VISUAL DEFICITS

It is well known that children with ‘delayed development’ have a much higher than average incidence of visual deficits, including visual impairment (VI) (i.e. uncorrectable low visual acuity; VA) (Copper &Schappert-Kimmijser, 1970; Warburg, 1975; Warburg et al., 1979; Tuppurainen 1983; Haussler 1996; Kwok et al. 1996; Chang et al., 2005; Nielsen et al, 2007a), and refractive errors and strabismus (Fletcher & Thompson 1961; Byron 1962; Bankes 1974; Woodruff, 1977; Tuppurainen 1983;Kwok et al. 1996; Chang et al., 2005;Nielsen et al, 2007b).The reason for this is varied and may reflect geneticcauses, chromosomal abnormalities, brain disorders, prematurity, perinatal insults, and infectious diseases. Nielsen et al (2007a) also reported ethnic differences with an over-representation of children originating from the middle-and far-east, and eastern Europe.

In the UK, the impact of VI on learning has been extensively reported on by the RNIB (Chanfreau&Cebulla, 2009; Bassett 2010). The generalfindingsfor children with only VI are that educational attainment falls behind from reception (age 4 years) and this attainment gap increases until Key Stage 3 (ages 11-14years), and remains the same thereafter. Reading, mathematics, and science are all affected.VI has other effects on school children including emotional well-being and social activities (Harris et al., 2014). When VI is associated with other disabilities, the effects of VI are considerably worse.

It should be noted that the effects of visual deficits on cognitive processing is generally poorly understood. Low IQ (‘developmental delay’) is often used as a measure, but this not adequate for ‘special’ learning difficulties where normal IQ is defined (or assumed) to be normal. However, the causal relationships among IQ (especially how it is assessed), VI, and learning difficulties (LD) are poorly understood. For example, in dyslexia the emphasis in the most recent 10 years has been on poor phonological deficits (Castles &Coltheart, 2004), even though the primary input is visual orthographic text. As pointed out by Whitney & Cornelissen (2007), poor phonological awareness may be the symptom rather than the cause of dyslexia. Abnormalities of visual processing (and eye movement control) may be at the root of dyslexia, especially in the young early reader.

We present below a range of known ‘clinical’ visual deficits that need to be considered as possibly contributory, or even causal in some instances, for cognitive processing. It is crucial to recognise that visual deficits in children in mainstream school may not be known to teachers for many reasons, including lack of medical assessment, socioeconomic causes, poor institutional communication between the educational and medical professions, different national systems in the EU, and statutory limitations. For example, in the UK, a colour vision deficit is not considered a disability, and is therefore not assessed at any age (in spite of 8% of boys being affected). Thus, it must be assumed that a child with LD could have one or more visual deficits.

This project was financed with support from the European Commission. This publication reflects the views only of the author, and the Commission cannot be held responsible for any use which may be made of the information contained therein.

SPECIFIC VISUAL DEFICITS

Most studies and reports on visual problems have focussed on either VI defined as low visual acuity (VA; poor resolution),refractive error (blur due to defocus), and strabismus (eye misalignment). These abnormalities can be quantified objectively and form the basis of any standard clinical assessment. There are many other visual deficits that also have impacts on vision, such as colour vision deficits, eye movement disorders and cerebral visual impairment (brain injury). These require specialist assessment and are not readily quantified by a single number (as in VA for example). Nevertheless, their impact may be more profound.

To understand this, it is important to recognise that much of visual processing (reading, looking at pictures, etc.) requires the control of a complex sequential process offoveation and processing visual content. Visual resolution is maximum at the retinal fovea (central vision), so that the image of a small feature must be imaged on the fovea ofeach eye. If the image is blurred due to poor optics (refractive error) or the retinal fovea is damaged (VI), the 'visual' image is poor and limits the ability for the child to detect or recognise the object. For example, the ability of children to recognise words decreases with smaller print size (Cornelissen et al. 1991) (with or without dyslexia).

