RadioGraphics

Brainstem Pathways for Horizontal Eye Movement: Pathologic Correlation with MR Imaging[1]

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TEACHING POINTS
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Horizontal eye movements are conducted by the medial rectus and the lateral rectus muscles, which are innervated by the oculomotor nerve (cranial nerve III) and the abducens nerve (cranial nerve VI), respectively. The oculomotor and the abducens nuclei are interconnected by a tract in the brainstem named the medial longitudinal fasciculus (MLF). Through the MLF, the actions of the oculomotor and the abducens nuclei are coordinated, generating conjugate horizontal eye movements. The disorders of horizontal eye movement that are caused by brainstem lesions are classified into three groups: *(a)* lateral gaze palsy, *(b)* internuclear ophthalmoplegia, and *(c)* one-and-a-half syndrome. Lateral gaze palsy is caused by a lesion involving the paramedian pontine reticular formation (PPRF) or the abducens nucleus. Internuclear ophthalmoplegia occurs as a result of a lesion involving the MLF. One-and-a-half syndrome is a combination of lateral gaze palsy and internuclear ophthalmoplegia and is caused by a lesion involving both *(a)* the ipsilateral PPRF or the ipsilateral abducens nucleus and *(b)* the ipsilateral MLF. The pathologic lesions depicted on magnetic resonance images were topographically well correlated with the brainstem pathways and each type of horizontal eye movement disorder. Most of the lesions were tiny acute infarctions and were found in the most posterior region of the pons, which corresponded to the location of the brainstem pathways. Therefore, awareness of the brainstem pathways controlling horizontal eye movement is important to avoid missing a small pontine lesion.

Abbreviations: MLF = medial longitudinal fasciculus, PPRF = paramedian pontine reticular formation

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Introduction

Horizontal eye movement is a product of the internuclear network in the brainstem. Conjoined ipsilateral lateral (or medial) gaze and contralateral medial (or lateral) gaze are created by the interaction of the ipsilateral abducens nucleus, the contralateral oculomotor nucleus, the interconnecting white matter tracts, and the extraocular muscles (1–3).

The pons is the primary site for the synthesis of the central signal that commands the components of horizontal eye movement to function as a cooperative unit (4). The interconnecting structure for the generation of horizontal eye movement is the paramedian pontine reticular formation (PPRF) (5). The signal from the PPRF transmits to the abducens nucleus and then projects (discharges) to the abducens nerve and simultaneously to the contralateral oculomotor nucleus via the medial longitudinal fasciculus (MLF) (1).

This neural pathway explains the mechanism of how different brainstem lesions can affect horizontal eye movement, causing diverse disorders. The main classes of horizontal eye movement disorders caused by brainstem lesions are *(a)* lateral gaze palsy, *(b)* internuclear ophthalmoplegia, and *(c)* one-and-a-half syndrome, a term that refers to the combination of ipsilateral lateral gaze palsy and internuclear ophthalmoplegia.

Teaching Point

Magnetic resonance (MR) imaging allows high spatial resolution and signal intensity contrast to be used to depict delicate anatomic structures of the neural pathways in the brainstem (6,7). Cranial nerves also can be superbly depicted with MR imaging; in particular, the cranial nerves surrounded by cerebrospinal fluid or the cavernous sinus can be depicted in detail by using three-dimensional high-resolution techniques, including the balanced turbo field-echo sequence, the constructive interference in steady-state sequence, or fast imaging employing steady-state acquisition (8–12). With these techniques, the compact pathologic lesions inducing the disorders of horizontal eye movement can be demonstrated, with topographic correlation.

The purpose of this article is to review the brainstem pathways controlling horizontal eye movement and the types of horizontal eye movement disorders and to correlate their MR imaging findings with the pathologic lesions. First, the anatomic structures and the brainstem pathways related to horizontal eye movement are reviewed: extraocular muscles, cranial nerves and their nuclei, PPRF, and MLF. Then the types of horizontal eye movement disorders are detailed, along with the pathologic lesions in the brainstem that can be demonstrated with MR imaging and correlated with the corresponding horizontal eye movement disorders topographically.

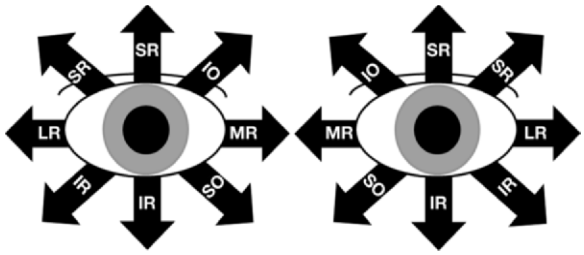


Figure 1. Schematic drawing of the extraocular muscles and their actions. Eye movements are conducted by six extraocular muscles for each eye. Vertical eye movements are produced by the superior rectus *(SR)* and the inferior rectus *(IR)* muscles. Horizontal eye movements are produced by the medial rectus *(MR)* and the lateral rectus *(LR)* muscles. The superior oblique *(SO)* and the inferior oblique *(IO)* muscles induce rotational eye movements. The arrows indicate the direction of the globe movement with the contraction of each extraocular muscle.

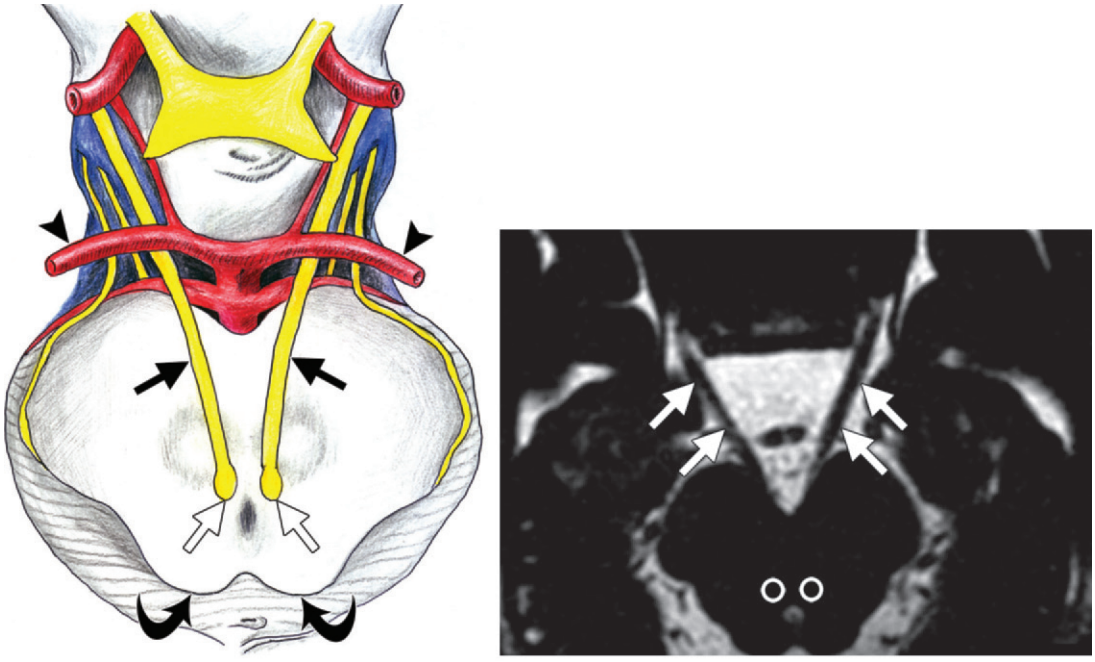
Anatomic Structures Related to Horizontal Eye Movement

