Chapter-12.

SUPRANUCLEAR CONTROL OF EYE MOVEMENTS

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ABSTRACT

Supranuclear control of eye movements refers to the higher-order neural mechanisms that coordinate voluntary and reflexive ocular movements by integrating cortical, subcortical, cerebellar, and brainstem networks. Unlike the ocular motor nuclei, supranuclear pathways regulate the planning, initiation, accuracy, and adaptation of saccades, smooth pursuit, vergence, fixation, and vestibulo-ocular reflexes. Key structures involved include the frontal and parietal eye fields, supplementary eye fields, superior colliculus, basal ganglia, cerebellum, and associated brainstem gaze centers, which work together to ensure precise binocular alignment and stable visual perception. Dysfunction of these pathways results in characteristic supranuclear gaze disorders, including gaze palsies, impaired saccades, pursuit deficits, and abnormalities of fixation, commonly observed in stroke, Parkinson's disease, progressive supranuclear palsy, multiple sclerosis, and traumatic brain injury. Understanding the neuroanatomy and physiology of supranuclear ocular motor control is fundamental for accurate neurological localization, clinical diagnosis, and the development of targeted neuro-optometric and neurorehabilitation strategies.

Keywords: Supranuclear control; Eye movements; Ocular motor system; Neuro-optometry.

12.1 INTRODUCTION

The supranuclear control of ocular movement encompasses the higher-order mechanisms by which the brain recruits, directs, and regulates ocular motor activity beyond the direct action of cranial nerve nuclei. While the ocular motor nuclei (3rd, 4th, and 6th cranial nerves) and their infranuclear pathways implement contraction of the extraocular muscles (EOM), supranuclear centres are responsible for the timing, planning, and coordination of movement in accordance with sensory input, cognitive demand, and spatial awareness (Leigh & Zee, 2015). These higher-order centres ensure that eye movements are synchronized, purposeful, and adaptable to environmental and behavioural context. Interruption of supranuclear pathways produces a wide range of clinically distinct ocular motor syndromes, highlighting their importance in neuro-optometry and clinical neuroscience (Leigh & Tomsak, 2005).

Cortical Control of Eye Movements: The cerebral cortex provides the primary initiation and volitional drive for ocular movements. The frontal eye fields (FEF), located in the prefrontal cortex, play a central role in generating voluntary saccades, primarily directing gaze contralaterally (Pierrot-Deseilligny et al., 1995). Damage to the FEF characteristically results in impaired initiation of saccades towards the opposite visual field. In contrast, the parietal eye fields are chiefly involved in reflexive and stimulus-driven saccades, integrating sensory input with spatial attention to rapidly reorient gaze (Pierrot-Deseilligny et al., 2003). The supplementary eye fields contribute to more complex functions such as memory-guided saccades and sequences of ocular movements required for tasks involving prediction and motor planning. Furthermore, the occipital cortex provides critical visual sensory input to guide smooth pursuits, ensuring accurate tracking of moving stimuli (Krauzlis, 2004).

Subcortical Structures and Integration: Underneath the cortical level, subcortical structures serve as crucial relay and integration centres. The superior colliculus (SC) integrates multimodal sensory inputs including auditory, visual, and somatosensory signals to generate reflexive

orienting saccades and rapid gaze shifts (Wurtz & Albano, 1980). The SC cooperates closely with cortical centres to mediate both voluntary and involuntary ocular movements. The basal ganglia deliver inhibitory control, preventing inappropriate saccades while enabling intended ones, and their dysfunction in conditions like Parkinson's disease results in hypometric, slowed, or absent saccades (Hikosaka et al., 2000). The thalamus functions as a relay hub, communicating cortical signals to the brainstem and superior colliculus, facilitating feedback mechanisms that fine-tune motor accuracy.

12.2 CEREBELLAR MODULATION OF EYE MOVEMENTS

The cerebellum is crucial for adaptive control, error correction, and fine-tuning of ocular motor behaviour (Ito, 2011). The flocculus and paraflocculus are important for smooth pursuit and for modulating the Vestibulo-ocular reflex (VOR), while the vermis and fastigial nuclei contribute to the accuracy and amplitude of saccades. Lesions in these areas lead to dysmetria saccades that overshoot or undershoot targets as well as gaze-evoked nystagmus and impaired pursuit movements (Robinson, 1981). The role of the cerebellum in recalibration ensures that ocular movements remain precise when visual demands change, highlighting its role in neural plasticity.

12.3 TYPES OF EYE MOVEMENTS GOVERNED BY SUPRANUCLEAR SYSTEMS

A. Saccades: Saccades are rapid ballistic movements that move the fovea onto objects of interest. Supranuclear pathways coordinate their amplitude, initiation, and direction. Cortical centres, mainly the FEF and SC, initiate saccades, while the cerebellum ensures accuracy (Leigh & Zee, 2015). Lesions affecting these centres result in delayed, hypometric, or absent saccades.

