Chapter-2.

DIPLOPIA AND CORTICAL SUPPRESSION MECHANISMS

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ABSTRACT

Diplopia, commonly known as double vision, is one of the most debilitating symptoms encountered in neuro-ophthalmology, arising from the inability of the visual system to fuse images from both eyes into a single, coherent perception. It is most frequently caused by ocular misalignment resulting from cranial nerve palsies, extraocular muscle disorders, or neurological diseases, although abnormalities in cortical mechanisms responsible for binocular fusion may also contribute. To reduce the visual confusion associated with persistent diplopia, the brain often initiates cortical suppression, an adaptive neurophysiological process that selectively inhibits visual input from one eye. While this compensatory mechanism alleviates the immediate perception of double vision, prolonged suppression can impair binocular vision, reduce stereoscopic depth perception, and lead to long-term sensory adaptations such as amblyopia or persistent binocular dysfunction. This chapter provides a comprehensive overview of the mechanisms underlying diplopia and cortical suppression, emphasizing their neurophysiological basis, clinical manifestations, diagnostic evaluation, and current therapeutic strategies, while highlighting the critical role of cortical plasticity in preserving and restoring visual function.

Keywords: Diplopia, Cortical Suppression, Binocular Vision, Neuro-Ophthalmology.

2.1 INTRODUCTION

Human vision is fundamentally binocular, relying on the coordinated function of both eyes and the central nervous system to produce a single, clear, and three-dimensional perception of the surrounding environment. Under normal physiological conditions, images formed on corresponding retinal points are transmitted through the visual pathways and integrated within the visual cortex, where sensory fusion occurs. This precise neural integration enables stereopsis, accurate depth perception, and efficient spatial orientation, all of which are essential for daily activities such as reading, driving, maintaining balance, and performing fine motor tasks. Diplopia, commonly known as double vision, occurs when this finely coordinated binocular system fails to fuse the visual inputs from both eyes into a single image. Instead of perceiving one object, the individual sees two separate images, resulting in visual confusion, impaired coordination, and reduced quality of life. Diplopia may be transient or persistent and can arise from abnormalities affecting the ocular motor system, orbital structures, cranial nerves, neuromuscular junction, or higher cortical centres involved in binocular processing. The most common cause of diplopia is ocular misalignment resulting from disorders of the extraocular muscles or the cranial nerves controlling eye movements. However, successful binocular vision depends not only on proper ocular alignment but also on intact cortical mechanisms responsible for sensory fusion. Consequently, lesions involving the visual cortex or higher-order visual pathways may also produce diplopia despite relatively preserved ocular motility. To minimize the disturbing perception of double vision, the brain frequently initiates an adaptive mechanism known as cortical suppression. Through selective inhibition of visual information from one eye, suppression allows the visual system to maintain a single percept despite persistent ocular misalignment. Although this adaptation provides immediate symptomatic relief, prolonged suppression compromises binocular vision, reduces stereoscopic depth perception, and may result in

long-term sensory abnormalities such as amblyopia or anomalous retinal correspondence. Understanding the interaction between ocular motor dysfunction and cortical adaptation is fundamental to modern neuro-optometry. Advances in neuroimaging, functional magnetic resonance imaging (fMRI), electrophysiology, artificial intelligence, and binocular vision rehabilitation have significantly expanded our understanding of cortical plasticity and its role in visual recovery. Consequently, contemporary management increasingly focuses not only on correcting ocular alignment but also on restoring binocular visual function through targeted rehabilitation strategies.

