

Article ▶ Vision Therapy in the Cerebral Palsy Population: Utilizing Vision Therapy Techniques to Remediate Motor and Sensory Deficits of the Visual System

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ABSTRACT

Background: Contrary to popular belief, cerebral palsy is not solely motor in nature. Sensory deficits are prevalent secondary to the location of damage resulting in the brain, specifically in the visual cortex. Ocular manifestations may be commonplace. The purpose of this case study was to assess the effect of vision therapy on the motor and sensory deficits of the visual system of a patient with mild cerebral palsy.

Case Report: The patient is an eight-year-old African-American male with mild cerebral palsy secondary to birth trauma. Baseline comprehensive visual efficiency and visual information processing examinations were conducted to assess all relevant deficiencies. The patient then received six months of office-based vision therapy. Progress evaluations were performed monthly, with a final evaluation and re-administration of the standardized testing following program completion. There were significant subjective and objective gains noted in both visual efficiency and visual information processing.

Conclusion: Office-based vision therapy was shown to significantly aid in the rehabilitation of both motor and sensory ocular/visual deficits present in a cerebral palsy patient. This case report suggests that, with appropriate learning paradigms and feedback, faulty neuronal connections have the capacity to become rehabilitated and strengthened owing to brain plasticity.

Keywords: cerebral palsy, cortical visual impairment, vision therapy, white matter damage of immaturity

Introduction

Developmental disabilities are prevalent in the United States, affecting an estimated 17% of children. Cerebral palsy (CP) is one of the major physical disabilities that affect functional development. Cerebral palsy is characterized by inability to control motor functions and has the potential to have a negative effect on the overall development of a child by affecting the child's ability to explore, speak, learn, and become independent.¹ The prevalence of CP varies little among developed countries. In the United States, it is about 1 per 1000, with approximately 4000 new cases annually.²

Causes of CP occur during the prenatal, perinatal, and postnatal time periods, making it difficult to determine the actual incident in many instances. The majority of cases appear to occur secondary to problems during intrauterine development, congenital disorders, asphyxia at any gestational age, and preterm birth. The most common cause of CP in the preterm population is injury to the periventricular white matter of the brain, resulting in intraventricular hemorrhage or periventricular leukomalacia. Periventricular white matter damage is highly predictive, with CP developing in 80% to 85% of infants.¹

Cerebral palsy has become an umbrella term of sorts that is familiar to most, but not clearly defined. While many agree that CP is a non-progressive disorder affecting both posture and movement as a result of a defect or insult to the immature brain, a universally accepted definition has been difficult to

agree upon. Debates about this static encephalopathy have been ongoing for more than 150 years as to what exactly the term 'cerebral palsy' encompasses. Past definitions have focused solely on a neurodevelopmental condition that is non-progressive in nature and results in motor impairment secondary to lesions occurring in the central nervous system. On July 11-13, 2004 at an international workshop in Bethesda, Maryland, an Executive Committee and selected leaders in the preclinical and clinical sciences met to review the term 'cerebral palsy' with the intent to provide a common conceptualization of CP for use by a broad international audience. A resultant report on the Definition and Classification of Cerebral Palsy was released in April 2006 offering up the following definition for international consensus and adoption:

"Cerebral palsy (CP) describes a group of permanent disorders of the development of movement and posture, causing activity limitation, that are attributed to non-progressive disturbances that occurred in the developing fetal or infant brain. The motor disorders of cerebral palsy are often accompanied by disturbances of sensation, perception, cognition, communication, and behavior; and by secondary musculoskeletal problems."³

This newly developed definition is the first not only to focus on the motor aspects of cerebral palsy, but also to bring attention to the additional neurodevelopmental disorders and impairments of sensation, perception, and cognition that tend to accompany it.