B. Smooth Pursuits: Smooth pursuit movements allow the eyes to maintain fixation on moving targets. These are controlled by cortical–cerebellar circuits, primarily involving the occipital cortex, FEF, and cerebellar flocculus (Krauzlis, 2004). Lesions produce broken or saccadic pursuits, causing difficulty in tracking dynamic objects.

C. Vergence: Vergence ocular movements align the eyes for near or distance targets. The supranuclear pathways for vergence involve midbrain structures near the oculomotor nucleus, modulated by cortical visual input (Shaikh et al., 2016). Dysfunction leads to convergence insufficiency or divergence paralysis, impairing depth perception (Das & Leigh, 2000).

D. Vestibulo-Ocular Reflex (VOR) Adaptation Although the VOR originates in the vestibular nuclei, its supranuclear control via cerebellar circuits ensures adaptation to changes in head movements and visual feedback (Ito, 2011). Lesions impairing this adaptation result in oscillopsia and imbalance.

12.4 CLINICAL MANIFESTATIONS OF SUPRANUCLEAR LESIONS

Lesions disrupting supranuclear pathways produce distinct clinical features that differ fundamentally from those caused by nuclear or infranuclear ocular motor dysfunction (Strupp & Kremmyda, 2013). Unlike direct cranial nerve palsies, supranuclear lesions often manifest as selective impairments in specific classes of ocular movements—saccades, pursuits, or vergence—while sparing others. This dissociation occurs because supranuclear centres are responsible for programming and coordinating motor commands rather than executing them (Leigh & Zee, 2015). Consequently, patients may retain basic ocular motility yet demonstrate profound deficits in the higher-order regulation of gaze. The clinical presentations differ according to the anatomical site of the lesion, offering powerful diagnostic clues for localizing neurological disease.

12.5 VOLUNTARY VS. REFLEXIVE DISSOCIATION

A hallmark of supranuclear ocular motor disorders is the dissociation between voluntary and reflexive movements. For instance, patients with frontal lobe lesions often cannot execute voluntary saccades towards the contralateral visual field but may still perform reflexive saccades to sudden stimuli (Pierrot-Deseilligny et al., 1995). Correspondingly, in vertical supranuclear palsy, voluntary upward or downgaze is reduced, yet the VOR may remain intact. This preservation of reflexive responses highlights that supranuclear pathways are disrupted, while brainstem and infranuclear circuits remain functional.

Saccadic Abnormalities: Supranuclear lesions often alter saccadic eye movements. Frontal eye field lesions cause delayed initiation of contralateral saccades, resulting in gaze deviation towards the side of the lesion in acute stages. Parietal lobe lesions impair reflexive and attentional saccades, often accompanied by hemispatial neglect (Pierrot-Deseilligny et al., 2003). Lesions of the superior colliculus may lead to a deficiency in express saccades and impaired reorienting to visual stimuli (Wurtz & Albano, 1980). Cerebellar dysfunction produces dysmetric saccades, manifesting as overshooting or undershooting of targets (Robinson,

1981). These anomalies significantly impair activities requiring rapid gaze shifts, like reading or scanning the environment.

Smooth Pursuit Deficits: Smooth pursuit movements are particularly susceptible to supranuclear damage. Lesions of the parieto-occipital junction result in impaired ipsilateral pursuit, producing broken, saccadic tracking instead of smooth ocular movements (Krauzlis, 2004). Cerebellar flocculus damage worsens pursuit deficiency by reducing gain, leading to lagging ocular movements relative to the moving stimulus. Clinically, patients often complain of difficulty following moving objects, blurred vision during dynamic activities, and visual fatigue.

Vergence Disorders: Vergence movements depend on supranuclear circuits within the midbrain, combined with cortical input (Shaikh et al., 2016). Lesions involving the pretectal region may cause convergence retraction nystagmus and impaired convergence, as seen in Parinaud's syndrome. Patients present with diplopia at near, asthenopia, and impaired depth perception. In some cases, divergence palsy occurs, leading to sustained esotropia and binocular diplopia at distance (Das & Leigh, 2000).

Vertical Gaze Palsy: Vertical supranuclear gaze palsy is a trademark of progressive supranuclear palsy (PSP) and midbrain lesions (Boxer et al., 2017). In PSP, patients initially develop slowing of vertical saccades, followed by restriction of voluntary vertical gaze, more distinct in downgaze (Bhidayasiri et al., 2001). Despite these deficiencies, the oculocephalic reflex often remains intact, confirming the supranuclear localization. This pattern is clinically different from nuclear or infranuclear lesions, where both voluntary and reflexive ocular movements are impaired.