2.2 PATHOPHYSIOLOGY OF DIPLOPIA

Diplopia develops when the normal mechanisms responsible for binocular vision fail to combine the images received from both eyes into a single, unified perception. Under physiological conditions, each eye captures a slightly different image of the same object due to their horizontal separation. These small retinal disparities are processed by the visual cortex and fused into a single image, enabling binocular vision, stereopsis, and accurate depth perception (Howard & Rogers, 2012). This process depends on precise ocular alignment, coordinated extraocular muscle function, intact neural pathways, and efficient cortical sensory fusion. When any component of this complex visual system is disrupted, binocular correspondence is lost, resulting in the perception of two separate images, a condition known as diplopia (von Noorden & Campos, 2002). The most common mechanism responsible for diplopia is ocular misalignment, in which the visual axes of the two eyes are no longer directed toward the same target. When the degree of ocular deviation exceeds the individual's fusional capacity, the brain is unable to merge the two retinal images, leading to persistent double vision. The direction of image separation depends on the pattern of ocular deviation and may be horizontal, vertical, oblique, or torsional. In many patients, the severity of diplopia varies with gaze direction, fixation distance, and the underlying pathology affecting ocular motility. Disorders affecting the ocular motor system represent the leading causes of binocular diplopia. Paralysis or weakness of the oculomotor (cranial nerve III), trochlear (cranial nerve IV), or abducens (cranial nerve VI) nerves disrupts the coordinated movement of the extraocular muscles, producing incomitant strabismus in which the magnitude of ocular deviation changes with different directions of gaze (Demer, 2015). Oculomotor nerve palsy may present with ptosis, pupillary abnormalities, and limitations in multiple directions of eye movement, whereas trochlear nerve palsy typically causes vertical and torsional diplopia that worsens during downward gaze. Abducens nerve palsy primarily results in horizontal diplopia due to impaired lateral rectus muscle function, with symptoms becoming more pronounced when looking toward the affected side. Neurological disorders involving the brainstem can also interfere with normal ocular coordination. Internuclear ophthalmoplegia (INO), commonly associated with multiple sclerosis in younger patients and ischemic stroke in older individuals, results from damage to the medial longitudinal fasciculus (MLF), the neural pathway responsible for coordinating conjugate horizontal eye movements. Lesions affecting this pathway impair communication between the abducens and oculomotor nuclei, producing impaired adduction of one eye, abducting nystagmus of the contralateral eye, and horizontal diplopia (Demer, 2015). In addition to neurogenic causes, restrictive orbital disorders can mechanically limit ocular motility and disrupt binocular alignment. Conditions such as thyroid eye disease, orbital fractures, extraocular muscle fibrosis, inflammatory orbital disease, and orbital tumors restrict normal eye movements, preventing the eyes from maintaining coordinated fixation. These mechanical restrictions frequently result in gaze-dependent diplopia and are often accompanied by pain, proptosis, eyelid abnormalities, or orbital swelling (von Noorden & Campos, 2002).

Although ocular motor abnormalities account for the majority of cases, diplopia is not solely a consequence of defective eye movements. Supranuclear and cortical mechanisms also play an important role in maintaining binocular vision. Even when ocular alignment remains relatively normal, lesions involving the occipital cortex, parietal cortex, or higher-order visual association areas may impair sensory fusion and binocular integration, resulting in persistent diplopia (Bridge et al., 2008). These cortical regions are responsible for integrating visual information from both eyes and constructing a unified perception of the external environment. Damage to these pathways disrupts the normal processing of binocular disparity and prevents effective cortical fusion. Higher-order visual processing within the dorsal ("where") and ventral ("what") visual pathways further contributes to stable binocular perception. The dorsal visual stream processes spatial localization, motion perception, and visuomotor coordination, whereas the ventral stream is involved in object recognition and visual identification. Dysfunction within either pathway can impair binocular integration despite normal retinal image formation and ocular alignment,

highlighting the important contribution of cortical processing to normal visual perception (Hess et al., 2014). Emerging evidence from functional neuroimaging and visual neuroscience has demonstrated that diplopia represents a complex interaction between peripheral ocular motor dysfunction and central sensory processing. Alterations in cortical excitability, binocular rivalry, and neural plasticity influence the brain's ability to adapt to ocular misalignment. Consequently, diplopia should be regarded not simply as a mechanical disorder of eye alignment but as a multifactorial neuro-ophthalmic condition involving coordinated abnormalities of ocular motor control, sensory fusion, and cortical visual processing. Understanding these mechanisms provides the foundation for accurate diagnosis and the development of targeted therapeutic strategies aimed at restoring binocular vision rather than merely eliminating the symptom of double vision.

2.3 CORTICAL SUPPRESSION MECHANISM

Cortical suppression is an adaptive neurophysiological response employed by the visual system to eliminate the disturbing perception of double vision when normal binocular fusion cannot be achieved. Rather than continuously perceiving two conflicting images, the brain selectively suppresses the visual information originating from one eye, thereby allowing a single, stable image to be perceived. This compensatory mechanism is particularly important in patients with persistent ocular misalignment, where sensory fusion is no longer possible. Although cortical suppression effectively relieves the immediate symptoms of diplopia, prolonged suppression may compromise binocular vision, reduce stereoscopic depth perception, and, in severe cases, contribute to permanent sensory deficits such as amblyopia (Schmidt & Kommerell, 1994; Harrad, 2006). The primary objective of cortical suppression is to maintain perceptual stability by resolving the sensory conflict created by misaligned retinal images.

