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CLINICAL FEATURES OF VISUAL AND OCULAR MOTOR DISORDERS

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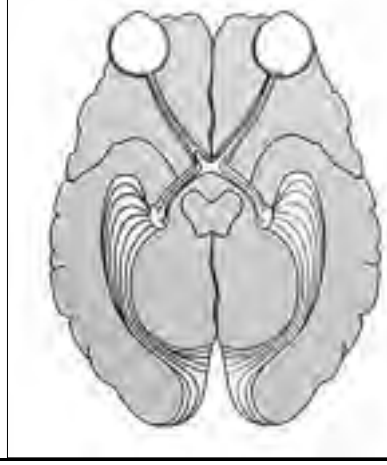
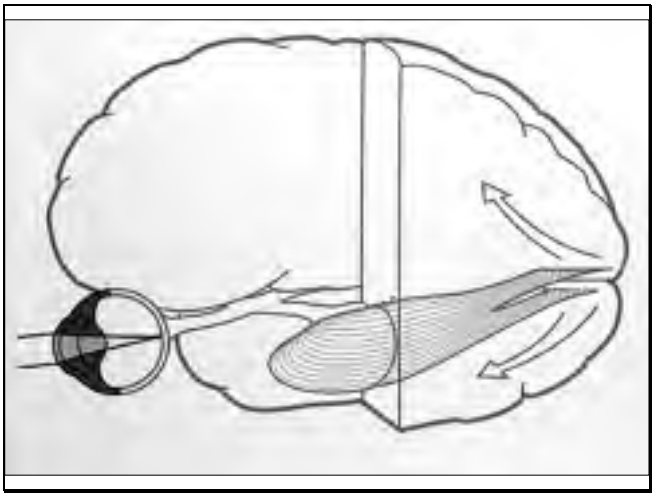
Departments of Ophthalmology and Neurology
University of Michigan Medical School



Here is the visual pathway seen in horizontal (axial) projection. It has three functional portions:
optical, retinocalcarine, and integrative.



The optical portion extends from the cornea to the retina. The retinocalcarine portion extends from the retina to the primary visual cortex. The integrative portion extends from the primary visual cortex to association parietal cortex for spatial vision and to association temporal cortex for recognition.

It is often very difficult to tell from the patient's description of visual difficulty where the defect lies.

Nikki Selewski

Most people who have optical problems will report that vision appears generally "blurred", but sometimes even defects (lesions) in the other portions of the visual pathway will give rise to this symptom.

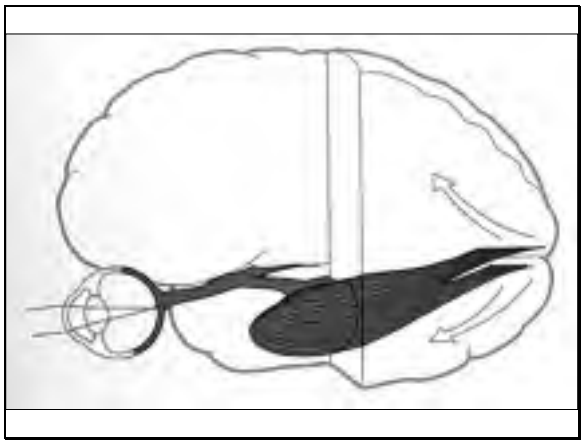
Optical defects arise from two sources: 1) uncorrected refractive errors; and 2) irregularities or opacities in the cornea or lens.

Nikki Selewski

Source Undetermined

One way to tell if the visual problem is optical is to see if the blurred vision disappears when a "multiple pinhole" is placed in front of the eye. If you wear glasses, you can see how this works by comparing your vision before and after "squinting" or before and after looking through a small hole made by your hand.

Refractive errors generally represent a mismatch between the optical elements of the eye and its axial length so that without optical aids, objects viewed at optical infinity are not properly focused on the retina.



This picture shows the retinocalcarine portion of the visual pathway. Defects here are diagnosed in three main ways: 1) looking at the retina and optic nerve with an ophthalmoscope; 2) checking for an afferent pupil defect; and 3) 1) performing visual field examination.

Ophthalmoscopic signs of retinocalcarine disease will be taken up in a later lecture.

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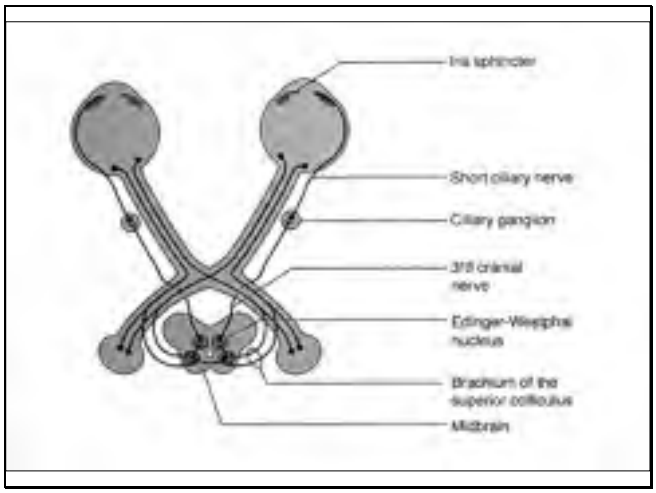