If, however, the image falls away from the fovea due to poor eye movement control, then a similar deficit will occur. Retinal signals are passed to the primary visual cortex (V1) where conscious seeing takes place, and are then processed by other parts of the brain where recognition and interpretation takes place (the ventral stream), and the guidance of movement takes place (the dorsal stream). Damage to different parts of the brain can lead to specific cognitive deficits (such as poor face recognition, poor letter recognition, poor reaching etc.). Once a specific feature has been processed, it is necessary to re-direct the fovea to the next visual feature (e.g. next word in a line of text), which requires saccadic eye movements. The whole procedure is integrated and usually subconscious, but any difficulty in any part of the process will interfere with cognitive vision and perception

In practice, it is difficult to separate the cause and effect of cognitive deficits. For example, poor eye movement control can make reading difficult, but genuine dyslexia can lead to atypical eye movements. In general, a child labelled with a SLD may have a sensory-motor deficit instead of, or in addition to, any apparent cognitive deficit. Visual screening in early childhood is very limited in the range of tests carried out. For example, in the UK colour vision deficits are not considered a disability and not screened for. There is also no certainty that any specific child has undergone screening anyway. Our default position is that a child may have visual deficits until shown otherwise. We next describe some of the more common visual deficits that need to be examined for. Any single or combination of deficits can lead to poor visual function and slow learning in a young child.

This project was financed with support from the European Commission. This publication reflects the views only of the author, and the Commission cannot be held responsible for any use which may be made of the information contained therein.

REFRACTIVE ERROR

Description

Refractive error (RE) (sometimes called ‘ametropia’) is defocus of the image at the retina, which leads to blurry vision and reduced visual acuity (VA). VA is usually measured as the size of the smallest letter (or other optotype) that can be recognised on a chart.RE is measured with a retinoscope when accommodation is temporarily paralysed (cyclopegia). When the image of a distant object falls behind the retina, the RE is hyperopia (or ‘hypometropia’) (long-sightedness). When the image forms in front of the retina the RE is myopia (short-sightedness). If the image formed is in focus at the retina, no RE is present (emmetropia). The prevalence of RE varies widely depending on ethnicity (Rudnicka et al 2010).

In children, the shape of the lens can be controlled to change the optical power of the eye (called ‘accommodation’). Thus, making the lens more spherical increases optical power and allows the image of near objects to be brought to a focus on the retina. Conversely, making the lens flatter brings far objects into focus. Thus, to some extent RE can be overcome by accommodation, but this can be incomplete and effortful (tiring).

RE can be corrected optically by spectacles or contact lenses and is therefore an avoidable cause of visual impairment. Nevertheless, uncorrected RE is the most common cause of poor visual acuity in school-age children.In a study of white children in Northern Ireland, Donoghue et al (2010) reported the prevalence of refractive error in two groups: 6-7year olds and 12-13 year olds. In the younger group, myopia was present in 2.8% and hyperopia in 26%, and in the older group myopia was present in 17.7% and hyperopia in 14.7%. However, even though spectacles had been prescribed, a quarter of them did not wear them at school. Thus, compliance is an important issue.

Effects

Uncorrected RE leads to loss of VA, blur, and low contrast sensitivity at high spatial frequencies.Myopia and hyperopia need to be considered separately. Myopic children are less affected for near work (such as a display) because the eye can accommodate, but even so, severe myopia can lead to very close work (bringing the display up very close). Hyperopic children may be able to accommodate near targets, but this can lead to eye strain and headache. Children with hyperopic RE tend to have poorer performance on visual cognitive tests (Atkinson et al., 2002), and poorer literacy (Williams et al., 2005). Hyperopia is more prevalent in children with LD than children with no LD (Rosner&Rosner, 1987).

Diagnostic

RE is diagnosed by refractionby a specialist.

UNCORRECTABLE LOW VISUAL ACUITY (VISUAL IMPAIRMENT).

This project was financed with support from the European Commission. This publication reflects the views only of the author, and the Commission cannot be held responsible for any use which may be made of the information contained therein.

