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Review Article

Unveiling the Shadows: A Comprehensive Review of Cortical Vision Blindness

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Abstract

Among the range of visual impairments, cerebral visual impairment is a significant challenge. Cortical visual blindness differs from ocular or optic nerve-related blindness in that it results from injury to the brain's occipital cortex, which is in charge of processing visual data from the eyes. Due to this illness, there is a communication breakdown between the brain and the eyes; although the eyes may continue to operate normally, the brain is unable to understand the visual information that the eyes provide. The genesis, pathophysiology, clinical symptoms, diagnostic techniques, and therapeutic methods for the management of cortical visual blindness are all thoroughly examined in this review. For those who are affected, cortical visual blindness continues to be a serious neurological disorder with far-reaching consequences. Technological developments in neuroimaging and rehabilitation continue to increase our knowledge of and ability to treat this illness, providing promise for better results and a higher standard of living. It emphasizes how crucial interdisciplinary cooperation is in the development of comprehensive care plans amongst neuroscientists, ophthalmologists, rehabilitation specialists, and educators.

Keywords: Cortical Visual Blindness; Cerebral Visual Impairment; Neuroimaging; Traumatic Brain Injury; Visual Rehabilitation

Abbreviations

CVI: Cortical Vision Impairment; VEP: Visual Evoked Potentials; ERG: Electroretinography; MRI: Magnetic Resonance Imaging; CT Scan: Computed Tomography Scan

Introduction

Vision loss caused by abrasions that affects geniculocalcarine fibers visual pathway is referred to as cortical or cerebral vision blindness (CB) [1,2]. Clinically, cortical vision blindness is characterized by binocular vision loss accompanied by a normal pupillary reflex and an abnormal ocular assessment. Cortical blindness is also known as cerebral visual impairment. Terms such as cortical vision impairment (CVI) or neurological vision impairment are used to describe vision loss resulting from injury to the central nervous system. These terms suggest that the primary impairment may affect not only the primary visual cortex but also other regions that support vision, including the optic radiations, visual cortex, and pathways leading to visual focus. It is clear that the term 'cortical blindness' should be reconsidered, as it may not fully represent the condition. The term is problematic because, in most cases, some residual vision persists and visual recovery is possible, and it can also have negative connotations for the child and their family. Many children with lesions in the visual cortex exhibit some remaining visual acuity, despite the implication of 'cortical vision impairment' that there is no cognitive visual awareness [3]. Consequently, when discussing these patients, we prefer to refer to them

as "cortically visually impaired" (CVI). This terminology highlights that infants with damage or defects in the optic radiations or visual cortex exhibit their deficiencies differently from adults, and it is not the same as the terminology used for adult patients. In some cases, sources may use the term 'cortical vision blindness' to describe certain forms of cortical visual impairment, emphasizing that these conditions may involve issues prior to the striate cortex and are cerebral in nature [4]. However, we advise against overusing the term 'total blindness' for these patients, as it can be misleading in the majority of cases involving affected infants".

Epidemiology

The epidemiology of childhood vision impairment remains poorly established, with few blind registries globally and varying definitions of blindness. Estimates of millions of blind children worldwide do not account for the many children suffering from vision problems. Ninety percent of blind children reside in developing nations, [5] where the main causes of vision loss are infections and malnutrition. According to Blind Babies Foundation Registry in Northern California, the prevalence of CVI has increased in developed nations [6], and it is also one of the main causes of vision impairment in children in these countries [5,7]. In five Nordic nations, a population-based prospective study found that the relative frequency of CVI as a cause of childhood blindness increased from 11% to 23% when comparing current findings with earlier prevalence studies [8]. This increase can be partly explained by advancements in medical care, which have enabled many premature infants and babies with severe brain injuries to survive [9]. Some disorders that used to cause blindness are now curable and typically do not cause visual impairment. For instance, the prevalence of impaired eyesight caused by congenital cataracts has significantly decreased due to advancements in detection and treatment. Amblyopia is a root cause of vision loss in one eye, but it seldom results in blindness in both.

At least 3.4% of children suffer from CVI. However, since many affected children remain undiagnosed, the actual figure might be higher. CVI affects nearly 50% of learning-disabled students enrolled in special education programs [10,11]. There are two cases of CVI for every 1,000 live births. Subcortical injury is more prevalent in preterm newborns, occurring in 19 out of every 1,000 live births for infants born between 20 and 27 weeks of gestation. For children between one and three years old, CVI is a very common cause of persistent visual loss. It affects about 50% of children diagnosed with visual impairments. Additionally, 56% of people with CVI have other impairments [11].