Extraocular Muscles

There are six extraocular muscles for each eye: the medial rectus, lateral rectus, superior rectus, inferior rectus, superior oblique, and inferior oblique muscles (13,14). Of these six extraocular muscles, the four rectus muscles (excepting the inferior and superior oblique muscles) have a tendinous origin from a common ring at the orbital apex and insert onto the sclera of the globe (15). The superior rectus and the inferior rectus muscles move the globe vertically, and the superior oblique and the inferior oblique muscles make torsional globe movements. The medial rectus and the lateral rectus muscles produce simple horizontal eye movements. The medial rectus muscle adducts the globe in the nasal (medial) direction. The lateral rectus muscle abducts the globe in the temporal (lateral) direction. The extraocular muscles and their actions are shown schematically in Figure 1.

Cranial Nerves and Their Nuclei

Each extraocular muscle is innervated by the corresponding cranial nerve, which originates from the individual cranial nerve nucleus. The levator palpebrae superioris muscles and the superior rectus muscles are innervated by the superior division of the oculomotor nerve (cranial nerve III). The medial rectus, the inferior rectus, and



a. b.

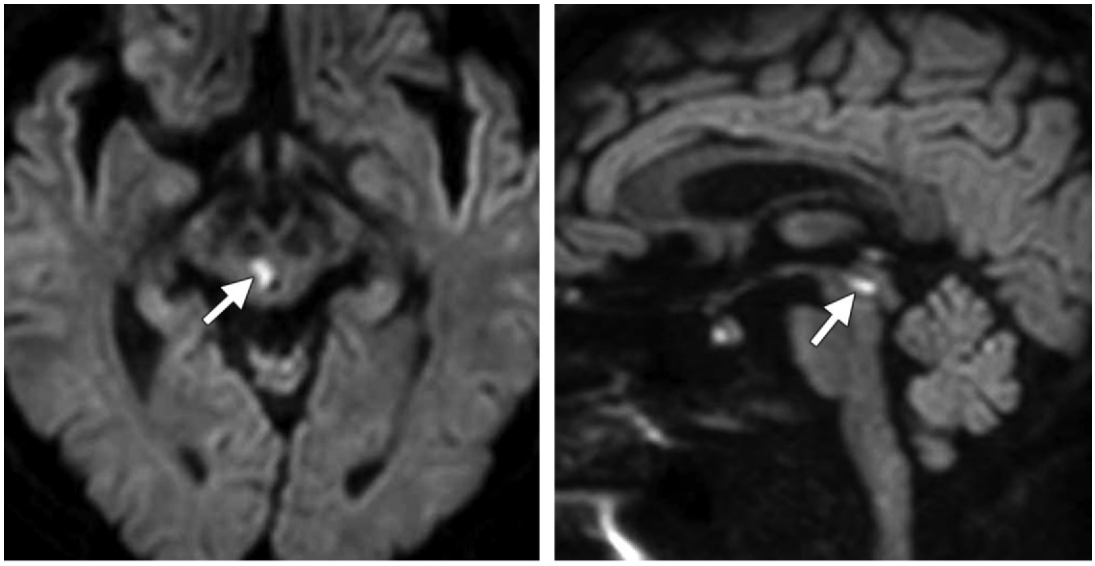
Figure 2. Locations of the oculomotor nerves (cranial nerves III) and oculomotor nuclei. **(a)** Drawing shows the oculomotor nerves (straight black arrows) originating from the oculomotor nuclei (white arrows) in relation to the adjacent structures: the superior colliculi (curved arrows) and the posterior cerebral arteries (arrowheads). **(b)** Axial reformatted thin-section T2-weighted MR image shows the oculomotor nerves (arrows) emerging from the medial side of the cerebral peduncle and traversing the interpeduncular cistern toward the cavernous sinus. Note the locations of the oculomotor nuclei (circles) in the midbrain.

the inferior oblique muscles are innervated by the inferior division of the oculomotor nerve (cranial nerve III) (16). The trochlear nerve (cranial nerve IV) innervates the superior oblique muscle. The lateral rectus muscle is innervated by the abducens nerve (cranial nerve VI) (17,18).

As previously stated, the medial rectus and the lateral rectus muscles move the globe medially and laterally, respectively. Therefore, conjugate horizontal eye movements are conducted by the oculomotor nerve (cranial nerve III) and the abducens nerve (cranial nerve VI). The following paragraphs provide a closer look at the oculomotor and the abducens nuclei and their nerves.

Oculomotor Nucleus and Nerve.—Each oculomotor nucleus is located in the pretectal area of the upper portion of the midbrain (mesencephalon). The pretectal area lies ventrolateral to the cerebral aqueduct at the level of the superior colliculus and the red nucleus (13,19). The oculomotor nerve (cranial nerve III) exits the brainstem medially to the cerebral peduncles and runs through the interpeduncular cistern between the posterior cerebral artery and the superior cerebellar artery (11,16). The nerve is close to the posterior communicating artery when it traverses the interpeduncular cistern (20). The nerve courses in the anteroinferior direction, pierces the dura mater, and passes through the cavernous sinus along its superolateral wall (21). The oculomotor nerve (cranial nerve III) is accompanied by a cerebrospinal fluid–filled dural cuff as it enters the superolateral roof of the cavernous sinus known as the oculomotor cistern (22). Then the nerve is divided into a superior division and an inferior division just posterior to the orbital apex. Escaping the intracranial space, both divisions of the oculomotor nerve (cranial nerve III) enter the orbital cavity via the superior orbital fissure.