Description

Uncorrectable low visual acuity reflects intrinsic anomalies/damage to the visual pathways that cannot be corrected by lenses. It is often called 'visual impairment' (but this usage is variable). VI may be due to a retinal or brain disorder (or both).In children with delayed development (IQ<=80),Nielsen at al. (2007a) found an incidence of 10.5% which correlated negatively with IQ, (reaching 22.4% for IQ<50). The causes of VI are manifold but most are pre-natal (with most being genetic) or peri-natal (with most due to prematurity). According to Nielsen et al, over half are secondary to brain damage, or cerebral visual impairment (CVI see below).

There are very many specific disorders that cause VI, and for a given diagnosis, each child is affected to a different degree. Thus, medical diagnoses are only rough guides to the disability experienced by the child. The commonality is, however, poor contrast sensitivity and low visual acuity (VA) ranging from mild visual loss to total blindness (rare). Note that there may be associated visual deficits, such as nystagmus, amblyopia.

Effect

As with RE, the main effect is low VA, poor contrast sensitivity at high spatial frequencies. However, there is no treatment. RE often occurs in addition to VI.

Diagnostic

VI is diagnosed by an ophthalmologist.

STRABISMUS & AMBLYOPIA

Description

Strabismus is a misalignment of the eyes (usually horizontal). In the UK it also known as "squint". Strabismus occurs in about 5% of schoolchildren. The most common type (60%) is when one 'turns in' (esotropia), but one eye may turn out (exotropia). The condition may start in infancy or later. Strabismus can usually be corrected by surgery. A common consequence of strabismus is amblyopia, where the vision of one eye fails to develop (cortically) normally. This is sometimes known as "lazy eye". Amblyopia can be treated by occlusion therapy (patching) where the normal eye is covered to force the poorer eye to develop, but treatment must start early otherwise the poor vision becomes permanent. Amblyopia can also be caused by unequal RE in each eye or when one is deprived of vision (such as cataract).

Effect

Strabismus and amblyopia usually only affects vision in one eye. If vision is the unaffected eye is normal, then behavioural vision is near-normal. However, most children have poor or no stereopsis, thus affecting depth perception. For

This project was financed with support from the European Commission. This publication reflects the views only of the author, and the Commission cannot be held responsible for any use which may be made of the information contained therein.

example, wearing 3-D glasses for a 3-D movie fails to give rise to the perception of depth.

Diagnostic

Diagnosed by a specialist.

COLOUR VISION DEFICIENCY

Description

Normal human colour vision is trichromatic and requires normal functioning of three cone pigments referred to as 'short', 'medium', and 'long' wavelength pigments (or 'blue', 'green', 'red' pigments'). When a pigment is deficient, the terms tritan (short), deutan (medium) and protan (long) are used. The genes for the L and M pigments are on the X chromosome; the S pigment is on chromosome 7. Mutations of the L and M pigments give rise to anomalous colour vision for medium/long wavelengths (red-green colours for a normal trichromat).

The vast majority of colour vision deficiencies are inherited, and occur independently of any other cognitive/brain problems. Most are X-linked recessive and affect about 8% of males and 0.5% females of European descent. Children with learning difficulties are just as likely to be affected as any other child.

The most common deficit is deuteranomaly, caused by an anomalous shifting of the medium wavelength to longer wavelengths. It affects 4.6% of males and 0.4% of females. Complete loss of medium wavelength function is called deuteranopia and affects 1.3% males and 0.01% females. In protanomaly, the L pigment is shifted to lower wavelength and affects 1.1% males, and 0.03% females, and loss of L pigment function is called protanopia affecting 1% males and 0.02% females. Abnormalities of the S pigment, called tritanopia, are much rarer (<0.02%) with autosomal dominant inheritance affecting males and females equally.

Effects

Colour vision is important for identifying objects and increasing contrast, especially in complex visual scenes. Colour vision is also used connotatively to convey a specific meaning or message (e.g. danger, traffic lights, etc.). The perception of colour depends on the relative absorption of the three pigments, so people with colour deficiency have a different perception of colours that can lead to difficulties in identifying and interpreting objects. For deutan and protan deficits, there is reduced (or absent) discriminability between long wavelengths, that is distinguishing between red – green colours.

This project was financed with support from the European Commission. This publication reflects the views only of the author, and the Commission cannot be held responsible for any use which may be made of the information contained therein.