Causes and pathogenesis

- **Hypoxic-Ischemic Brain Damage:** The most frequent cause of juvenile cerebral or cortical visual impairment is unquestionably hypoxic-ischemic brain damage [12]. A hypoxic-ischemic brain damage is the most frequent reason fir cerebral injury in children. Hypoxic-ischemic brain damage is the most common reason for cerebral injury in children. In term newborns, watershed zones in the cerebral cortex, particularly between the flow of the middle and anterior cerebral arteries and the posterior and medial cerebral arteries, are commonly affected. Parasagittal regions infarct due to hypoxia-induced loss of vascular flow autoregulation, which results in hypoperfusion of these watershed territories. Most commonly, though not exclusively, the striate cortex is impacted. Additionally, more anterior structures like the parietal and temporal cortex, as well as the corresponding and frequently occipital areas of vision, are implicated. Preterm neonates are less likely than term infants to experience hypoxia-ischemia-related parasagittal infarctions. In preterm babies, the germinal matrix in the deep white matter of the periventricular region is affected, particularly when the damage occurs between 24 and 34 weeks of pregnancy [13]. This area becomes vulnerable to hypoxia-ischemia-related bleeding in the capillaries. The damage, which is thought to be caused by free radicals and a reduction in antioxidation, is specific to the area around the ventricles because immature oligodendrocytes and subplate neurons there are more susceptible to ischemia than mature oligodendrocytes [14]. The pathophysiology appears to be much more complex, even though the optic radiations pass directly between the ventricles, which accounts for some of the visual involvement. The development of visual connections between the cortex and thalamus depends on the circuits created by these subplate neurons".
- **Congenital hydrocephalus:** The most prevalent cause of vision loss in children suffering from hydrocephalus is optic atrophy [15]. Third ventricular dilatation, chiasmal traction, vascular effects on the visual pathways, post-papilledema atrophy, sequential papillomacular bundle atrophy, or related developmental abnormalities, acting alone or in combination, may cause this condition.

- **Meningitis:** In the past, the most common causes of cortical vision blindness or cerebral visual impairment were thought to be meningitis and hydrocephalus. Recent studies indicate that 11.8% to 15% of cerebral visual impairment cases are caused by infections [7,16]. Haemophilus influenza is the most prevalent pathogen causing CVI because it can damage the occipital brain. Additional causal agents linked to ocular and cerebral vision issues include the herpes simplex virus, meningococci, and pneumococci. Visual impairment usually manifests late in the course of the infection, [17] and numerous neurologic consequences are frequently experienced in conjunction with it. A variety of factors, such as thrombophlebitis, arterial occlusion, hypoxic-ischemic damage, venous sinus thrombosis, and hydrocephalus, may result from infection-related brain injury.
- **Trauma:** Another contributory factor of pediatric CVI is head trauma, which can cause either temporary or permanent damage [18]. The most frequent cause of post-traumatic CVI is shaken baby syndrome. Sudden loss of vision in children can occur as a result of minor traumas and may be followed by migraines, disorientation, sleepiness, vomiting, or seizures.
- **Epilepsy:** In children with epilepsy, central visual impairment is primarily caused by infantile spasms [19]. The exact pathophysiology causing visual impairment in patients with infantile spasms is unknown. Additionally, several seizure medications can temporarily exacerbate vision problems, resulting in diplopia and oscillopsia. One particular infantile spasm medication, Vigabatrin, has been linked to retinal toxicity-related peripheral visual loss.
- **Shunt Failure**: Shunt failure [20], which can result in occipital lobe abnormalities and ischemia; infections such as bacterial meningitis, encephalitis, neonatal herpes simplex, and congenital toxoplasmosis; maternal use of amphetamines and cocaine during pregnancy; and metabolic diseases (most neurodegenerative diseases can impair cortical vision) are additional causes of CVI. Risks associated with cardiac treatment (heart attacks and open-heart surgery have been linked to CVI), twin pregnancies, epilepsy, and central nervous system developmental abnormalities also contribute to CVI.
- **Ocular Abnormalities**: Refractive errors, strabismus, and other ophthalmological abnormalities are also linked to CVI and must be treated [21] (e.g., with corrective lenses) to optimize any remaining vision. Children with CVI have been observed to have optic nerve atrophy, which results in visual impairment. When prenatal hypoxia–ischemia occurs in preterm infants, visual field development may be delayed. It is possible that these children have cerebrally derived strabismus.