Instead of attempting to fuse two incompatible images, the visual cortex temporarily inhibits neural signals from the eye producing the conflicting image. This selective inhibition enables the individual to function without constant visual confusion, although it comes at the expense of normal binocular integration. The extent and duration of suppression vary depending on several factors, including the patient's age, duration of ocular deviation, degree of ocular misalignment, underlying neurological condition, and the capacity of the visual system for cortical adaptation.

A. Neurobiological Basis of Cortical Suppression

The neurobiological mechanisms underlying cortical suppression involve complex inhibitory interactions within the central visual pathways. Experimental studies using electrophysiology, functional magnetic resonance imaging (fMRI), and magnetoencephalography (MEG) have demonstrated that suppression primarily originates within the primary visual cortex (V1), where binocular information from both eyes is first integrated (Harrad, 2006; Thompson & Hess, 2015). Under normal conditions, neurons within V1 receive balanced input from both eyes and combine these signals to produce a unified binocular percept. However, when persistent retinal disparity exceeds the brain's capacity for sensory fusion, inhibitory neural circuits selectively reduce the responsiveness of neurons receiving input from the affected eye. This inhibitory process extends beyond the primary visual cortex into higher-order visual association areas, including V2, V3, V4, and the extrastriate cortex, where more complex visual attributes such as shape, motion, contrast, colour, and depth perception are processed (Hess et al., 2014). Consequently, suppression influences not only image perception but also several higher-order visual functions that depend on coordinated binocular input. Functional neuroimaging studies have consistently demonstrated reduced cortical activation in regions corresponding to the suppressed eye, while electrophysiological investigations reveal altered neural synchrony and delayed interocular signal transmission, further supporting the role of cortical inhibition in suppressing conflicting visual information (Bridge et al., 2008). Recent advances in neuroscience have also highlighted the contribution of neuroplasticity to cortical suppression. The visual cortex possesses the remarkable ability to reorganize neural networks in response to persistent sensory conflict. Through adaptive synaptic modifications and changes in cortical excitability, the brain gradually learns to suppress the conflicting retinal image, thereby restoring a stable visual experience despite continued ocular misalignment. This adaptive capacity explains why some patients experience spontaneous reduction of diplopia over time, whereas others continue to suffer persistent double vision depending on the extent of cortical reorganization.

B. Retinotopic Specificity of Suppression

Cortical suppression is not a generalized inhibition of vision from one eye but rather a highly localized

and retinotopically organized process. Instead of suppressing the entire visual field of the affected eye, the brain selectively inhibits only those retinal regions that generate conflicting images. These localized areas of reduced visual sensitivity are known as suppression scotomas and correspond precisely to the retinal locations responsible for diplopia (Schmidt & Kommerell, 1994). This spatial selectivity allows unaffected regions of the retina to continue contributing to visual perception while minimizing sensory conflict.

The size, location, and intensity of suppression scotomas are not fixed and may change according to gaze direction, fixation distance, attentional demands, illumination, and the surrounding visual environment. During different visual tasks, the cortex dynamically adjusts the extent of suppression to optimize visual performance. Such flexibility reflects the highly adaptive nature of the binocular visual system and its ability to balance visual clarity with binocular function. The characteristics of suppression differ considerably between children and adults because of differences in cortical plasticity. In individuals who develop strabismus during early childhood, suppression often becomes broad, stable, and deeply established.

Since the immature visual cortex is highly plastic, prolonged suppression during critical periods of visual development may interfere with normal binocular maturation, resulting in amblyopia, loss of stereopsis, and permanent binocular vision deficits (Holmes & Clarke, 2006; Hess et al., 2014). Conversely, adults who develop acquired diplopia generally retain a more mature binocular visual system. Their suppression is usually partial, intermittent, and less extensive because adult cortical plasticity is comparatively limited (Levi et al., 2015). As a result, adults are more likely to remain symptomatic with persistent diplopia, although targeted rehabilitation and vision therapy can often enhance cortical adaptation and improve binocular function.

Overall, cortical suppression represents a sophisticated adaptive mechanism that reflects the remarkable capacity of the visual cortex to preserve perceptual stability in the presence of ocular misalignment. While it provides immediate relief from double vision, prolonged suppression may compromise binocular visual function. Understanding the neural basis and retinotopic organization of suppression is therefore essential for developing effective diagnostic strategies and individualized neuro-optometric rehabilitation aimed at restoring normal binocular vision rather than simply eliminating the symptom of diplopia.

