In press, Neuroscience and BioBehavioral Reviews

The inter-relationships between cerebral visual impairment, autism and intellectual disability.

Chokron S.[1,2,3], Kovarski K.[1,2,3], Zalla T.[4], Dutton G.N.[5]

1. Fondation Ophtalmologique A. de Rothschild, Unité Vision et Cognition, Paris, France
2. CNRS (Integrative Neuroscience and Cognition Center, UMR 8002), Paris, France
3. Université Paris Descartes, Sorbonne Paris Cité, Paris, France
4. Institut Jean Nicod, CNRS, Ecole Normale Supérieure, Paris, France
5. Department of Vision Science, Glasgow Caledonian University, Glasgow, UK

Corresponding author
Sylvie Chokron, PhD
CNRS (Integrative Neuroscience and Cognition Center, UMR 8002), Paris, France
Centre Universitaire des Saints Pères
45 Rue des Saints Pères
75006, Paris, France

Declaration of conflicting interest
The authors declare that there are no conflicts of interest.

Abstract

From birth, vision guides our movement, facilitates social interaction and accords recognition and understanding of the environment. In children, vision underpins development of these skills, and is crucial for typical development. Deficits in visual processing may lead to impairment of cognitive, motor, and social development, placing children at risk of developing features of autism. Severe early onset visual dysfunction accords the greatest risk. Cerebral Visual Impairment (CVI) can lead to disorders of cognitive and social development that resemble Autism Spectrum Disorder (ASD). Similarly, children who appear primarily affected by cognitive and social developmental disorders, can manifest a range of visual and perceptual deficits that may be contributory to their disorder. This dual perspective highlights the need for links between impaired vision and neurodevelopmental disorders to be identified and acted upon by means of applying appropriate social and educational strategies. There is good evidence to show that targeted systematic screening for visual and perceptual impairments, and implementation of long-term management approaches, is now required for all at risk children.

Keywords: CVI; Blindness; ASD; Intellectual Disability; Interaction, Cognitive development

Introduction

Early sensory, cognitive and emotional experiences contribute to brain development by shaping the construction and subsequent refinement of neuronal-circuitry. For this reason, visual as well as hearing impairments can profoundly limit social, cognitive and motor development (Tamis-LeMonda et al., 1996). In particular, visual recognition and memory during infancy are crucial for the development of a great number of skills (Herschkowitz et al., 1997). Indeed, before being able to interact or communicate with others, infants experience a visual relationship with the environment (de Hevia et al., 2014). From birth, vision is particularly involved in the development of guidance of motor functions, postural control, grasping, visual search and recognition of shapes and objects. For instance, infants aged between 5 and 18 months use their vision to begin to reach out, grasp and manipulate small objects placed in front of them (Braddick and Atkinson, 2013). Vision is also required to identify and explore social cues such as faces, that represent preferential stimuli in newborns (Johnson et al., 1991), with early face processing being crucial at first for establishing and maintaining relationships, and later for the understanding of others' intentions. Vision is thus strongly involved in early cognitive and social development. However, whereas the diagnosis of ophthalmic disorders has improved significantly in recent years, visual impairments due to neurological lesions often remain underestimated and underdiagnosed. Nevertheless, in industrialized nations the Cerebral (or Cortical) Visual Impairments (CVI) have become the leading cause of severe visual impairment in children (Dutton and Bax, 2010; Kong et al., 2012), and represent one of the major contributors to developmental disorders (Watson et al., 2003). Although often neglected in clinical practice, the link between sensory impairments, developmental disorders and learning disabilities is yet well documented.

For instance, early onset visual and/or hearing deficits may impair the development of crucial adaptive skills (Kekelis and Andersen, 1984; Rogers and Puchalski, 1988). Disturbances of visual processes have thus been found to be closely associated with a range of neurodevelopmental and psychiatric conditions (Jeyabalan and Clement, 2016), placing early emotional, cognitive and adaptive skills at risk (Ek et al., 1998). Without early diagnosis and intervention, visual impairments can thus severely compromise child development and quality of life (Chadha and Subramanian, 2011), and can also contribute to the manifestation of autistic-like traits (Fazzi et al., 2019; Fazzi et al., 2007). Moreover, the deleterious consequences of CVI on cognitive and social development can easily be confounded with other primary conditions such as Autism Spectrum Disorder (ASD) or Intellectual Deficiency (ID). Indeed, the distinction between CVI, ID and ASD/autistic traits has become increasingly difficult to make for two reasons. First, in recent years, both ASD and ID have both been associated with anomalies of visual and perceptual function (Little, 2018; Simmons et al., 2009) and second, the adverse impact of CVI on cognitive development and social relationships is now established (Chokron and Dutton, 2016; Fazzi et al., 2015; Pawlenko et al., 2014).

The present review seeks to highlight the relationships between visual function, social development and cognitive functions, through comparison of the manifestations of CVI with those of ASD and ID, as these are frequent neurodevelopmental disorders manifesting both visual deficits and cognitive and social interaction disabilities. These conditions present important similarities, thus warranting further investigation to elicit visual deficits, whose appropriate management can accord significant benefit.

1. Cerebral Visual Impairment (CVI)

1.1. Definition

From their in depth review of the salient evidence based literature, Sakki et al. (2018) have recently proposed that CVI should be defined as 'a verifiable visual dysfunction which cannot be attributed to disorders of the anterior visual pathways or any potentially co-occurring ocular impairment'. The term CVI thus describes damage (with structural lesions) or malfunction (due to non-structural impairment) of the visual pathways, including the lateral geniculate bodies, the optic radiations, the occipital cortices and the visual association areas, as well as the tectum and thalamus (Bauer et al., 2014). Additionally, associated disorders of eye movement control are commonly observed (Chokron and Dutton, 2016). As in children with cerebral palsy, brain imaging may show no visible structural abnormality. As a consequence, owing to the heterogeneity of expression and to the association of co-occurring disturbances, the definition of CVI is still the subject of debate (Fazzi et al., 2007; Good, 2009; Ortibus et al., 2011). CVI includes deficits in primary visual functions, and also, all cognitive and motor aspects of vision. As the impairments of visual perception and cognition due to CVI and their impact have yet to be fully understood, they are currently being studied to provide understanding of how they may contribute to the complex puzzle of disorders of learning, developmental coordination, and social-interaction (Chokron and Dutton, 2016; Fazzi et al., 2019; Lehman, 2012; Lueck and Dutton, 2015; Lueck et al., 2019).

It is therefore important to highlight the known semiology of CVI so as to avoid any confusion with other neurodevelopmental conditions with similar features.

1.2. Semiology

Depending on the lesion site, side and size, CVI can include primary visual deficits as well as more complex deficits of visual cognition, such as impairment in visual analysis, exploration, visual attention and/or visual memory, as well as deficits of visual search and visual guidance of limb and body movement. In children with CVI, these primary and higher-level visual deficits can be either isolated or occur in combination. This semiology of CVI is presented below.

1.2.1. Primary visual deficits including visual field defects

Visual pathway dysfunctions can impair the primary visual functions of visual acuity, contrast sensitivity and color vision (Zihl and Dutton, 2015), and can cause a range of homonymous visual field defects for which the location and extent depend directly on the topography of the post-chiasmatic brain lesion present (Figure 1) (Brodsky, 2010).

/Insert Figure 1/

1.2.2. Deficits in visual recognition

While middle temporal lobe dysfunction can limit perception of fast movement (Zihl and Heywood, 2016), lesions affecting the ventral stream pathways between the occipital lobes and the image libraries in the temporal lobes impair recognition of objects, people and route finding (i.e. visual agnosia) (Dutton and Jacobson, 2001) ; Pawlenko et al., 2014). Affected children can present with difficulties interpreting what they see, yet still be able to name objects by using their other sensory modalities such as touch and hearing. Importantly, temporal cortex impairments can also affect recognition of complex stimuli such as faces and/or facial expressions, potentially prejudicing the social cognition domain (Dutton and Bax, 2010; Zihl and Dutton, 2015), and can lead to detectable homonymous contralateral upper visual field deficits (Figure 1).

1.2.3. Deficits in spatial cognition

Lesions and dysfunction affecting the dorsal stream pathways, occurring between the occipital lobes and the posterior parietal lobes, lead to impairments of visual exploration, visual attention, spatial organization, representation and visuo-motor coordination (Macintyre-Beon et al., 2010; Philip and Dutton, 2014). Posterior parietal lobe dysfunction can disrupt non-conscious moment-to-moment mapping of the environment, limiting both visual search and visual guidance of movement (Goodale, 2011). Hemispatial neglect in children and adults results from unilateral posterior parietal damage (Laurent-Vannier et al., 2006; Laurent-Vannier et al., 2003), and it is related to difficulties in reacting to, or interacting with stimuli presented on the side of the body contralateral to the lesion (Bartolomeo and Chokron, 2002), with the left attentional visual field usually being affected to greater degree (Ting et al., 2011). The affected person can behave as if half of her surrounding space, in relation to the body, does not exist (Laurent-Vannier et al., 2006; Laurent-Vannier et al., 2003), but may manifest a compensatory body turn to enable the otherwise unattended foot to be seen while walking or to render all the food to be attended to while sitting at table. Auditory and tactile attention tend also to be deficient, with diminished capacities of searching, scanning, hearing and motor function in relation to the affected side of the body (Philip and Dutton, 2014),

Spatial organization and representation in children with CVI can be evaluated through several tests including drawing and copying geometric shapes, arranging cubes, doing puzzles, and spatial reasoning (Cavezian et al., 2010; Chokron et al., 2004; Dulin et al., 2008).

Impaired peripheral and/or central vision affect visuo-motor coordination, while the impaired visual guidance of movement, or optic ataxia that accompanies simultanagnosia in those with bilateral posterior parietal pathology results from impaired parallel processing of the elements of the visual scene (Chokron and Dutton, 2016).