The locations of the oculomotor nerves and the oculomotor nuclei are shown in Figure 2. Pathologic lesions in the oculomotor nucleus (Fig 3) may cause not only a horizontal eye movement disorder but also a vertical eye movement disorder because the oculomotor nerve (cranial nerve III) innervates the superior rectus, the inferior rectus, and the inferior oblique muscles. The lesion can also induce ptosis that is due to the dysfunction of the levator palpebrae superioris muscle.



a. b.

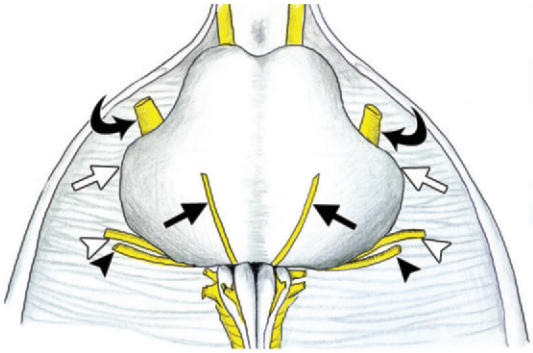
Figure 3. Vertical diplopia in a 67-year-old man. Axial **(a)** and sagittal **(b)** diffusion-weighted MR images show a small acute infarction (arrow) in the right posterior portion of the midbrain anterolateral to the cerebral aqueduct, a location that corresponds to the location of the right oculomotor nucleus.

Abducens Nucleus and Nerve.—The abducens nucleus is located in the facial colliculus on the floor of the fourth ventricle at the level of the mid to lower portion of the pons (13,16). The abducens nerve (cranial nerve VI) has a long cisternal course (20), which makes evaluation of the nerve in the subarachnoid space easy and accurate (23). The nerve exits the brainstem from the medullopontine sulcus just above the pyramids and runs in the superolateral direction in the prepontine cistern (24). Then the nerve pierces the dura mater and courses along its dural sleeve in the petroclival venous confluence known as the Dorello canal (25). This canal can be viewed as a cerebrospinal fluid–filled invagination into the petroclival dura mater (18). After the abducens nerve (cranial nerve VI) leaves the Dorello canal, it passes through the cavernous sinus (26). The nerve is medially located within the cavernous sinus—medial to the oculomotor and trochlear nerves (cranial nerves III and IV)—and is laterally adjacent to the cavernous internal carotid artery (26). The abducens nerve enters the orbital cavity via the superior orbital fissure.

The locations of the abducens nerves and the abducens nuclei are shown in Figure 4. Pathologic lesions in the abducens nucleus (Fig 5) can cause complete lateral gaze palsy. The detailed mechanism of lateral gaze palsy is discussed in the section "Lateral Gaze Palsy."

Paramedian Pontine Reticular Formation

The PPRF is the last supranuclear relay involved in conjugate horizontal eye movement (27). The PPRF is located in the pontine tegmentum near the abducens nucleus and is provided with neural inputs from the cortex (13). The PPRF receives afferent impulses from the frontal eye field and from the parietal eye field; the emanated impulse travels along the internal capsule and the cerebral peduncle, then decussates to the opposite side at the level of the midbrain, and finally innervates the PPRF (28). The PPRF contains excitatory "burst" neurons that produce the pulse transmitted to the ipsilateral abducens nucleus (Fig 6) (29).



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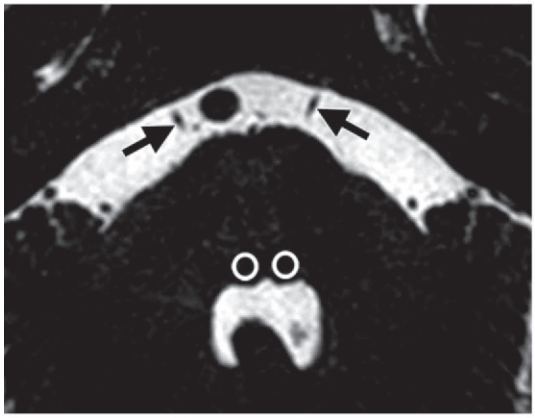

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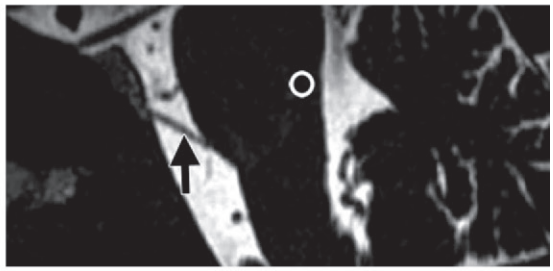

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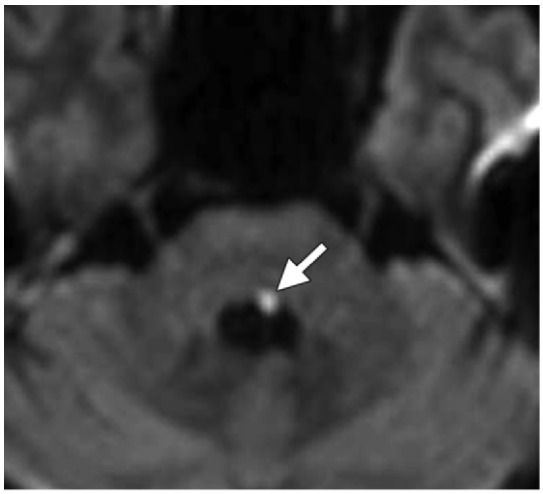
Figure 4. Locations of the abducens nerves (cranial nerves VI) and abducens nuclei. **(a)** Drawing shows the abducens nerves (straight black arrows) emerging from the medullopontine sulcus and their relation to the adjacent structures: the pons (white arrows), trigeminal nerves (curved arrows), facial nerves (cranial nerves VII) (black arrowheads), and vestibulocochlear nerves (cranial nerves VIII) (white arrowheads). **(b, c)** Axial thin-section **(b)** and sagittal reformatted **(c)** T2-weighted MR images show the abducens nerves (arrows) traversing the prepontine cistern in a superolateral direction toward the cavernous sinus. Note the locations of the abducens nuclei (white circles) in the facial colliculus area of the pons.



