Detecting the serious visual disorders of childhood

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Abstract

Vision is important throughout childhood, leading early developmental progress, supporting academic achievement and enhancing the general quality of life. This article is directed towards the early identification of the serious ocular problems which may arise in children. General screening and assessment are discussed, including important pointers in the history and examination. Selected topics include the baby who cannot see, refractive errors and strabismus, nystagmus, cloudy or prominent eyes, and trauma.

Keywords amblyopia; congenital ocular abnormalities; eye examination; paediatric vision disorders; screening; strabismus

Learning objectives

After reading this article you should be able to:

- in order to examine a child's eyes successfully, the emphasis has to be on gaining trust first. Explaining the fun and games with child banter and nursery songs can all help tremendously
- remember vision impairment in babies can present as 'failure to bond' rather than a direct concern regarding the ability to see
- remind parents of the importance of attending the vision screening assessment around school entry age

Introduction

To be able to see the world in all its glory is a truly wonderful thing. For the new born baby, already fascinated by faces, sight is critically important in leading normal general development. A toddler's gradual exploration of their environment is captivating for any parent to behold. As childhood progresses all too quickly and schooling ensues, having a good set of eyes affords the individual with the best opportunity of reaching their true potential. Reduced vision not only tends to impair the quality of one's life, it can affect academic performance, choice of occupation and socio-economic status.

The aim of this article is to raise awareness in the importance of detecting childhood eye problems as early as possible, as they initially present. Eye disorders are relatively common, so what should general healthcare professionals seeing children be on the watch out for? The correct identification of a visual disorder at an early stage will have a significant benefit; conversely, failing to pick up on the clues can have a huge impact in terms of potential life years of visual morbidity.

Screening and general assessment

As part of the neonatal examination, paediatricians undertake a vital structural assessment of ocular integrity and check for the potentially serious ocular infection of ophthalmia neonatorum.

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Using the direct ophthalmoscope, corneal transparency and the presence of clear ocular media are confirmed with the dual observations of shiny clear anterior segment details and equally bright red reflexes. This can sometimes prove difficult in cases such as those with dark ocular pigmentation or small pupils. If there is any doubt of structural ocular pathology or cataract a referral to a paediatric ophthalmologist should be made.

Colobomas of the inferior iris, retina and choroid are relatively rare. They are the result of an embryonic failure in closure of the inferior choroidal fissure, itself created by optic vesicle invagination. Such structural mal-development can be unilateral or bilateral, associated with a smaller microphthalmic eye, and occasionally seen with other systemic congenital abnormalities. The degree of any visual impairment is dependent on the extent of the defect. Rarer still are colobomas within the eyelid and the cranio-facial syndromes which result in proptosis. These cause the instant problem of potentially sight-threatening corneal exposure. Hydrogel dressings and lacrilube ointment protection will be required before surgical correction.

Ophthalmia neonatorum presents with the signs of a profuse purulent discharge from the eyes, marked conjunctival chemosis, and redness and swelling of the eyelids. A baby may be blinded within 24 h of onset if either a gonococcus or pseudomonas species are responsible for the infection, as these can rapidly penetrate the cornea. Chlamydial ophthalmia usually presents a few days after birth and carries a risk of pneumonitis. Herpes simplex may show classic vesicles on the surrounding eyelid. It is imperative to take immediate swab specimens and commence systemic and frequent topical antibiotics whilst awaiting laboratory confirmation of the causative organism.

The neonatal screening is shortly followed up by the general practitioner 6–8 week checks. Here, as well as addressing any general eye or vision concerns, the presence of bilateral red reflexes and fixing and following behaviour is mandated. It is well to remember that a visually impaired baby may appear impassive because of the lack of fixation. This may present as a bonding issue or maternal post-natal depression.

Vision screening services offered thereafter vary throughout the country. The current gold standard is an orthoptic led screening programme undertaken at 4–5 years of age. The opportunistic screening provided at other times by all other observant healthcare professionals, family and friends plays an additional important role.

