Neuroplasticity as a Proposed Mechanism for the Efficacy of Optometric Vision Therapy & Rehabilitation

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Abstract

Optometric vision therapy and rehabilitation have been used to successfully treat a wide range of visual disorders for over 80 years. However, it was not until the past few decades that the scientific basis supporting its efficacy was beginning to be understood. This review article discusses current neuroplasticity concepts, the supporting research, their therapeutic applications in other disciplines, and their implications to optometry. It can be hypothesized that vision therapy techniques strengthen synaptic connections through Hebbian learning and induce cortical reorganization to maximize visual efficiency. This can provide a theoretical basis for the efficacy of vision therapy, while neuroimaging can provide an objective basis.

Key Words

brain injury, neuroplasticity, vision rehabilitation, vision therapy

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INTRODUCTION

Vision therapy (VT) has been an integral part of the optometric profession since the late 1920s. To date, there are numerous scientific articles demonstrating the effectiveness of VT in treating oculomotor dysfunction, non-strabismic binocular disorders, strabismus, amblyopia, accommodative disorders and visual information processing disorders.[1-5] There are also various articles addressing the success of VT in reducing symptoms attributable to visual dysfunction following an acquired brain injury.[6-8] Nonetheless, many skeptics of optometric VT exist in the medical community. These skeptics include some ophthalmologists and pediatricians who maintain that the majority of behavioral management approaches in VT are not evidence-based, and thus are not endorsed by their specialties.[9-11] A significant exception is that of VT in treating convergence insufficiency. Recent results from randomized clinical trials conducted by the Convergence Insufficiency Treatment Trial Group give increased evidence of the efficacy of visual therapy for this condition.[12-14] Until similar randomized multicenter clinical trials are published on the treatment of other visual disorders, VT will continue to be considered an "investigational intervention" according to the medical model, and thus is excluded from coverage by some health care insurance policies.[15-17]

Although scientific evidence is the primary criterion for evaluating a treatment, the theoretical basis of the treatment must also be considered. The mechanism for producing change needs to be consistent with principles of science.[18,19] Recent advances in neuroscience provide the framework for understanding the efficacy of VT, and may bridge the gap between treatment models of organic and functional disorders.[20] This review will present some current concepts on neuroplasticity, examine its therapeutic applications in neurorehabilitation, and discuss its implications in optometric VT and rehabilitation.

Neuroplasticity Concepts

Contrary to age-old assumptions that changes in neural pathways are possible only during critical periods of development, modern neuroscience views the brain as a dynamic structure throughout life. This view has its origins in Hebb's hypothesis from half a century ago that the synaptic connections between two neurons strengthen from repeated and persistent stimulation.[21] This results in learning and memory of that behavior at the level of the synapse. Throughout the past few decades, there has been an abundance of research on neuroplasticity. However, it was not until the development of non-invasive functional neuroimaging in the 1990s that the scientific community really began to embrace the concept of an adult, plastic brain.[22,23] At a microscopic level, modifications in synaptic firing, presynaptic neurotransmitter vesicle release, dendritic arborization, axonal sprouting and astrocyte activity have been identified in the adult nervous system.[24] Neurogenesis within the olfactory bulb, the dentate gyrus, and even the cerebral cortex in the adult primate has also been reported.[25-27] At a macroscopic level, there is ample evidence of behaviorally-induced and experience-dependent cortical reorganization.[28] These changes play a critical role not only in development, but also in learning, aging and the process of recovery following acquired brain injury.[29] Current thinking about cortical plasticity suggests

that there are four major types of functional neuroplastic changes demonstrated in humans: homologous area adaptation, compensatory masquerade, cross-modal reassignment and map expansion.[30]

Homologous area adaptation occurs when a damaged brain region is compensated for by transferring the function to unaffected brain regions. This type of plasticity is limited to early stages in human development, and can result in "crowding" of the new brain area. One example in the literature is a case of a 17-year-old boy who sustained a right parietal lobe injury in a motor vehicle accident when he was 7 months old.[31] Although the right parietal lobe is a structure that supports visuospatial skills, the patient had no difficulty with these skills; rather, he exhibited impaired arithmetic and language skills, which are primarily functions of the left parietal lobe. Functional MRI findings led researchers to conclude that an interhemisphere transfer of spatial function from right to left parietal lobe occurred in infancy. This transfer then led to an acquired left parietal lobe dysfunction secondary to competition for left hemisphere representation between spatial and verbal functions.[30]