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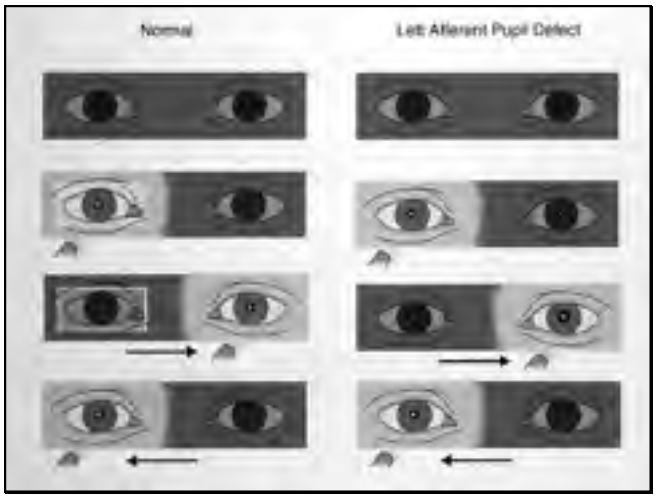
A second way to look for disease in the retinocalcarine pathway is to perform the swinging light test for a relative afferent pupil defect (RAPD).
The basis of this test is the papillary reflex pathway, shown here. Note that the input (afferent) limb is identical with the pathway for vision. But there is a fork from the optic tract to the dorsal midbrain, where input is distributed equally to both sides of the brain stem. The signal then goes to the parasympathetic Edinger-Westphal nuclei whose axons join the somatic axons of cranial nerve 3 and synapse in the ciliary ganglion and then travel on to the iris sphincter.



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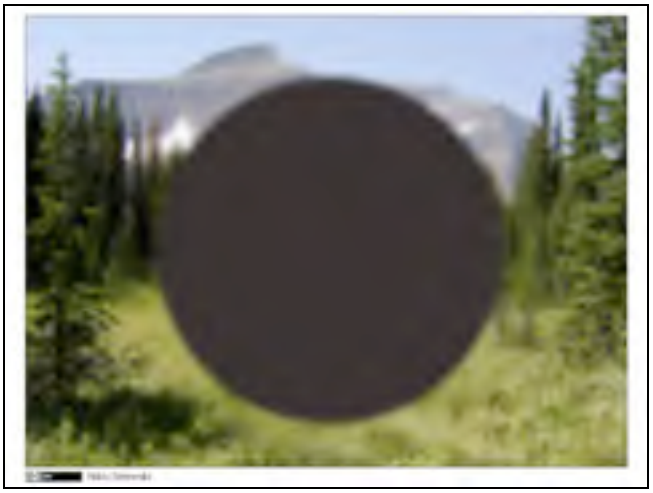
The swinging light test aims to diagnose a RAPD. In the normal subject (left column), light shined first on the right eye, then on the left eye, and back on the right eye, produces no change in pupil size. In a patient with a left RAPD from a left optic nerve lesion (right column), light shined on the left eye produces a dilation of the left pupil. When it is swung back to shine on the right eye, the pupil constricts.

RAPDs come almost exclusively from optic nerve lesions. The point is that the lesion must be asymmetric in the two eyes. It is either unilateral or if bilateral, much more severe in one eye than the other.