History

Parental observations and concerns usually hold the key. Pointers to a potential visual defect frequently include persistently holding objects and books up to the face, only ever sitting close to the television, general clumsiness, behaviour problems and failure to reach developmental milestones. Any observer noticing an abnormally moving or mal-aligned squinting eye, even intermittently, should never be dismissed. Squints can be very variable in nature and not always instantly obvious or reproducible on gross inspection. Ensure general health and development are on track and that there are no motor or neurological concerns.

A family history of requiring spectacles from a young age, lazy eye or squint is important and if there are any particular concerns with the child an early referral should be made. Other potential heritable ocular conditions (congenital cataract, retinitis pigmentosa, juvenile glaucoma, retinoblastoma), or systemic metabolic or genetic diseases (collagen synthesis disorders, phakomatoses) with ocular manifestations are usual prompts to initiate screening.

Vision testing

Age appropriate tests and great experience are required to accurately determine vision in children, even more-so those with developmental delay or complex learning disability where it may be difficult to interpret visual behaviour.

The assessment of pre-verbal children warrants close observation of visual behaviour. How well does the child survey their surroundings, make eye contact, and both fix and follow on an attention grabbing small toy? Particularly important for the detection of unilateral amblyopia is the reaction when either eye is occluded: varying degrees of protestation from outright distress to trying to look around occurs on having the normal eye covered; the weaker eye is covered without any objection. Based on the behavioural premise that toddlers will look for a picture or pattern rather than at a plain surface, preferential looking techniques such as Cardiff cards can provide a quantitative measure of visual acuity. From the age of two most children have sufficient language skills to name simple pictures such as in the Kay pictures test. From three upwards matching letters employing a key card is possible. For the detection of subtle amblyopia it is better to employ the more sensitive tests such as the crowded LogMAR.

Corneal reflections and the cover test

Checking that the corneal reflections of a pen torch light are symmetrical is a basic assessment of ocular alignment. In a small or uncooperative child this may be all that is possible initially.

The cover test relies upon the direct observation for any refixation movements induced by covering each eye individually. Maintaining attentive visual fixation is best achieved by using an age appropriate target. A small picture or toy works well for the younger child but detailed targets such as a letter on a reduced Snellen chart stick are preferable for those old enough to be suitably engaged. On covering one eye observe carefully the opposite eye for any movement which would positively indicate a true strabismus. An inwards movement indicates a prior divergent position (exotropia), an outwards movement a prior convergent position (esotropia); a downwards movement signifies a prior elevated position (hypertropia), an upwards movement a prior depressed position (hypotropia).

Ocular motility

The extra-ocular movements should be observed for fullness and symmetry with a pen torch or toy following across the nine cardinal positions of gaze. Restriction or under-action of the eyes can be graded on a scale of limitation as -1 (25%) to -4 (100%). The doll's head manoeuvre or swinging baby test both invoke the vestibular-ocular reflex and can help differentiate between a true restriction and a pseudo-limitation as seen in a large angle esotropia. An over-acting extra-ocular muscle shows the oppositely graded tendency ($+1$ to $+4$) for the corneal limbus border to partially disappear under the eyelid margin.

General observations regarding head and eyelid positions

Various combinations of head turns and tilts can be the result of cervical torticollis, but if the neck moves freely without limitation an ocular cause for a potential compensatory head posture (CHP) should be sought. Oculo-motor imbalance as occurs vertically in congenital superior oblique palsy and horizontally in Duane's syndrome are frequent causes of CHP. One of the features of congenital nystagmus is the null zone, an eccentric position of gaze where the nystagmus is dampened, the CHP allowing forward gaze with optimal visual acuity.

Ptosis can be of cosmetic concern, but also presents a significant barrier to normal visual development which can result in marked secondary amblyopia. Congenital levator dystrophy (where the lid neither elevates nor depresses well), Marcus-Gunn jaw-winking synkinesis, congenital Horner's syndrome and third nerve palsy are easily recognized.

Specific vision problems in childhood

The baby who can't see

In young babies, wandering gaze with no fixation is a very worrying presentation warranting urgent referral. Wherever there has been a history of prematurity, difficult birth, hypoxia, special baby care admission, early developmental delay or epilepsy, there is the possibility of cortical visual impairment. In economically developed countries, this type of brain damage is now the commonest cause of poor vision in childhood.