The features of CVI may be compounded by associated disorders of eye movement control and their negative visual consequences (Boot et al., 2012; Lueck and Dutton, 2015). Because of the complexity and extent of the visual cortex, the CVIs are highly heterogeneous and do not relate to a specific diagnostic group. Instead, every affected child has their own unique and complex clinical profile of visual disabilities.

1.3. Epidemiology

Recent developments in the treatment of sight threatening paediatric eye conditions including retinopathy of prematurity, cataract and glaucoma, have decreased the prevalence of ocular visual impairment, while in industrialized nations, advances in neonatal medicine have increased survival rates of both prematurely born infants, and those who develop neurologic lesions during or shortly after birth. This has led to neurologic disorders becoming the commonest cause of impairment of vision in children (Boot et al., 2010; Dutton and Bax, 2010; Kong et al., 2012). Yet the International Classification of Disease (ICD 10) does not include CVI as a cause of blindness and visual impairment in children. This can result in governments and administrative bodies and institutions failing to recognise this multifaceted condition. Three to 4% of children aged between 4 and 6 years may have an identifiable visual and/or attentional deficit as a sequel to a possible neurologic lesion or dysfunction sustained around the time of birth (Cavezian et al., 2010; Kocur and Resnikoff, 2002; Pascolini and Mariotti, 2012; Rahi et al., 2003; Vitale et al., 2006). While unrecognised visual dysfunctions due to CVI can be inappropriately categorised as Learning Difficulties (Chokron and Démonet, 2010; Lueck and Dutton, 2015; Lueck et al., 2019). Optic nerve hypoplasia and optic atrophy have in the past been considered isolated diagnoses, but are now known to be associated with cerebral visual pathway and cortex lesions in some cases (Zeki et al., 1992). Such brain lesions can also cause focal retinal ganglion cell layer atrophy (Lennartsson et al., 2014) due to retrograde transsynaptic degeneration from the brain pathology back into the retina (Jacobson et al., 1997). Yet singular diagnoses of isolated optic atrophy, or optic nerve hypoplasia may be given, leading to the underlying cerebral lesions going unrecognized (Zeki et al., 1992). Although CVI is now considered integral with cerebral palsy (Bax et al., 2007; Rosenbaum et al., 2007), the fact that visual impairment is often a hidden disability can also contribute to missed diagnosis. Moreover, affected children and caregivers may be unaware of the impairments in visual functioning or may present with difficulties in dealing with related cognitive and learning difficulties (van den Broek et al., 2017). In some cases, CVIs can be identified for the first time only years after onset (Neves et al., 2016; Werth and Schadler, 2006). Similarly, a behavioral diagnosis can also be given (Chokron and Dutton, 2016), without the underlying visual cause being identified (Boot et al., 2010).

1.4. Cognitive and behavioral consequences due to CVI

Early acquired or congenital visual impairments affect many cognitive processes and may induce severe and detrimental consequences with respect to all aspects of early development (Dale and Sonksen, 2002; Do et al., 2017; Iverson et al., 1994; Sonksen and Dale, 2002), including impaired self-initiated mobility and optimal posture, as well as delayed motor development (Chokron and Dutton, 2016; Fazzi et al., 2007).

Learning difficulties due to failure to cater for CVI can thus resemble a primary intellectual deficit (Boot et al., 2012; Flanagan et al., 2003). Several signs can closely resemble a range of neurodevelopmental disorders such as ASD or Attention Deficit Hyperactivity Disorder (ADHD) (Fazzi et al., 2019; Jure et al., 2016; Kiani et al., 2019; Koller, 2012; Mukaddes et al., 2007).

Children without sight or with severe visual impairments (Bigelow, 1990) and those who lack early stimulation (Rutter et al., 1999) are at risk of developmental issues (Flanagan et al., 2003), some of which overlap with ASD (Fazzi et al., 2019; Hobson et al., 1999; Kiani et al., 2019). The greater the visual deprivation the greater the developmental impact, but for those who gain basic vision before 16 months of age, typical intellectual development is more likely (Dale and Sonksen, 2002; Sonksen and Dale, 2002). Thus, rapid developmental progress is possible when appropriate measures are taken and these need to be provided at the earliest opportunity (Pawlenko et al., 2014).

CVI can impair several areas of cognitive development and impact learning, especially if undetected and not treated appropriately (Fazzi et al., 2015; Flanagan et al., 2003). For example, object permanence, a cognitive developmental milestone acquired through vision (Huang and Spelke, 2015), is also crucial to develop early notions of causality as well as the motivation to approach and to interact with people and objects (Fazzi et al., 2015). Visual impairment, however, may lead to reduced mental imagery and visual conceptualization, implicating difficulties in understanding and interacting with the social environment (Dulin et al., 2008), unless other sensory modalities compensate for the lack of visual information (Brambring and Asbrock, 2010).

1.4.1. Motor skills

Vision is naturally involved in interaction with the environment, and is necessary to recognize objects and to bring about visually guided reach and grasp, for example. In the course of child development, vision precedes action, and during his/her first months the infant experiences a visual relationship with the outside world before being able to voluntarily act within it (Itier and Batty, 2009). Indeed, as Lipsitt and Spiker (1967) point out, the newborn infant is both nonverbal and motorically immature with a limited behavioral repertoire, although his/her visual perception is already at work (Chokron, 2012; de Hevia et al., 2014). As Braddick and Atkinson (2013) explain, infants aged between 5 and 18 months show an almost compulsive response to reach out, grasp, and manipulate any small object placed in front of them. As these authors discuss, this is a striking motor behaviour, but it is also a visual behaviour reliant upon intact functioning of the dorsal and ventral streams of the visual system (Milner and Goodale, 2008). It is thus not surprising that motor skills can be adversely impacted by visual impairments. In particular, impairments of dorsal stream function are known to implicate a wide range of motor skills (e.g. hand-body movements, visuo-motor coordination, and grasping) (Chokron and Dutton, 2016). In addition to these difficulties, individuals with CVI can also present motor particularities such as stereotyped behaviors and mannerisms (Fazzi et al., 1999; Fazzi et al., 2019; Molloy and Rowe, 2011). As a matter of fact, many studies have repeatedly brought to light evidence for specific visual as well as motor deficits in children with CVI, who have survived preterm birth and/or neonatal asphyxia (Atkinson and Braddick, 2007; Azria et al., 2016; Birtles et al., 2012; Braddick and Atkinson, 2013; Carey and Geldman, 1991; Dutton and Bax, 2010; Dutton and Jacobson, 2001; Johansson et al., 2014; Khetpal and Donahue, 2007; Lueck and Dutton, 2015; Maalouf et al., 1999; Marlow et al., 2007; Soul and Matsuba, 2010) .

1.4.2. Attention

As visual perception is a major contributor to attention (Bartolomeo and Chokron, 2002; Chokron et al., 2004), many children with CVI manifest attentional deficits. Deficits in selective attention, orientation of attention in space, and joint attention have been observed (Chokron and Dutton, 2016; Fazzi et al., 2015).

Evaluating Theory of Mind (ToM) in children with visual deficits can be challenging because many tests used to assess ToM rely on visual capacities (e.g. false belief tasks). However, Green et al. (2004) were able to assess ToM understanding in 18 sighted children as well as in 18 children with congenital profound visual impairment, who had had no access to visual information throughout their development. Children from the two groups were matched individually by chronological age, verbal IQ and verbal mental age. Six of the children with congenital profound visual impairment failed one or more of the false belief tasks, whereas all the sighted children passed all of the tasks. Verbal IQ and verbal mental age were found to differ in children with good and poor performances on the false belief tasks, with the findings indicating that poor ToM performance could be associated with visual disorders as well as with lower verbal IQ.

Indeed, because visual impairments can affect the development of ToM, purely due to visual and perceptual deficits, some children with CVI may be diagnosed with ToM deficits, despite these difficulties in interpreting others’ intentions and reactions merely reflecting the deleterious effect of CVI on the understanding of the social context owing to not being able to see facial expressions, for example. As a matter of fact, in a recent study, the introduction and use of reliable tools has identified similar impairments in ToM capacities in blind children as compared to control peers (Bartoli et al., 2019), thus suggesting that the visual nature of the tests or the stimuli should systematically be considered and taken into account.

1.4.3. Language

From birth, the relationship between the newborn and its environment is initially predominantly visual, becoming verbal later. Some studies have shown that newborns can imitate the shape of the mouth at birth of somebody saying ‘I’ or ‘O’, thus showing audio-visual integration capacities (Coulon et al., 2013) with intact mirror cell capacity (Khalil et al., 2018). Social interactions depend not only upon sharing verbal information, but also on the implicit ability to detect, analyze and potentially mimic visual cues from the eyes, facial expressions, body language, posture and movements (Farroni et al., 2002; Kovacs et al., 2010; Zalla et al., 2013). Language production and comprehension difficulties are often described in children with ID and ASD, but the co-occurrence of visual impairment, intellectual disability and ASD has a possible compounding effect (see Mosca et al., 2015). Nevertheless, language can be delayed in children with CVI (Erickson and Hatton, 2007; Fazzi et al., 2015; Tadic et al., 2010), as acknowledged by parents, who often describe several difficulties in interacting with their child, whose ability to respond to stimuli is altered (McConachie, 1990; McConachie and Moore, 1994; Sonksen, 1985, 1989). How can children with impaired vision make sense of and learn from vocabulary referring to what they do not see? Thus, communication can be profoundly affected, as children with CVI may manifest delay in language acquisition and these language deficiencies can in turn lead to reciprocal parental language compensations (van den Broek et al., 2017). Accordingly, it has been shown that the language styles of parents with children with CVI, aged between 2 to 5 years differ from those of parents of sighted children, probably as adaptive responses to atypical child behaviors (Erin, 1990; Kekelis and Andersen, 1984). Although it is acknowledged that children with CVI and blind children may present language delay (see Kiani et al., 2019; Pérez-Pereira and Conti-Ramsden, 1999; Pérez-Pereira and Conti-Ramsden, 2005; Tadic et al., 2010), important compensations can be observed when caregivers pay particular attention to the communication domain (Webster and Roe, 1998). Indeed, if caregivers are aware of the child's visual deficit, their language ideally needs to become matched to the world the child can comprehend (e.g. radio language: language not referred to any visual input).