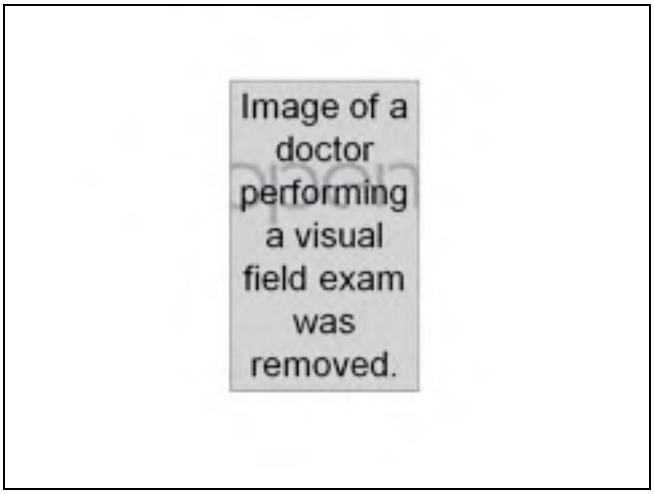


Nikki Selewski

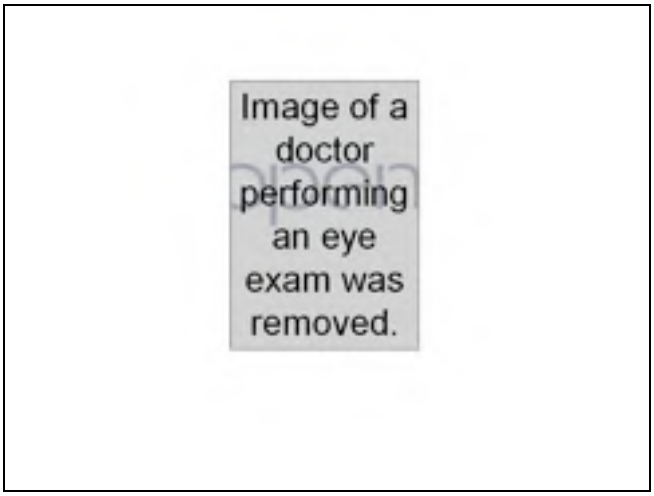
The third way of diagnosing disease within the retinocalcarine pathway is by doing visual field examination. The basis of this technique is that lesions in this part of the visual pathway nearly always produce discrete, or localized, defects in the visual field. The shape of the visual field defect often predicts WHERE in the retinocalcarine pathway the responsible lesion lies.
For example, this is a central scotoma. It foretells a lesion of the retina or optic nerve.

Here is a hemianopic defect. It foretells a lesion of the optic chiasm or retrochiasmal portion of the pathway.

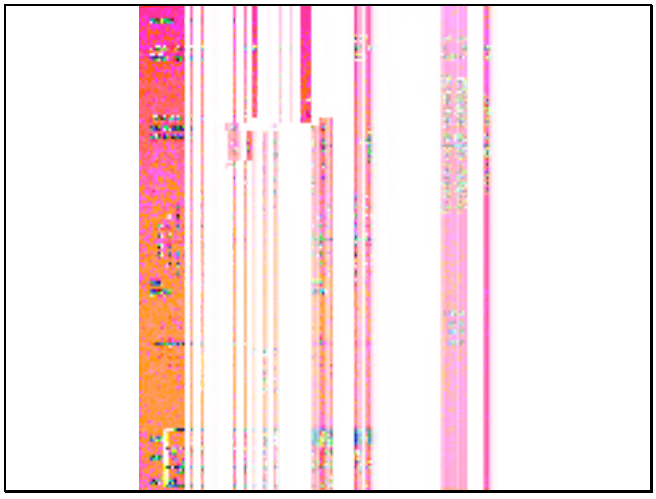
Nikki Selewski



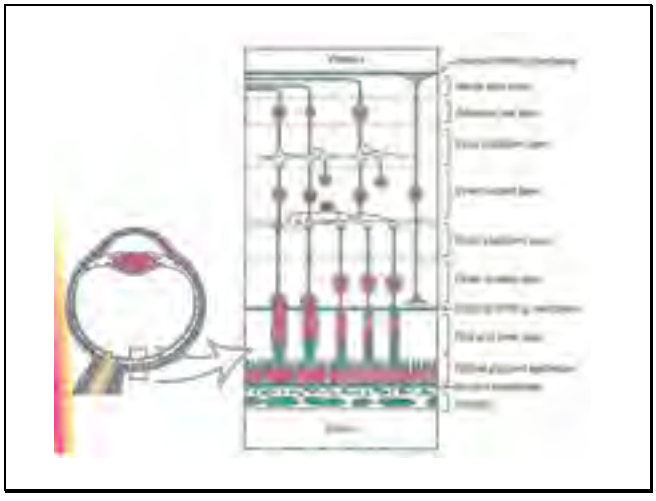
Visual fields can be tested informally by having the patient close one eye at a time and look directly at your open eye. You display one or two vertical fingers briefly within each of the four quadrants of the visual field and ask the patient to identify how many fingers she sees. This is an insensitive method useful only for detecting very large defects.



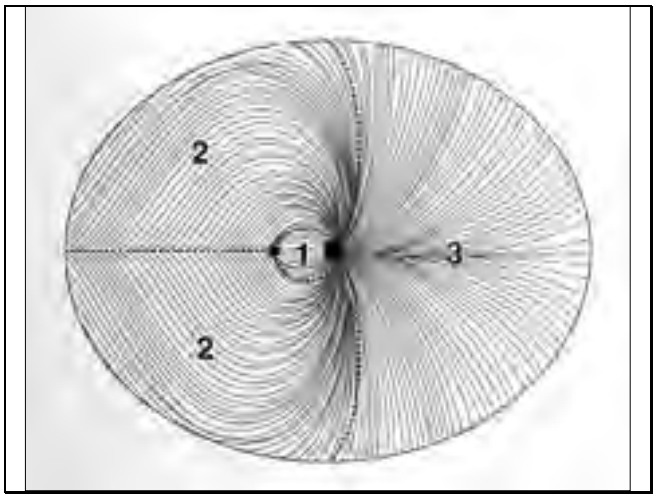
Formal methods of visual field testing are more sensitive and reliable. In the kinetic perimetry, the patient looks at a central dark spot and signals whether she sees a *moving* white spot projected onto a part of the bowl. Most current formal perimetry uses consecutive displays of a *stationary* spot ("static perimetry") because it is more sensitive.