The phenomenon of delayed visual maturation presents similarly, as the seemingly blind baby. It is believed to be caused by late myelination within the occipital lobes. Ultimately, it can only be diagnosed by exclusion and retrospectively when the vision finally (and thankfully) 'switches on'.

There are a myriad of blinding congenital disorders affecting the cornea, lens, retina, and optic nerve which can also present early, often then with the emerging development of horizontal pendular nystagmus. A detailed ophthalmological examination may uncover the pathology. In the fairly common clinical situation where the eye examination appears normal, however, non-improvement of vision by 6 months of age warrants further investigation with the joint input of ophthalmologist and paediatrician. This should include visual system electro-diagnostic investigations and brain MR imaging.

The earlier a baby's vision problem can be picked up the better so that appropriate advice and support can be sought from the relevant teams for the visually impaired. These vary from area to

area but include Vision Assessment Teams who work with community paediatrics, ophthalmology departments, educational services (teachers for the visually impaired) and social care.

Children with vision impairment should be assessed for their developmental progress by such professionals with knowledge in this area. The usual milestones do not apply. Other complex syndromal and learning disabilities are also common, where the developmental delay may not be associated primarily with their visual problems but will be accentuated by them.

Refractive errors

Refractive errors are extremely common in adults and children, with a prevalence of around one in ten. Undetected refractive errors in children leads in a significant proportion to the development of strabismus, defects in binocularity, or amblyopia (commonly referred to as lazy eye — but more accurately considered a defect in the central visual pathways). Prompt identification and correction of the refractive error with spectacles is the ideal goal.

The commonest refractive error encountered in children is hypermetropia, or long-sightedness. According to the level of prescription required and accommodative effort, the individual's visual system has the poor choice between being either blurred with straight eyes, or accommodating to bring the image clearer but invoking the convergence associated with the near response. This excess accommodation stresses fusional mechanisms trying to hold the images from each eye together, which can fail and decompensate into a constant convergent strabismus.

Emerging myopia is more frequent at the onset of adolescence, but can occur in young children where there is a family history. High myopia with an absence of vitreous suggests Stickler syndrome, where the risk of retinal detachment is high necessitating ophthalmic screening.

Anisometropia is the condition where a difference in optical power between the eyes puts the stronger prescription eye at continuous visual disadvantage. It is a common and disabling disorder of normal binocular single vision development. Secondary unilateral amblyopia is common and may lead to strabismus; if binocularity is not completely lost and the eyes remain straight, the amblyopia may become dense and often go undetected until a visual screening assessment around the commencement of schooling.

The blurred image caused by uncorrected astigmatism, where the optical focus is different across two planes of the rugby-ball shaped eye, is also a frequent cause of amblyopia and strabismus.

High refractive errors of all types can cause bilateral amblyopia and maybe associated with various ocular and systemic conditions (Table 1).

Disorders associated with refractive errors in childhood

Ocular conditions	Systemic diseases & syndromes
Cataract	Albinism
Colobomas	Alport syndrome
Glaucoma	Alagille syndrome
Microphthalmia	Bassen-Kornzweig syndrome
Ptosis	Cerebral palsy
Retinal dystrophy	Crouzon syndrome
Retinitis pigmentosa	Down syndrome
Retinopathy of prematurity	Ehlers-Danlos syndrome
	Fabry's disease
	Flynn-Aird syndrome
	Laurence-Moon-Bardet-Biedel
	Learning disability
	Noonan syndrome
	Rett syndrome
	Sensori-neural deafness
	Senior-Loken syndrome
	Stickler syndrome
	Marfan syndrome
	WAGR syndrome
	X-linked learning disability

Table 1

Strabismus

All infants should have straight eyes with synchronized full eye movements by 6 months of age. Immaturity of the rapidly developing ocular motor systems prior to this can occasionally be responsible for temporary disturbances in ocular alignment; such fleeting deviations (most commonly esotropia) are usually noted by the parents to be lessening over time.