The diagnosis of CP is made largely through clinical observations. Common markers of CP include abnormal muscle tone (hypo- and hypertonic), an increase in deep tendon reflexes, delayed developmental milestones, and an abnormal neurologic examination. However, while development is delayed, it is extremely rare to note regression of motor function. A definitive diagnosis of CP is not an easy task. The Surveillance of Cerebral Palsy in Europe (SCPE) network, consisting of 22 centres from 15 countries, comprised child neuro-paediatricians, child rehabilitation doctors, and epidemiologists who presented the SCPE criteria at a meeting in Oxford in 1999. They found that a CP diagnosis must rely on clinical picture and history. It was agreed upon that age 5 years was the optimal age for confirmation of diagnosis and that no upper age limit should exist. However, it is useful to isolate CP cases of post-neonatal origin, which they defined as cases arising from an etiological event 27 completed days after birth.⁴

Classification is another daunting task. One common form of subdivision relies upon the extent of motor involvement and identification of the limbs involved. Quadriplegia occurs when both arms and both legs are involved, while diplegia occurs when only the lower half of the body is affected. Hemiplegia involves only one side of the body and is the most common form occurring in children born at term.² Cerebral palsy can then be broken down into subtypes based upon abnormal pattern of movement and lesion location. Spastic CP shows increased tone and pathological reflexes (prolonged or an inability to stop spontaneously) and generally occurs from a lesion in the pyramidal cells and tracts. Dyskinetic CP, from basal ganglia lesions, presents with involuntary and extraneous movement causing misdirection and becoming habituated. This subtype can be further split into chorea (rapid, involuntary, jerky movements) and athetoid (slow, writhing, contorting movements). Ataxic CP, occurring from cerebellar lesions, is characterized by low tone, equilibrium disturbance, and loss of orderly muscle coordination.² It is important to note that while these classifications exist, many cases of cerebral palsy result in a mixed form and are ultimately difficult to classify. To date, lesion size and impairment severity have not been statistically correlated.⁵

Effective management of CP is crucial to maximize overall child development. While this is a lifelong condition with no known cure, technology, therapy, and education provided by a team of highly trained professionals can aim to improve functional abilities of the child, allowing them to perform to their fullest potential. Optometrists are a major part of this team and are in an excellent position to identify all ocular manifestations, whether sensory or motor. Vision therapy is underutilized and can aid in rehabilitating the visual system through repetitive motor activities and the strengthening of neuronal pathways in the brain. Ultimately, the visual system will be optimized, and integration between the visual and motor systems can be improved.

The purpose of this case study was to assess the benefits of vision therapy on the ocular/visual manifestations of cerebral palsy. I believe that CP is a head-to-toe motor control issue leading to an increased prevalence of ocular motor control abnormalities. Because CP is highly motor in nature, repetitive ocular/visual motor activities will be required to develop a sense of 'muscle memory.' In addition, I suspect that the neurodevelopmental impairments go beyond being solely motor in nature. The hypothesis is that the plasticity of the brain will allow us to rehabilitate and strengthen faulty neuronal input and feedback signals that are additionally hampering visual efficiency and visual information processing.

Ocular/Visual Manifestations

In the past, a diagnosis of CP culminated from a collection of signs, symptoms, and historical information. However, with the development of neuroimaging techniques such as computerized tomography and magnetic resonance imaging, greater insight has been gained into the pathophysiology of CP. This allows for a better understanding of this largely motor disorder and the spectrum of findings associated with it, specifically the vast array of ocular/visual manifestations.

The spectrum of visual disorders in children with CP encompasses a wide array of both central and peripheral problems. Though originally thought to be strictly motor in nature, recent research has found that children with CP exhibit a unique ocular/visual profile, with deficiencies spanning from the anterior ocular structures back to the visual cortex in the occipital lobe of the brain.⁶

In patients with CP, the best average visual acuity for either eye was 20/50.² This reduction seemed to stem from a variety of factors, including, but not limited to, poor cooperation and understanding of the test, cortical visual impairment, nystagmus, and optic atrophy. In a study conducted between January 2002 and June 2005, a sample of 129 children with CP underwent ophthalmic examination. Of the children examined, 74.5% presented with a significant refractive error, with 33% being hyperopic, 6% myopic, 6% astigmatic, and 29% with a mixed refractive error.⁶ Although this particular study found hyperopia to be the most common, studies have not come to a general consensus regarding refractive profiles. There is also wide discrepancy regarding the prevalence of strabismus in cerebral palsy. This same study found 88.2% of the 129 children manifesting strabismus.⁶ The statistics vary with regard to the direction of the turn, with variability of the angle ranging from moderate esotropia to moderate exotropia. This lack of binocularity in turn results in compromised depth perception, which can greatly hinder locomotion and navigation.