This is the typical display of data generated by static perimetry. It gives the "thresholds" of how well the patient saw in many different positions in the visual field. The physician's job is to see if the abnormally high thresholds (relatively poor vision areas) add up to a bona fide defect and if that defect has a shape that permits localization of the responsible lesion within the retinocalcarine pathway.



Lesions of the outer retina (photoreceptors, bipolar cells and layers in between) cause visual field defects whose shape corresponds to the area that is damaged.



Lesions of the inner retina (retinal ganglion cells and nerve fiber layer) and the optic nerve cause visual field defects that obey the organization of the retinal nerve fiber layer, shown here. Notice that there are three functional compartments: 1) maculopapillar; 2) arcuate; and 3) nasal radial.

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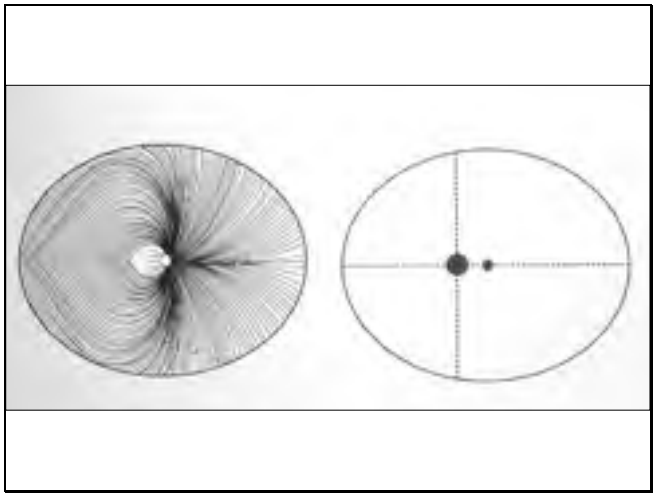

Lesions of the maculopapillar compartment (retinal disorders, toxic, metabolic, inflammatory optic nerve disorders) may produce a central scotoma, shown here.

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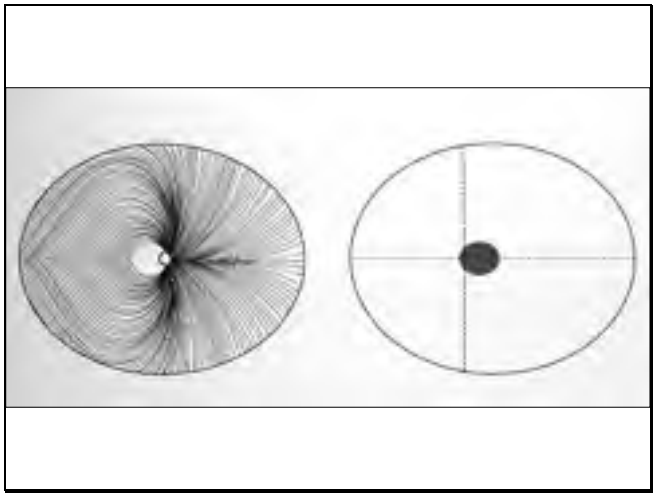

Toxic, metabolic, and inflammatory optic neuropathies often produce a cecocentral scotoma, shown here.

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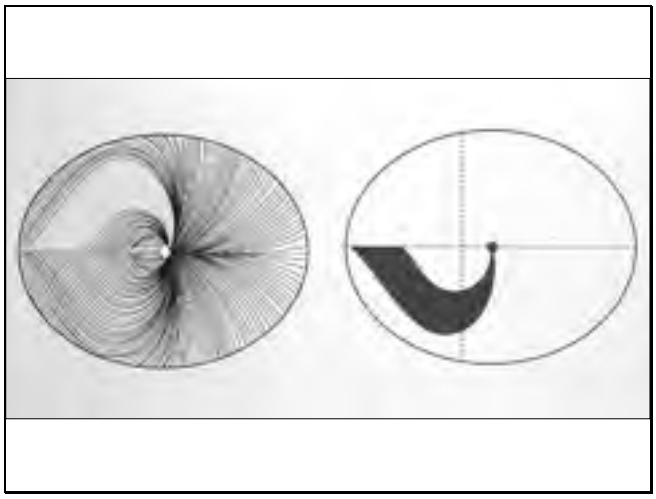

Ischemia, glaucoma, longstanding papilledema (from increased intracranial pressure), and congenital dysplasia often produce arcuate defects, shown here.

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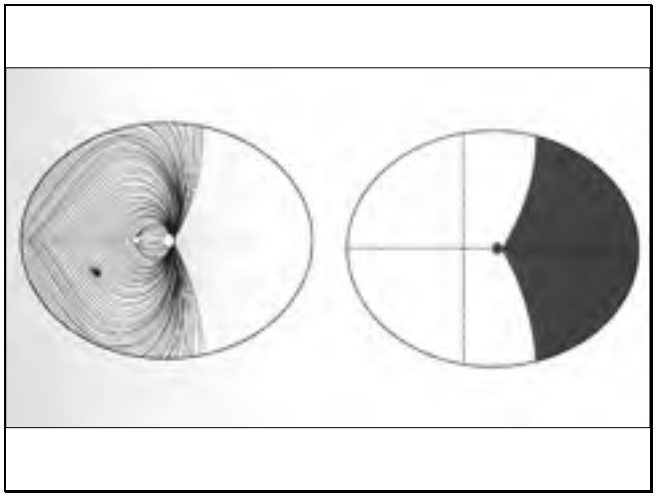

Temporal wedge defects, which result from damage to the nasal radial nerve fiber bundles, are relatively rare. But they often result from congenital dysplastic lesions of the optic nerve.