Quite often, there is an appearance of convergent squint but this is only simulated by broad epicanthic folds. In so-called pseudo-strabismus, the corneal light reflections are symmetrical; the cover test shows no deviation and prism tested motor fusion reflexes ultimately prove binocular single vision to be present.

The prevalence of true strabismus in children is 4%. Strabismus is primarily categorized according to the direction of deviation and secondarily by whether the deviation is apparent (manifest) or occasionally controlled and hidden (latent). Manifest strabismus can have cosmetic and functional consequences, such as amblyopia and non-binocularity. Whenever the angle of latent strabismus is too large to control, symptoms of decompensation such as eyestrain (asthenopia) and double vision may occur. The treatment of strabismus can be a very involved process over many years, and may include spectacles, occlusion amblyopia therapy, orthoptic exercises, botulinum toxin and formal squint surgery.

Congenital strabismus includes esotropia and exotropia. The former typically has an onset within the first few months of life, thus is more correctly referred to as essential infantile esotropia. Its exact cause is unknown, but possibly represents a primary dysfunction of central binocular processing. Congenital exotropia is seen less frequently, requiring careful exclusion for potential serious causes for uni-ocular visual loss as would occur in sensory exotropia.

Strabismus developing in later childhood often occurs with unrecognized refractive errors. Uncorrected hypermetropia initially causing intermittent episodes of accommodative esotropia is particularly common in children between the ages of two and four. In the opposite direction, myopia may decompensate a latent divergent squint tendency, more often in slightly older children. Intermittent exotropia is the commonest form of

divergent squint affecting children, typically upwards of 1 year of age. Parents and teachers observe one eye to spontaneously drift outwards when tired or not concentrating; monocular eye closure especially in bright light may also be reported.

Paralytic strabismus in children includes congenital and acquired causes of third, fourth and sixth cranial nerve palsies. Of these, it is congenital superior oblique palsy that is seen most frequently. The varying (incomitant) vertical strabismus induced is maximal when the affected eye adducts and classically results in a compensatory head tilt towards the shoulder of the normal eye. An eye failing to abduct more usually represents the congenital cranial dis-innervation disorder of Duane's retraction syndrome, rather than sixth nerve palsy which is quite rare in childhood. Duane's syndrome is confirmed clinically with the additional observation of narrowing palpebral fissures on adduction caused by the retracting globe as both medial and lateral recti abnormally co-contract.

All professionals involved with children need to recall that although strabismus has many important and treatable ocular causes, it can also rarely represent the harbinger of serious intracranial diseases. Red flags for hydrocephalus or brain tumour are a recent onset of double vision or an acquired paralytic strabismus. Referral for strabismus should thus not be delayed.

Nystagmus

An infant presenting with repetitive oscillations of one or both eyes can be a cause of much concern for parents and physician alike. Nystagmus results from a bewildering array of disorders which induce dysfunctional stability of the ocular movements. Investigation may uncover an ocular or neurological structural cause, or by a process of elimination suggest an idiopathic aetiology assumed to be a primary motor system defect. Such congenital idiopathic nystagmus is usually of a horizontal pendular type, is present at birth and may be inherited.

The nystagmus caused by poor vision of any cause (sensory deprivation) usually develops later, around 3–6 months of age. It also typically has a horizontal pendular character, but sometimes has rotary and jerk components especially on lateral gaze.

Although rare, it is vitally important to exclude congenital cataracts as a cause of nystagmus. Bilateral amblyopia may already be significant and not completely reversible even with urgent cataract surgery at this late stage.

Albinism is a frequent cause of infantile onset nystagmus. Not always is the case the obvious snow-white haired individual with oculo-cutaneous albinism. Blue eyed true blondes, strawberry blondes and even brunettes may have just the ocular form: identifying the sometimes subtle trans-illumination defects at the slit-lamp in a darkened room should be the ophthalmologist's first task. Visual evoked potentials as part of electro-physiological testing provides diagnostic confirmation revealing the associated higher proportion of crossing fibres within the optic chiasm (the visual input to one eye stimulates more of the contralateral occipital cortex than usual).