Clinical manifestation and Diagnosis of CVI

Visual function varies significantly in children with CVI, and this variability can be caused by both external and internal factors [22]. Due to individual differences in visual processing and the diversity of brain injury, CVI manifests as a wide range of visual disorders. The most typical characteristics include decreased or limited social gaze, transient fixations that recur regularly, atypical reactions to light, such as photophobia or light staring, and loss of visual field (e.g., hemianopic deficiency, inferior altitudinal loss, and widespread constriction). Pediatric CVI is also characterized by reduced contrast sensitivity, which may be influenced by the spatial frequency of the visual input; relative sparing of color vision [23], greater latency during visually guided eye movements, such as saccades and fixations [24]; and turning away from an object during a visually guided reach, which may result from central vision-related visual field abnormalities. A child with CVI may also exhibit delayed reactions to visual stimuli and a tendency to view items with their peripheral vision or other specialized fields of vision. Because the ocular system appears normal, CVI is not always easily noticeable, and children with CVI due to antenatal or neonatal damage may not recognize that their field of view is abnormal. This represents an actual inability of the child to understand that their vision is distorted, rather than a true case of anosognosia, which is a lack of awareness of one's own condition. Therefore, CVI is not a conscious symptom, and as a result, it is frequently misdiagnosed. Often, it is the negative effects on interactions, behavior, or learning that alert parents, educators, and clinicians to the presence of a problem. This likely contributes to the underdiagnosis of CVI and the confusion it causes with other conditions such as autism, learning disabilities, or coordination disorders [25].

When diagnosing and treating cortical visual impairment (CVI), taking a detailed history is a crucial step [26] in the process. Practitioners can identify the various etiologies and contributing factors behind CVI by carefully examining prenatal, perinatal, medical, developmental, familial, and social histories. Children with CVI

who do not have any abnormalities in their anterior visual pathway during an eye exam may still exhibit poor visual behavior. In clinical settings, most optometrists and ophthalmologists evaluate visual acuity using a behavioral scale [27]. To avoid visual congestion, children with CVI often prefer to observe objects up close. CVI can significantly impact visual acuity, which measures how clear or sharp a child's vision is. A child's visual acuity can vary greatly and even fluctuate from day to day due to CVI. Standard visual acuity tests may not be effective for children with CVI, as they often rely on verbal cues or have difficulty understanding and following instructions. Instead, objective measurements such as visual evoked potentials (VEPs) or preferential gazing tests (like Teller Acuity Cards) may be used [28]. Even if their baseline visual acuity is within normal ranges, children with CVI may have trouble identifying faces, objects, or patterns. Even if their baseline visual acuity falls within normal ranges, children with CVI may have difficulty recognizing faces, objects, or patterns. They may also exhibit the "crowding effect", [23] where they struggle to identify a target among various visual stimuli. When presented with complex visual input, such as viewing an object against a patterned background instead of a solid one, these children frequently struggle. This phenomenon is known as the "crowding effect." Roman-Lantzy developed the CVI Range, which is the most commonly used visual functioning scale in the educational sector for children with CVI [29]. A child is assigned a score between 1 and 10 based on subjective observations of their ability to perceive different visual elements (colors, movement, visual complexity, etc.). Based on the CVI phase the patient is currently experiencing, different adaptive methods are recommended. These scores correspond to three distinct stages (levels of severity) of the condition. The CVI Range is recommended as a tool to guide therapy involving visual stimulation and to track the effectiveness of treatment [30].

- **Phase I:** A child typically sees in one color (often red or yellow), requires movement to localize an item, views slowly, and shows a preference for a specific visual area. They may ignore human faces and focus on objects in simple settings. Their ability to concentrate on vision is hindered by sounds and other stimuli. They might habitually stare at objects such as fans, lights, or windows. If something is unfamiliar, they may notice it from across the room. Additionally, when they touch an object, they may not look at it.
- **Phase II:** The child may begin to recognize two or three colors but will still need movement to focus on an object, and their preferences for the visual field may decrease. In these cases, a child's delay in response may worsen and persist when they are tired, anxious, or overstimulated. They stop staring at lights and start to focus on faces and objects that are farther away. As they focus, they begin to reach for recognizable items.
- **Phase III:** The child no longer shows a significant preference for any particular color, object, or visual field; delays in looking and touching are resolved. They can tolerate auditory stimuli from the environment or musical toys. However, complex scenes and unusual visuals may still pose challenges.