C. Developmental and Adaptive Considerations

The capacity of the visual system to adapt to diplopia varies considerably with age, reflecting differences in cortical plasticity and the stage of visual development. During early childhood, the visual cortex possesses a remarkable ability to reorganize in response to abnormal binocular input. When persistent ocular misalignment occurs during this critical period of visual maturation, the brain often develops cortical suppression as a protective adaptation to eliminate the confusing perception of double vision. By selectively suppressing the image from the deviating eye, the developing visual system maintains a single visual percept and minimizes sensory conflict. Although this adaptive response effectively prevents persistent diplopia, it also interferes with normal binocular visual development. Prolonged suppression during childhood may lead to reduced stereopsis, impaired binocular integration, and the development of amblyopia, particularly if appropriate intervention is delayed (Holmes & Clarke, 2006).

The immature visual system demonstrates a high degree of neuroplasticity, allowing extensive cortical reorganization in response to altered visual experience. While this flexibility enables adaptation to ocular misalignment, it may also establish long-lasting sensory changes that become increasingly difficult to reverse in later life. Consequently, early diagnosis and timely management of childhood strabismus and diplopia are essential to preserve binocular vision and prevent permanent sensory deficits. The adult visual system exhibits considerably reduced cortical plasticity. Adults who develop diplopia secondary to cranial nerve palsies, neurological disorders, trauma, or orbital disease generally experience persistent awareness of double vision because the mature visual cortex has a limited capacity to establish complete suppression. Instead, suppression in adults is often partial, intermittent, and highly adaptable, providing temporary relief without completely eliminating binocular input (Bavelier et al., 2010).

Although this limited suppression helps preserve stereoscopic vision and binocular function, it also leaves many patients symptomatic, particularly during visually demanding activities such as reading, driving, or prolonged near work. The differences between childhood and adult adaptation highlight the delicate balance between maintaining visual comfort and preserving normal binocular function. Understanding these developmental variations is

fundamental for selecting age-appropriate therapeutic strategies and optimizing long-term visual rehabilitation.

2.4. CLINICAL MANIFESTATIONS

The clinical presentation of diplopia varies depending on the underlying cause, severity of ocular misalignment, duration of the disorder, and the effectiveness of cortical adaptation. Patients typically describe seeing two images of a single object that may be separated horizontally, vertically, obliquely, or torsionally, depending on the affected extraocular muscle or cranial nerve (von Noorden & Campos, 2002). Symptoms may worsen in specific directions of gaze, during prolonged visual tasks, or when viewing distant or near objects. Many individuals also experience blurred vision, eye strain, headaches, dizziness, impaired balance, and difficulty judging distances, all of which significantly affect daily activities and quality of life. The extent of symptoms largely depends on the brain's ability to compensate through cortical suppression. Partial suppression often provides intermittent relief from double vision but may result in fluctuating visual clarity, reduced contrast sensitivity, decreased reading speed, and increased visual fatigue, particularly under low-light conditions or during prolonged concentration (Hess et al., 2014). Patients frequently report intermittent diplopia that becomes more pronounced with fatigue or reduced attention.

Ccomplete cortical suppression effectively eliminates the perception of double vision by functionally inhibiting visual input from one eye. While this adaptation restores single vision, it comes at the cost of binocular function, leading to loss of stereopsis, impaired depth perception, and reduced spatial awareness (Schmidt & Kommerell, 1994). Such individuals often rely predominantly on monocular visual cues for navigation and object localization. In patients with longstanding childhood strabismus, the brain may develop Anomalous Retinal Correspondence (ARC), a complex sensory adaptation in which non-corresponding retinal points are neurologically remapped to facilitate binocular single vision despite persistent ocular deviation. Although ARC reduces the perception of diplopia and allows some degree of binocular cooperation, it represents a deeply established cortical adaptation that may complicate subsequent orthoptic rehabilitation or surgical correction of strabismus (Burian & von Noorden, 1974). The diversity of clinical manifestations reflects the complex interaction between ocular motor abnormalities, cortical sensory adaptation, and the brain's capacity for neuroplastic reorganization.