Retinal dystrophies may be signalled by photosensitivity, high refractive errors and pigmentary disturbances of the macular region of the retina. The ocular examination may appear normal, however, and only abnormalities on the electro-retinogram are apparent further signifying the essential role electro-physiological testing plays in the diagnosis of childhood nystagmus.

The condition of foveal hypoplasia may elude diagnosis until the child is old enough to sit still for an OCT scan (ocular coherence tomography) which confirms in cross section absence of the normal foveal depression.

Manifest latent nystagmus is associated with defects in the development of binocularity, and therefore it is frequently seen with strabismus, amblyopia and vision reducing ocular pathology. The complex of failed binocular vision also encompasses over-acting inferior obliques (eyes elevate in adduction) and dissociated vertical deviation (an eye elevates in reduced light conditions, such as behind an occluder during the cover test). In manifest latent nystagmus, the latent component is accentuated by covering each eye in turn whereby the nystagmus uniquely increases in intensity with the fast jerk phase towards the side of the seeing eye. Its importance is firstly in recognition to avoid unnecessary neurological investigation, and secondly to ensure visual acuity is measured with both eyes open as this will be much better than that with each eye individually.

Although rare, any nystagmus with onset after 6 months of age could well be neurological in origin and necessitates urgent detailed clinical evaluation and appropriate neuro-imaging. Posterior fossa structural abnormalities and tumours, and the various neuro-degenerative and inflammatory conditions may present with jerk nystagmus; there are usually other associated symptoms and signs. The curious combination of acquired asymmetrical nystagmus with head nodding and tilting represents the triad of spasmus nutans; although it may represent a benign and self-limiting condition, neuro-imaging is now recommended as there are frequent reports of parasellar and hypothalamic tumours.

Cloudy, big or prominent eyes

The normal shiny and crystal clear cornea allows an observer to see the iris in full detail. Any of a number of ocular and systemic conditions can result in opacification of the cornea and give the appearance of a cloudy eye (Table 2).

Congenital glaucoma is rare, having a prevalence of 0.01%. It classically presents with the enlarged buphthalmic hazy oedematous cornea and excessive watering, affecting one or both eyes (Figure 1).

If there is any doubt a child might have larger than normal or cloudy eye(s), the horizontal corneal diameter should be measured grossly with a ruler – 11.5 mm is normal. Anything larger is suspicious of either glaucoma pressure driven growth or the isolated developmental condition of megalocornea. Bilateral large eyes can be particularly difficult to spot.

Juvenile onset glaucoma may be associated with a positive family history, Axenfeld–Reiger syndrome, neurofibromatosis or Sturge–Weber syndrome – the corneas tend to be clear as in adult chronic glaucoma and screening is required.

Congenital abnormalities of the cornea itself can result in opacification. These are seen within the anatomic spectrum of anterior segment dysgenesis and cellular pathophysiology of congenital hereditary endothelial dystrophy.

Nutritional deficiency and any of blunt, sharp or thermal injury can also disrupt the clarity of the cornea. Where a consistent history is lacking, non-accidental injury should be suspected.

Causes of corneal opacity

Congenital	Acquired
Anterior segment dysgenesis	Band keratopathy
Birth trauma from forceps	Cystinosis
Dermoids	Epidermolysis bullosa
Glaucoma	Exposure keratopathy
Hereditary corneal dystrophies	Foreign body
Infection	Inflammatory keratitis
	Ichthyosis
	Keratoconus hydrops
	Metabolic disorders
	Mucopolysaccharidosis
	Microbial keratitis
	Neurotrophic keratopathy
	Trauma
	Vitamin deficiency

Table 2

Prominence of an eye caused by proptosis is rare in children (Figure 2). It will usually be accompanied with other signs related to orbital pathology, such as lid swelling, reduced extra-ocular motility or optic nerve dysfunction. Slowly developing proptosis is usually caused by an intra-orbital dermoid cyst. In the acute setting of an unwell febrile child, orbital cellulitis secondary to infection within the paranasal sinuses is the usual cause. Orbital cellulitis can develop from the much more commonly encountered pre-septal (peri-orbital) cellulitis if antibiotic treatment is delayed. Progression to severe complications such as blindness, neurological deficit and even death can occur quite rapidly. Emergency admission to hospital for intra-venous antibiotics is required. Optimal management occurs under the shared care of paediatrics, ENT and ophthalmology. Very occasionally, a child presenting with acute proptosis but without fever has either an orbital rhabdomyosarcoma or bleeding within a lymphangioma (chocolate cyst).