Compensatory masquerade is the reorganization of existing synaptic pathways to use an alternative cognitive strategy to perform a task. This type of plasticity is harnessed when the original strategy cannot be used efficiently due to brain injury. A second, usually less efficient pathway, will then be used to perform a task. For example, instead of relying on an intuitive sense of direction to navigate between two places, a person with an acquired brain injury and impaired spatial sense can resort to another strategy for spatial navigation, such as memorizing landmarks. This is based on the assumption that the brain injury spares a process that can be used to compensate for the impairment. The increased cognitive demand in using an alternative strategy results in a change in neuronal networks.[30]

Cross-modal reassignment involves the acceptance of a new sensory input into a brain structure that has been deprived of its main input. For example, through electrophysiological and neuroimaging studies, the primary visual cortex in individuals who became blind in childhood have been shown to process somatosensory input. This indicates a shift in function from processing visual stimuli to processing tactile stimuli.[32] However, activation of the visual cortex occurs only during discrimination of meaningful geometric forms such as Braille letters, and not homogenous fields of tactile stimuli.[30] This type of plasticity has been observed in blind subjects whose onset of vision loss was before 16 years of age.[33]

Map expansion results in the enlargement of a functional cortical region in response to repeated behavior or frequent exposure to a stimulus—also termed "use-dependent cortical reorganization." For example, violinists show an enlargement of the left hand representation in their sensorimotor cortex due to greater left hand fingering movements as compared to right hand bowing movements on the violin.[34,35] Similarly, the sensorimotor cortical representation of the reading finger is expanded in blind Braille readers.[36] This form of cortical plasticity demonstrates that function can alter structure. It is present through life and is continuously modified by experience.[37]

These four types of neuroplasticity demonstrate ***equipotentiality*** and ***vicariation*** in the adult central nervous system. Equipotentiality refers to the concept that an anatomical area of the brain has the ability to perform disparate functions, while vicariation refers to the idea that redundant neural systems are able to operate under abnormal conditions. Contemporary research suggests that these mechanisms may be the basis of documented recovery for various neurological conditions, whether degenerative, psychiatric, developmental, vascular or traumatic in origin. [23]

Therapeutic Applications in Neurorehabilitation

ln the past decade, there has been a paradigm shift in neurorehabilitation due to an increased understanding of the brain's striking capacity for reorganization and recovery of function. Traditionally, the purpose of rehabilitation was to teach substitution strategies. This was because most clinicians believed that there was no possibility of recovery, regeneration or repair once a functional area in the brain was damaged.[34,38] Rehabilitation was generally only offered in the acute stages following a brain injury because it was assumed to be futile for deficits that have been present for longer than one year. Those who did not accept the precise relationship between structure and function were considered "out-of-touch with scientific reality."[23] Today, it is no longer possible to refute the numerous studies which demonstrate some degree of repair and regeneration under the appropriate clinical conditions. The current view is that it is possible to rehabilitate chronic conditions through the application of neuroplasticity principles to maximize recovery of function.

One of the most active areas of research in rehabilitation is the treatment of patients with acquired brain injury. Each year in the United States, approximately 1.4 million individuals sustain a traumatic brain injury, and 795,000 individuals experience a stroke.[39,40] Due to the aging population and improved medical care, there are many more survivors of these brain injuries, and an increased incidence of chronic neurological deficits from these conditions. A major deficit that is experienced by individuals with acquired brain injury is hemiparesis.

Since the late 1990s, a neurorehabilitation technique termed constraint-induced movement therapy, or CI therapy, has been used to treat upper extremity hemiparesis in stroke patients. CI therapy involves a two-to-three week course of repetitive training of the paretic arm along with restraint of the non-impaired arm. This is accomplished by doing up to six hours of task-oriented activities daily with the non-affected arm in a sling, or hand placed in a mitt for most waking hours. Individualized training programs are based upon the selection of increasingly difficult tasks as improvement is noted.[41]

Results from multiple well-designed randomized controlled studies indicate that CI therapy produced statistically significant and clinically relevant improvements in arm motor function that persisted for at least one year following therapy.[41,42] These improvements were not influenced by age, sex or initial level of paretic arm function, and were seen in both acute and chronic strokes. Subjects who had undergone CI therapy reported improved daily function and quality of life compared to the control groups. Additional studies indicate that improvements in neurological function corresponded with changes in the brain.[43-48] Profuse increases in gray matter in both sensory and motor areas—contralateral and ipsilateral to the affected arm—were noted, as well as bilateral increases in the hippocampus. These findings suggest that CI therapy induces use-dependent cortical reorganization to treat motor deficits in stroke patients.