Impairment of the oculomotor system is also extremely prevalent and likely motor in nature. This is typically evident by abnormalities of fixation, smooth pursuit, and saccadic movements. Additional abnormal ocular movements have also been documented, including abduction deficits, overaction of

the inferior oblique, and dissociated vertical deviations. Many children with CP develop compensatory strategies such as brusque head movements, blinking, and hyperfixation to adapt to these motor deficiencies.⁶ Accommodation has also been found to be erratic and generally reduced in this population, resulting in an inability to sustain focus on near work.⁷

From the vast array of studies done on the ocular manifestations of cerebral palsy, we can deduce that while many deficits are motor in nature, there is significant support showing that the sensory system also contributes. MRI scans of the brain demonstrate pathophysiology that provides evidence of lesions in the visual cortex that can interfere with both visual perception and the processing of sensory information required for motor output.⁸

A European collaborative study of CP examined over 350 MRI scans from a clinical population of 430 cases and reported staggering statistics regarding lesion location and the link between visual impairments and CP, far beyond being solely motor in nature. Children with spastic diplegia exhibited a significant prevalence of white matter damage of immaturity (WMDI), including both periventricular leukomalacia (PVL) and periventricular hemorrhage. In fact, seventy-one percent of the children with spastic diplegia proved to have WMDI.⁸ This finding is extremely important because WMDI is actually the cause of another group of disorders in childhood and more commonly causes visual disability rather than motor disability.⁸ Damage of this nature to the posterior aspect of the cerebrum interferes with both the ventral and dorsal streams of the visual cortex. Ocular/visual manifestations vary based upon exact lesion location, with the dorsal stream moderating motor function while the ventral stream is highly associated with visual perception. With this information, it is reasonable to conclude that some proportion of spastic diplegics exhibit central visual problems, and more importantly, the vision problems in CP are just as much sensory as they are motor.

In some cases of spastic quadriplegia, we also see signs of white matter damage of immaturity, but most damage is found in the cortical and subcortical areas of the brain. Children with hemiplegia show damage restricted to one side of the brain in the form of strokes or asymmetrical periventricular leukomalacia.⁸

The literature shows that 60 to 70% of children with CP also manifest cerebral visual impairment (CVI),⁶ a visual deficit caused by damage to the retrogeniculate visual pathways in the absence of any major ocular disease. As discussed above, the periventricular white matter damage that underlies CP frequently involves the optic radiations, occipital cortex, and visual associative areas, explaining the prevalence of this overlap. In light of this information, visual perception and processing can be considered common symptoms in many cases of CP. One study of 105 children with CP, ranging in age between 6 and 15, used the Motor Free Visual Perception Test to assess the integrity of visual perception, more specifically looking at spatial relationships, visual discrimination, figure-ground

perception, visual closure, and visual memory. It was found that perceptual age in CP falls far below chronological age. Sixty children were found to have perceptual abilities less than those of a 6-year-old, and 28 children had perceptual abilities equal to someone between 6 and 7.5 years of age. Seventeen children appeared with perceptual abilities better than those of a 7.5-year-old child.⁹

Difficulties with visual-motor integration reflect altered sensory-motor interaction in CP. Saavedra et al. examined the complex coordination of the eye, head, hand, and trunk required when reaching to grasp an object of interest. They found that children with CP needed more concurrent head movement when shifting their gaze and had significant difficulty isolating eye, hand, and head movements. They concluded that coupled eye, head, and hand movements may be the default output of the CNS early in development, and the ability to isolate the effectors may be necessary in order to gain feedback control of motor actions. Results of this study suggest that a primary deficit across all diagnostic groups in CP may be the inability to isolate the various effectors.¹⁰ This may also be linked to the persistence of primitive reflexes in CP.¹¹

Additionally, extensive lesions of the brain can ultimately lead to visual field deficits, with lesion location dictating the type and severity of the loss. When insult to the visual system occurs early during gestation and is relatively limited, brain plasticity may be capable of compensating, and the field will be normal. This, however, is not always the case. When field loss does occur, it can affect both orientation and mobility, adding insult to injury on an already weakened motor system. Unfortunately, it is likely that visual field deficits go undetected in many of these children because assessment can be hampered by an inability to understand or to cooperate during formal testing. Confrontation fields tend to underestimate prevalence due to a failure to identify scotomas within a functional field and relative deficits.

