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Neuroradiology in the Ocular Motility Disorders :
I. Supranuclear Pathway[1]

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The supranuclear control of eye movement involves the pathway extending from the cerebral cortex to the ocular motor nuclei located in the brain stem. This paper describes the normal supranuclear pathway, which controls eye movement. We also include magnetic resonance imaging findings of the typical ocular manifestations caused by disorders involving the supranuclear pathway, providing the anatomic explanations for certain clinical signs.

Index words : Brain, anatomy
Brain, abnormalities
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Movement of the eye is a complex phenomenon regulated by a high level of coordination between sensory and motor functions which center stationary or moving objects in the central retina (fovea). The securing and stabilization of object images on the fovea during head movement are the basic functions of eye movements (1). Recent advances in the techniques of magnetic resonance (MR) imaging have opened a new horizon for the evaluation of ocular motility disorders; pathologies once considered beyond the scope of imaging are now frequently visualized. Knowledge of the basic pathway and physiology involved in eye movement provides a better understanding of the ocular motility disorders. The supranuclear control of eye movement involves the pathway extending from the cerebral cortex to the ocular motor nuclei located in the brain stem. In this paper, in addition to describing the normal supranuclear pathway of eye movement, we demonstrate MR imaging findings of the typical ocular manifestations caused by disorders involving the supranuclear pathway, providing the anatomic explanations for certain clinical signs.

Classification of Eye Movement

Many theories and assumptions have attempted to explain the mechanism of eye movement. For the sake of simplicity, we assume that there are two independent major subsystems involved in the control of eye movements, namely version and vergence (1). The version subsystem controls all conjugate movements, while the vergence subsystem controls all those that are disconjugate. The former encompasses two distinct eye movements, saccades (fast eye movements) and pursuit (slow eye movements), and the latter mediates vergence eye movements. While all three movements share a common neural pathway from the ocular motor nuclei to the extraocular muscles, each is subject to a different supranuclear neural control.

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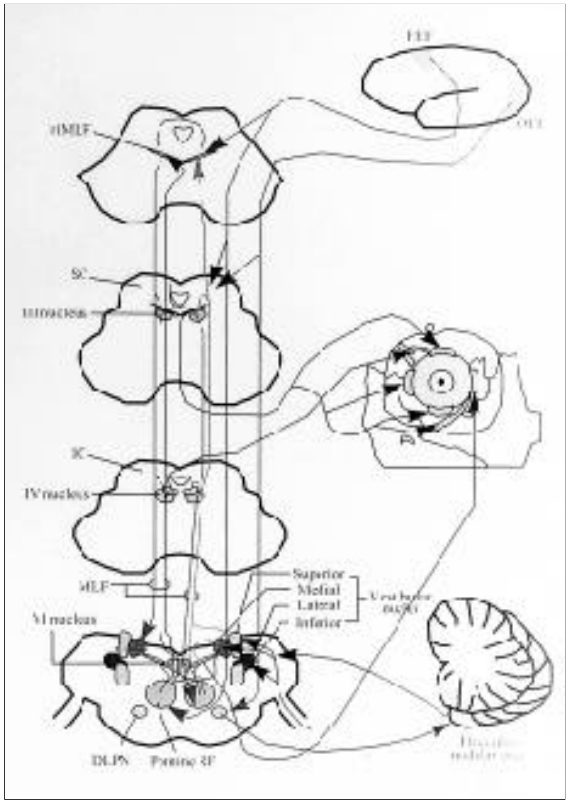

Fig. 1. Diagram of overall supranuclear, nuclear, and infranuclear pathways of eye movement control.
DLPN = dorsolateral pontine nucleus ; IC = inferior colliculus ; MLF = medial longitudinal fasciculus ; OEF = occipital eye fields ; RF = reticular formation ; FEF = frontal eye fields ; riMLF = rostral interstitial nucleus of medial longitudinal fasciculus ; SC = superior colliculus.