The basis of CI therapy is to reverse "learned non-use"–a concept from basic somatosensory deafferentation research

with animals. This research involves surgical abolishment of a sensory nerve to determine the functional and structural adaptations that follow. Monkeys who have this procedure in one arm demonstrate disuse of the affected arm, which leads to a decrease in the brain's cortical representation of that body part. However, monkeys who are trained to consistently use the affected limb (often through forced use by tying down the non-affected limb) have a return of function to their once useless limb.[42] There is an expansion in the representational zone of that body part corresponding to the recovery of function. This finding shows the relationship between structure and function in the induction of neuroplasticity. Such studies provide crucial insight as to why the traditional physical and occupational therapy technique of encouraging the use of the unaffected arm to do the work of both arms hinders an individual's recovery of movement and function of the affected arm.[41]

In 2006, the American Stroke Association proclaimed the use of CI therapy to be "at the forefront of a revolution."[49] Due to its success, CI therapy has been expanded to treat upper and lower extremity paresis in patients affected by traumatic brain injury, spinal cord injury, hip fracture, cerebral palsy, hemisphrectomy and other neurological conditions.[50-52] The shift away from braces, assistive devices, wheelchairs and other compensatory mechanisms to training recovery of function is slowly being adopted in various clinical settings.[38]

The success of CI therapy has inspired clinical researchers to incorporate its principles into other rehabilitation modalities. One example is improving language functions through constraint-induced aphasia therapy. This program was founded on the following concepts: prevention of compensatory communication (e.g. gesturing), use of an appropriate massed-practice technique that focused on patients' communication needs, and incorporation of more difficult language tasks as patients improve.[53] Instead of a conventional six-month course of speech and language therapy, constraint-induced aphasia therapy intensifies the therapy by condensing 30 hours of therapy into two weeks. Involving the patient's support networks and using computer-based training programs at home during this period also adds to the intensity level. A pilot study that was published in 2005 showed that the majority of patients who completed the therapy had significant improvements in language functions. This was irrespective of age, severity or duration of aphasia. In addition, patients' language skills remained stable at the six-month post-therapy follow-up.[54] As a result of these findings, a recent review article concluded that it is reasonable to assume that the concepts supporting CI therapy could be applied to other cognitive impairments after neurological damage. Some of the deficits listed were attention, spatial neglect, memory and other perceptual deficits.[55] Future research will be needed to better determine what factors enhance recovery in these various disorders and how to effectively integrate basic science principles into clinical practice.

Implications in Optometric Vision Therapy & Rehabilitation

The scientific community is gradually embracing the notion that rehabilitation of motor, sensory and cognitive impairments can alter brain reorganization and result in functional recovery.[23] Therefore, the training and rehabilitation of functional visual disorders through repetitive, targeted visual rehabilitative techniques should not be a foreign concept. Based on what is now known about neuroplasticity, it can be inferred that the mechanism for the efficacy of VT and rehabilitation is through strengthening synaptic connections and inducing cortical reorganization to maximize visual efficiency. These neuroplastic changes are taking place through VT techniques such as:

- appreciating physiological diplopia on the Brock string as an anti-suppression technique
- making a motor response to a visual mismatch by localizing the clown's nose with a pointer on the vectogram
- placing pegs into the pegboard rotator while coordinating the movement to the beat of a metronome, or
- visually recalling the last parquetry block pattern in order to make the correct match.

The principles emphasized in successful VT are the same principles used by other rehabilitation specialties to maximize recovery of function through induction of neuroplasticity. These include: repetition, motivation, loading, multi-sensory integration and feedback.[56] The difference is that optometrists were utilizing many of these principles before they came into vogue and before there was repeatable empirical and clinical data to support the efficacy of the techniques. One example is the introduction of amblyopia therapy in 1743 by George L. de Buffon, who covered the better seeing eye and suggested the use of prescription glasses for the worse seeing eye.[57] These concepts were adapted by optometrists during the 20th century. Currently, amblyopia is treated by "constraining" the use of the non-amblyopia eye through occlusion, penalization or pharmacologic therapy so that the amblyopic eye can recover function.[58] Usually, near activities are prescribed to stimulate the visual pathway.[59] This is conceptually similar to CI therapy–using passive constraint in conjunction with active therapy.

Currently, the active therapy approach for amblyopia is to improve ocularmotor, accommodative, binocular and perceptual skills. This approach–part of the basic optometric regimen–is not commonly employed by some ophthalmologists who believe that it is no more advantageous than the passive approach (i.e. patching).[60] This is despite recent results from the pilot study by the Pediatric Eye Disease Investigator Group suggesting that near activities are beneficial in treating amblyopia.[61] However, confirmation of these results in a formal randomized clinical trial is needed before treatment protocols are likely to change. To gain the same level of acceptance obtained by other neurorehabilitation treatments, brain imaging research is needed.