1.4.4. Social interaction

CVI can disrupt social interaction as it can interfere with many processes needed for communication (Dutton and Bax, 2010; Pawlenko et al., 2014). Accordingly, children and adolescents with CVI present a higher risk of developing socio-emotional difficulties (Huurre and Aro, 1998; Ortibus et al., 2011; Papadopoulos et al., 2011). The mechanisms of the development of social communication difficulties and autistic traits in visually impaired children have yet to be fully understood, but current evidence and clinical experience suggest that some signs of these conditions might be common (Sonksen and Dale, 2002).

Visually impaired children tend to acquire the use of possessive pronouns like 'my' or 'mine' later (at 36.5 months on average) than typically sighted children (at 19 months on average) (Sonksen and Dale, 2002). The same applies to the personal pronoun acquisition (i.e. "I") for which first use occurs later and/or is altered (Erin, 1990), while typically developing children progressively learn this distinction through visual contact and by reciprocated sharing of visually perceived facial information. Interestingly, impaired vision limits the distinction between the ‘self’ and the ‘other’, potentially affecting early development of inter-subjectivity, a foundational aspect of interpersonal relationships and language related uses (van den Broek et al., 2017).

Interacting with a child who does not access, track, nor recognize visual stimuli, or does not return smiles represents an important challenge to shaping early interactions. Parents of children with severe visual impairment might have limited resources to stimulate and interpret their child’s interest (Jan et al., 1987; Rogers and Puchalski, 1988; van den Broek et al., 2017), and to motivate the development of movement, especially until the child learns how to compensate via understanding of auditory information and tactile exploration (Dale and Sonksen, 2002; Sonksen et al., 1984).

It is often noticed by families and school professionals that some children with CVI and severe ID can be fearful of their surroundings. This highlights that additional efforts to help affected children to explore the environment are necessary.

Several potential reasons why CVI could impair social interaction are shown in Table 1.

Disorders of vision can lead to face and facial expression recognition deficits and to abnormal eye contact (e.g. gaze avoidance, poor eye contact). If the child’s caregivers are unaware of these difficulties, and misinterpret the lack of response as a sign of lack of motivation, severe social interaction disability can result. For some children with CVI, early onset disorders of recognition and analysis can be severe and lead to isolation and withdrawal. Conversely, clinical experience suggests that informed families, who learn to adapt their communication to overcome the visual disturbances can help in reducing such behaviors.

Blindness or severe visual impairment can preclude children from learning many aspects of social interaction, including language and emotion conveyed by crucial visual information such as facial expressions and gestures (Buimer et al., 2018).

1.5. CVI and autistic traits

Congenital or early onset CVI and blindness, regardless of the etiology are often associated with autistic traits, with a reported prevalence between 25% (Garcia-Filion and Borchert, 2013) and 50% (Jure et al., 2016; Kiani et al., 2019). Studies indicate that absent or reduced visual perception from birth or early childhood represents a risk factor for the development of autistic-like conditions (Jure et al., 2016). Children who suffer from a severe socio-visual deprivation, manifest severe cognitive deficits, akin to those of children with CVI, suggesting that lack of vision and social deprivation may have similar effects and can lead to an autistic-like profile (Rutter et al., 1999).

In line with this concept, recent studies provide evidence that CVI has such an important impact on social skills that many affected children can additionally be diagnosed as having a pervasive developmental disorder (Fazzi et al., 2019), such as ASD (Pawlenko et al., 2014). As a consequence, it appears necessary to systematically screen for signs of CVI in all at risk infants (e.g. born preterm or in an adverse neurological context) (Macintyre-Beon et al., 2013) and children, as early as possible, to provide proper care and to prevent and reduce severe social interaction and/or cognitive and/or behavioral disorders (Cavezian et al., 2010; Dammeyer, 2012; Urqueta Alfaro et al., 2018). There is also an urgent need to develop a schedule to identify social communication difficulties, including autistic traits, in children with visual impairments regardless of their origin (ocular or cerebral) (Absoud et al., 2011). However, it should be noted that available observational tools partly rely on visual behaviors (e.g. eye contact, joint attention, pointing, language, action, face and emotion recognition) and therefore may be a potential source of bias between visual deficits and social interaction deficits (Absoud et al., 2011). In addition, as we describe below, the risk of confusion between CVI and ASD is increased by the presence of visual deficits in children with ASD.

1.6. Autism and visual functioning impairments

ASD is a neurodevelopmental disorder characterized by disturbances in the social domain, with difficulties in communication and social interaction, and by stereotyped behaviors and restricted interests (APA, 2013). Within this second group of characteristics, hyper- and hypo-reactivity to sensory input are now considered diagnostic features. In the visual modality, visual fascination with lights or movements are often reported in children with ASD, but other atypical visual behaviors are also often observed, such as lateral glances or reduced looking at faces (Coulter, 2009; Hellendoorn et al., 2014; Mottron et al., 2007). The fact that children with profound disabilities and little or no visual behavior, start to look around for the first time when surrounded by the uniform environment of a tent, which excludes all clutter from competing for visual attention, adds weight to the concept that simultanagnostic vision is responsible (Little and Dutton, 2015), meriting further investigation. Thus, while autistic traits are often found in individuals with CVI, visual disturbances are also found in individuals with ASD, suggesting that the relationships between CVI and ASD in their various forms need to be further investigated.

In accordance with both hyper- and hypo-sensory symptoms and signs being described along with several signs of atypical vision (Simmons et al., 2009), ASD is characterized by an important heterogeneity of expression which makes it particularly challenging to investigate the visual profiles in those affected. ASD is well recognized as comprising a range of multi-dimensional disorders with no single cause (Happé and Frith, 2006; Happe et al., 2006), and often involving numerous concurrent conditions including ID, language disorder and ADHD (Gillberg and Fernell, 2014). Although the impact of visual deficits on social and cognitive development is well recognized in children with CVI this is currently far from being the case in children with ASD. However, the growing interest in the atypical expression of sensory inputs in ASD suggests that basic visual functions might play a crucial role in other impaired domains (Macintyre-Beon et al., 2010; Pellicano et al., 2005; Spencer et al., 2000; Thye et al., 2017).

1.7. From peripheral disturbances to visual perception in ASD

Ophthalmic deficits are commonly identified in subjects with ASD, especially in those with comorbid ID (Little, 2018). Ophthalmic disturbances include severe refractive error, strabismus, and amblyopia (Ikeda et al., 2013; Khanna et al., 2020; Little, 2018; Mouridsen et al., 2017), but as impaired central visual function is also known to contribute to the atypical development of autism (Bakroon and Lakshminarayanan, 2016; Brown et al., 2017; Freeman, 2010; Simmons et al., 2009) atypical vision from the earliest stages of visual processing, to higher-level impairments, should be considered as contributory factors.

Optical error at birth is common and the development of normal optics (emmetropisation) and the ability to focus between distance and near progressively takes place. This dual process is probably in part contingent upon typical development of the visual brain to create clear mental imagery, so that injury to the visual brain can impair these processes (Lueck and Dutton, 2015; McClelland et al., 2006; Saunders et al., 2010). Refractive error and impaired accommodation can be found in children with autism (Anketell et al., 2018; Khanna et al., 2020; Little, 2018), but with spectacle correction, children with autism tend to have normal visual acuities (Anketell et al., 2015; Koh et al., 2010), so that reduced visual acuities in a child with autistic signs should lead to suspicion of a low vision as a cause (Bakroon and Lakshminarayanan, 2016; Simmons et al., 2009). Basic visual disorders cannot therefore be imputed directly from autistic behaviors, and complete and specific visual examination is required.

In addition, some individuals with ASD may demonstrate some visual functional skills superior to those with typical development, such as shorter response times in target detection from amongst distracting stimuli (Mottron et al., 2006; Plaisted and Davis, 2005; Shirama et al., 2017) or faster saccades (Kovarski et al., 2019). Some authors have also found superior performance in ASD individuals compared to typical participants in their analyses of overlapping figures and in constructional tasks, such as the Kohs block design test (Caron et al., 2006; Shah and Frith, 1993). These various tasks require local feature analysis rather than a global strategy, indicating that some individuals with autism may show a bias toward local visual processing, but to the detriment of overall scene processing (Mottron and Burack, 2001; Mottron et al., 2006). However, due to the large heterogeneity in the autistic population, it remains impossible to compare the results of these different studies. In addition, in these studies, it is difficult to disentangle better performance, from local processing bias or lower performance when processing global stimuli.

According to a recent neuroanatomical study among two young adults with CVI due to periventricular white matter pathology from birth (Bauer et al., 2014), limited visual parallel processing observed when global vision is pathologically altered (such as in simultanagnosia) could be explained by paucity of the neuronal tracts between the higher visual centers in the posterior parietal lobes where the scene is mapped and the frontal lobes where selective attention is accorded, (the superior longitudinal fasciculus)(Milner and Goodale, 2008). Thus, there is a need to distinguish between the 'bias toward local visual processing' described in autism, and impaired visual mapping in the posterior parietal lobes causing simultanagnostic vision, from difficulty with global visual processing, because it is clearly important to differentiate these two diagnostic labels. In similar vein, in prediction tasks, visuo-spatial, and social reasoning tasks, individuals with ASD tend to apply bottom-up processing guided by sensory input, whereas typical subjects use top-down processing modulated by the frontal cortex, guided by learned knowledge and prior experience (Chambon et al., 2017; Kovarski et al., 2019; Pellicano and Burr, 2012; Thillay et al., 2016). This atypical visual perception may relate to atypical brain connectivity, especially between the primary visual cortex and extrastriate areas (Bird et al., 2006; Simard et al., 2015; Takesaki et al., 2016) and could explain disturbances in processing both simple stimuli and complex information such as faces.