Normal Supranuclear Pathway (Fig. 1)

Fast Eye Movements(Saccades)

The visual stimulus for fast eye movements (saccades) is target displacement in space (1). Reticular formation in the pons and midbrain plays a critical role in the generation of all types of saccades. The paramedian pontine reticular formation (PPRF) contains neurons essential for horizontal saccades, while the rostral interstitial nucleus of the medial longitudinal fasciculus (riMLF), located in the rostral midbrain, is essential for vertical saccades. The PPRF receives inputs from the contralateral frontal eye fields (FEF, Brodmann area 8) by way of the anterior limb of the internal capsule and the medial portion of the cerebral peduncle. This pathway decussates in the lower midbrain and upper pons. The riMLF probably receives input from both the FEF and the PPRF. The superior colliculi receive visual inputs from both the posterior cortical areas and the FEF via direct and indirect (basal ganglia) pathways, and probably play a role in both spontaneous and reflexive saccades. Voluntary saccades are initiated in the FEF, which receive inputs from the supplemental motor and posterior cortices (2). The pars reticulata of the substantia nigra inhibits the superior colliculi, suppressing excessive spontaneous saccades, while neurons in the caudate nu-

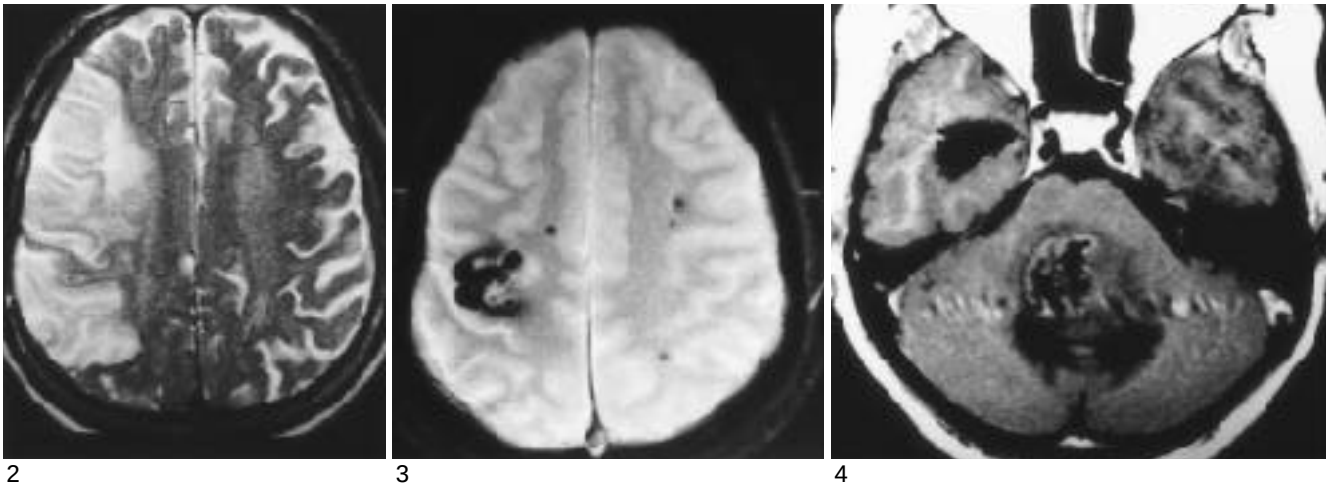

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Fig. 2. Saccadic palsy caused by acute cerebral infarction. Axial T2-weighted MR image shows large cerebral infarction in the right MCA territory. The patient's eyes were deviated to the right side and the patient had leftward conjugate gaze palsy.

Fig. 3. Saccadic palsy caused by frontal lobe hemorrhage. Axial gradient-echo MR image shows multiple hemorrhagic foci in the brain with the largest being located just anterior to the right central sulcus in a 42-year-old man with seizure. His eyes were deviated to the left side and he had rightward conjugate gaze palsy.

Fig. 4. Horizontal gaze palsy caused by a grade II pontine astrocytoma. Enhanced axial T1-weighted MR image shows a mass with peripheral rim-like enhancement in the right pontine tegmentum, and the patient had horizontal rightward gaze palsy.

cleus projecting to the pars reticulata of the substantia nigra inhibit substantia nigra cells.

Slow Eye Movement(Pursuit)

The major stimulus for slow eye movement (pursuit) is a fixated target that moves(1). Clinical and experimental observations have suggested that the occipital visual cortex (Brodmann area 17) contains neurons that respond to a moving visual stimulus and project to the middle temporal and medial superior temporal visual areas with some influence from the adjacent parietal cortex. From these extrastriate visual areas neurons descend to the dorsolateral pontine nuclei and subsequently to the contralateral flocculus and dorsal vermis of the cerebellum, from which projections relay to the ipsilateral vestibular nuclei and finally to the ocular motor nuclei. If unilateral, pursuit abnormality usually occurs in the direction ipsilateral to the side of a lesion (2, 3).