The Modified Clinical Examination

The prevalence of visual problems in children with CP tends to go under-reported as a result of difficulties with examination. Motor impairment and, in some circumstances, cognitive impairment hinder the optometrist's ability to obtain both objective and subjective data in the examination room. Undiagnosed ocular/visual disorders can lead to learning difficulties secondary to poor tracking when reading, binocular instability, impaired accommodation, and visual perceptual dysfunctions. In addition, productive visual exploration is crucial to foster overall motor improvement; thus, a poor visual system contributes to a poorer global prognosis for the child. As an optometrist, it is important to modify the examination in order to obtain the necessary data for proper diagnosis and execution of treatment.

Appropriate positioning of the patient is imperative to minimize stress on general and ocular muscle tone. Supportive seating, and potentially testing while in a supine position, may

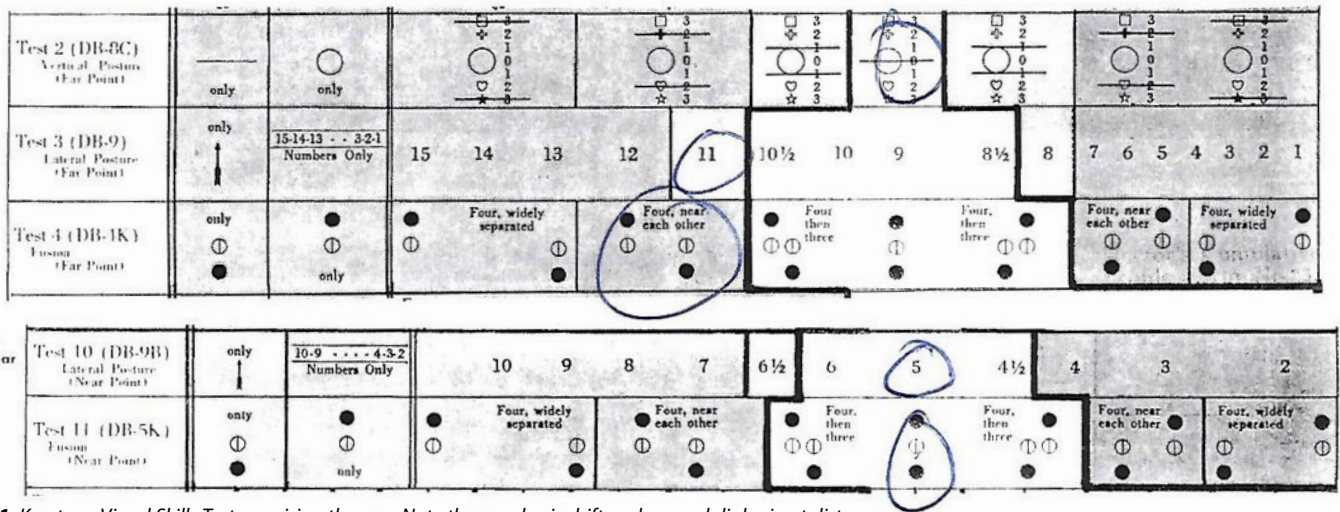


Figure 1: Keystone Visual Skills Test pre-vision therapy. Note the exophoric drift and crossed diplopia at distance.

be required in certain circumstances. Additionally, it may be necessary to rely more on objective rather than subjective data. Function questionnaires filled out by caregivers and other support staff may also be useful.