Electroretinography (ERG) in CVI is used to differentiate it from retinal disorders. In CVI, the retina and optic nerve are typically structurally and functionally normal [25]. Therefore, a normal ERG result can support the diagnosis of CVI, indicating that the visual impairment is due to cortical or subcortical brain dysfunction rather than retinal pathology. In some cases, patients with CVI might also have mild retinal anomalies. ERG can provide a baseline assessment of retinal function, helping to understand the extent to which retinal issues might contribute to the overall visual impairment. This comprehensive understanding is crucial for developing tailored intervention strategies that address both retinal and cortical aspects. Patients with visual impairment may have coexisting retinal conditions that could complicate the diagnosis. ERG helps exclude retinal pathologies such as retinitis pigmentosa, cone-rod dystrophies, or other hereditary retinal disorders. By ruling out these conditions, clinicians can more confidently attribute the visual impairment to cortical dysfunction.

Although there is disagreement on the value of Visual Evoked Potentials (VEP) in the diagnosis and prognosis of children with cerebral visual impairment, several researchers have evaluated the precision of flash and pattern VEP responses. VEP could be useful for tracking the recovery of vision. After studying VEPs following fetal hypoxia, McCulloch and colleagues [31] found a substantial correlation between long-term visual prognosis and normal, aberrant, or nonexistent visual evoked potentials during the first few months after birth. Regardless of the etiology, Taylor and colleagues [32] discovered that flash VEP recordings were helpful in

forecasting visual prognosis in children who were cortically blind. For many of these patients, follow-up investigations with pattern VEPs proved useful in tracking their recovery. Furthermore, a positive visual outcome was associated with children suffering from severe cortical blindness who had a normal VEP.

Neuroimaging investigations

Premature newborns may sustain a variety of brain injuries due to hypoxia and hypoperfusion, ranging from mild to severe. However, once the newborn is discharged from the hospital, CT (computed tomography) scans and MRI (magnetic resonance imaging) are more informative and reliable than ultrasonography [33] for assessing the extent of white matter damage and related conditions. MRI and CT scans can reveal ventriculomegaly, characterized by an abnormal enlargement of the trigone and body of the lateral ventricle, reduced periventricular white matter volume, and significant atrophy affecting the sulci and surrounding areas near the ventricles, often with minimal intervening white matter [34].

Magnetic resonance imaging will demonstrate increased signal levels in the periventricular white matter area and prolonged myelitis. It should be noted that sagittal MRI can identify thinning of the corpus callosum caused by transcallosal fiber loss [35]. Infants who are born prematurely and have experienced severe underweight episodes or heart failure exhibit a distinct pattern of brain damage. The injury primarily affects the brainstem nuclei and deep gray matter, although periventricular injury may also occur. The cerebral cortex, cerebellum, and thalamus are often significantly damaged. MRIs performed many months after the injury will show small thalami, brainstem, and cerebellum, which are typically accompanied by diminished cerebral white matter. This group has a low survival rate; however, if they do survive, they may experience athetosis, quadriparesis, severe seizure conditions, and mental impairment [36]. Periventricular leukomalacia is becoming a prevalent cause of cortical visual impairment as survival rates for very preterm infants improve. In comparison, the frequency of encephalitis caused by hypoxic-ischemic damage in newborns seems to be declining. However, ten to fifteen percent of brain dysfunction in term newborns is due to perinatal hypoxic-ischemic damage. In infants with low to severe hypoxic-ischemic events, damage typically occurs in the watershed zones, which are located between the posterior and middle cerebral arteries, as well as between the middle and anterior cerebral arteries. These events cause distinct, often infarction-like, cystic lesions in the border zones between major circulatory areas. Consequently, infarctions most commonly develop in the parieto-occipital and frontal regions. During the acute phase of damage, either ultrasonography or CT scans can be accurate in identifying the extent of injury. Therefore, when evaluating these patients, MRI is preferred. After an infant recovers from severe hypoxic-ischemic damage, MRI [37] examinations may reveal cerebral atrophy and shrinkage in the white matter adjacent to the stroke area, such as ventricular enlargement on both sides, gyral abnormalities, ulegyria due to cortical loss, and wedge-like infarcts in the watershed zones. Term newborns who experience significant hypoxic-hypoperfusion episodes or cardiocirculatory arrest often exhibit a specific pattern of brain damage. Unfortunately, the majority of these children do not survive.