A variety of deficits in facial processing can be identified in autistic subjects. These include poor memory for faces (Bakroon and Lakshminarayanan, 2016; Simmons et al., 2009), a decrease in the facial inversion effect (Hobson et al., 1988), reduced visual scanning, especially in the eye region of the face (Klin et al., 2002) and difficulties in recognition conveyed emotion (Ashwin et al., 2007; Lozier et al., 2014), as well as limited ability to extract important cues from faces such as the emotional content (Adolphs et al., 2001). These anomalies in face processing appear early during the development of autistic children and represent a crucial sign of ASD (Dawson et al., 2005). However, although faces are social stimuli, they are also visual stimuli, and so the relation between visual and social interaction deficits need to be further investigated to characterize the potential differentiation and links between CVI and autism.

Individuals with autism tend to have difficulties in processing social visual stimuli compared to non-social visual stimuli (Zalla et al., 2016). However, recent research has led to controversial results, as both social and non-social visual detection is impaired in individuals with ASD (Guillon et al., 2014; Johnson et al., 2016; Kovarski et al., 2019), suggesting a broad disruption of visual functioning, rather than a specific social deficit (Robertson and Baron-Cohen, 2017; Thye et al., 2017). Accordingly, higher oculomotor functions and visual attention can also be atypical (Chokron et al., 2014; Johnson et al., 2016; Kovarski et al., 2019). In line with a recent meta-analysis, individuals with ASD display a range of higher oculomotor disorders including anomalies of saccadic eye movement, deficits in saccadic inhibition, and difficulty tracking moving visual targets (Johnson et al., 2016). Eye movements have also been found to be atypical in children and adults with ASD, being either slower and/or less precise (Johnson et al., 2016) or also faster (Kovarski et al., 2019). Their performance is also subject to greater variation, suggesting anomalies in lower eye movement control (Zalla et al., 2018).

Importantly, undetected ophthalmic or oculomotor deficits may have led to biased results in a range of studies that have employed observation of eye movements to assess vision in subjects with autism. Moreover, tests of social cognition, ToM, facial recognition and language are standard practice, yet the nature of the subject's visual functioning integrity is crucial to ascertain in this context. For example, deficits in visual attention (e.g. joint attention) in children with ASD could partly explain the social interaction deficits (Sonksen and Dale, 2002; Swettenham et al., 1998). Faced with the frequency of visual dysfunction in subjects with ASD, it is also possible that underlying oculomotor deficits (Shishido et al., 2019) or CVI could significantly contribute to the clinical picture of ASD and to associated deficits (i.e. ID). In particular, in some children with ASD, oculomotor deficit refers to apraxia of gaze, which is the absence or lack of controlled, voluntary and targeted eye movements. A systematic neurovisual examination in these children would likely elicit the extent to which children with ASDs are able to voluntarily direct their gaze toward different targets. In fact, apraxia of gaze that accompanies the simultanagnosia and optic ataxia indicative of a pattern of Balint syndrome in children (Philip et al., 2016) may be the cause of some of the anomalies of gaze hitherto described.

Despite the increasing interest in visual disturbances in autism (see Robertson and Baron-Cohen, 2017; Simmons et al., 2009), it is important to note that the lower end of the spectrum of severity is not sufficiently represented in vision research studies (Brown et al., 2017). Such bias is partly due to difficulties in testing those presenting mild to severe ID, implicating that visual disturbances might be underestimated in a subgroup of low functioning persons with ASD as well as in persons with only ID (Khanna et al., 2020).

2. Visual perception and ID

Similarly to ASD, visual impairments are also frequently associated with ID (Boot et al., 2012; Warburg, 1994, 2001), and can have complex and severe detrimental effects on most aspects of early childhood development, if appropriate early intervention is not implemented (Aslan et al., 2013; Couser et al., 2017). The resulting developmental disorders in children with CVI can take the form of language delay, and/or of attentional deficit, and/or of executive functional deficits and/or of learning disabilities and/or of deficits in social interaction (Lueck et al., 2019), and are very similar to those observed in children with autism (Charman et al., 1997; Wing and Gould, 1979), as well to those seen in visually deprived children (Rutter et al., 1999).

Dale and Sonksen (2002) have described 102 children with visual impairment occurring in the first 16 months of life due to various causes (ophthalmological and cortical). Eleven children among this group displayed overall delay with severe learning disability and autistic signs. Interestingly, of these eleven children, ten presented with severe visual impairments.

Early acquisition of basic visual function appeared to have a protective effect on intellectual development (Dale and Sonksen, 2002). Etiology and lesion locations differed within the set of visually impaired children, indicating that the severity of sensory deprivation rather than the nature of the lesion might influence development. Conversely, Stiers and Vandenbussche (2004) found a dissociation between nonverbal intelligence performance and visual perceptual abilities. However, their study was performed among 57 children who were neonatally at risk for CVI due to prematurity and/or birth asphyxia, but none of them exhibited visual field defects. However, as mentioned above, the severity of the visual deprivation seemed to determine the relative importance of the developmental delay.

A high incidence of visual perception deficits has been identified in both patients with signs of autism and in those with ID, as also acknowledged by the DSM-5 (APA, 2013). Several studies, mirroring the research in autism, have looked into the frequency of visual deficits in a population of subjects with ID, and found an increased prevalence of visual impairment (Boot et al., 2012; Carvill, 2001), including disturbances in visual orienting behavior, spatial cognition and oculomotor functioning (Boot et al., 2012; Giuliani and Schenk, 2015).

Interestingly, Battaglia (2011) underlines that the relationship between sensory deficits and ID is frequent, and that these are also observed in multiple conditions including ASD, the fragile X syndrome, tuberous sclerosis complex associated with ASD, Rett syndrome, 22q13.3 deletion syndrome, familial dysautonomia, Williams syndrome, Down syndrome and Prader-Willi syndrome, but also in other contexts such as a history of neonatal distress, or perinatal brain lesions often accompanying premature birth (Couser et al., 2017; Geldof et al., 2015; Krinsky-McHale et al., 2014). The role of sensory disorders in the evaluation of other functions is often underestimated, meaning that in some cases difficulties in completing some specific tests (e.g. IQ tests) might be due to sensory deficits rather than to a specific difficulty (Carvill, 2001). Crucially, clinicians who provide psychometric testing should receive training in seeking and detecting signs of sensory deficits (Yeates, 1991).

The prevalence of visual deficit varies depending on the degree of severity sought and the methods employed (questionnaire or visual evaluation) (Carvill and Marston, 2002; Cavezian et al., 2010; Chokron, 2015). Most studies have found that profound visual deficits are much more prevalent in ID subjects (from 3% to 28% for total or profound visual impairment) (Carvill, 2001; Carvill and Marston, 2002) than in typically developing subjects.

3. Overlap between CVI and ASD and ID

In addition to common visual disturbances, CVI and ASD share anomalies in other domains that might represent, at least in part, consequences of abnormal visual functioning.

Here, we show that impairments of visual functioning might be related to disturbances in the socio-emotional domain, including language delay, and atypical use of language such as pronoun reversal

(Overweg et al., 2018) or ToM delay. In addition, stereotyped behaviors and mannerisms are also considered as frequent consequences of reduced vision, suggesting that impairments of vision hamper the overall development of the child.

Importantly, ID is a frequent comorbidity of ASD. It is still difficult to understand whether visual disturbances are related to ASD, or rather to ID (being itself an associated condition). It is also possible that at least in some cases, disturbances in visual functioning might be responsible for autistic-like profiles, sometimes leading to an official diagnosis (Kiani et al., 2019).

Conclusion and future directions

Taken together, increasing evidence underlines the crucial role of vision in cognitive and social development. As a matter of fact, ASD and ID are found to co-occur with an array of visual deficits (Kiani et al., 2019). This frequent association raises the question of the extent to which some autistic signs could in some cases actually be the consequence of visual disorders.

Visual processing and social cognition in ASD are either inappropriately considered too difficult to assess, or, of greater concern, less relevant to those with additional ID. Nevertheless, it is crucial to evaluate visual functioning (e.g. eye disorders and CVI) in children with neurodevelopmental disorders for whom the visual disability may be, at least partly, a remediable underlying contributory cause.

A more thorough understanding of the relationships between vision, cognitive and social development is thus urgently warranted, to recognize need for, and to provide salient interventions, and to prevent cognitive, communication and social interaction difficulties in patients with CVI. Moreover, further research in this area could lead to a better understanding on how to disentangle neurodevelopmental disorders proper, from developmental disorders due to impairments of visual functioning.

The present review aims to help shed light on the complex interplay between CVI, ASD and ID, and on the degree to which each condition might contribute to the other. The differential diagnosis between visual and developmental deficits (e.g. ASD and/or ID) is a crucial issue (Chokron and Dutton, 2016; Dutton et al., 2017; Lueck et al., 2019) and would prevent confusing visual impairment on the one hand, with its detrimental effects upon development on the other. Systematic screening for visual deficits in at-risk children with or without social interaction and/or cognitive deficits is a pressing matter (Graignic-Philippe et al., 2014; Petkovic et al., 2016). Evaluation tools are currently available for systematic testing of these children (Cavezian et al., 2010; Chokron, 2015; Chokron and Zalla, 2017). There is also an urgent need for clinical tools that can help with the identification of social communication difficulties and autistic traits in young children with visual impairment (Absoud et al., 2011), by ensuring that all test materials used are calibrated to avoid artefactual detection of anomalies, by being rendered visible despite low vision. Optical disorders of the eyes are more common and must be sought in all children with multiple disabilities and those with CVI. When detected, optimal spectacle correction for near and distance is essential to avert preventable learning impairments (Das et al., 2010). Moreover, early screening of infants with significant brain injury is also crucial (Das et al., 2010; Ventura et al., 2017).