Nystagmus and Oscillations: Supranuclear pathology may also manifest as abnormal eye oscillations. Cerebellar lesions often cause gaze-evoked nystagmus, rebound nystagmus, and ocular flutter (Strupp & Kremmyda, 2013). These involuntary oscillations reflect a failure of the cerebellum to stabilize gaze-holding neural networks (Ito, 2011). Patients experience oscillopsia, reduced visual acuity, and imbalance.

Complex Syndromes: Several neurological disorders produce characteristic clusters of supranuclear eye movement anomalies. In progressive supranuclear palsy, patients present with early postural instability, vertical gaze palsy, and impaired saccades (Boxer et al., 2017). In Parkinson's disease, hypometric saccades, impaired pursuits, and convergence insufficiency are common, reflecting basal ganglia dysfunction (Hikosaka et al., 2000). In cerebellar degenerations, gaze-holding failure, dysmetric saccades, and pursuit deficiency dominate the clinical picture (Leigh & Tomsak, 2005). Recognition of these syndromes' aids in differential diagnosis and guides neuroimaging and management approaches.

12.6 CONCLUSION

Supranuclear control of ocular movements shows a sophisticated correlation between cortical, sub cortical and brainstem structures, allows the eyes to move in a purposeful, accurate and adaptable manner. Disturbance at any level produces unique clinical syndromes that provide diagnostic clues to underlying neurological disease. A comprehensive understanding of supranuclear mechanisms is therefore important for neuro optometric evaluation, as it allows clinicians to differentiate between central and peripheral ocular motor dysfunction and to implement targeted rehabilitation approaches.

12.7 BIBLIOGRAPHY

1. Leigh, R. J., & Zee, D. S. (2015). The neurology of eye movements (5th ed.). Oxford University Press.
2. Pierrot-Deseilligny, C., Müri, R. M., Ploner, C. J., Gaymard, B., Demeret, S., & Rivaud-Péchoux, S. (2003). Decisional role of the parietal cortex in reflexive and voluntary saccades. Annals of Neurology, 53(3), 405–406.
3. Pierrot-Deseilligny, C., Rivaud, S., Gaymard, B., Müri, R., & Vermersch, A. I. (1995). Cortical control of saccades. Annals of Neurology, 37(5), 557–567. https://doi.org/10.1002/ana.410370504
4. Krauzlis, R. J. (2004). Recasting the smooth pursuit eye movement system. Journal of Neurophysiology, 91(2), 591–603. https://doi.org/10.1152/jn.00801.2003
5. Wurtz, R. H., & Albano, J. E. (1980). Visual-motor function of the primate superior colliculus. Annual Review of Neuroscience, 3, 189–226. https://doi.org/10.1146/annurev.ne.03.030180.001201
6. Hikosaka, O., Takikawa, Y., & Kawagoe, R. (2000). Role of the basal ganglia in the control of purposive saccadic eye movements. Physiological Reviews, 80(3), 953–978. https://doi.org/10.1152/physrev.2000.80.3.953
7. Ito, M. (2011). The cerebellum: Brain for an implicit self. FT Press.
8. Robinson, D. A. (1981). The use of control systems analysis in the neurophysiology of eye movements. Annual Review of Neuroscience, 4, 463–503.

https://doi.org/10.1146/annurev.ne.04.030181.002335
9. Leigh, R. J., & Tomsak, R. L. (Eds.). (2005). Eye movement disorders in clinical practice. Oxford University Press.
10. Strupp, M., & Kremmyda, O. (2013). Central ocular motor disorders, including gaze palsy and nystagmus. Journal of Neurology, 260(5), 1380–1397. https://doi.org/10.1007/s00415-012-6787-9
11. Boxer, A. L., Yu, J. T., Golbe, L. I., Litvan, I., Lang, A. E., & Höglinger, G. U. (2017). Advances in progressive supranuclear palsy: New diagnostic criteria, biomarkers, and therapeutic approaches. The Lancet Neurology, 16(7), 552–563. https://doi.org/10.1016/S1474-4422(17)30157-6
12. Shaikh, A. G., Zee, D. S., Crawford, J. D., & Jinnah, H. A. (2016). Disorders of vergence eye movements. Current Opinion in Neurology, 29(1), 91–96. https://doi.org/10.1097/WCO.0000000000000274
13. Das, V. E., & Leigh, R. J. (2000). Visual–vestibular interaction in vergence eye movement. Experimental Brain Research, 130(4), 484–492. https://doi.org/10.1007/s002219900273
14. Ghasia, F. F., & Shaikh, A. G. (2015). Neural circuits for binocular vision and their adaptation in strabismus. Current Opinion in Neurobiology, 33, 195–203. https://doi.org/10.1016/j.conb.2015.05.004
15. Bhidayasiri, R., Riley, D. E., Somers, J. T., Lerner, A. J., Büttner-Ennever, J. A., & Leigh, R. J. (2001). Pathophysiology of slow vertical saccades in progressive supranuclear palsy. Neurology, 57(11), 2070–2077. https://doi.org/10.1212/wnl.57.11.2070