Rehabilitation and treatment

As of now, there is no accepted treatment for cerebral palsy in children. However, neonatologists work to reduce the prevalence of CVI (as well as other disorders) by managing extremely premature births and addressing prenatal hypoxic-ischemic injury. The primary goal of rehabilitation for those with cortical visual impairment (CVI) is to address impairments, stimulate and enhance visual function, and empower individuals to make the most of their vision [29]. Strategies in this rehabilitation include personalized visual stimulation and training, compensatory methods to improve contrast and simplify surroundings, saccadic compensation training, and a multidisciplinary approach involving neurologists, ophthalmologists, and rehabilitation specialists [34]. Rehabilitation for CVI also considers the emotional impact of the condition and provides support to both the patient and their family. Targeted visual stimuli, such as lights, shapes, and contrasting colors, are presented to help with visual acuity, visual field awareness, and object recognition. The stimuli are customized to match the individual's unique visual deficiencies and strengths. Techniques such as simplifying the environment with fewer patterns, improving contrast, and reducing distractions can benefit individuals with CVI-related issues like simultanagnosia (difficulty recognizing multiple items at once) and prosopagnosia (facial blindness). Saccadic compensation training aims to enhance quick eye movements, or saccades, to address visual field abnormalities. A multidisciplinary approach [7] is essential for addressing the complex visual, cognitive, and functional

demands of the individual, requiring coordination between ophthalmologists, neurologists, and rehabilitation specialists. Cortical vision blindness can vary from mild to severe and may fluctuate significantly; however, with early intervention, some improvement is achievable over time. Listed below are some of the potential therapies used to treat this condition: Visual Stimulation Therapies: This therapy helps to connect the brain and ocular circuits. It involves repeatedly presenting visual tasks on a regular basis, allowing individuals with CVI to associate non-impaired abilities (such as spatial and temporal orientation) with the visual stimuli.

- **Sensory Integrative Therapies:** This technique benefits children who have difficulty processing sensory information. Sensory integration therapy addresses the five primary senses: sound, sight, smell, touch, and taste. It also targets the functioning of the vestibular sense, thermoception (temperature sense), interoception, and proprioception (body position sense). It is commonly included in an occupational therapy plan, as children with CVI often face challenges with visual perception and the integration of other senses, which can help improve their overall sensory processing and communication.
- **Occupational Therapy:** Individuals with cerebral visual impairment can benefit from occupational therapy as it aids them in performing daily routine tasks while boosting their self-confidence. Occupational therapists help these individuals learn to use their vision effectively, incorporating non-visual strategies to perform various tasks independently.
- **Speaking Therapy:** This therapy is typically used to address language and speech difficulties but can also be part of a CVI treatment plan. Speech therapists help children improve their speaking, swallowing, and feeding skills, which may be affected by visual impairment.

The findings from neuroimaging investigations guide the development of personalized rehabilitation strategies. For example, if MRI identifies specific cortical regions afflicted by CVI, rehabilitation programs might be designed to treat these specific deficiencies. Techniques such as sensory substitution and adaptive technologies are designed based on the insights gained from neuroimaging. Continuous neuroimaging examinations during the rehabilitation process track changes in brain activity and structure, allowing revisions to the rehabilitation plan to better match the patient's changing needs. The therapy of CVI is intimately related to neuroimaging findings and rehabilitation outcomes. Imaging investigations are used to identify specific forms of cortical injury, and pharmacological medications and cognitive therapy are chosen accordingly. For example, if neuroimaging reveals that visual circuits are maintained but functionally degraded; interventions may focus on improving brain connections or compensating with cognitive methods. The use of neuroimaging data guarantees that therapies are accurately targeted and customized to the individual's neuroanatomical profile, hence increasing the overall efficacy of the therapeutic approach.