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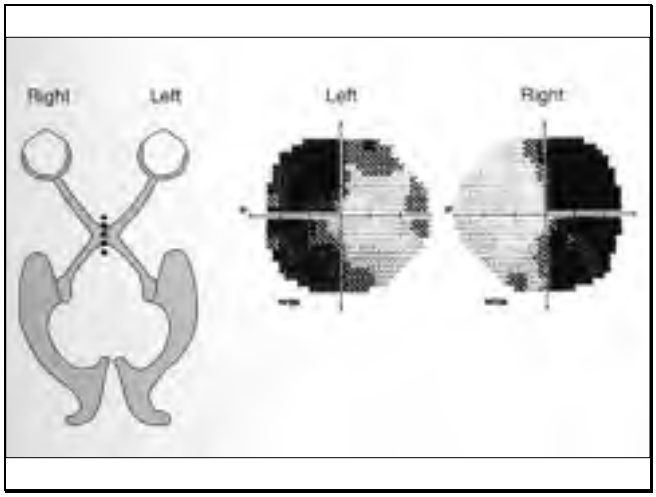

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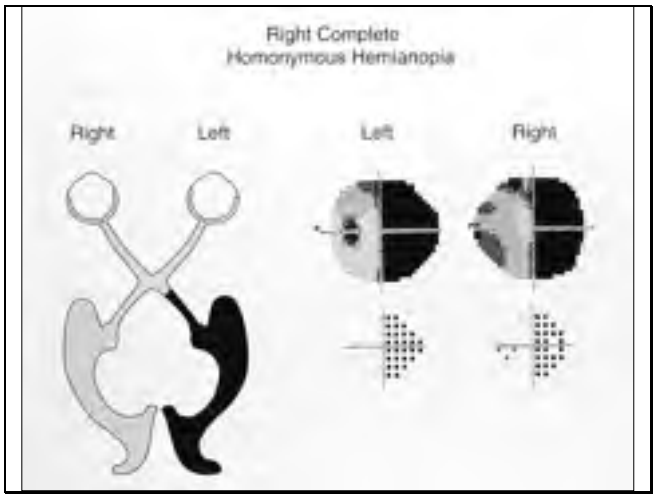
When a lesion affects the optic chiasm, the visual field defect is called a bitemporal hemianopia. Note that the nasal border of the defect in the visual field of both eyes is more-or-less aligned to the vertical meridian passing through fixation. It is that feature that defines a hemianopic defect. It reflects the fact that axons of retinal ganglion cells have become sorted here so that those coming from the nasal retina cross to the opposite optic tract and those coming from temporal retina do not cross. The crossing fibers are always preferentially injured (for reasons that are not fully understood).



Lesions that affect the retinocalcarine pathway posterior to the chiasm (retrochiasmally) produce hemianopic defects on the same side of visual space, or homonymous hemianopias. Although there are some special features of homonymous hemianopias that may allow you to tell where within the retrochiasmal pathway the lesion lies, we will not take up that subject here.

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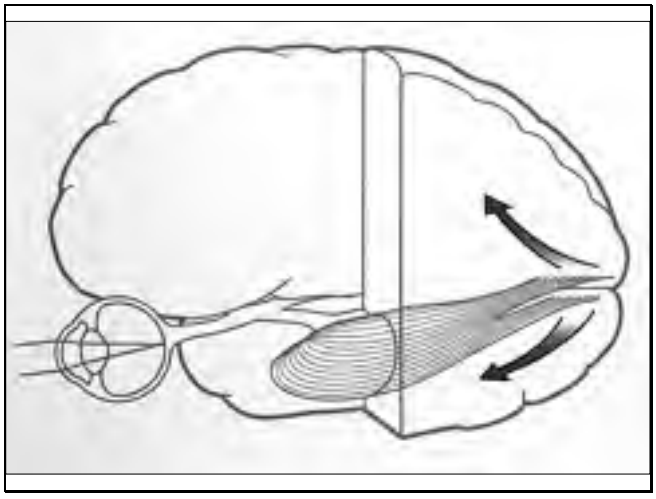

The integrative visual pathway has two components: 1) parietal, and 2) temporal, as shown here by the two arrows.