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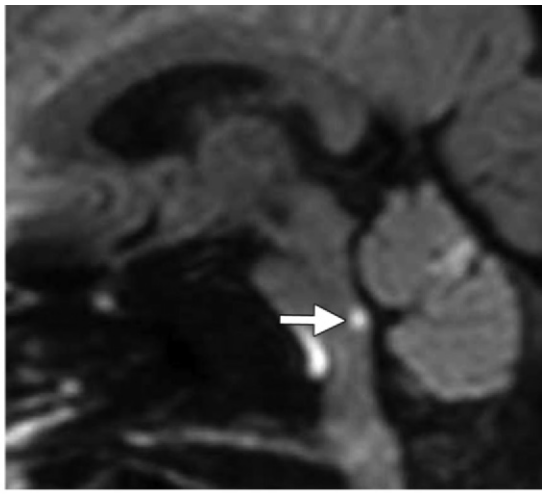

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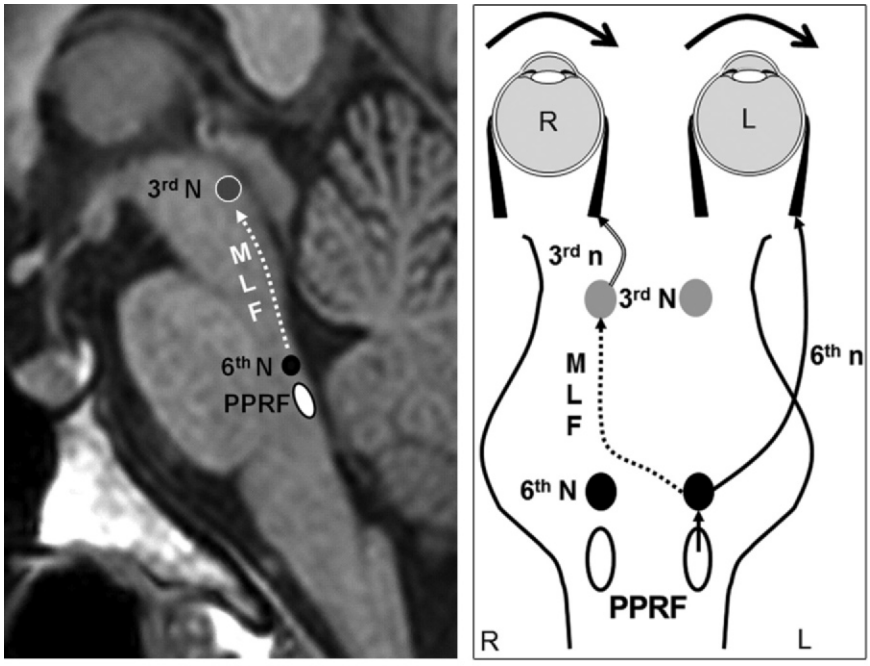
Figure 5. Horizontal diplopia in a 44-year-old man. Axial **(a)** and sagittal **(b)** diffusion-weighted MR images show a small acute infarction (arrow) in the left posterior portion of the pons in the facial colliculus area that corresponds to the location of the left abducens nucleus.

Figure 6. Brainstem pathways of horizontal eye movement. **(a)** Sagittal T1-weighted MR image shows the pathway from the PPRF (white oval) to the abducens nucleus (*6th N*, black circle) and the oculomotor nucleus (*3rd N*, gray circle) through the MLF (dashed arrow). **(b)** Diagram shows signal (solid straight arrow) from one of the PPRFs (white ovals) activating the ipsilateral abducens nucleus (*6th N*, black circles) as a horizontal gaze center. The signal is transmitted to the ipsilateral abducens nerve (*6th n*, cranial nerve VI) and contracts the ipsilateral lateral rectus muscle. The contralateral oculomotor nucleus (*3rd N*, gray circles) is simultaneously activated through the MLF (dashed arrow) from the abducens nucleus and transmits the signal to the contralateral oculomotor nerve (*3rd n*, cranial nerve III), which results in conjugate horizontal eye movement because of the contraction of the contralateral medial rectus muscle.



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Medial Longitudinal Fasciculus

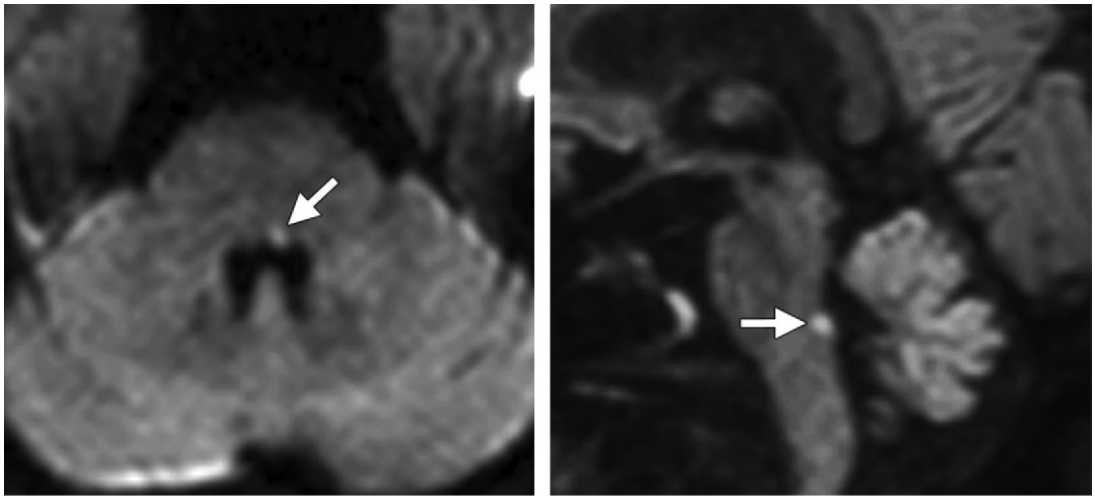
The MLF is the longitudinal white matter tract that is located in the posterior part of the pons just anterior to the fourth ventricle (Fig 6) (30). The MLF contains neural fibers that interconnect the oculomotor, the trochlear, the abducens, and the vestibular nuclei. The MLF coordinates the actions of the oculomotor and the abducens nuclei; this interaction produces bilateral conjugate horizontal eye movements.

