging of the retina are useful in abusive head trauma ?<br>a. Indirect ophthalmoscope<br>b. Retcam<br>c. Non mydriatic camera<br>d. -optos<br>e. Optical coherence tomography<br>f. Neuro imaging | | |
| **9. Documentation:** What are the methods used to document/record eye findings?<br>(i) Standardized methods of recording eye findings | | |
| **If you have selected any of the above questions, please continue to SECTION B** | | |
| **10.** Study relates to none of the areas listed above (does not address key questions) | | Please provide further detail in SECTION C, then proceed to SECTION E |

## SECTION B.
## Evidence Type (study design):

Please only tick the evidence type which applies to the data set that is of interest to the Update. This is **your** opinion on Evidence Type, even if this differs from what has been stated in the study.

| | Yes | | Yes |
|---|---|---|---|
| Case series | | Cross-sectional uncontrolled before and after study | |
| Case study | | Prospective cohort/longitudinal | |
| Case-control | | Retrospective cohort study | |
| Randomised controlled trial | | Interrupted time series | |
| Systematic review | | | |

## SECTION C.
## Exclusion Criteria

**Study does not address any of the key questions – go straight to SECTION F**

- If you selected SECTION A option 10 please work through the criteria below and tick any that apply
- If none of the criteria listed apply, please provide a brief explanation of your decision under option 10

The study is EXCLUDED

**Study addresses one or more of the key questions**

- If you specified that the study addresses one or more of the key questions in SECTION A please work through relevant criteria below and tick any which apply

If you select any of the criteria the study should be EXCLUDED

**Exclusion Criteria Yes Comment**

**Adult data or mixed child and adult data where child data could not be extrapolated**

**Consensus statement or personal practice**

**Ophthalmic examination performed by non-ophthalmologist**

4. AHT – ranking of abuse within study of -5

**or mixed ranking where cases ranked 1-4 could not be extracted**

5. Study exclusively addresses retinal findings in association with:
   - prior ophthalmic surgery
   - solid mass lesions of the eye (e.g. retinoblastoma) or brain
   - RH in association with eye disease ROP, retinal telangiectasia, diabetes

**Expert opinion**

7. If you have specified that the STUDY DOES NOT ADDRESS ANY KEY QUESTION (SECTION A – option 10), but this was NOT for any of the above criteria, please briefly detail your reasoning here:

**If the study is EXCLUDED, please go directly to SECTION E.**

## SECTION D.
## Quality Criteria

| Study Quality Criteria | Yes | Comment |
|---|---|---|
| How have the authors defined abuse? (Please state criteria used) | | |
| What ranking of abuse criteria would you apply? (Rank 1 to 5) | | |
| Have the authors actively excluded abuse from the non-abused group? (accidental and/or organic cause) | | |

## SECTION D.
## Methodological Quality Criteria

**Please complete section relevant to study type chosen**

| Questions to assist with the critical appraisal of all studies | | | | |
|---|---|---|---|---|
| **Please tick the appropriate column** | **Yes** | **No** | **Unclear/ not known** | **Not applicable** |
| 1. Were the aims of the study clearly stated? | | | | |
| 2. Was the study clearly focused in terms of population and outcomes? | | | | |
| 3. Have authors included demographic information? | | | | |
| 4. Was the choice of study method appropriate? | | | | |
| 5. Are the inclusion criteria explicit? | | | | |
| 6. Have the results of the study been clearly presented? | | | | |
| 7. Have study objectives been met? | | | | |
| 8. Are all the figures in the table and the text consistent? | | | | |
| 9. Were all important outcomes/results considered? | | | | |
| 10. Are conclusions clear and unbiased? | | | | |

**If you have completed this table please go to next table and complete it if it is relevant to the study type if not go to SECTION F.**

| Questions to assist with the critical appraisal comparative studies | | | | |
|---|---|---|---|---|
| **Please tick the appropriate column** | **Yes** | **No** | **Unclear/ not known** | **Not applicable** |
| 11. Is the population appropriate? | | | | |
| 12. Is confounding and bias considered? | | | | |
| 13. Were the statistical methods used appropriately? | | | | |
| 14. Were all important outcomes/results considered? | | | | |
| Overall, do you think that this study is significantly methodologically flawed? | | | | |
| Comments | | | | |

**If you have completed this table go to SECTION F, if not please go to next table and complete it if it is relevant to the study type.**

| Questions to assist with the critical appraisal of a randomised controlled trial | | | | |
|---|---|---|---|---|
| **Please tick the appropriate column** | **Yes** | **No** | **Unclear/ not known** | **Not applicable** |
| 1. Is the trial relevant to the needs of the study? | | | | |
| 2. Did the trial address a clearly focussed issue in terms of the population, intervention and outcome? | | | | |
| 3. Were the assignments of children/ families to intervention randomised? | | | | |
| 4. Were all the patients who entered the trial properly accounted for at its conclusion? | | | | |
| 5. Were patients, health/social care workers and study personnel 'blind' to treatment? | | | | |
| 6. Were the groups similar at the start of the trial? | | | | |
| 7. Aside from the intervention, were the groups treated equally? | | | | |
| 8. Have the results of the study been clearly presented? | | | | |
| 9. Are the data in the tables or graphs and the text consistent? | | | | |
| 10. Were the statistical methods used appropriately? | | | | |
| 11. Were all important outcomes/ results considered? | | | | |
| Overall, do you think that this study is significantly methodologically flawed? | | | | |
| Comments | | | | |