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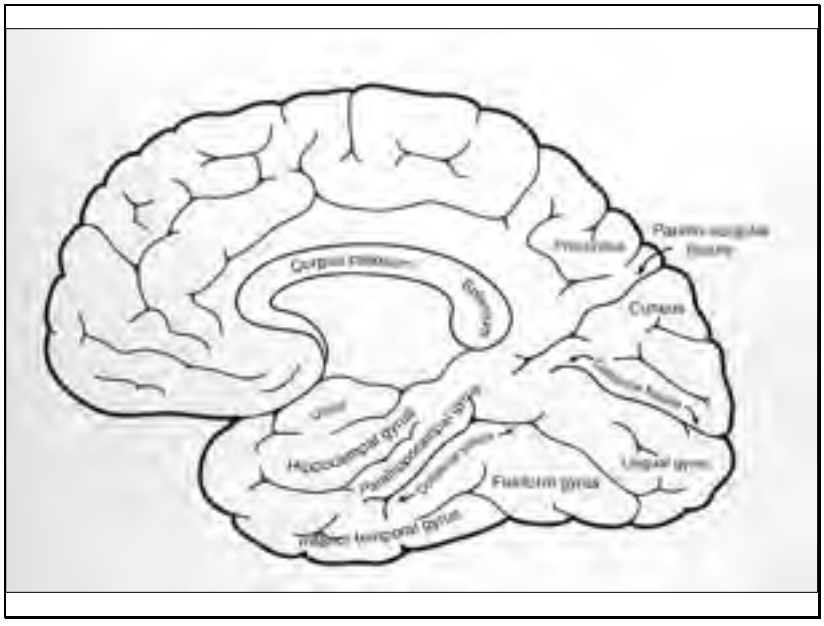

In this medial view of a brain hemisphere, you see some of the gyri involved in visuoparietal and visuotemporal integrative pathways.

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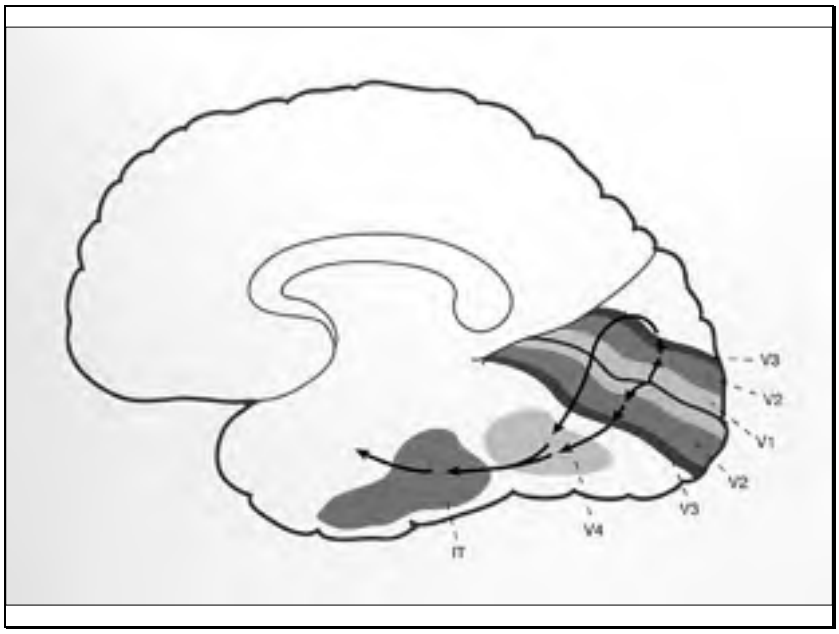
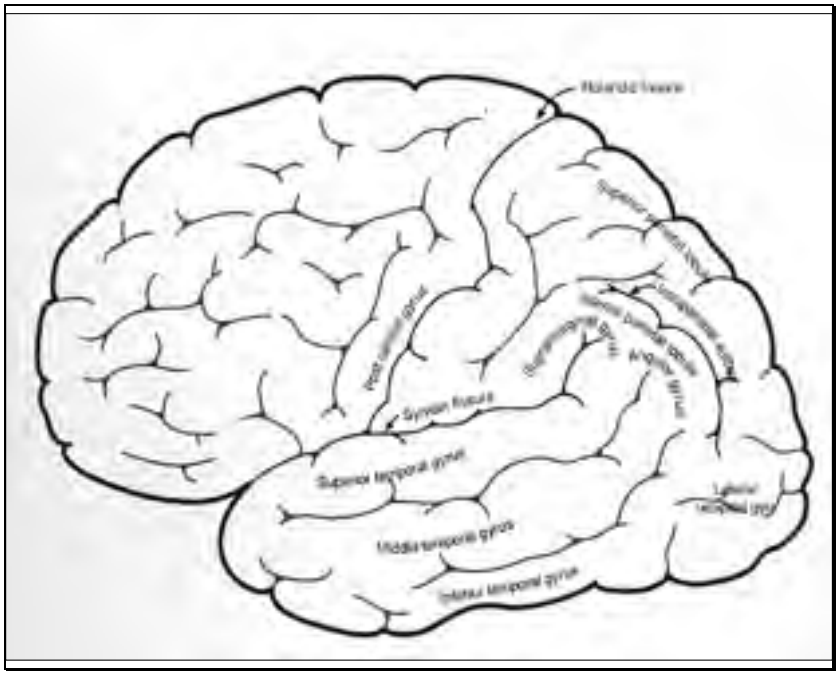
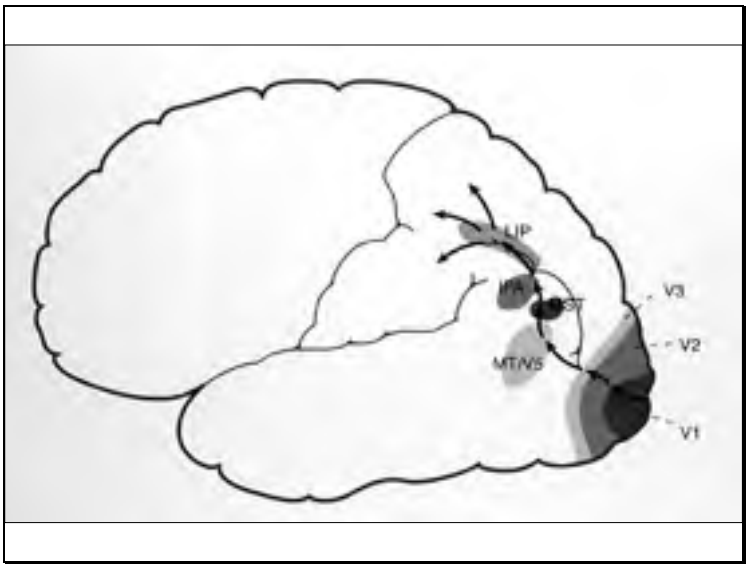