From a practical standpoint, seeking out and taking a deeper look into visual deficits in patients with cognitive and social disorders and inversely, studying cognitive and social disorders in children with visual deficit, and the way they are related to each other, would afford a better understanding and more precise description of these differing clinical entities. Additionally, this should reduce social interaction difficulties or even prevent them, through implementation of specific visible stimulation of visual analytic processing, and optimized day to day living conditions. This approach could promote the development of early and suitable family life and therapy with increased effectivity.

From an experimental and clinical point of view, the visual modality is often used to test many skills and functions that are critical in ASD and ID (e.g. ToM, IQ, socio-emotional capacities). In this way, lower level visual functioning should be systematically controlled for before testing the child, in order to avoid any artefactual confusion caused by a visual deficit and its deleterious impact upon psychometric assessments that require typical vision.

Vision is needed to guide movement, to access information and to interact socially, and for the young child, it is also needed to acquire these skills. Children cannot learn from what they do not see. In similar vein, in prediction tasks, visuo-spatial, and social reasoning tasks, the performance of some children with ASD and/or ID can in part be the result of specific limitations of vision. Skilled profiling of vision is needed to ensure that each element that is not seen, can be learned through salient alternative approaches.

Figure 1

Visual field deficits related to damage to the brain (reproduced from Lueck and Dutton (2015), Copyright held by G.N. Dutton).

Acknowledgments

The authors gratefully acknowledge the research support and generosity of the Fondation Orange and Fondation de France as well as the Rothschild Foundations (Geneva, Paris) allowing them to use preliminary research results in preparing this review

References

Absoud, M., Parr, J.R., Salt, A., Dale, N., 2011. Developing a schedule to identify social communication difficulties and autism spectrum disorder in young children with visual impairment. Dev. Med. Child Neurol. 53, 285-288.

Adolphs, R., Sears, L., Piven, J., 2001. Abnormal processing of social information from faces in autism. J. Cogn. Neurosci. 13, 232-240.

Anketell, P.M., Saunders, K.J., Gallagher, S.M., Bailey, C., Little, J.A., 2015. Brief Report: vision in children with Autism Spectrum Disorder: what should clinicians expect? J. Autism Dev. Disord. 45, 3041-3047.

Anketell, P.M., Saunders, K.J., Gallagher, S.M., Bailey, C., Little, J.A., 2018. Accommodative function in individuals with Autism Spectrum Disorder. Optom. Vis. Sci. 95, 193-201.

APA, 2013. Diagnostic and Statistical Manual of Mental Disorders (DSM-5). American Psychiatric Association, Washington, DC.

Ashwin, C., Baron-Cohen, S., Wheelwright, S., O'Riordan, M., Bullmore, E.T., 2007. Differential activation of the amygdala and the 'social brain' during fearful face-processing in Asperger Syndrome. Neuropsychologia 45, 2-14.

Aslan, L., Aslankurt, M., Aksoy, A., Altun, H., 2013. Preventable visual impairment in children with nonprofound intellectual disability. Eur. J. Ophthalmol. 23, 870-875.

Atkinson, J., Braddick, O., 2007. Visual and visuocognitive development in children born very prematurely. Prog. Brain Res. 164, 123-149.

Azria, E., Kayem, G., Langer, B., Marchand-Martin, L., Marret, S., Fresson, J., Pierrat, V., Arnaud, C., Goffinet, F., Kaminski, M., Ancel, P.Y., group, E.s., 2016. Neonatal mortality and long-term outcome of infants born between 27 and 32 weeks of gestational age in breech presentation: The EPIPAGE cohort study. PLoS One 11, e0145768.

Bakroon, A., Lakshminarayanan, V., 2016. Visual function in autism spectrum disorders: a critical review. Clin. Exp. Optom. 99, 297-308.

Bartoli, G., Bulgarelli, D., Molina, P., 2019. Theory of mind development in children with visual impairment: the contribution of the adapted comprehensive test ToM storybooks. J. Autism Dev. Disord.

Bartolomeo, P., Chokron, S., 2002. Orienting of attention in left unilateral neglect. Neurosci. Biobehav. Rev. 26, 217-234.

Battaglia, A., 2011. Sensory impairment in mental retardation: a potential role for NGF. Arch. Ital. Biol. 149, 193-203.

Bauer, C.M., Heidary, G., Koo, B.B., Killiany, R.J., Bex, P., Merabet, L.B., 2014. Abnormal white matter tractography of visual pathways detected by high-angular-resolution diffusion imaging (HARDI) corresponds to visual dysfunction in cortical/cerebral visual impairment. J. AAPOS 18, 398-401.

Bax, M.C., Flodmark, O., Tydeman, C., 2007. Definition and classification of cerebral palsy. From syndrome toward disease. Dev. Med. Child Neurol. Suppl. 109, 39-41.

Bigelow, A., 1990. Relationship between the development of language and thought in young blind children. Journal of Visual Impairment & Blindness 84, 414-419.
Bird, G., Catmur, C., Silani, G., Frith, C., Frith, U., 2006. Attention does not modulate neural responses to social stimuli in autism spectrum disorders. Neuroimage 31, 1614-1624.
Birtles, D.B., Atkinson, J., Anker, S., Braddinck, O., Groppo, M., Cowan, F., Rutherford, M., 2012. Bimanual coordination in young children born preterm: effects of white matter injury. Dev. Med. Child Neurol. 54, 162-163.
Boot, F.H., Pel, J.J., Evenhuis, H.M., van der Steen, J., 2012. Factors related to impaired visual orienting behavior in children with intellectual disabilities. Res. Dev. Disabil. 33, 1670-1676.
Boot, F.H., Pel, J.J., van der Steen, J., Evenhuis, H.M., 2010. Cerebral Visual Impairment: which perceptive visual dysfunctions can be expected in children with brain damage? A systematic review. Res. Dev. Disabil. 31, 1149-1159.
Braddick, O., Atkinson, J., 2013. Visual control of manual actions: brain mechanisms in typical development and developmental disorders. Dev. Med. Child Neurol. 55 Suppl 4, 13-18.
Brambring, M., Asbrock, D., 2010. Validity of false belief tasks in blind children. J. Autism Dev. Disord. 40, 1471-1484.
Brodsky, M.C., 2010. Pediatric Neuro-Ophthalmology. Springer-Verlag New York, New York.
Brown, A.C., Chouinard, P.A., Crewther, S.G., 2017. Vision research literature may not represent the full intellectual range of Autism Spectrum Disorder. Front. Hum. Neurosci. 11, 57.
Buimer, H.P., Bittner, M., Kostelijk, T., van der Geest, T.M., Nemri, A., van Wezel, R.J.A., Zhao, Y., 2018. Conveying facial expressions to blind and visually impaired persons through a wearable vibrotactile device. PLoS One 13, e0194737.
Carey, S., Geldman, R., 1991. Biology and knowledge: structural constraints on development. Erlbaum, Hillsdale, NJ.
Caron, M.J., Mottron, L., Berthiaume, C., Dawson, M., 2006. Cognitive mechanisms, specificity and neural underpinnings of visuospatial peaks in autism. Brain 129, 1789-1802.
Carvill, S., 2001. Sensory impairments, intellectual disability and psychiatry. J. Intellect. Disabil. Res. 45, 467-483.
Carvill, S., Marston, G., 2002. People with intellectual disability, sensory impairments and behaviour disorder: a case series. J. Intellect. Disabil. Res. 46, 264-272.
Cavezian, C., Vilayphonh, M., de Agostini, M., Vasseur, V., Watier, L., Kazandjian, S., Laloum, L., Chokron, S., 2010. Assessment of visuo-attentional abilities in young children with or without visual disorder: toward a systematic screening in the general population. Res. Dev. Disabil. 31, 1102-1108.
Chadha, R.K., Subramanian, A., 2011. The effect of visual impairment on quality of life of children aged 3-16 years. Br. J. Ophthalmol. 95, 642-645.
Chambon, V., Farrer, C., Pacherie, E., Jacquet, P.O., Leboyer, M., Zalla, T., 2017. Reduced sensitivity to social priors during action prediction in adults with autism spectrum disorders. Cognition 160, 17-26.
Charman, T., Swettenham, J., Baron-Cohen, S., Cox, A., Baird, G., Drew, A., 1997. Infants with autism: an investigation of empathy, pretend play, joint attention, and imitation. Dev. Psychol. 33, 781-789.