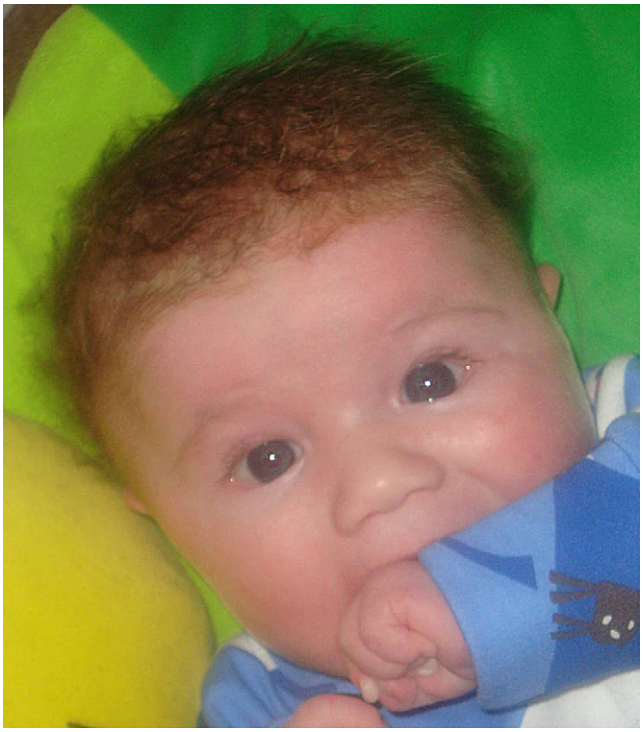

Figure 1 Early left congenital glaucoma with enlargement of the cornea. Parents noticed an asymmetry from birth, but found it difficult to pin-point and incorrectly ascribed it to a squint. The cornea became oedematous and hazy shortly after this photograph was taken. Glaucoma surgery was arranged.

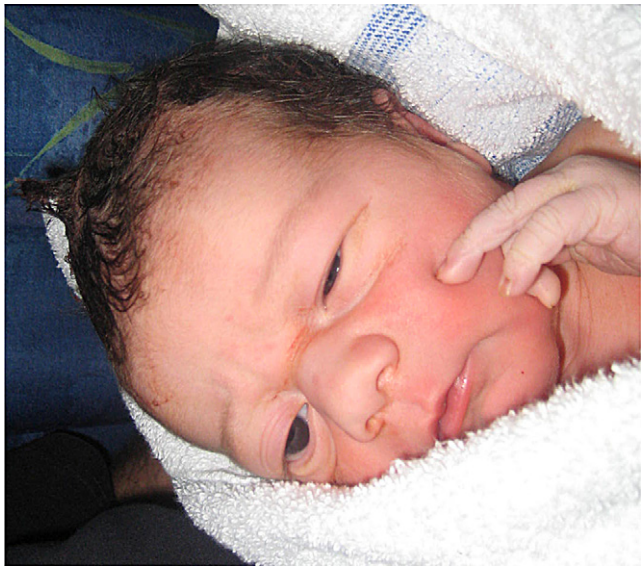

Figure 2 Marked right proptosis in a new born baby. Neuro-imaging showed a large orbit-expanding mass. A biopsy proved this to be juvenile xanthogranuloma, rarely described in this location. The lesion rapidly regressed on systemic steroids, with the only sequelae of unilateral mild enophthalmos and high myopia.