Identifying additional neurological conditions associated with CVI is a crucial part of treatment. Children with CVI often experience various motor issues, such as abnormalities in ocular motor function. Caregivers' observations are essential; they frequently notice that children with CVI may stare at lights (seen in 60% of these children) [38], espond to highly contrasted colors or objects in a darkroom, and show particular reactions to certain parts of their visual field. Addressing the underlying causes and helping individuals adapt to their condition are key components of treating cortical visual impairment. Rehabilitation and optimizing residual vision are often primary goals, as cortical visual impairment results from damage to the brain's visual processing centers. If cortical blindness is due to a treatable condition, such as inflammation or infection, doctors may prescribe anti-inflammatory medications or antibiotics. Stem cell therapy [39] and various visual stimulation programs have also been suggested as beneficial for children with CVI. Technological advancements have significantly improved the independence of those who are cortically blind. Screen readers, voice-activated assistants, and smartphone apps designed for visually impaired users can assist with a range of functions, including reading, communication, navigation, and entertainment.

Discussion

This neurological condition can have a substantial impact on a person's day-to-day activities, affecting various areas such as social relationships, education, mobility, and communication [40]. Understanding these effects is essential for formulating effective support and intervention strategies aimed at enhancing the quality of life for those suffering from cortical blindness. A patient with CVI will have difficulty navigating unknown places. While they may be able

to navigate their homes without assistance due to familiarity with their surroundings, navigating unfamiliar environments is particularly challenging for someone who is blind, especially if they have lost all vision [7]. Relocating items without first notifying or asking the blind person is something that those who live with or visit should avoid doing. Commercial premises can be improved for blind users with tactile tiles, but unfortunately, this is often not the case. This presents a significant challenge for blind individuals who might want to visit the location. A primary goal for disabled individuals is to achieve independence. A blind person can lead an independent life by using specifically designed adaptive tools. There is a lot of flexible technology available to help blind individuals live independently, but it is often difficult to find in local stores or markets. The refreshable Braille display is an excellent example of such a device [41]. Blind people often have to search extensively and put in a lot of effort to obtain each piece of equipment that will help them become more independent. While being amiable and supportive is commendable, overly helpful people can sometimes make things more difficult for blind individuals. Many people are so eager to assist someone with a disability that they forget to ask whether the person actually needs help. Even if a blind person seems to be moving slowly from your perspective, you shouldn't rush to assist without first speaking with them [42]. You can end up creating issues for a blind person. Although most blind individuals develop their own strategies for recognizing and organizing at least their own clothing, it remains a challenging process. Since many blind people rely on the shape and texture of objects for recognition, folding laundry can be difficult. Matching and organizing socks, in particular, becomes a challenging task. This difficulty arises because color perception is extremely challenging for those who are completely blind.

It can be challenging for blind individuals to find well-written books in accessible formats. Additionally, despite the abundance of information and reading material available online, blind people often face difficulties accessing the Internet [21]. A blind person can use screen reading software, but navigating the Internet can be challenging if websites are not designed with accessibility in mind. Blind individuals rely on picture descriptions to understand the content conveyed through photographs. Living in a world designed for the sighted presents a number of typical challenges. Small errors can occur, such as tripping over a chair that has not been properly pushed under a desk or breaking a glass left at the edge of a table. However, sighted individuals often mistakenly assume that such errors are due to the visual impairment itself, rather than the inaccessibility of the environment. Low vision or blindness does not reflect an individual's intelligence or happiness. The inability of a sighted observer to imagine the surroundings without vision does not imply that blind people lead unhappy or miserable lives due to their impairment [43]. The world of work is significantly different for the blind. Given the scarcity of accessible employment and workspaces, it is understandable why hiring a visually impaired individual might be perceived as a risk by some companies. This situation not only severely impairs their economic independence but also has a substantial impact on their emotional health and self-worth. People who are visually impaired or blind often struggle to support themselves due to the limited options available to them.

Conclusion

In industrialized countries, cerebral visual impairment (CVI) is the most common cause of pediatric visual impairment; in poor countries, its prevalence is rising. The diagnosis and characterization of visual impairments in children with CVI might be difficult due to neurologic comorbidities. There isn't a specific treatment for CVI at the moment, and a lot of the rehabilitation strategies are untested. If given the proper evaluation and assistance, children and youth with CVI should gradually improve in their visual functioning. Compared to ocular vision impairments, CVI necessitates a totally different approach to instructional assistance and material/environmental adjustments.

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