The abducens nucleus, which is activated by the excitatory signal from the PPRF, projects (discharges) two types of neurons to intermediate conjugate horizontal eye movement (30). One type of neuron forms the ipsilateral abducens nerve (cranial nerve VI), which projects to the ipsilateral lateral rectus muscle. By the action of the abducens nerve (cranial nerve VI) and the lateral rectus muscle, lateral movement of the ipsilateral eye is performed. At the same time, the other type of neuron forms the abducens interneuron, which decussates to the contralateral pons. The impulse is conveyed through the MLF to the contralateral oculomotor nucleus in the midbrain, which successively activates the oculomotor nerve (cranial nerve III), innervating the contralateral medial rectus muscle. Therefore, the excitatory signal from the PPRF produces simultaneous contraction of the ipsilateral lateral rectus muscle and the contralateral medial rectus muscle.

Teaching Point

To sum up, the brainstem pathways for horizontal eye movements start from the abducens nucleus as the horizontal gaze center taking signal from the PPRF. These pathways continue to the ipsilateral abducens nerve (cranial nerve VI) and the contralateral oculomotor nerve (cranial nerve III) through the MLF and end in conjugate horizontal eye movement in the ipsilateral direction to the side of the abducens nucleus.

Figure 7. Left lateral gaze palsy in a 58-year-old woman. **(a, b)** Axial **(a)** and sagittal **(b)** diffusion-weighted MR images show a small acute infarction (arrow) in the left pontine facial colliculus area where the abducens nucleus is located. **(c)** Diagram shows the mechanism for lateral gaze palsy: A pathologic lesion (large dark gray circle) involves one of the PPRFs (white ovals) or one of the abducens nuclei (*6th N*, black circles). × = interruptions of signal transmission or eye movement that are a result of the lesion, *3rd n* = contralateral oculomotor nerve (cranial nerve III), *6th n* = ipsilateral abducens nerve (cranial nerve VI), *3rd N* = oculomotor nuclei (small gray circles). **(d)** Top to bottom: Photographs of the patient's rightward, midline, and leftward horizontal gaze show that the patient has impaired abduction of the left eye and impaired adduction of the right eye with leftward gaze. With rightward gaze, the movement of both eyes is intact.



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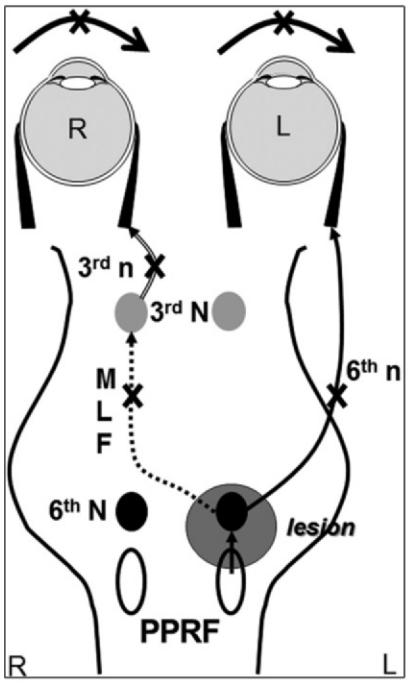

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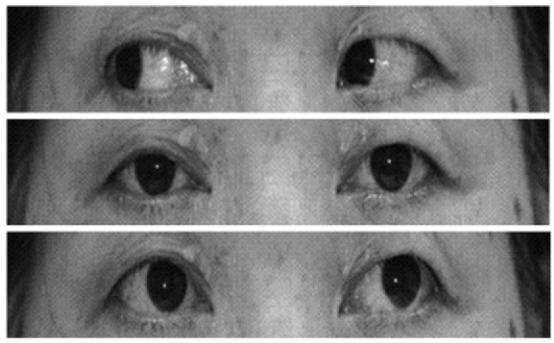

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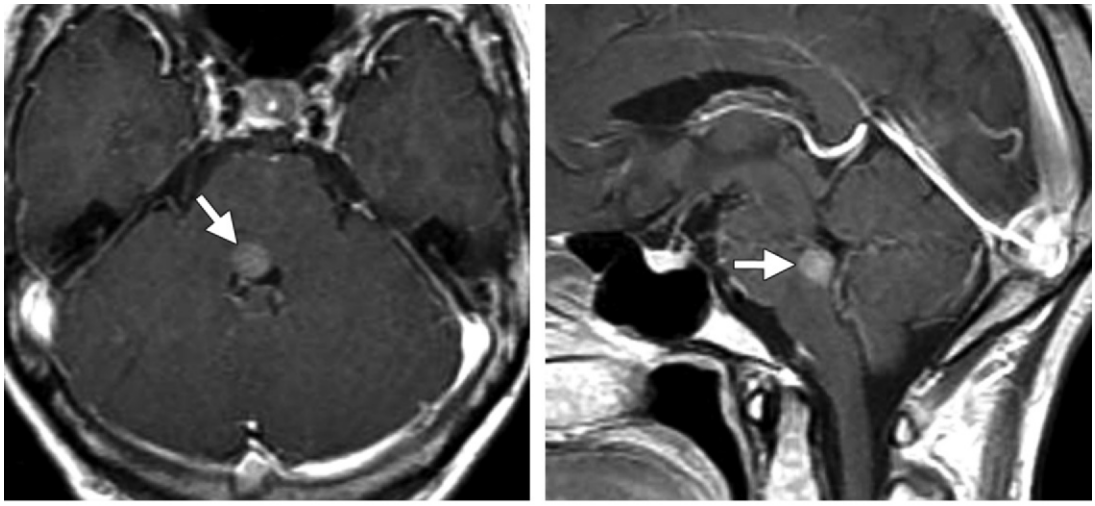
Disorders of Horizontal Eye Movement

Lateral Gaze Palsy

Lateral gaze palsy implies paresis of the abduction of the ipsilateral eye and conjugate paresis of the adduction of the contralateral eye when the patient tries to gaze in the ipsilateral direction. Lateral gaze palsy is caused by a pathologic lesion involving the PPRF or the abducens nucleus (Figs 7, 8) (31). Common causes are pontine infarctions (32) and, occasionally, space-occupying lesions in the pons, such as a brainstem glioma (33) or a pontine

Teaching Point

Figure 8. Right lateral gaze palsy in a 51-year-old woman. Axial **(a)** and sagittal **(b)** contrast-enhanced T1-weighted MR images show a small enhancing nodule (arrow) in the right facial colliculus area where the abducens nucleus is located. The presumed diagnosis was a metastasis from breast cancer.