Diagnostics

Difficult to diagnose, and not routine in the UK.

NYSTAGMUS

Description

Nystagmus is an eye movement disorder characterised by involuntary oscillation of the eyes. Oscillations may be horizontal, vertical, or torsional (rotary) and may affect one eye or both eyes. There are many types of nystagmus, but they are usually classified as 'developmental' or 'acquired'. Developmental nystagmus has an onset in early infancy and is usually associated with congenital visual deficits, whereas acquired nystagmus can have an onset from infancy (very rare) to adulthood and is associated with neurological disease.In schoolchildren, the vast majority of nystagmus is developmental and can be subdivided into two types: Latent nystagmus (LN) (also known as Fusion Maldevelopment Nystagmus Syndrome) and Infantile nystagmus (IN) (also known as infantile nystagmus syndrome, or congenital nystagmus.

LN is strongly associated with early-onset strabismus; ~99% of cases of LN have strabismus, and ~50% of infants with early onset strabismus develop LN. Given that 1% of infants develop early-onset strabismus, the incidence of LN is 0.5% (1:200), but many cases go unreported (Harris 2014). A peculiarity of LN is that it becomes manifest or worse when one eye is occluded. LN is horizontal (sometimes with an additional torsional component). LN occurs with unequal vision from birth (see amblyopia), and most cases the nystagmus is always manifest to some degree since vision in one eye is worse (equivalent to partial occlusion).

IN occurs in ~0.3% of infants, and is associated in 70% of cases with bilateral visual deficits (e.g. albinism, cataracts, aniridia, and many more). In 30% no underlying visual defect can be identified, and the nystagmus is labelled as "idiopathic" IN (Harris 2014). IN is usually horizontal and conjugate (same in both eyes), but there are exceptions.

Effects

Even though the nystagmus causes the fovea to be constantly moving across the point of regard in a visual scene, children do NOT normally experience the visual world as oscillating (called "oscillopsia"). IN often increases in intensity when the child is under stress, and oscillopsia may then occur, but it is not a serious problem (unlike acquired nystagmus).

This project was financed with support from the European Commission. This publication reflects the views only of the author, and the Commission cannot be held responsible for any use which may be made of the information contained therein.

In terms of VA, it is difficult to distinguish between the cause and effect of developmental nystagmus.Recent data suggests that, even in idiopathic IN, the moving eyes do not reduce VA. Rather, VA is already poor due either to an unknown retinal problem or amblyopia induced by the nystagmus(Dunn et al., 2014). VA is typically 0.3 logMar, but ranges widely. Thus, in this respect, developmental nystagmus should be viewed as an uncorrectable cause of poor VA.

Nystagmus has other important and unique effects, however.The intensity of nystagmus in both LN and IN depends on the gaze direction (the direction of the eyes relative to the head), and typically there is a regionof least nystagmus called a “null region”. For many the null region is not straight ahead, but to one side or up or down. The child will then adopt an abnormal head posture to compensate by bringing the null region to bear on the region of visual interest. For example, a null region in right gaze is compensated by turning the head to the left.

Foveating a peripheral visual target is usually achieved by a saccadic eye movement, sometimes accompanied with a head movement. Thus, in normal viewing of a visual scene different visual targets are sequentially processed rapidly by an alternating sequence of fixations and saccades (called “visual scanning”).Nystagmus prevents precise foveation, so that viewing a peripheral target takes longer, which is colloquially known as the “time-to-see” phenomenon. It depends on the precise waveform of the oscillations, and is exacerbated by stress (Jones et al., 2013). Thus, nystagmus should be viewed as causing processing delays, although they may not have a cognitive component *per se*, but simply reflect delay in foveation.

Diagnostics

Nystagmus can usually be seen clinically, but eye movement recording is necessary to identify the type of nystagmus.