Chokron, S., 2015. Evaluation of visuo-spatial abilities (EVA): a simple and rapid battery to screen for CVI in young children, in: Lueck, A.H., Dutton, G.N. (Eds.), Impairment of vision due to disorders of the visual brain in childhood: a practical approach. American Foundation for the Blind (AFB) Press, Huntington, WV.
Chokron, S., A., 2012. Comment voient les bébés ? Le Pommier, Paris.
Chokron, S., Colliot, P., Bartolomeo, P., 2004. The role of vision in spatial representation. Cortex 40, 281-290.
Chokron, S., Démonet, J.F., 2010. Approche neuropsychologique des troubles des apprentissages. De Boek, Marseille.
Chokron, S., Dutton, G.N., 2016. Impact of cerebral visual impairments on motor skills: implications for developmental coordination disorders. Front. Psychol. 7, 1471.
Chokron, S., Pieron, M., Zalla, T., 2014. Troubles du spectre de l'autisme et troubles de la fonction visuelle : revue critique, implications théoriques et cliniques. L'Information Psychiatrique 90, 819-826.
Chokron, S., Zalla, T., 2017. Troubles de la fonction visuelle, troubles de l'interaction et développement cognitif. Revue de neuropsychologie, neurosciences cognitives et cliniques 9, 35-44.
Coulon, M., Hemimou, C., Streri, A., 2013. Effects of seeing and hearing vowels on neonatal facial imitation. Infancy 18, 782-796.
Coulter, R.A., 2009. Understanding the visual symptoms of individuals with autism spectrum disorder (ASD). Optometry and Vision Development 40, 164-175.
Couser, N.L., Masood, M.M., Aylsworth, A.S., Stevenson, R.E., 2017. Ocular manifestations in the X-linked intellectual disability syndromes. Ophthalmic Genet. 38, 401-412.
Dale, N., Sonksen, P., 2002. Developmental outcome, including setback, in young children with severe visual impairment. Dev. Med. Child Neurol. 44, 613-622.
Dammeyer, J., 2012. Children with Usher syndrome: mental and behavioral disorders. Behav Brain Funct 8, 16.
Das, M., Spowart, K., Crossley, S., Dutton, G.N., 2010. Evidence that children with special needs all require visual assessment. Arch. Dis. Child. 95, 888-892.
Dawson, G., Webb, S.J., McPartland, J., 2005. Understanding the nature of face processing impairment in Autism: Insights from behavioral and electrophysiological studies. Dev. Neuropsychol. 27, 403-424.
de Hevia, M.D., Izard, V., Coubart, A., Spelke, E.S., Streri, A., 2014. Representations of space, time, and number in neonates. Proc. Natl. Acad. Sci. U. S. A. 111, 4809-4813.
Do, B., Lynch, P., Macris, E.M., Smyth, B., Stavrinakis, S., Quinn, S., Constable, P.A., 2017. Systematic review and meta-analysis of the association of Autism Spectrum Disorder in visually or hearing impaired children. Ophthalmic Physiol. Opt. 37, 212-224.
Dulin, D., Hatwell, Y., Pylyshyn, Z., Chokron, S., 2008. Effects of peripheral and central visual impairment on mental imagery capacity. Neurosci. Biobehav. Rev. 32, 1396-1408.
Dutton, G.N., Bax, M., 2010. Visual impairment in children due to damage to the brain. Wiley-Blackwell.
Dutton, G.N., Chokron, S., Little, S., McDowell, N., 2017. Posterior parietal visual dysfunction: an explanatory review. Vision Development & Rehabilitation 3, 10-22.

Dutton, G.N., Jacobson, L.K., 2001. Cerebral visual impairment in children. Semin Neonatol 6, 477-485.
Ek, U., Fernell, E., Jacobson, L., Gillberg, C., 1998. Relation between blindness due to retinopathy of prematurity and autistic spectrum disorders: a population-based study. Dev. Med. Child Neurol. 40, 297-301.
Erickson, K.A., Hatton, D., 2007. Literacy and visual impairment. Semin. Speech Lang. 28, 58-68.
Erin, J.N., 1990. Language samples from visually impaired four and five-year olds. Journal of Childhool Communication Disorders 13, 181-191.
Farroni, T., Csibra, G., Simion, F., Johnson, M.H., 2002. Eye contact detection in humans from birth. Proc. Natl. Acad. Sci. U. S. A. 99, 9602-9605.
Fazzi, E., Lanners, J., Danova, S., Ferrarri-Ginevra, O., Gheza, C., Luparia, A., Balottin, U., Lanzi, G., 1999. Stereotyped behaviours in blind children. Brain Dev. 21, 522-528.
Fazzi, E., Micheletti, S., Galli, J., Rossi, A., Gitti, F., Molinaro, A., 2019. Autism in children with cerebral and peripheral visual impairment: fact or artifact? Semin. Pediatr. Neurol.
Fazzi, E., Molinaro, A., Hartmann, E., 2015. The potential impact of visual impairment and CVI on child development., in: Lueck, A.H., Dutton, G.N. (Eds.), Vision and the brain: understanding Cerebral Visual Impairment in children. AFB Press, American Foundation for the Blind, New York.
Fazzi, E., Signorini, S.G., Bova, S.M., La Piana, R., Ondei, P., Bertone, C., Misefari, W., Bianchi, P.E., 2007. Spectrum of visual disorders in children with cerebral visual impairment. J. Child Neurol. 22, 294-301.
Flanagan, N.M., Jackson, A.J., Hill, A.E., 2003. Visual impairment in childhood: insights from a community-based survey. Child Care Health Dev. 29, 493-499.
Freeman, R.D., 2010. Psychiatric considerations in cortical visual impairment, in: Dutton, G.N., Bax, M. (Eds.), Visual impairment in children due to damage to the brain. Wiley-Blackwell, London.
Garcia-Filion, P., Borchert, M., 2013. Optic nerve hypoplasia syndrome: a review of the epidemiology and clinical associations. Curr. Treat. Options Neurol. 15, 78-89.
Geldof, C.J., van Wassenaer-Leemhuis, A.G., Dik, M., Kok, J.H., Oosterlaan, J., 2015. A functional approach to cerebral visual impairments in very preterm/very-low-birth-weight children. Pediatr. Res. 78, 190-197.
Gillberg, C., Fernell, E., 2014. Autism plus versus autism pure. J. Autism Dev. Disord. 44, 3274-3276.
Giuliani, F., Schenk, F., 2015. Vision, spatial cognition and intellectual disability. Res. Dev. Disabil. 37, 202-208.
Good, W.V., 2009. Cortical visual impairment: new directions. Optom. Vis. Sci. 86, 663-665.
Goodale, M.A., 2011. Transforming vision into action. Vision Res. 51, 1567-1587.
Graignic-Philippe, R., Dayan, J., Chokron, S., Jacquet, A.Y., Tordjman, S., 2014. Effects of prenatal stress on fetal and child development: a critical literature review. Neurosci. Biobehav. Rev. 43, 137-162.
Green, S., Pring, L., Swettenham, J., 2004. An investigation of first-order false belief understanding of children with congenital profound visual impairment. Br. J. Dev. Psychol. 22, 1-17.

Guillon, Q., Hadjikhani, N., Baduel, S., Roge, B., 2014. Visual social attention in autism spectrum disorder: insights from eye tracking studies. Neurosci. Biobehav. Rev. 42, 279-297.
Happé, F., Frith, U., 2006. The weak coherence account: detail-focused cognitive style in autism spectrum disorders. J. Autism Dev. Disord. 36, 5-25.
Happe, F., Ronald, A., Plomin, R., 2006. Time to give up on a single explanation for autism. Nat. Neurosci. 9, 1218-1220.
Hellendoorn, A., Langstraat, I., Wijnroks, L., Buitelaar, J.K., van Daalen, E., Leseman, P.P., 2014. The relationship between atypical visual processing and social skills in young children with autism. Res. Dev. Disabil. 35, 423-428.
Herschkowitz, N., Kagan, J., Zilles, K., 1997. Neurobiological bases of behavioral development in the first year. Neuropediatrics 28, 296-306.
Hobson, R.P., Lee, A., Brown, R., 1999. Autism and congenital blindness. J. Autism Dev. Disord. 29, 45-56.
Hobson, R.P., Ouston, J., Lee, A., 1988. What's in a face? The case of autism. Br. J. Psychol. 79 (Pt 4), 441-453.
Huang, Y., Spelke, E.S., 2015. Core knowledge and the emergence of symbols: The case of maps. J Cogn Dev 16, 81-96.
Huurre, T.M., Aro, H.M., 1998. Psychosocial development among adolescents with visual impairment. Eur. Child Adolesc. Psychiatry 7, 73-78.
Ikeda, J., Davitt, B.V., Ultmann, M., Maxim, R., Cruz, O.A., 2013. Brief report: incidence of ophthalmologic disorders in children with autism. J. Autism Dev. Disord. 43, 1447-1451.
Itier, R.J., Batty, M., 2009. Neural bases of eye and gaze processing: the core of social cognition. Neurosci. Biobehav. Rev. 33, 843-863.
Iverson, J.M., Capirci, O., Caselli, M.C., 1994. From communication to language in two modalities. Cognitive Development 9, 23-43.
Jacobson, L., Hellstrom, A., Flodmark, O., 1997. Large cups in normal-sized optic discs: a variant of optic nerve hypoplasia in children with periventricular leukomalacia. Arch. Ophthalmol. 115, 1263-1269.
Jan, J.E., Groenveld, M., Sykanda, A.M., Hoyt, C.S., 1987. Behavioural characteristics of children with permanent cortical visual impairment. Dev. Med. Child Neurol. 29, 571-576.
Jeyabalan, N., Clement, J.P., 2016. SYNGAP1: Mind the Gap. Front. Cell. Neurosci. 10, 32.
Johansson, A.M., Domellof, E., Ronnqvist, L., 2014. Long-term influences of a preterm birth on movement organization and side specialization in children at 4-8 years of age. Dev. Psychobiol. 56, 1263-1277.
Johnson, B.P., Lum, J.A., Rinehart, N.J., Fielding, J., 2016. Ocular motor disturbances in autism spectrum disorders: Systematic review and comprehensive meta-analysis. Neurosci. Biobehav. Rev. 69, 260-279.
Johnson, M.H., Dziurawiec, S., Ellis, H., Morton, J., 1991. Newborns' preferential tracking of face-like stimuli and its subsequent decline. Cognition 40, 1-19.
Jure, R., Pogonza, R., Rapin, I., 2016. Autism Spectrum Disorders (ASD) in blind children: very high prevalence, potentially better outlook. J. Autism Dev. Disord. 46, 749-759.
Kekelis, L.S., Andersen, E.S., 1984. Family communication styles and language development. Journal of Visual Impairment & Blindness 78, 54-65.