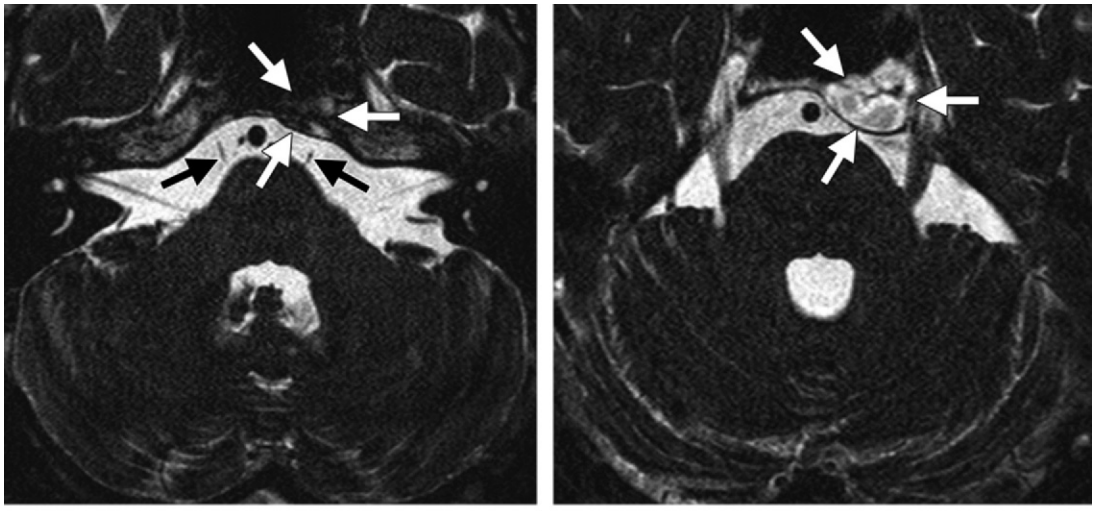
a. b.

metastasis (27). If the neural signal output fails to discharge from the PPRF or the abducens nucleus, the abducens nerve (cranial nerve VI) cannot convey the excitatory signal to the ipsilateral lateral rectus muscle, and this in turn causes paresis of the abduction of the ipsilateral eye (34). At the same time, no neural signal output can be transmitted to the MLF, which is supposed to convey the excitatory signal to the contralateral oculomotor nucleus, and this lack of signal has the effect of denervation of the contralateral medial rectus muscle, thus causing paresis of the conjugate adduction of the contralateral eye.

Abducens Nerve Palsy

Peripheral abducens nerve palsy is distinct from lateral gaze palsy (33). If any pathologic lesion involves only the abducens nerve (cranial nerve VI) itself—not the abducens nucleus or the PPRF—the lesion impairs only the abduction of the ipsilateral eye by affecting the ipsilateral lateral rectus muscle (Fig 9). The abducens nerve (cranial nerve VI) is vulnerable to various injuries because of its long intracranial course: The nerve can be damaged by stretching injury caused by *(a)* intracranial masses, including tumorous conditions such as metastasis, or *(b)* increased intracranial pressure; nerve damage can also result from lesions or trauma of the base of the skull and even from sellar lesions (33). Tumors originating from the abducens nerve (cranial nerve VI), such as schwannoma, can be another source of abducens nerve palsy (35). Conjugate adduction of the contralateral eye remains intact because the pathway from the abducens nucleus or the PPRF to the contralateral oculomotor nucleus via the MLF is not damaged. Therefore, isolated abducens nerve palsy affects only abduction of the ipsilateral eye and does not result in conjugate horizontal eye movement when the patient gazes in the ipsilateral direction.

Figure 9. Left peripheral abducens nerve palsy in a 19-year-old man. **(a, b)** Axial thin-section T2-weighted MR images (**a** obtained more caudad than **b**) show an enchondroma of the clivus (white arrows) compressing the left abducens nerve (cranial nerve VI) at the Dorello canal. Both abducens nerves (black arrows in **a**) are well depicted in the prepontine cistern. **(c)** Diagram shows the mechanism for the peripheral abducens nerve palsy: A pathologic lesion (large dark gray circle) involves the left abducens nerve (6^{th} *n*, cranial nerve VI). × = interruptions of signal transmission or eye movement that are a result of the lesion, 6^{th} *N* = abducens nuclei (black circles), 3^{rd} *n* = contralateral oculomotor nerve (cranial nerve III), 3^{rd} *N* = oculomotor nuclei (small gray circles). **(d)** Top to bottom: Photographs of the patient's rightward, midline, and leftward horizontal gaze show that the patient has impaired abduction of only the left eye with leftward gaze; the other ocular movements are preserved.



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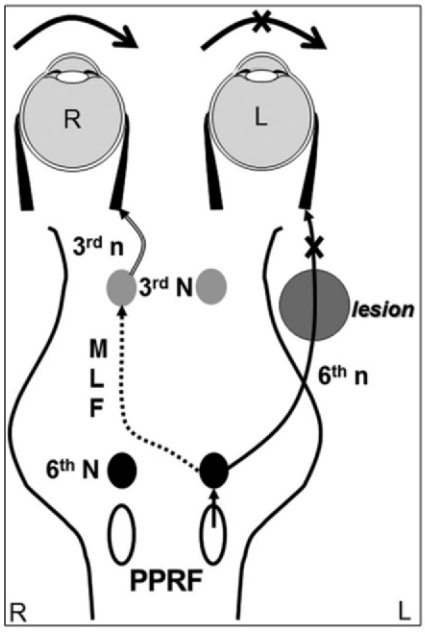

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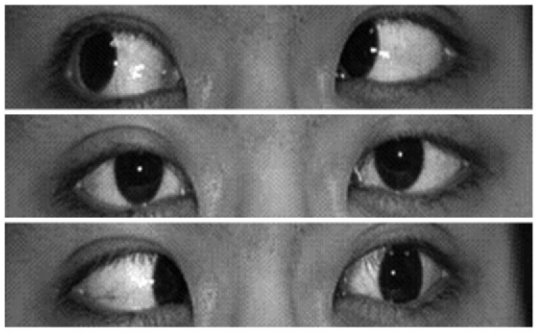