Case Study

An eight-year-old male, NC, was first seen in September 2013 for a comprehensive visual efficiency and visual information processing examination. He was delivered via Caesarian section following a nipped umbilical cord during an amniocentesis procedure. He required three blood transfusions at birth and stabilization of blood pressure. At the age of 18-20 months, NC was diagnosed with mild CP secondary to trauma sustained at birth. His static encephalopathy led to a generalized left-sided hemiparesis, mild hypotonia, mildly delayed fine motor/graphomotor/self-care skills, and mild sensory processing difficulties. He was receiving speech and occupational therapy two times per week. His ocular history was significant for strabismus surgery to reduce exotropia in 2010. NC's parents reported the following signs: tiring easily, poor reading, poor comprehension, omitting words, rereading lines, right/left confusion, letter reversals, red eyes, and a general dislike of reading.

Pre-Vision Therapy Optometric Findings

NC was initially evaluated over three consecutive office visits in September 2013. Distance Snellen visual acuity was 20/20- OD and OS with a very mild amount of astigmatism found upon refraction. On the sensorimotor examination, NC exhibited a 6-8 prism diopter constant left exotropia at distance, with crossed diplopia at distance present on Keystone Visual Skills testing (Figure 1). Red Maddox Rod testing at near revealed a 9 prism diopter exo drift. Compensating base out ranges were severely reduced ($x/12/2$), showing that NC had a very low tolerance for convergence with almost no ability to recover fusion once it was broken. No stereopsis was perceived either on Random Dot Stereograms or non-Random Dot Wirt Circles, indicating an absence of both global and local

stereoscopic awareness. An open-view autorefractor was used to assess focusing at near. NC exhibited unstable accommodation in both eyes when viewing a letter at 40 cm. Pursuits and saccades were disjointed due to difficulty crossing the midline; thus, large compensatory head movements were initiated.

The visual processing examination also showed global delays. We administered the Test of Visual Perceptual Skills, 3rd edition (TVPS-3). To interpret the scores, the 50th percentile rank is average for one's age level, and the 25th percentile is low average. NC's scores on the subtests were as follows:

Subtest	Percentile Rank
Visual Discrimination	<1
Visual Memory	<1
Spatial Relations	.16
Form Constancy	.63
Sequential Memory	.25
Figure Ground	.63
Visual Closure	.50

Visual-motor integration ability was evaluated several ways. On the Wold Sentence Copy test, NC was asked to copy a standardized sentence from far to near. This test was used qualitatively, and his word spacing and alignment was considered very poor (Figure 2). He was also tested using the Van Orden Binocular Behavior Pattern test, where he was asked to look through a stereoscope while tracing geometric figures and drawing lines using both hands. He demonstrated a below-adequate ability to integrate his visual and motor systems. NC was then tested on the Gardner Reversal Frequency test. He made 29 errors, which is well below one standard deviation for his expected age level. Finally, we tested NC's reading fluency using the Test of Silent Word Reading Fluency (TOSWRF). This is a standardized timed test where he must insert lines between sequences of letters when he recognizes a word. NC scored in the 32nd percentile for his age, with an age equivalent of 7.6.