Here you see the waystations in the visuotemporal pathway. They are named after homologous regions in macaque brain.



In this lateral view of a brain hemisphere, you see some of the gyri involved in visuoparietal and visuotemporal integrative pathways.



Here you see the waystations in the visuoparietal pathway. They are named after homologous regions in macaque brain.

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Ocular Motor System

1. Move the Eyes from One Equidistant Target to Another ("Saccades")
2. Move the Eyes from Distant to Near Target or Vice Versa ("Vergence")
3. Keep the Eyes on Target When the Target Moves ("Pursuit")
4. Keep the Eyes on Target When the Head Moves ("Vestibulo-ocular")

The ocular motor system is designed to move the eyes from one target to another and to keep the eyes tracking a moving target. To move the eyes from one target to another, the saccadic system is used. To keep the eyes tracking a moving target, the pursuit system is used. To refixate or follow a target moving toward or away from one's eyes, the vergence system is used. To keep the eyes on a target when one's head moves, the vestibulo-ocular system is used.

Saccades

1. Voluntary
   a. Move eyes to target seen in peripheral field ("visually guided")
   b. Move eyes to unseen target ("non visually guided")
2. Involuntary
   a. Rapid eye movements of sleep
   b. Fast phases of nystagmus
   c. Random

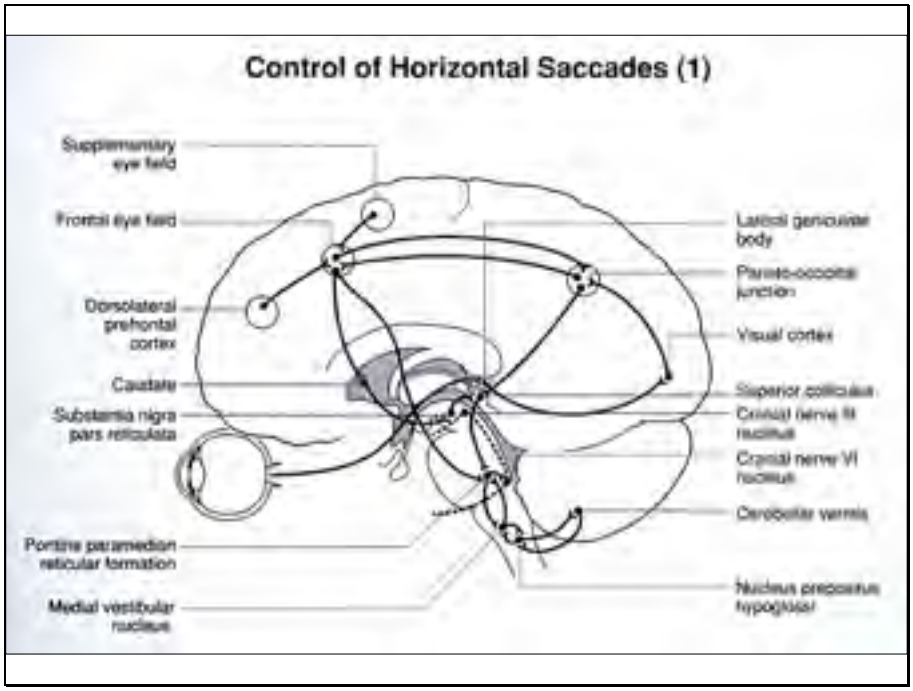
There are two classes of saccades: voluntary and involuntary. Voluntary saccades can occur to a seen target or an unseen target. Involuntary saccades occur during sleep, as the fast phases of nystagmus, and randomly.