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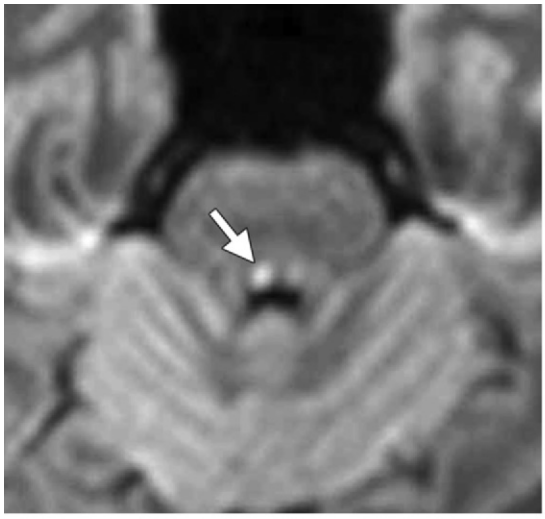
Figure 10. Internuclear ophthalmoplegia in a 40-year-old man. **(a, b)** Axial **(a)** and sagittal **(b)** diffusion-weighted MR images show a small acute infarction (arrow) in the right posterior portion of the pons where the MLF is located. **(c)** Diagram shows the mechanism for internuclear ophthalmoplegia: A pathologic lesion (large dark gray circle) involves the MLF. × = interruptions of signal transmission or eye movement that are a result of the lesion, 6^{th} *n* = abducens nerve (cranial nerve VI), 6^{th} *N* = abducens nuclei (black circles), 3^{rd} *n* = oculomotor nerve (cranial nerve III), 3^{rd} *N* = oculomotor nuclei (small gray circles). **(d)** Top to bottom: Photographs of the patient's rightward, midline, and leftward horizontal gaze show that the patient has impaired adduction of the right eye with leftward gaze. With rightward gaze, the movement of both eyes is intact.



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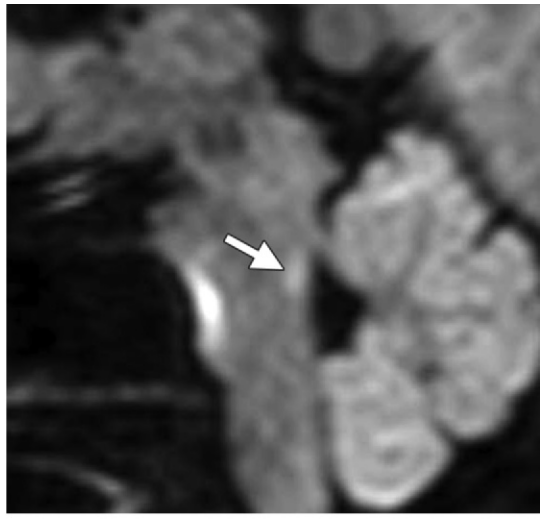

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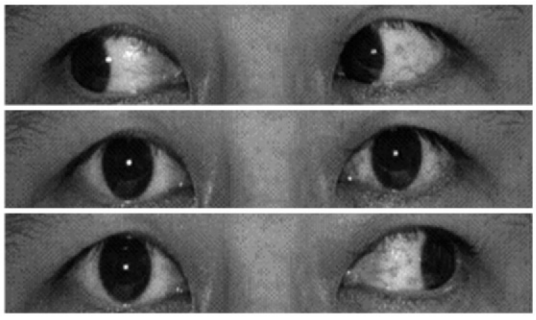

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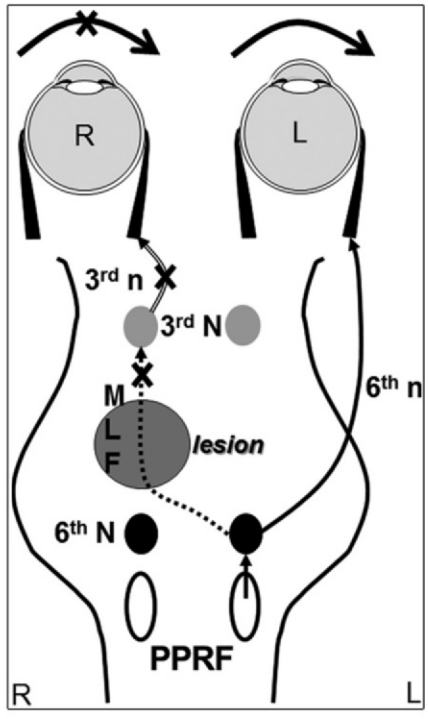

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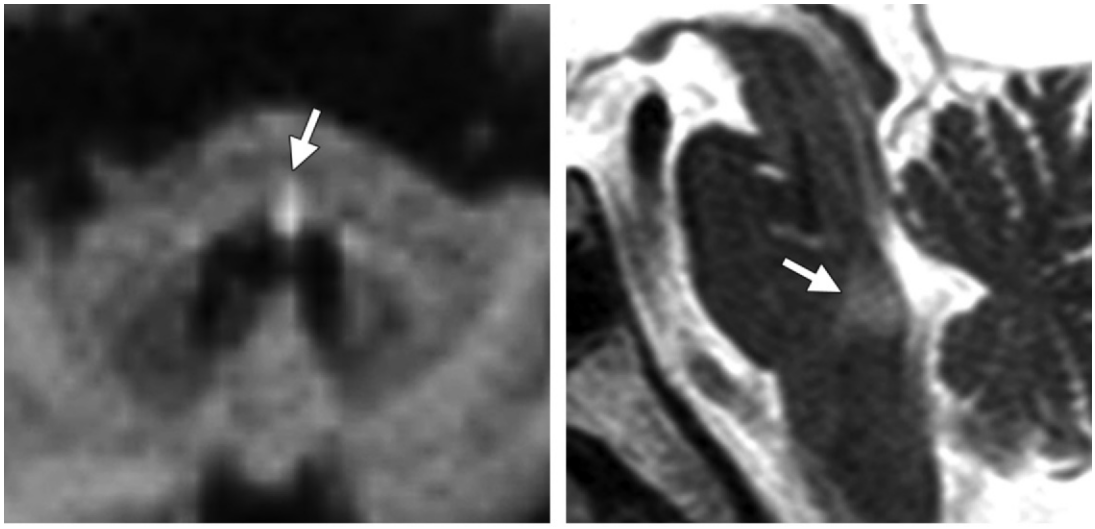
Internuclear Ophthalmoplegia

Internuclear ophthalmoplegia is characterized by the impairment of adduction of the ipsilateral eye and the preservation of abduction of the contralateral eye when the patient tries to gaze in the contralateral direction (36). Internuclear ophthalmoplegia is induced by a lesion involving the MLF in the brainstem; the most common cause is a pontine infarction (Fig 10). However, the disorder can also occur as a result of a demyelinating disease such as multiple sclerosis, which is prevalent in Europe and North America (37).