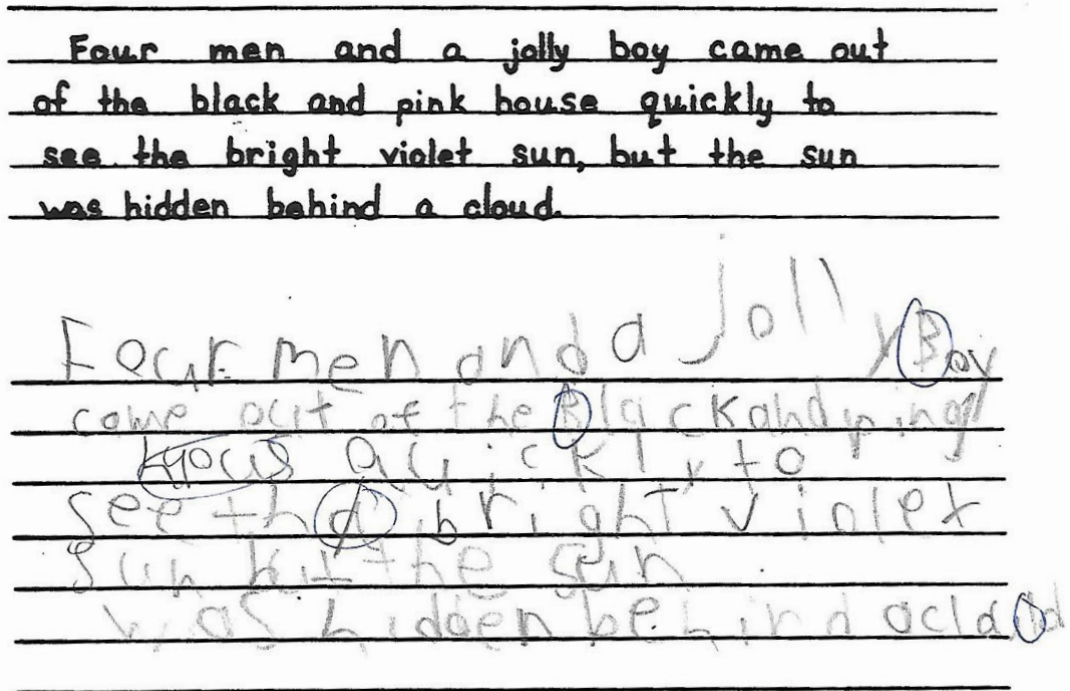


Figure 2: Wold Sentence Copy pre-vision therapy

Overview of Vision Therapy

NC completed six months of weekly, office-based vision therapy, reinforced by daily at-home activities that were assigned following each session. The focus of his treatment program was to increase his convergence ranges to stabilize binocularity, to improve eye tracking, and to stabilize focusing at near. Additionally, we aimed to target his deficiencies in visual information processing with an emphasis on laterality and directionality, visual discrimination, visual and sequential memory, spatial relations, and visual-motor integration. Representative activities included Brock string, vectograms, slap tap, the Sanet Vision Integrator (SVI), the Neuro-Vision Integrator (NVR), Marsden ball, and parquetry blocks.

Throughout the course of NC's vision therapy program, we found that certain modifications were required to accommodate for global deficiencies secondary to his CP. It is important to keep in mind that each patient is different. While these techniques worked for NC, different modifications may be required to optimize performance for other children.

Since NC exhibited only mild left-sided hemiparesis, he did not require an additional support system to maintain his posture when seated or walking. In fact, we found that he had the most difficulty attending to activities when we asked him to sit in one spot. It became evident that NC performed best when provided with an object to hold in his hands or when placed on a balance board or balance beam. Being motorically engaged seemed to have an arousal or orienting effect on his performance, allowing him better to execute on his visual activities. Examples include standing on a balance board while tracking a Marsden ball or holding a stuffed animal

while working on gross peripheral fusion of vectograms at distance. While this technique worked with our patient, in more moderate and severe cases of cerebral palsy, it may be necessary to have the patient seated for all activities to stabilize gross motor control, which in turn will stabilize fine motor control of the eyes.

To address many of his perceptual deficiencies, NC worked best with parquetry blocks that he could hold and manipulate. This again seemed to assist in engaging him in the activity and provided him with motor feedback. To address spatial relations, we used an 8.5" x 11" piece of plastic as his "board." He was directed to align the shapes on this board exactly where he saw them on the page (e.g., if a green square was in the left inferior quadrant of the page, a green square should be placed in the left inferior quadrant of the board). The parquetry blocks were also used to address figure-ground issues. NC was asked to pull out all of the yellow triangles from the box of assorted colors and shapes. This was turned into a competition when he attempted to beat his previous time by performing the task as quickly and accurately as possible. Finally, NC was shown a simple design constructed of two or three parquetry blocks for 3 seconds. The blocks were then covered, and he was asked to recreate the design from his visual memory.

When working on eye teaming, we focused first on tasks at near then slowly progressed to intermediate and far distances where NC struggled the most with binocularity. We were able to establish good convergence ranges at near and found that he developed the capability to appreciate Random Dot Stereopsis, though processing was slow. Due to his lag

time in stereoscopic localization, we needed to give NC extra time to observe a target before he was able accurately to appreciate depth and to provide a SILO (small-in, large-out) response. When asked to view a stereopsis target on the Vision Therapy System 3, we found that NC was unable to detect any depth within the ten second allotted period prior to a change in the stimulus. However, when we paused the program and encouraged him to take his time while viewing, he was able accurately to detect which objects appeared to pop out at him.