SACCADE INITIATION FAILURE

Description

Saccade initiation failure (SIF) is a difficulty in triggering saccades(Harris et al., 1996). SIF is also known as “ocular motor apraxia”, or “congenital ocular motor apraxia”, but the condition is not a true apraxia. SIF is distinct from “dyspraxia”. SIF is often considered rare, but this reflects the difficulty in recognising the problem. SIF is associated with a very wide range of clinical conditions from brain malformations, perinatal disorders, neurodegenerative conditions, and a variety of syndromes(Cassidy et al, 2000). The most typical findings on brain imaging are cerebellar malformations and white matter

This project was financed with support from the European Commission. This publication reflects the views only of the author, and the Commission cannot be held responsible for any use which may be made of the information contained therein.

disorders, but in many cases no underlying neurological cause can be found(Shawkat et al, 1995).

Regardless of the underlying neurology, most children with SIF also have other issues. Developmental delay is common with late walking and poor motor coordination. Expressive (articulatory) speech delay is very common and usually requires speech therapy. Other eye movement abnormalities are also common including poor smooth pursuit, inaccurate saccades (saccade dysmetria), strabismus, and also nystagmus less commonly.

To overcome the saccadic deficit, many children develop compensatory strategies such as a peculiar head-thrusting and/or blinking which helps to trigger saccades. However this is not universal, and in any case, they are not easy to detect.

Effects

The main effect of SIF is the difficulty in shifting gaze from one visual object to another. For most children, this prolongs fixation on the current object and delays refixations. For the severely affected, refixations may be virtually impossible and the child appears not to look at or scan the visual scene. Indeed, infants with severe SIF appear blind.

Reading and scanning pictures become difficult and can be misinterpreted by parents, teachers, and psychological assessorsas indicating dyslexia, learning difficulties, and even low IQ (Jan et al. 1998). For example, performance on IQ tasks that require many saccades (such as comparing pictures) can be below normal but incorrectly interpreted as low visuo-spatial IQ, which when coupled with speech difficulties can indicate low general IQ, even though cognitive function may be normal. Currently, it is not known how many children with LD actually have SIF.

Diagnostics

SIF is difficult to test for, and diagnosis is usually made in a specialist paediatric ophthalmology centre. Eye movement recording will show long saccade latencies and prolonged fixations. Without eye movement recording, SIF appears similar to the time-to-see phenomenon in nystagmus.

For cognitive visual tasks, it is important to gauge their demand for saccadic eye movements separately from their cognitive content (e.g. comparing look-alike pictures for differences).

Reading may also be slow because of the difficulty in fixating the next word along the line, rather than a problem in comprehension. This is particularly important for left-right languages (rather than vertical languages),as SIF usually only affects horizontal saccades. Children with SIF will sometimes turn

This project was financed with support from the European Commission. This publication reflects the views only of the author, and the Commission cannot be held responsible for any use which may be made of the information contained therein.

a book horizontally in order to read ‘down’ the line. Others will move the book jerkily to the left to reduce the need to make saccades (potentially a very useful diagnostic via hand-held tablets).

There is a compelling overlapof SIF with the general condition of developmental coordination disorder (DCD), (viz: oral/speech dyspraxia and motor dyspraxia). Even the use of head movements instead of eye movements has been emphasised: see http://edbydesign.com/learning/learning-disabilities/dyspraxia.html.

CEREBRAL VISUAL IMPAIRMENT

Description

Cerebral visual impairment (CVI) (sometime called “cortical visual impairment”) refers to children who have visual problems in processing visual information that is NOT due to problems with the eyes *per se* (although children with CVI may also have ocular problems). We consider eye movement disorders as separate from CVI.

In schoolchildren, CVI usually originates from birth (pre-natal or peri-natal). In particular, there is a strong association of CVI with premature or traumatic birth (e.g. periventricular leukomalacia, perinatal asphyxiation). Postnatal head trauma (e.g. physical abuse, head injury) can also lead to CVI but is less common.CVI may also follow brain tumour, meningitis, hydrocephalus or encephalitis.

Effects

CVI can be due to damage to the visual cortex and associated cortices involved in processing visual information (as well as other areas of the brain). Damage to the visual cortex (V1) can result in a range of VI from complete cortical blindness to reduced visual acuity (with normal eyes), and various degrees of field defects in which various regions of the visual field are ‘blind’.