The basic concepts of neuroplasticity provide a sound theoretical basis for the efficacy of active VT and rehabilitation. It can be deduced from cortical plasticity research that these interventions create new neural pathways in the primary and extrastriate cortices, and reduce the amount of suppression that maintains the amblyopic state. Recent neuroimaging studies with amblyopic patients show a decrease in gray matter volume in the visual cortex, the ventral temporal and parietal-occipital area. This is consistent with observations that amblyopic patients have abnormalities in spatial vision, contrast sensitivity function, vernier acuity, and impaired contour detection.[62,63] Stimulating the magnocellular and parvocellular pathways through repetitive, targeted techniques can perhaps increase the gray matter in these areas and subsequently decrease spatial and object processing deficits.

For many years, it was believed that amblyopia was only amenable to treatment during the "critical period"—up until sev-

en or eight years old—when plasticity is at its height. However, recent publications have demonstrated that effective treatment can occur at any age due to evidence of residual plasticity in the adult amblyopic visual cortex.[64] There are cases of improved visual function of the amblyopic eye in adults after central vision loss or enucleation of the good eye.[65-67] Other studies have suggested that VT and task repetition can produce substantial improvements in visual acuity and binocular vision for both children and adults.[68-70] The notion that plasticity in the visual processing of adults with amblyopia exists can be used as a basis to improve the visual function of patients who may have been previously told that it was too late for treatment.[71]

The concept that age is not a contraindication in amblyopia therapy can be applied to other functional vision problems, such as oculomotor dysfunction, strabismus, non-strabismic binocular vision disorders and visual-perceptual deficits. Dr. Oliver Sacks brought this connection between VT and neurorehabilitation at any stage of life to national attention in his award-winning article from the June 19, 2006 issue of *The New Yorker*.[72] His article details how, at the age of 48, Susan Barry ("Stereo Sue"), who had three strabismus surgeries as a child, developed stereoscopic depth perception for the first time while participating in an optometric VT program. As a neurobiologist, this experience was particularly enlightening for Dr. Barry and forced her to reexamine the subject of adult neuronal plasticity and rehabilitation; she no longer teaches her neurobiology students the conventional wisdom that the human brain is only malleable during the critical period and loses the capacity for reorganization in adulthood.[73] Examples like this illustrate that age is not the limiting factor to success in therapy. Depending on the diagnosis, the age of the patient may point to a guarded prognosis with treatment, but the idea of training an older individual is no longer deemed inconceivable.

Perhaps what is more important than age of the patient is the patient's attentional ability during therapy. Recent research has shown that attention plays a critical role in modulating change.[74] When subjects attend to the sensation of fingertip vibrotactile stimulation, increased activation on functional imaging is seen in the primary somatosensory cortex. This is as opposed to when they do not attend to the stimulus.[75] These results suggest that the physiological response of a primary cortical area can be modified through active attention to the relevant task or stimulus. Additionally, these findings explain why there is poorer functional recovery in subjects whose attention circuits in the frontal lobe were damaged from a brain injury.[34] Nevertheless, a frontal lobe injury should not be viewed as a contraindication in therapy. This is because neuroplastic changes can still take place through vicariation mechanisms, as there is no single attention center in the brain. Even still, the patient must demonstrate motivation throughout the rehabilitation process to produce the attentional behavior needed to induce cortical plasticity. This can be applied to the amelioration of vision dysfunctions. It serves as a reminder that varying the therapeutic techniques and increasing the level of difficulty as patients improve are important methods to maintain the brain's attention. This effectively strengthens the synaptic pathways and leads to the best outcomes after therapy. The role of attention may help explain why an individual who attempts unstructured and relatively unattended activation of sensorimotor and perceptual systems through unsupervised home therapy (e.g. pencil push-ups) does not achieve meaningful improvement in their visual symptoms. This provides further basis as to the efficacy of an optometric VT program that emphasizes an awareness of the process.

Conclusion

The principles revealed by neuroplasticity research can be applied to virtually all aspects of optometric VT and rehabilitation**.** Recent advances in neuroscience provide a theoretical foundation for why VT works and how age-old VT techniques tap into neuroplasticity to ameliorate functional visual problems.

Since approximately 70% of all sensory input fibers to the brain is related to vision and visual processing, optometrists can play a major role in the rehabilitation of visually-related deficits.[76,77] By emphasizing awareness of the process, feeling tones, visualization, feedback, intersensory integration, problem solving and repetition, a comprehensive learning experience is created to maintain long-lasting functional improvement. Future research should include additional randomized clinical trials to confirm the efficacy of the treatment of various functional visual disorders. In addition, non-invasive neuroimaging should be obtained to assess the changes in neural architecture. In time, cortical plasticity research may play a role in further validating a treatment modality that has improved the visual efficiency of countless patients.

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