During all ocular motor therapy, NC had an extremely difficult time dissociating his eye movements from his head movements. We found that the simple act of placing a stuffed animal on his head helped to stabilize his head, allowing adequate isolation of the eyes. Additionally, we utilized the head sensor with the Neuro-Vision Rehabilitator, encouraging him to maintain head alignment in order to limit the negative auditory feedback when head movement was excessive.

The overall goal of the program was to improve or to eliminate visual deficiencies that hindered NC's ability to excel both in educational instruction and globally in orientation and mobility in everyday life. NC was provided with both the Home Therapy System and the Perceptual Therapy System II to work on at home. These provided great reinforcement of the eye teaming, focusing, tracking, and perceptual skills that were developed in-office.

Post-Vision Therapy Optometric Findings

After six months of vision therapy, NC experienced significant visual improvements. His once constant left exotropia at distance had become intermittent in nature, with a preference for right eye fixation. Cover testing at near revealed a mild exophoria. Base out ranges at near were expanded to an above-average range of x/30/16. NC was subjectively noting less of an eye turn and required less compensatory head movement when tracking and reading. His accommodative accuracy had also greatly improved. NC tested positive on the Random Dot E and correctly answered several Random Dot Stereopsis targets at 250 seconds. At peak performance, stereoscopic depth perception improved to accurate identification of 7 out of 10 Wirt Circles.

NC also made significant gains in select areas of weak visual information processing. Upon repeat administration of the Test of Visual Perceptual Skills, 3rd edition (TVPS-3), NC showed significant improvements in the following areas:

<u>Subtest</u>	<u>Percentile Rank</u>
Visual Discrimination63
Visual Memory25
Spatial Relations.	>99
Sequential Memory84

Repeat administration of the Gardner Reversal Frequency test, though still below normal for NC's age, demonstrated an improvement from 29 errors to 20 errors. His reading fluency using the Test of Silent Word Reading Fluency (TOSWRF) improved, placing him in the 47th percentile, with an age equivalent of 8.6. NC also made strides in the area of visual-motor integration, with demonstration of more control on the Van Orden Binocular Behavior Pattern test. Though NC still exhibits some deficiencies in the areas of visual information processing, he has developed a sound foundation of visual skills upon which to continue to build. As a result of the progress made up to this point, NC will continue to receive an additional two months of vision therapy to further his gains.

Discussion

It is imperative to dispel the past notion that CP manifests itself primarily through movement disorders, with few other associated findings outside of the traditional motor impairments. In this case study, we investigated prevalent ocular manifestations in a child with CP. We concur with current research that a visual perceptual disorder is an integral part of the clinical picture of cerebral palsy and should not solely be viewed as an associated finding.

Visual information processing is an extremely complex cerebral activity that involves a large portion of the central nervous system. It is evident that lesions sustained either during gestation or early in life are disrupting the structure and function of the visual cortex. Damage in this area of the brain has become so predominant in these patients that all forms of cerebral palsy seem to exhibit specific motor and neuro-ophthalmic profiles.

In light of this information, it is crucial that children with CP see a developmental optometrist annually. Early assessment and accurate detection of visual disorders is paramount to foster a better global prognosis. A comprehensive ocular health examination is not adequate. Both visual efficiency and visual information processing need to be evaluated, and when indicated, proper referral to an office that specializes in vision therapy is vital.

Conclusion

This case study demonstrated that office-based vision therapy does aid significantly in rehabilitating the ocular manifestations of cerebral palsy. There were significant gains made in both how efficiently the eyes take in information and how this information is then processed, stored, and utilized to integrate other systems. I propose that CP is a head-to-toe motor control issue resulting in deficiencies of the visual system. Though perceived to be mainly motor in nature, many cerebral lesions found in these patients are occurring in the visual cortex. As a result, we find not only deficiencies in visual efficiency, but in visual information processing as well. Through a variety of office-based vision therapy techniques, we were able successfully to use repetitive ocular motor activities

to develop a sense of muscle memory to enhance both tracking and eye teaming. In addition, neuroplasticity allowed us to strengthen many of the faulty neuronal input and feedback signals that were affecting visual perception and motor control.

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