When associated visual areas are affected, CVI can also lead to cognitive difficulties in:

1. Recognising objects, such as faces (prosopagnosia)
2. Spatial orientation and visually guided behaviour
3. Depth perception
4. Movement perception (akinetopsia)
5. Simultaneous perception (simultanagnosia), and figure-ground separation.

This project was financed with support from the European Commission. This publication reflects the views only of the author, and the Commission cannot be held responsible for any use which may be made of the information contained therein.

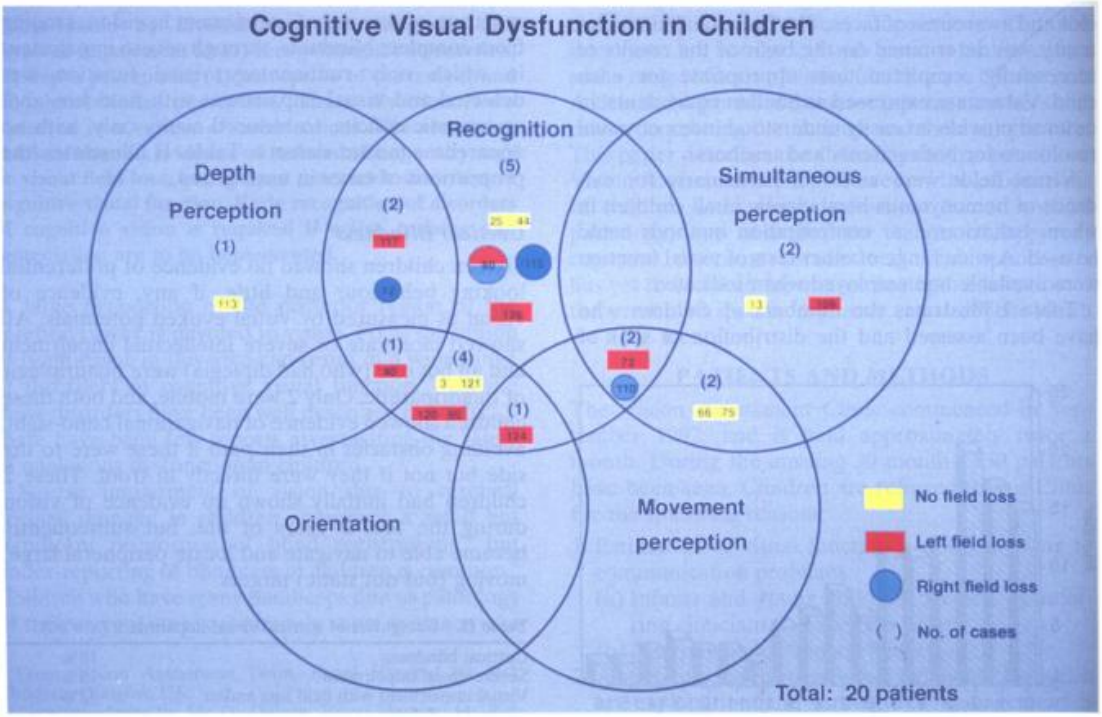

Figure 1From Dutton et al. (1996)

Simultanagnosia is the inability to see more than one object at the same time, and consequently patients have difficulty in seeing the 'overall' picture. According to Dutton et al. (1996), face recognition was more problematic for groups of well-known individuals rather than one-to-one, suggesting a simultanagnostic component as well.

Diagnostics

There is no simple way to identify CVI. It is usually based on poor behavioural vision with normal eyes. However, this is inadequate when there is an additional ocular problem, or when there are only cognitive deficits. It is also notoriously difficult to predict CVI from medical history or from MRI images.

There is clearly an overlap with CVI and LDs, but the prevalence of some degree of CVI in children with LD is not known.Some important signs that suggest CVI are:

a) Not reaching for objects to one side, or looking away from an object/person in order to see them (hemianopia).
b) Seeing only objects if they are still (akinetopsia), or for some children when objects do move.
c) Poor responses to faces (prosopagnosia)
d) Difficulty in responding to objects with a 'busy' background (simultanagnosia).