2.5. DIAGNOSTIC APPROACHES

The accurate diagnosis of diplopia and cortical suppression requires a comprehensive evaluation that combines detailed clinical history, ocular examination, binocular vision assessment, psychophysical testing, neuroimaging, and electrophysiological investigations. A systematic diagnostic approach not only identifies the underlying cause of diplopia but also evaluates the degree of sensory adaptation and cortical involvement, enabling individualized treatment planning. Clinical examination begins with assessment of ocular alignment, extraocular muscle function, head posture, and binocular visual performance. The cover-uncover test and alternate cover test remain the gold-standard clinical methods for detecting both manifest and latent strabismus by evaluating ocular deviation under dissociated viewing conditions (von Noorden & Campos, 2002). Measurement of ocular deviation using prism cover testing further quantifies the magnitude of misalignment. The Maddox rod test is particularly valuable for determining the direction, magnitude, and characteristics of diplopic image separation, assisting in localization of the affected ocular motor pathway. Assessment of sensory adaptation is equally important. Worth Four-Dot Testing and the Bagolini Striated Lens Test evaluate binocular fusion and cortical suppression under conditions that closely resemble natural viewing. These tests help differentiate between normal binocular vision, suppression, and diplopia while assessing the extent of sensory adaptation. The synoptophore provides detailed quantitative analysis of binocular vision by measuring fusion ranges, mapping suppression scotomas, and identifying Anomalous Retinal Correspondence (ARC), making it an essential instrument in orthoptic assessment (Burian & von Noorden, 1974).

Electrophysiological investigations provide objective evidence of cortical function. Visual Evoked Potentials (VEPs) frequently demonstrate reduced response amplitudes or prolonged latencies from the suppressed eye, reflecting diminished cortical responsiveness resulting from interocular inhibition (Harrad, 2006). Advances in neuroimaging have further enhanced understanding of cortical suppression. Functional Magnetic Resonance Imaging (fMRI) demonstrates decreased activation within the primary visual cortex and extrastriate visual areas corresponding to the suppressed eye, while magnetoencephalography (MEG) reveals altered neural synchrony and

disrupted binocular signal integration (Bridge et al., 2008). Together, these diagnostic techniques provide a comprehensive assessment of both ocular motor dysfunction and cortical sensory processing, facilitating accurate diagnosis and appropriate management.

2.6 MANAGEMENT STRATEGIES

The management of diplopia aims not only to relieve the distressing symptom of double vision but also to preserve or restore normal binocular vision whenever possible. Successful treatment requires careful identification of the underlying cause, assessment of cortical adaptation, and selection of individualized therapeutic strategies that address both motor and sensory components of the disorder. Prismatic correction remains one of the most commonly used non-surgical treatments for symptomatic diplopia. Prisms alter the direction of incoming light, shifting the retinal image into the patient's fusional range and allowing the visual cortex to merge images from both eyes into a single percept (von Noorden & Campos, 2002). Fresnel prisms, which are lightweight and removable, are particularly useful in acute or fluctuating ocular deviations, whereas ground-in prisms are generally prescribed for stable, long-term deviations requiring permanent correction. When prism therapy is ineffective or contraindicated, occlusion therapy may be employed to eliminate the conflicting image. Complete occlusion using an eye patch or opaque lens immediately abolishes diplopia but sacrifices binocular vision and stereopsis. Consequently, partial occlusion techniques, including Bangerter filters, frosted lenses, or translucent occlusive films, are often preferred because they reduce sensory conflict while preserving peripheral awareness and some degree of binocular function (Schmidt & Kommerell, 1994).

Modern neuro-optometric management increasingly emphasizes binocular rehabilitation rather than prolonged suppression. Contemporary vision therapy utilizes evidence-based techniques such as dichoptic training, perceptual learning, computerized binocular exercises, and virtual reality-based rehabilitation to reduce cortical suppression, strengthen binocular fusion, improve vergence control, and restore stereoscopic vision (Polat et al., 2004; Hess et al., 2014). These therapies exploit the principles of neuroplasticity by encouraging balanced stimulation of both eyes and promoting reorganization within the binocular visual cortex. In patients with persistent ocular misalignment resulting from cranial nerve palsy, restrictive strabismus, or congenital ocular motor disorders, strabismus surgery may be required to restore anatomical alignment of the eyes. Surgical correction improves ocular positioning and expands the potential for binocular fusion; however, optimal functional recovery often depends on postoperative orthoptic rehabilitation to prevent recurrence of cortical suppression and facilitate restoration of normal binocular visual processing (von Noorden & Campos, 2002). The integration of optical correction, neurorehabilitation, and surgical management therefore provides a comprehensive approach that addresses both the motor and sensory components of diplopia, ultimately improving visual function and patient quality of life.