Khalil, R., Tindle, R., Boraud, T., Moustafa, A.A., Karim, A.A., 2018. Social decision making in autism: On the impact of mirror neurons, motor control, and imitative behaviors. CNS Neurosci. Ther. 24, 669-676.
Khanna, R.K., Kovarski, K., Arsene, S., Siwiaszczyk, M., Pisella, P.J., Bonnet-Brilhault, F., Batty, M., Malvy, J., 2020. Ophthalmological findings in children with autism spectrum disorder. Graefes Arch. Clin. Exp. Ophthalmol.
Khetpal, V., Donahue, S.P., 2007. Cortical visual impairment: etiology, associated findings, and prognosis in a tertiary care setting. J. AAPOS 11, 235-239.
Kiani, R., Bhaumik, S., Tyrer, F., Bankart, J., Miller, H., Cooper, S.A., Brugha, T.S., 2019. The relationship between symptoms of autism spectrum disorder and visual impairment among adults with intellectual disability. Autism Res.
Klin, A., Jones, W., Schultz, R., Volkmar, F., Cohen, D., 2002. Visual fixation patterns during viewing of naturalistic social situations as predictors of social competence in individuals with autism. Arch. Gen. Psychiatry 59, 809-816.
Kocur, I., Resnikoff, S., 2002. Visual impairment and blindness in Europe and their prevention. Br. J. Ophthalmol. 86, 716-722.
Koh, H.C., Milne, E., Dobkins, K., 2010. Contrast sensitivity for motion detection and direction discrimination in adolescents with autism spectrum disorders and their siblings. Neuropsychologia 48, 4046-4056.
Koller, H.P., 2012. Visual processing and learning disorders. Curr. Opin. Ophthalmol. 23, 377-383.
Kong, L., Fry, M., Al-Samarraie, M., Gilbert, C., Steinkuller, P.G., 2012. An update on progress and the changing epidemiology of causes of childhood blindness worldwide. J. AAPOS 16, 501-507.
Kovacs, A.M., Teglas, E., Endress, A.D., 2010. The social sense: susceptibility to others' beliefs in human infants and adults. Science 330, 1830-1834.
Kovarski, K., Siwiaszczyk, M., Malvy, J., Batty, M., Latinus, M., 2019. Faster eye movements in children with autism spectrum disorder. Autism Res. 12, 212-224.
Krinsky-McHale, S.J., Silverman, W., Gordon, J., Devenny, D.A., Oley, N., Abramov, I., 2014. Vision deficits in adults with Down syndrome. J Appl Res Intellect Disabil 27, 247-263.
Laurent-Vannier, A., Chevignard, M., Pradat-Diehl, P., Abada, G., De Agostini, M., 2006. Assessment of unilateral spatial neglect in children using the Teddy Bear Cancellation Test. Dev. Med. Child Neurol. 48, 120-125.
Laurent-Vannier, A., Pradat-Diehl, P., Chevignard, M., Abada, G., De Agostini, M., 2003. Spatial and motor neglect in children. Neurology 60, 202-207.
Lehman, S.S., 2012. Cortical visual impairment in children: identification, evaluation and diagnosis. Curr. Opin. Ophthalmol. 23, 384-387.
Lennartsson, F., Nilsson, M., Flodmark, O., Jacobson, L., 2014. Damage to the immature optic radiation causes severe reduction of the retinal nerve fiber layer, resulting in predictable visual field defects. Invest. Ophthalmol. Vis. Sci. 55, 8278-8288.
Lipsitt, L.P., Spiker, C.C., 1967. Advances in child development and behavior. Accademic Press, London.

Little, J.A., 2018. Vision in children with autism spectrum disorder: a critical review. Clin. Exp. Optom.
Little, S., Dutton, G.N., 2015. Some children with multiple disabilities and cerebral visual impairment can engage when enclosed by a 'tent': Is this due to Balint syndrome? British Journal of Visual Impairment 33, 66-73.
Lozier, L.M., Vanmeter, J.W., Marsh, A.A., 2014. Impairments in facial affect recognition associated with autism spectrum disorders: a meta-analysis. Dev. Psychopathol. 26, 933-945.
Lueck, A.H., Dutton, G.N., 2015. Impairment of vision due to disorders of the visual brain in childhood: A practical approach., in: Lueck, A.H., Dutton, G.N. (Eds.), Vision and the Brain: Understanding Cerebral Visual Impairment in Children. AFB Press, American Foundation for the Blind, New York.
Lueck, A.H., Dutton, G.N., Chokron, S., 2019. Profiling children with cerebral visual impairment using multiple methods of assessment to aid in differential diagnosis. Semin. Pediatr. Neurol.
Maalouf, E.F., Duggan, P.J., Rutherford, M.A., Counsell, S.J., Fletcher, A.M., Battin, M., Cowan, F., Edwards, A.D., 1999. Magnetic resonance imaging of the brain in a cohort of extremely preterm infants. J. Pediatr. 135, 351-357.
Macintyre-Beon, C., Ibrahim, H., Hay, I., Cockburn, D., Calvert, J., Dutton, G.N., Bowman, R., 2010. Dorsal stream dysfunction in children. A review and an approach to diagnosis and management. Curr. Pediatr. Rev. 6, 166-182.
Macintyre-Beon, C., Young, D., Dutton, G.N., Mitchell, K., Simpson, J., Loffler, G., Bowman, R., Hamilton, R., 2013. Cerebral visual dysfunction in prematurely born children attending mainstream school. Doc. Ophthalmol. 127, 89-102.
Marlow, N., Hennessy, E.M., Bracewell, M.A., Wolke, D., 2007. Motor and executive function at 6 years of age after extremely preterm birth. Pediatrics 120, 793-804.
McClelland, J.F., Parkes, J., Hill, N., Jackson, A.J., Saunders, K.J., 2006. Accommodative dysfunction in children with cerebral palsy: a population-based study. Invest. Ophthalmol. Vis. Sci. 47, 1824-1830.
McConachie, H., 1990. Early language development and severe visual impairment. Child Care Health Dev. 16, 55-61.
McConachie, H.R., Moore, V., 1994. Early expressive language of severely visually impaired children. Dev. Med. Child Neurol. 36, 230-240.
Milner, A.D., Goodale, M.A., 2008. Two visual systems re-viewed. Neuropsychologia 46, 774-785.
Molloy, A., Rowe, F.J., 2011. Manneristic behaviors of visually impaired children. Strabismus 19, 77-84.
Mosca, R., Kritzinger, A., van der Linde, J., 2015. Language and communication development in preschool children with visual impairment: A systematic review. S. Afr. J. Commun. Disord. 62, e1-e10.
Mottron, L., Burack, J., 2001. Enhanced perceptual functioning in the development of autism, in: Burack, J., Charman, T., Yirmiya, N., Zelazo, R.R. (Eds.), The development of autism: Perspectives from theory and research. Erlbaum, Mahwah, NJ, pp. 131-148.

Mottron, L., Dawson, M., Soulieres, I., Hubert, B., Burack, J., 2006. Enhanced perceptual functioning in autism: an update, and eight principles of autistic perception. J. Autism Dev. Disord. 36, 27-43.
Mottron, L., Mineau, S., Martel, G., Bernier, C.S., Berthiaume, C., Dawson, M., Lemay, M., Palardy, S., Charman, T., Faubert, J., 2007. Lateral glances toward moving stimuli among young children with autism: Early regulation of locally oriented perception? Dev. Psychopathol. 19, 23-36.
Mouridsen, S.E., Rich, B., Isager, T., 2017. Eye disorders among adult people diagnosed with infantile autism in childhood: a longitudinal case control study. Ophthalmic Epidemiol. 24, 332-335.
Mukaddes, N.M., Kilincaslan, A., Kucukyazici, G., Sevketoglu, T., Tuncer, S., 2007. Autism in visually impaired individuals. Psychiatry Clin. Neurosci. 61, 39-44.
Neves, A., Carvalheira, F., Campos, J., Alfaiate, P., Campos, A., Paulo, J., Sousa, C., 2016. Right Homonymous Hemianopia: a clinical case report of schizencephaly. Case Rep Ophthalmol 7, 16-20.
Ortibus, E.L., De Cock, P.P., Lagae, L.G., 2011. Visual perception in preterm children: what are we currently measuring? Pediatr. Neurol. 45, 1-10.
Overweg, J., Hartman, C.A., Hendriks, P., 2018. Children with autism spectrum disorder show pronoun reversals in interpretation. J. Abnorm. Psychol. 127, 228-238.
Papadopoulos, K., Metsiou, K., Agaliotis, I., 2011. Adaptive behavior of children and adolescents with visual impairments. Res. Dev. Disabil. 32, 1086-1096.
Pascolini, D., Mariotti, S.P., 2012. Global estimates of visual impairment: 2010. Br. J. Ophthalmol. 96, 614-618.
Pawlenko, T., Chokron, S., Dutton, G.N., 2014. Considerations in Behavioral Diagnoses of CVI: Issues, Cautions, and Potential Outcomes, in: Hall Lueck, A., Dutton, G.N. (Eds.), Impairment of vision due to disorders of the visual brain in childhood: a practical approach. AFB, USA.
Pellicano, E., Burr, D., 2012. When the world becomes 'too real': a Bayesian explanation of autistic perception. Trends Cogn Sci 16, 504-510.
Pellicano, E., Gibson, L., Maybery, M., Durkin, K., Badcock, D.R., 2005. Abnormal global processing along the dorsal visual pathway in autism: a possible mechanism for weak visuospatial coherence? Neuropsychologia 43, 1044-1053.
Pérez-Pereira, M., Conti-Ramsden, G., 1999. Language development and social interaction in blind children. Taylor & Francis, London.
Pérez-Pereira, M., Conti-Ramsden, G., 2005. Do blind children show autistic features?, in: E., P. (Ed.), Autism and blindness: Current findings and reflections. Whurr, London, pp. 99-127.
Petkovic, M., Chokron, S., Fagard, J., 2016. Visuo-manual coordination in preterm infants without neurological impairments. Res. Dev. Disabil. 51-52, 76-88.
Philip, S.S., Dutton, G.N., 2014. Identifying and characterising cerebral visual impairment in children: a review. Clin. Exp. Optom. 97, 196-208.
Philip, S.S., Mani, S.E., Dutton, G.N., 2016. Pediatric Balint's Syndrome Variant: A Possible Diagnosis in Children. Case Rep Ophthalmol Med 2016, 3806056.
Plaisted, K., Davis, G., 2005. Examining magnocellular processing in autism. Cahiers de Psychologie Cognitive 23, 172-179.