This project was financed with support from the European Commission. This publication reflects the views only of the author, and the Commission cannot be held responsible for any use which may be made of the information contained therein.

e) Looking for an object and then purposefully looking away before reaching for it (optic ataxia).

IMPLICATIONS FOR VISUAL DISPLAYS

To maximise a child's performance it is necessary to adapt a visual display to the child's visual abilities. Assuming no eye movement recording ability, the main visual dimensions that need to be adapted and hence 'diagnosed' are:

1) Colour content
2) Minimum object size
3) Sensitivity to object density
4) Response time allowance
5) Pointing accuracy
6) Ability to recognise/discriminate faces

[7 Care with objects in depth (disparity)]

Interestingly, in a recent design of therapeutic visual games for children with CVI, Waddington et al. (2015) employed adapted control of (2), (3), and (4). They also recommended san-serif fonts (e.g. Arial), voiceover or audio feedback when the cursor or finger (touchscreen) is over a usable icon, and simplifying or removing visual backgrounds.

It is important to recognise that these adaptations are not independent of each other. For example, a visual object with low visual contrast (from the child's perspective) takes longer to respond to. Using red vs. green objects for diagnosing minimum object size would be counterproductive for a colour blind child. A child with simultanagnosiawill not perform well on any task with a crowded display. A child with prosopagnosia will not perform well on a task that requires the discrimination of faces, and so on. Given that a child's abilities are unknown at the outset, care is needed in the 'diagnostic' tests and their sequence of administration.

REFERENCES

Atkinson J, Anker S, Nardini M, Braddick O, Hughes C, Rae S, Wattam-Bell J, Atkinson S (2002) Infant vision screening predicts failures on motor and cognitive tests up to school age. Strabismus 10:187-198.

This project was financed with support from the European Commission. This publication reflects the views only of the author, and the Commission cannot be held responsible for any use which may be made of the information contained therein.

Bankes JLK (1974): Ocular problems of mentally handicapped children. Aust J Ophthalmol 2: 71–74.

Bassett, P. (2010). Educational progress of young blind and partially sighted pupils.Statsconsultancy for RNIB.

Byron HM (1962): Ophthalmic survey of 162 mentally retarded children. N Y State JMed 62: 3102–3104.

Cassidy L, Taylor D, Harris CM. (2000) Abnormalsupranuclear eye movements in the child - a practical guide to examination and interpretation Survey of Ophthalmology 44:479-506.

Chanfreau, J., &Cebulla, A. (2009).Educational attainment of blind and partially sighted pupils. London: RNIB.

Chang YS, Shih MH, Tseng SH, Cheng HC &Teng CL (2005): Ophthalmologic abnormalities in high school students with mental retardation in Taiwan. J Formos Med Assoc 104: 578–584

Copper AC &Schappert-Kimmijser J (1970):The causes of blindness in 156 visually and mentally defective children. Ophthalmologica 160: 302.

Cornellisen P, Bradley L, Fowler S, Stein J (1991) What children see affects how they read. Dev Med Child Neurol 33:755-762.

Donoghue L, McClelland JF, Logan NS, Rudnicka AR, Owen CG, Saunders KJ (2010) Refractive error and visual impairment in school children in Northern Ireland. British Journal of Ophthalmology 94:1155-1159.

Dunn MJ, Margrain TH, Woodhouse JM, Ennis FA, Harris CM, Erichsen JT (2014) Visual acuity in infantile nystagmus in the absence of image motion. *Investigative Ophthalmology and Visual Science*55:2682-6.

Dutton G, Ballantyne J, Boyd G, Bradnam M, Day R,McCulloch D, Mackie R, Phillips S, Saunders K. Corticalvisual dysfunction in children: a clinical study. Eye 1996;10: 302–309.

Fletcher MC & Thompson MM (1961): Eye abnormalities in the mentally defective. Am J MentDefic 66: 242–244.

Harris CM, Shawkat F, Russell-Eggitt I, Wilson J, Taylor D. (1996) Intermittent horizontal saccade failure ("ocular motor apraxia") in children. British Journal of Ophthalmology 80: 151-158.