The white pupil

The white pupil (leucocoria) has many potential causes (Table 3), including top of the list cataract and the rare eye cancer retinoblastoma (Figure 3). Unfortunately, digital cameras aimed from a temporal angle of 15 degrees over the optic nerve head are now-a-days commonly producing pseudo-leucocoria as an artefact;

Causes of leucocoria

Cataract
Chorio-retinal scars (toxocara, toxoplasmosis)
Chorio-retinal coloboma
Coat's disease
Digital camera artefact
Posterior hyperplastic primary vitreous
Retinoblastoma
Retinal detachment
Severe intermediate uveitis

Table 3

Figure 3 Left leucocoria. A detailed eye examination proved completely normal in this case. Digital camera artefact leucocoria can only be diagnosed by exclusion of potentially serious ocular pathology. It is caused where the camera flash angles eccentrically over the optic nerve head; straighter photographs have normal bilateral red reflexes. All cases of leucocoria should be referred urgently.

nevertheless, such cases should be urgently examined thoroughly by an ophthalmologist before drawing any conclusion.

Congenital cataracts are rare, with a prevalence of 0.06%, but a common cause of leucocoria. Less dense cataracts cause a reduction in the normal brightness of the red reflex. The aetiology includes familial, infective, metabolic, syndromal, focal lenticular developmental abnormality (posterior lenticonus) and idiopathic. The cataracts may be unilateral or bilateral. Collaborative efforts are crucial in successful management, commencing with early diagnosis by neonatologists and general practitioners. Thorough general paediatric and ophthalmic assessments and early cataract surgery between 6 and 12 weeks are mandated to obtain good visual results; late treatment misses the critical periods of visual development with consequential sensory deprivation amblyopia and nystagmus.

Acquired cataract may also cause leucocoria in later childhood, most commonly secondary to chronic uveitis or trauma. Any secondary amblyopia occurring before visual maturity at approximately age seven will be less severe and more reversible with occlusion therapy compared with congenital cataract cases.

In any child presenting with either leucocoria or strabismus secondary to poor vision in one eye (sensory strabismus) the background concern should always be retinoblastoma. This rare sight- and life-threatening ocular cancer affects around 50 young children per year in the UK. The tumour is usually in just one eye, but in a third of cases it is bilateral. Whilst 90% cases are sporadic, the remainder are associated with a genetic fault (loss of function of the Rb tumour suppressor gene on chromosome 13) and a positive family history. It can spread throughout the eye, before invading the orbit, optic nerve and brain. It metastasizes to bone and bone marrow. Management takes place in specialist centres in London and Birmingham. The treatment for retinoblastoma offers one of the highest success rates of all childhood malignancies: combinations of surgery, cryotherapy, chemotherapy and radiotherapy can lead to cure in 95% of cases.

Trauma

Serious eye injuries occurring in children can be easily missed by the uninitiated. Left to their own devices children can get up to all kinds of mischief and may be too afraid to confess the exact mechanism of injury. It is wise to keep a very high index of suspicion. Non-accidental abuse injury is raised as a possibility where the parental history is not consistent or is not in keeping with the observed injury.

In witnessed sharp injury the immediate difficulty is in obtaining an adequate examination in a distressed child. In such a situation, penetrating eye injury will need to be very carefully excluded. There is also the possibility of retained foreign bodies within seemingly innocuous lid lacerations, ranging from the small to the surprisingly large. Small foreign bodies can present later with localized infection. Larger foreign bodies have been known to transverse the orbit and cause brain injury. If there is any doubt an urgent referral to an ophthalmologist should be made; quite often an examination under general anaesthetic is required.

Mild blunt injury causes ocular contusion, inflammation and micro-hyphaema which may raise the intra-ocular pressure. Moderate-to-severe deformational forces can easily disrupt the delicate intra-ocular contents and lead to macro-hyphaema, secondary glaucoma, cataract and retinal detachment. ◆

FURTHER READING

American Academy of Ophthalmology Pediatric Ophthalmology/Strabismus Panel. Preferred Practice Pattern® Guidelines. Pediatric eye evaluations. Available at. San Francisco, CA: American Academy of Ophthalmology, http://www.aao.org/ppp, 2007.

Ansons AM, Davies H. Diagnosis and management of ocular motility disorders. 3rd Edn. Oxford: Blackwell Science Ltd, 2001.

Orbis Telemedicine Cybersight. Available at http://www.cybersight.org.

Taylor D, Hoyt CS. Paediatric ophthalmology and strabismus. 3rd Edn. Edinburgh: Elsevier Saunders, 2005.

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