Rahi, J.S., Cable, N., British Childhood Visual Impairment Study, G., 2003. Severe visual impairment and blindness in children in the UK. Lancet 362, 1359-1365.
Robertson, C.E., Baron-Cohen, S., 2017. Sensory perception in autism. Nat. Rev. Neurosci. 18, 671-684.
Rogers, S.J., Puchalski, C.B., 1988. Development of object permanence in visually impaired infants. Journal of Visual Impairment & Blindness 82, 137-142.
Rosenbaum, P., Paneth, N., Leviton, A., Goldstein, M., Bax, M., Damiano, D., Dan, B., Jacobsson, B., 2007. A report: the definition and classification of cerebral palsy. Dev. Med. Child Neurol. Suppl. 109, 8-14.
Rutter, M., Andersen-Wood, L., Beckett, C., Bredenkamp, D., Castle, J., Groothues, C., Kreppner, J., Keaveney, L., Lord, C., O'Connor, T.G., 1999. Quasi-autistic patterns following severe early global privation. English and Romanian Adoptees (ERA) Study Team. J. Child Psychol. Psychiatry 40, 537-549.
Sakki, H.E.A., Dale, N.J., Sargent, J., Perez-Roche, T., Bowman, R., 2018. Is there consensus in defining childhood cerebral visual impairment? A systematic review of terminology and definitions. Br. J. Ophthalmol. 102, 424-432.
Saunders, K.J., Little, J.A., McClelland, J.F., Jackson, A.J., 2010. Profile of refractive errors in cerebral palsy: impact of severity of motor impairment (GMFCS) and CP subtype on refractive outcome. Invest. Ophthalmol. Vis. Sci. 51, 2885-2890.
Shah, A., Frith, U., 1993. Why do autistic individuals show superior performance on the block design task? J. Child Psychol. Psychiatry 34, 1351-1364.
Shirama, A., Kato, N., Kashino, M., 2017. When do individuals with autism spectrum disorder show superiority in visual search? Autism 21, 942-951.
Shishido, E., Ogawa, S., Miyata, S., Yamamoto, M., Inada, T., Ozaki, N., 2019. Application of eye trackers for understanding mental disorders: Cases for schizophrenia and autism spectrum disorder. Neuropsychopharmacol Rep 39, 72-77.
Simard, I., Luck, D., Mottron, L., Zeffiro, T.A., Soulieres, I., 2015. Autistic fluid intelligence: Increased reliance on visual functional connectivity with diminished modulation of coupling by task difficulty. Neuroimage Clin 9, 467-478.
Simmons, D.R., Robertson, A.E., McKay, L.S., Toal, E., McAleer, P., Pollick, F.E., 2009. Vision in autism spectrum disorders. Vision Res. 49, 2705-2739.
Sonksen, P.M., 1985. A developmental approach to sensory disabilities in early childhood. Int. Rehabil. Med. 7, 27-32.
Sonksen, P.M., 1989. Constraints upon parenting: experience of a paediatrician. Child Care Health Dev. 15, 29-36.
Sonksen, P.M., Dale, N., 2002. Visual impairment in infancy: impact on neurodevelopmental and neurobiological processes. Dev. Med. Child Neurol. 44, 782-791.
Sonksen, P.M., Levitt, S., Kitsinger, M., 1984. Identification of constraints acting on motor development in young visually disabled children and principles of remediation. Child Care Health Dev. 10, 273-286.
Soul, J., Matsuba, C., 2010. Common etiologies of cerebral visual impairment, in: Dutton, G.N., Bax, M. (Eds.), Visual impairment in children due to damageto the brain. Clinics in developmental medicine. Mac Keith Press, London.

Spencer, J., O'Brien, J., Riggs, K., Braddick, O., Atkinson, J., Wattam-Bell, J., 2000. Motion processing in autism: evidence for a dorsal stream deficiency. Neuroreport 11, 2765-2767.
Stiers, P., Vandenbussche, E., 2004. The dissociation of perception and cognition in children with early brain damage. Brain Dev. 26, 81-92.
Swettenham, J., Baron-Cohen, S., Charman, T., Cox, A., Baird, G., Drew, A., Rees, L., Wheelwright, S., 1998. The frequency and distribution of spontaneous attention shifts between social and nonsocial stimuli in autistic, typically developing, and nonautistic developmentally delayed infants. J. Child Psychol. Psychiatry 39, 747-753.
Tadic, V., Pring, L., Dale, N., 2010. Are language and social communication intact in children with congenital visual impairment at school age? J. Child Psychol. Psychiatry 51, 696-705.
Takesaki, N., Kikuchi, M., Yoshimura, Y., Hiraishi, H., Hasegawa, C., Kaneda, R., Nakatani, H., Takahashi, T., Mottron, L., Minabe, Y., 2016. The contribution of increased gamma band connectivity to visual non-verbal reasoning in autistic children: a MEG study. PLoS One 11, e0163133.
Tamis-LeMonda, C.S., Bornstein, M.H., Baumwell, L., Melstein Damast, A., 1996. Responsive parenting in the second year: specific influences on children's language and play. Early Development and Parenting 5, 173-183.
Thillay, A., Lemaire, M., Roux, S., Houy-Durand, E., Barthelemy, C., Knight, R.T., Bidet-Caulet, A., Bonnet-Brilhault, F., 2016. Atypical brain mechanisms of prediction according to uncertainty in autism. Front. Neurosci. 10, 317.
Thye, M.D., Bednarz, H.M., Herringshaw, A.J., Sartin, E.B., Kana, R.K., 2017. The impact of atypical sensory processing on social impairments in autism spectrum disorder. Dev. Cogn. Neurosci. 29, 151-167.
Ting, D.S., Pollock, A., Dutton, G.N., Doubal, F.N., Ting, D.S., Thompson, M., Dhillon, B., 2011. Visual neglect following stroke: current concepts and future focus. Surv. Ophthalmol. 56, 114-134.
Urqueta Alfaro, A., Morash, V.S., Lei, D., Orel-Bixler, D., 2018. Joint engagement in infants and its relationship to their visual impairment measurements. Infant Behav. Dev. 50, 311-323.
van den Broek, E.G.C., van Eijden, A., Overbeek, M.M., Kef, S., Sterkenburg, P.S., Schuengel, C., 2017. A systematic review of the literature on parenting of young children with visual impairments and the adaptions for Video-Feedback Intervention to Promote Positive Parenting (VIPP). J Dev Phys Disabil 29, 503-545.
Ventura, L.O., Lawrence, L., Ventura, C.V., Dutton, G.N., Marinho, P., Ferro, P.F., Gois, A.L., Dias, N.C., Ventura, L., Moore, C.A., Hyvarinen, L., 2017. Response to correction of refractive errors and hypoaccommodation in children with congenital Zika syndrome. J. AAPOS 21, 480-484 e481.
Vitale, S., Cotch, M.F., Sperduto, R.D., 2006. Prevalence of visual impairment in the United States. JAMA 295, 2158-2163.
Warburg, M., 1994. Visual impairment among people with developmental delay. J. Intellect. Disabil. Res. 38 (Pt 4), 423-432.
Warburg, M., 2001. Visual impairment in adult people with intellectual disability: literature review. J. Intellect. Disabil. Res. 45, 424-438.
Watson, C.S., Kidd, G.R., Homer, D.G., Connell, P.J., Lowther, A., Eddins, D.A., Krueger, G., Goss, D.A., Rainey, B.B., Gospel, M.D., Watson, B.U., 2003. Sensory, cognitive, and linguistic

factors in the early academic performance of elementary school children: The Benton-IU project. J. Learn. Disabil. 36, 165-197.
Webster, A., Roe, J., 1998. Children with visual impairments: Social interaction, language and learning. Routledge, London.
Werth, R., Schadler, G., 2006. Visual field loss in young children and mentally handicapped adolescents receiving vigabatrin. Invest. Ophthalmol. Vis. Sci. 47, 3028-3035.
Wing, L., Gould, J., 1979. Severe impairments of social interaction and associated abnormalities in children: epidemiology and classification. J. Autism Dev. Disord. 9, 11-29.
Yeates, S., 1991. Hearing loss in adults with learning disabilities. BMJ 303, 427-428.
Zalla, T., Fernandez, L.G., Pieron, M., Seassau, M., Leboyer, M., 2016. Reduced saccadic inhibition of return to moving eyes in autism spectrum disorders. Vision Res. 127, 115-121.
Zalla, T., Labruyere, N., Georgieff, N., 2013. Perceiving goals and actions in individuals with autism spectrum disorders. J. Autism Dev. Disord. 43, 2353-2365.
Zalla, T., Seassau, M., Cazalis, F., Gras, D., Leboyer, M., 2018. Saccadic eye movements in adults with high-functioning autism spectrum disorder. Autism 22, 195-204.
Zeki, S.M., Hollman, A.S., Dutton, G.N., 1992. Neuroradiological features of patients with optic nerve hypoplasia. J. Pediatr. Ophthalmol. Strabismus 29, 107-112.
Zihl, J., Dutton, G.N., 2015. Cerebral Visual Impairment in Children. Springer-Verlag, Wien.
Zihl, J., Heywood, C.A., 2016. The contribution of single case studies to the neuroscience of vision. Psych J 5, 5-17.