**If you have completed this table go to SECTION F, if not please go to next table and complete it if it is relevant to the study type.**

| Questions to assist with the critical appraisal of a systematic review | | | | |
|---|---|---|---|---|
| **Please tick the appropriate column** | **Yes** | **No** | **Unclear/ not known** | **Not applicable** |
| 1. The study addresses a clearly focused question and describes the inclusion and exclusion criteria of the review | | | | |
| 2. The literature search is sufficiently rigorous to identify all the relevant studies and includes at least two databases | | | | |
| 3. The quality of the study has been assessed and reported | | | | |
| 4. Sufficient details of each of the included studies are presented | | | | |
| 5. An adequate description of the methodology used to analyse the data is included, and the methods used are appropriate to the question | | | | |
| Overall, do you think that this study is significantly methodologically flawed? | | | | |
| Comments | | | | |

**If you have completed this table go to SECTION F.**

## SECTION E.
## Final Decision

| **Please tick the appropriate column** | **Yes** | **No** | **Comment** |
|---|---|---|---|
| Is the study included? | | | |
| If the study is excluded, should it be added under 'other useful references'? | | | |
| If excluded please state level of evidence and grade | | | |
| Key points meriting inclusion (list strengths) | | | |
| Weaknesses, potential confounders and study limitations (if study is INCLUDED) | | | |
| Additional comments | | | |

**If study is included, please go to SECTION F.**

## SECTION F.
## Data extraction

| | |
|---|---|
| Number of children<br>(per group for comparative) | |
| Age (range or average)<br>(per group for comparative) | |
| Male to female ratio or percentage<br>(per group for comparative) | |
| Findings | |
| Ranking of abuse | |
| Other comments | |
| Level of Evidence and Grade | |

**Thank you for completing this review**
Please return by email to Patrick.Watts@wales.nhs.uk

If you are not able to return by email, please post to:
Patrick Watts, Department of Ophthalmology, University Hospital of Wales, Cardiff CF14 4XW

# Appendix 4

**OPHTHALMOLOGY PATIENT RECORD**
**- FOR SUSPECTED PAEDIATRIC HEAD TRAUMA**

The ROYAL COLLEGE of OPHTHALMOLOGISTS

**HISTORY** _____

PATIENTS DETAILS

| Visual Acuity | Right eye | Left eye |
|---|---|---|

**OCULAR MOTILITY**

If possible to assess

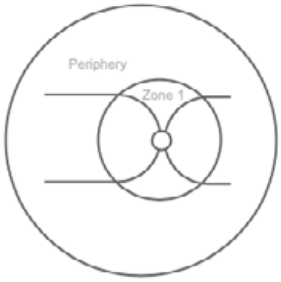
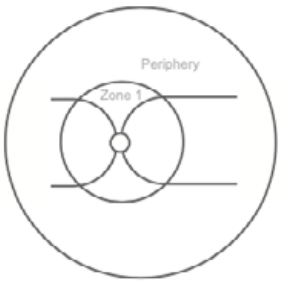
☐ ☐ ☐ Right eye ☐ ☐ ☐ ☐ ☐ ☐ Left eye ☐ ☐ ☐

| SUBCONJUNCTIVAL HAEMORRHAGES | | | |
|---|---|---|---|
| Right eye | | Left eye | |
| Yes | No | Yes | No |
| ANTERIOR SEGMENT | | | |
| Right eye | | Left eye | |

**Pupil size and Pupillary reflexes**

**PERIOCULAR BRUISING:** (mark areas of bruising)

Circle single or multiple appropriate responses if present or enter free text

| **FUNDUS** Circle if present | **RIGHT** EYE | | | | **LEFT** EYE | | | |
|---|---|---|---|---|---|---|---|---|
| **Retinal Haemorrhages** | YES | | NO | | YES | | NO | |
| **NUMBER** of Retinal haemorrhages | Few (1-10) | Many (11-20) | Too numerous to count | | Few (1-10) | Many (11-20) | Too numerous to count | |
| **LOCATION** of retinal haemorrhages | Pre retinal | Intraretinal | Subretinal | Multilayered | Pre retinal | Intraretinal | Subretinal | Multilayered |
| **DISTRIBUTION** of retinal haemorrhages | Posterior Pole Few/many/ too numerous to count (Zone 1 – ROP classification) | | Periphery Few/many/too numerous to count (outside Zone 1) | | Posterior Pole Few/many/ too numerous to count (Zone 1 – ROP classification) | | Periphery Few/many/too numerous to count (outside Zone 1) | |
| **SIZE** of retinal haemorrhages | Small (< 1dd) | Medium 1-2dd | Large >2dd | | Small (< 1dd) | Medium 1-2dd | Large >2dd | |
| **MORPHOLOGY** of haemorrhages White centered etc | | | | | | | | |
| **Macula Retinoschisis** | Yes / No |  | | | Yes / No |  | | |
| **Perimacular folds** | Yes / No | | | | Yes / No | | | |
| **Optic disc** (findings) oedema | Yes / No | | | | Yes / No | | | |



Summary

Management plan

Pupils dilated with Phenylephrine 2.5% ☐ Tropicamide 1% ☐ 0.5% ☐

**Fundus examined with**
Indirect ophthalmoscope (and 20d / 28d / 30d / 2.2d)
Photography ☐
Wide angle ☐ → *Contact* ☐ → *Non contact* ☐
Other ☐
OCT ☐

Name and signature

Date and time of examination

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