Vergence Eye Movement

The stimulus for vergence eye movement is target displacement or motion along the visual z axis(toward or away from the observer) (1). Motor cells involved in the vergence system are intermixed in the midbrain reticular formation, from where commands are transmitted to the ocular motor nuclei (2).

Supranuclear Pathology Resulting in Ocular Motility Disorders

Horizontal Version Abnormalities

Unilateral saccadic palsy usually results from an acute destructive frontal stroke, which causes deviation of the eyes toward the side of the lesion and impaired gaze toward the contralateral side (Fig. 2). It is usually associated with contralateral hemiparesis or hemianopsia (2). If the opposite hemisphere is intact, saccadic palsy is transient and normal function is usually regained in days to weeks. In contrast, irritative frontal lesions secondary to focal seizure activity or acute cerebral hemorrhage cause tonic deviation of the eyes to the contralateral side and gaze is impaired toward the same side of the lesion (Fig. 3). Bilateral lesions of the frontomesencephalic system cause saccadic palsy in both directions. In this situation, patients rely on vestibulo-ocular reflex to change fixation (3). A similar manifestation can be observed in congenital or acquired diseases such as multiple sclerosis, Huntington's disease, Wilson's disease, and spinocerebellar degeneration (2, 3). Unilateral pursuit paresis occurs with posterior hemispheric disease and manifests as saccadic pursuit in the direction ipsilateral to the side of the lesion; it is usually accompanied by contralateral homonymous hemianopsia (2). Bilateral saccadic pursuit may accompany the use of sedative drugs, inattention, fatigue, or impaired consciousness, and is also seen in cases involving diffuse cerebral, cerebellar or

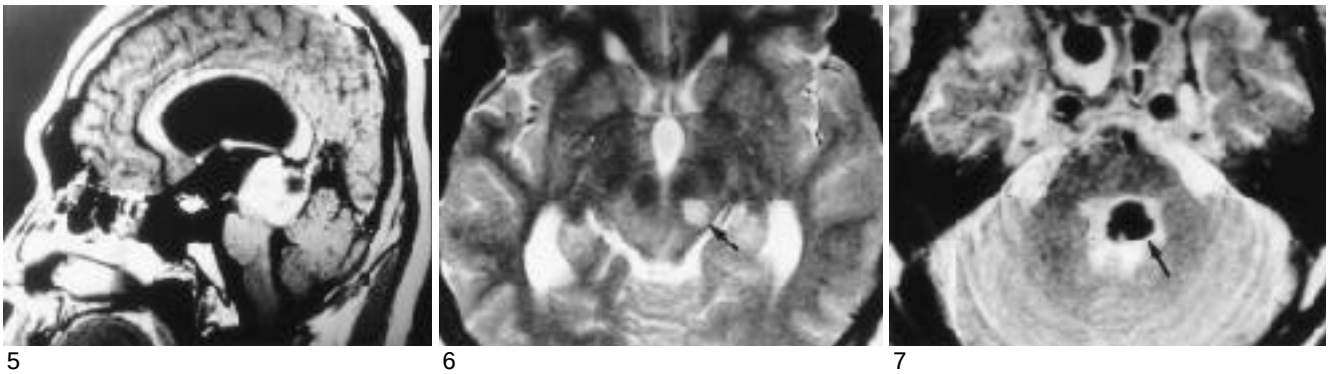

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Fig. 5. Vertical gaze palsy caused by pineal germinoma (Parinaud's syndrome) .Enhanced sagittal T1-weighted MR image shows a well enhanced pineal mass which causes compression of the midbrain resulting in patient's upgaze palsy.

Fig. 6. Internuclear ophthalmoplegia caused by type 2 neurofibromatosis. Axial T2-weighted MR image shows an oviod mass with high signal intensity in the rostral midbrain (arrow) just posterior to the left red nucleus. After gadolinium infusion, the lesion was well enhanced (not shown). Presumptive diagnosis is glioma.

Fig. 7. One-and-a-half syndrome caused by pontine hemorrhage. Axial T2-weighted MR image shows acute hemorrhage with dark signal intensity (arrow) in the very area of left abducens nerve nucleus. The hemorrhage extended up to the ipsilateral midbrain (not shown).