2.7 FUTURE DIRECTIONS

The future of research and clinical management of diplopia is increasingly focused on restoring normal binocular vision by harnessing the brain's inherent capacity for neuroplasticity. Rather than relying solely on compensatory strategies such as cortical suppression or optical correction, contemporary approaches aim to reactivate suppressed visual pathways, strengthen binocular integration, and promote long-term functional recovery. Advances in neuroscience, neuroimaging, artificial intelligence, virtual reality, and non-invasive brain stimulation are reshaping the understanding of cortical adaptation and opening new possibilities for personalized neuro-optometric rehabilitation. One promising area of investigation involves non-invasive brain stimulation (NIBS) techniques, including Transcranial Magnetic Stimulation (TMS) and Transcranial Direct Current Stimulation (tDCS). These techniques modulate cortical excitability by delivering controlled magnetic or electrical stimulation to specific regions of the visual cortex. Preliminary studies suggest that combining brain stimulation with conventional binocular vision therapy may enhance cortical plasticity, reduce interocular suppression, improve sensory fusion, and accelerate visual rehabilitation in selected patients with persistent diplopia (Bavelier et al., 2010). Although these methods remain largely investigational, they represent a promising adjunct to conventional neuro-optometric treatment.

Virtual Reality (VR) and Augmented Reality (AR) technologies are also emerging as innovative tools for binocular rehabilitation. These immersive environments enable precise control of binocular disparity, vergence demand, contrast balance, and visual stimuli, allowing clinicians to design individualized therapeutic exercises that gradually stimulate suppressed cortical pathways. By providing engaging and interactive rehabilitation

environments, VR-based therapy has shown encouraging results in improving binocular fusion, stereopsis, vergence control, and visual comfort in patients with diplopia and other binocular vision disorders. The integration of eye-tracking technology further allows continuous monitoring of ocular alignment and treatment progress during rehabilitation. Recent advances in artificial intelligence (AI) and computational neuroscience are further transforming the management of diplopia. Machine learning algorithms can analyze complex clinical, imaging, and eye movement data to identify patterns of ocular misalignment, predict treatment outcomes, and recommend personalized rehabilitation protocols. Computational models of binocular vision are enhancing our understanding of cortical sensory integration and enabling the development of adaptive therapeutic strategies tailored to each patient's unique visual deficits. Such precision-based approaches have the potential to improve treatment efficacy while reducing rehabilitation time.

Another rapidly evolving field is neuropharmacology, where researchers are investigating pharmacological agents capable of temporarily modulating cortical excitability and synaptic plasticity. When combined with structured visual training, these agents may enhance the brain's capacity for binocular reintegration by facilitating synaptic remodeling within the visual cortex. Although clinical evidence remains limited, future pharmacological interventions may complement existing rehabilitation strategies and improve recovery in patients with chronic suppression or longstanding binocular dysfunction. Advances in functional neuroimaging, including functional magnetic resonance imaging (fMRI), diffusion tensor imaging (DTI), and magnetoencephalography (MEG), continue to improve understanding of the neural mechanisms underlying binocular vision and cortical suppression. These technologies provide valuable insights into cortical reorganization, allowing clinicians to monitor treatment-induced changes in neural activity and objectively evaluate rehabilitation outcomes. As imaging techniques become increasingly sophisticated, they are expected to play an important role in developing individualized treatment strategies based on each patient's pattern of cortical adaptation. Collectively, these innovations represent a paradigm shift in the management of diplopia. The emphasis is gradually moving away from merely suppressing symptoms toward actively restoring normal binocular function through targeted neurorehabilitation. Continued interdisciplinary collaboration among neuro-ophthalmologists, optometrists, neuroscientists, biomedical engineers, and rehabilitation specialists will be essential to translate these emerging technologies into routine clinical practice and further improve visual outcomes for patients with binocular vision disorders.

2.8 CONCLUSION

Diplopia is a complex neuro-ophthalmic disorder resulting from disruption of ocular alignment, binocular fusion, or cortical visual processing. Cortical suppression serves as an adaptive mechanism to eliminate double vision by inhibiting conflicting visual input; however, prolonged suppression may impair stereopsis and binocular function. Advances in neuroimaging, vision therapy, neuro-optometric rehabilitation, and emerging technologies such as artificial intelligence, virtual reality, and non-invasive brain stimulation are transforming the management of diplopia by promoting cortical plasticity and binocular restoration. Continued research into the neural mechanisms of binocular vision will further enhance personalized therapeutic strategies and improve visual outcomes and quality of life for affected patients.

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