Harris CM (2013) Infantile (congenital) nystagmus. Optometry Today56 (July 12): pp.48-53

Harris CM (2013) Latent nystagmus.Optometry Today54 (May 17): pp.49-53.

Haussler M, Bartels H &Strassbourg HM (1996): Multihandicapped blind and partially sighted children in South Germany. I. Prevalence, impairments and ophthalmologic findings. Dev Med Child Neurol 38:1068–1075.

This project was financed with support from the European Commission. This publication reflects the views only of the author, and the Commission cannot be held responsible for any use which may be made of the information contained therein.

Jan JE, Kearney S, Groenveld M, Sargent MA, Poskitt KJ. (1998)Speech, cognition, and imaging studies in congenital ocularmotor apraxia.Developmental Medicine& Child Neurology 40: 95–99.

Jones PH, Harris CM, Woodhouse JM, Margrain TM, Ennis F, Erichsen JT (2013) The impact of stress on the visual function of patients with infantile nystagmus syndrome. *Investigative Ophthalmology and Visual Science*54:7943-51.

Kwok SK, Ho PCP, Chan AKH, Gandhi SR & Lam DSC (1996): Ocular defects in children and adolescents with severe mental deficiency. J Intellect Disabil Res 40: 330–335.

McQuaid RD &Arvidsson J (1992): Vision examination of children in Riyadh's handicapped children house. J Am OptomAssoc 63: 262–265.

Nielsen LS, Skov L, Jensen H. (2007a) Visual dysfunctions and ocular disorders inchildren with developmental delay. I Prevalence, diagnoses and aetiology of visual impairment. Acta Opthalmologica Scandinavica ;85:149-56.

Nielsen LS, Skov L, Jensen H. (2007b) Visual dysfunctions and ocular disorders in children withdevelopmental delay. II Aspects of refractive errors, strabismus and contrast sensitivity. Acta Opthalmologica Scandinavica 85:419-26.

Rosner JRosner J (1987) Comparison of visual characteristics in children with and without learning difficulties. Am J OptomPhysiol Opt 64: 531-533.

Rudnicka AR, Owen CG, Nightingale CM, Cook DG, Whincup PH (2010) Ethnic differences in the prevalence of myopia and ocular biometry in 10- and 11-year-old children: the child heart and health study in England (CHASE). Investigative Ophthalmology& Visual Sciences 51: 6270-6276.

Shawkat FS, Kingsley D, Kendall B, Russell-Eggitt I, Taylor DSI, Harris CM. (1995) Neuro-radiological and eye movement correlates in children with intermittent saccade failure: "ocular motor apraxia". Neuropediatrics 26: 298-305.

Tuppurainen K (1983): Ocular findings among mentally retarded children in Finland. ActaOphthalmolScand 61: 634–644.

Waddington, J., Linehan, C., Gerling, K., Hicks, K., & Hodgson, T. (2015).Participatory design of therapeutic video games for young people with neurological vision impairment. In: CHI 2015, 18-24 April 2015, Seoul, Republic of Korea.

Warburg M (2001): Visual impairment in adult people with intellectual disability: literature review. J Intellect Disabil Res 45:424–438.

Warburg M, Frederiksen P &Rattleff J (1979): Blindness among 7700 mentally retarded children. In: Smith V & Keen J(eds). Visual Handicap in Children.

Whitney C, Cornelissen P (2007) Letter-position encoding and dyslexia.In Visual Factors in Reading (eds. PL Cornelissen and C Singleton) Blackwell Publishing; UK.

This project was financed with support from the European Commission. This publication reflects the views only of the author, and the Commission cannot be held responsible for any use which may be made of the information contained therein.

Williams WR, Latif AHA, Hannington L, Watkins DR (2005) Hyperopia and educational attainment in a primary school cohort. Archives of Diseases in Childhood 90: 150-153.

Woodruff ME (1977): Prevalence of visual and ocular anomalies in 168 non-institutionalized mentally retarded children. Can J Public Health 68: 225–232.

This project was financed with support from the European Commission. This publication reflects the views only of the author, and the Commission cannot be held responsible for any use which may be made of the information contained therein.