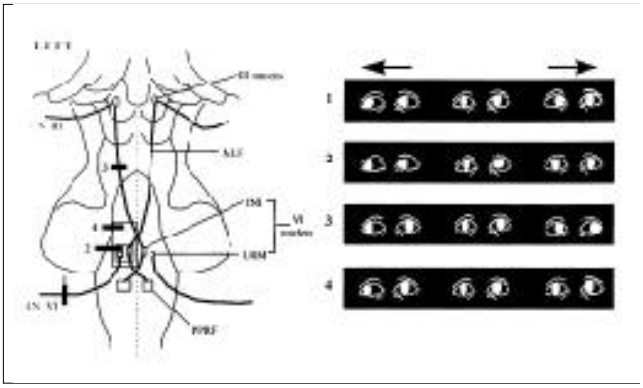

Fig. 8. Diagrams showing deficits of horizontal gaze following damage to the abducens/MLF system. Lesions labeled 1-4 on the left diagram can produce the characteristic ocular manifestations shown on the right diagram: 1, abducens palsy caused by lesion in left abducens nerve; 2, lateral gaze palsy caused by lesion in left abducens nucleus; 3, internuclear ophthalmoplegia caused by lesion in left MLF; and 4, one-and-a-half syndrome caused by lesion in left abducens nerve nucleus and left MLF.INI = intranuclear interneuron; LRM = lateral rectus motor neuron; MLF = medial longitudinal fasciculus; P-PRF = paramedian pontine reticular formation.frequency encoding pulse, PE - phase encoding pulse)

brain stem disease (2). The saccadic and pursuit subsystems converge anatomically at subthalamic and upper brain stem levels (2). The PPRF is believed to be the final prenuclear anatomical pathway of ipsilateral saccades. All unilateral PPRF lesions are associated with ipsilateral gaze paralysis, which is usually persistent. Excitatory projections from the PPRF mediating horizontal saccades are directed to the ipsilateral abducens nucleus. From synapses in intranuclear interneurons located in the abducens nucleus, they are transmitted to the contralateral MLF which, in turn, conveys impulses to the medial rectus subnucleus. Axons carrying horizontal vestibulo-ocular and pursuit signals also connect with internuclear interneurons (2). Lesions that are small enough to be confined to the PPRF may, therefore, spare the vestibulo-ocular pathway, while those that involve the abducens nucleus frequently impair the vestibulo-ocular reflex (Fig. 4).

Vertical Gaze Abnormalities

Vertical gaze palsy typically occurs in diseases involving the midbrain and thalamus. While projections for downward gaze are more or less directly dorsolateral from the riMLF, where they are primarily arranged laterally, to the oculomotor and trochlear nuclei, those for upward gaze are more sophisticated, reflecting the fact that upward gaze palsy result from lesions variously placed in the midbrain. The critical structures involved are the riMLF, the interstitial nucleus of Cajal, the posterior commissure, the pretectal area and the dorsal periaqueductal gray matter (2, 3). Dorsal midbrain (Parinaud 's) syndrome is the most well-known abnormality affecting vertical gaze with pineal region tumors and midbrain infarction as a leading cause (Fig. 5). In addition to upgaze palsy, the syndrome is frequently accompanied by mydriasis, light-near dissociation, lid retraction, lid lag and convergence-retraction nystagmus.

Internuclear Ophthalmoplegia (INO)

The MLF conveys saccadic, vestibulo-ocular, and pursuit impulses to the ipsilateral medial rectus subnucleus (2). Unilateral disruption of the MLF prevents the ipsilateral medial rectus from adduction during horizontal versions; it is commonly accompanied by nystagmus of the abducting eye and is known as internuclear ophthalmoplegia (INO) (Fig. 6). Most cases of INO are exophoric in the primary position, without symptomatic diplopia. In bilateral INO, exotropia is occasionally seen. This is the so-called WEBINO (wall-eyed bilateral INO) syndrome, which might be caused by simultaneous involvement of the medial rectus subnuclei. Lesions that are large enough to involve an MLF as well as the ipsilateral abducens nucleus or the PPRF result in so-called one-and-a-half syndrome, in which condition ipsilateral horizontal gaze palsy occurs in combination with INO during gaze towards the opposite side (3-6). Abduction of the opposite eye is the only spared movement in this condition (Fig. 7). Deficits of horizontal gaze following damage to the various sites of the abducens/MLF system are demonstrated in Figure 8.

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안구운동에 관여하는 핵상경로는 대뇌 피질로부터 뇌간에 위치한 안구운동핵에 이르는 신경통로를 의미한다. 이 임상화보에서는 안구운동을 조절하는 정상 핵상경로와 이를 침범하는 질환에 의하여 야기되는 특징적인 안구운동 장애의 자기공명영상 소견을 해부학적 근거와 더불어 기술하였다.