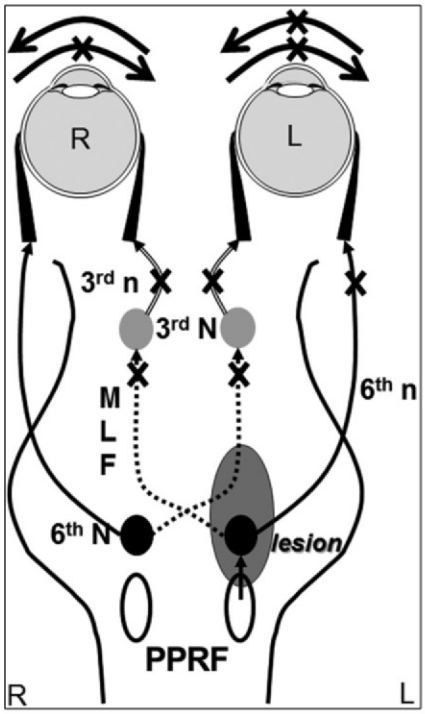
Teaching Point

If one side of the MLF does not function properly, neural signal transmission from the contralateral abducens nucleus is interrupted, which results in a failure to activate the oculomotor nucleus on the same side as the affected MLF. Therefore, adduction performed by the medial rectus muscle is impaired on the same side as the affected MLF, and this impairment leads to internuclear ophthalmoplegia.

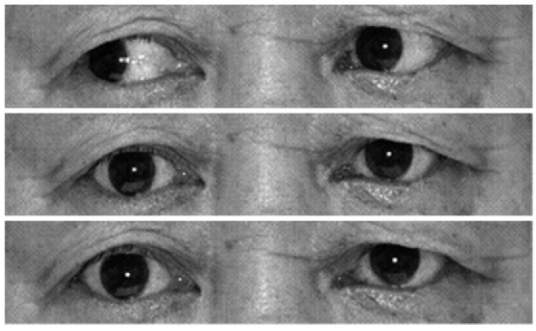
Figure 11. One-and-a-half syndrome in a 63-year-old man. **(a, b)** Axial diffusion-weighted **(a)** and sagittal T2-weighted **(b)** MR images show an acute infarction (arrow) in the facial colliculus area of the left posterior portion of the pons. The infarction may involve both the PPRF (or the abducens nucleus) and the MLF. **(c)** Diagram shows the mechanism for one-and-a-half syndrome: A pathologic lesion (dark gray oval) in the dorsal pontine tegmentum involves both the ipsilateral PPRF (or the abducens nucleus) and the ipsilateral MLF. × = interruptions of signal transmission or eye movement that are a result of the lesion, 6^{th} *N* = abducens nuclei (black circles), 6^{th} *n* = ipsilateral abducens nerve (cranial nerve VI), 3^{rd} *n* = oculomotor nerves (cranial nerves III), 3^{rd} *N* = oculomotor nuclei (small gray circles). **(d)** Top to bottem: Photographs of the patient's rightward, midline, and leftward horizontal gaze show that the patient has impaired movement of both eyes with leftward gaze. With rightward gaze, the patient has impaired adduction of the left eye, while the abduction of the right eye is intact.



a. b.



c.



d.

One-and-a-Half Syndrome

One-and-a-half syndrome consists of lateral gaze palsy in one direction and internuclear ophthalmoplegia in the other direction with a contralateral gaze (38). Therefore, the only horizontal eye movement preserved is abduction of the contralateral eye.

Teaching Point

One-and-a-half syndrome occurs with lesions in the dorsal pontine tegmentum that impair both *(a)* the ipsilateral PPRF or the abducens nucleus and *(b)* the ipsilateral MLF (39). The well-known causes for one-and-a-half syndrome are *(a)* infarctions (Fig 11); *(b)* demyelinating disease, including multiple sclerosis; *(c)* hemorrhage; *(d)* aneurysm or vascular malformation; and *(e)* neoplasm, including metastasis (38,40). The lesions causing one-and-a-half syndrome are somewhat larger than those causing lateral gaze palsy and internuclear ophthalmoplegia

because the lesions have to involve the PPRF or the abducens nucleus and have to extend superiorly to the MLF.

Conclusions

The disorders of horizontal eye movement that are caused by brainstem lesions are classified into three groups: *(a)* lateral gaze palsy, *(b)* internuclear ophthalmoplegia, and *(c)* one-and-a-half syndrome. Lateral gaze palsy is caused by a lesion involving the PPRF or the abducens nucleus. Internuclear ophthalmoplegia results from a lesion involving the MLF. One-and-a-half syndrome (a combination of lateral gaze palsy and internuclear ophthalmoplegia) is caused by a lesion involving both the ipsilateral PPRF (or the ipsilateral abducens nucleus) and the ipsilateral MLF.

Awareness of the brainstem pathways generating conjugate horizontal eye movement helps to diagnose the small pontine lesion. In particular, because a tiny acute infarction in the brainstem is the most common cause of horizontal eye movement disorders, a diffusion-weighted pulse sequence should be added to the MR imaging protocol in elderly patients.

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Brainstem Pathways for Horizontal Eye Movement: Pathologic Correlation with MR Imaging

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Page 48
The main classes of horizontal eye movement disorders caused by brainstem lesions are *(a)* lateral gaze palsy, *(b)* internuclear ophthalmoplegia, and *(c)* one-and-a-half syndrome, a term that refers to the combination of ipsilateral lateral gaze palsy and internuclear ophthalmoplegia.

Pages 52
To sum up, the brainstem pathways for horizontal eye movements start from the abducens nucleus as the horizontal gaze center taking signal from the PPRF. These pathways continue to the ipsilateral abducens nerve (cranial nerve VI) and the contralateral oculomotor nerve (cranial nerve III) through the MLF and end in conjugate horizontal eye movement in the ipsilateral direction to the side of the abducens nucleus.

Page 53
Lateral gaze palsy is caused by a pathologic lesion involving the PPRF or the abducens nucleus (Figs 7, 8) (31).

Page 56
Internuclear ophthalmoplegia is induced by a lesion involving the MLF in the brainstem; the most common cause is a pontine infarction (Fig 10).

Page 57
One-and-a-half syndrome occurs with lesions in the dorsal pontine tegmentum that impair both *(a)* the ipsilateral PPRF or the abducens nucleus and *(b)* the ipsilateral MLF (39).