Saccades

Test by having patient fixate a stationary target and then look from one target to another

Note whether eyes are still during fixation, and note speed, amplitude, accuracy of refixations, and if oscillations are present

In testing for saccades, first watch the patient's eyes as they fixate a stationary target. There should be no "intrusive" eye movements. Then have the patient look from one stationary target (your finger or light) to another. Watch for speed, amplitude, accuracy of refixations, and oscillations.



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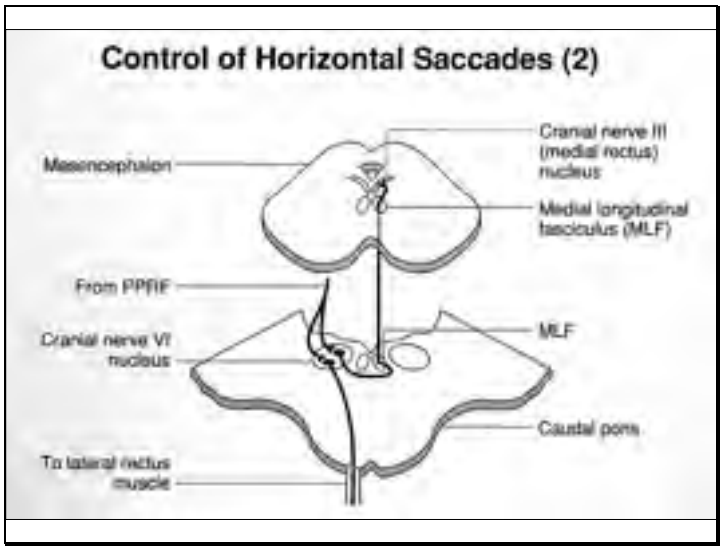
This schematic shows the pathways involved in generating horizontal saccades. There are two centers: parieto-occipital and frontal. The parieto-occipital center generates visually-guided voluntary saccades; the frontal center generates non visually-guided voluntary saccades, but each center does both. Involuntary saccades (nystagmus fast phases, rapid eye movements of sleep) are probably generated in the brain stem.



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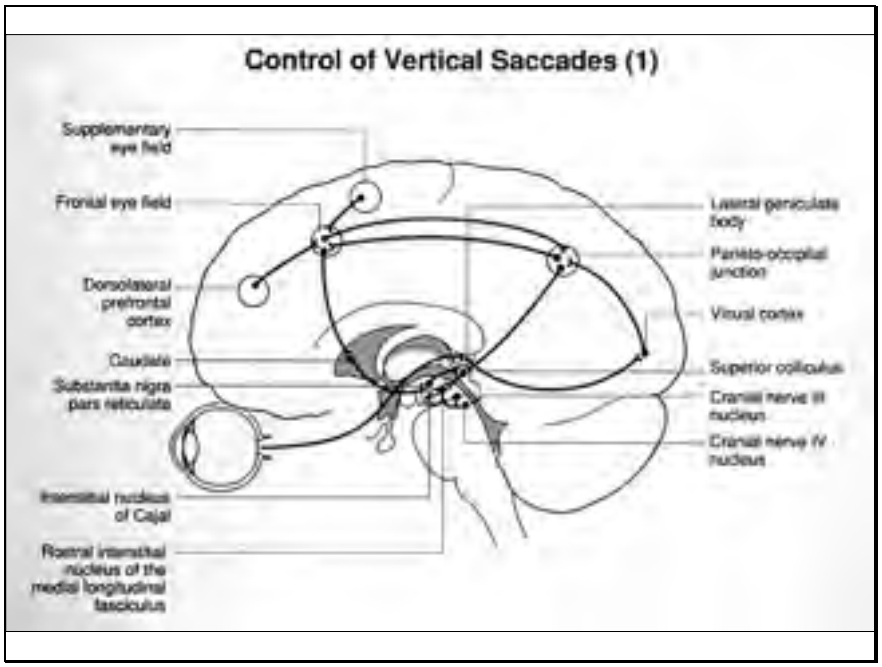
This schematic shows the brain stem pathway for horizontal saccades. From the cerebral centers, the descending pathways cross in the upper brain stem and end in the contralateral pontine paramedian reticular formation (PPRF). From there, they go to the sixth cranial nerve nucleus. From there, the signal is sent via abducens motor neurons to the ipsilateral lateral rectus muscle, and via abducens interneurons through medial longitudinal fasciculus (MLF) to contralateral medial rectus subnucleus of cranial nerve 3.

Within the PPRF are burst cells responsible for the "pulse" of innervation that gets the eyes moving. Another neural network is responsible for the "step" of innervation that maintains the eyes in an eccentric gaze position against the drag of the orbital soft tissues that forces the eyes back toward the center. The pulse and step of saccades are modified by a network involving the medial vestibular nucleus (MVN), nucleus prepositus hypoglossi (NPH), and cerebellar vermis. The role of this network is to make sure that actual and desired eye position are identical.



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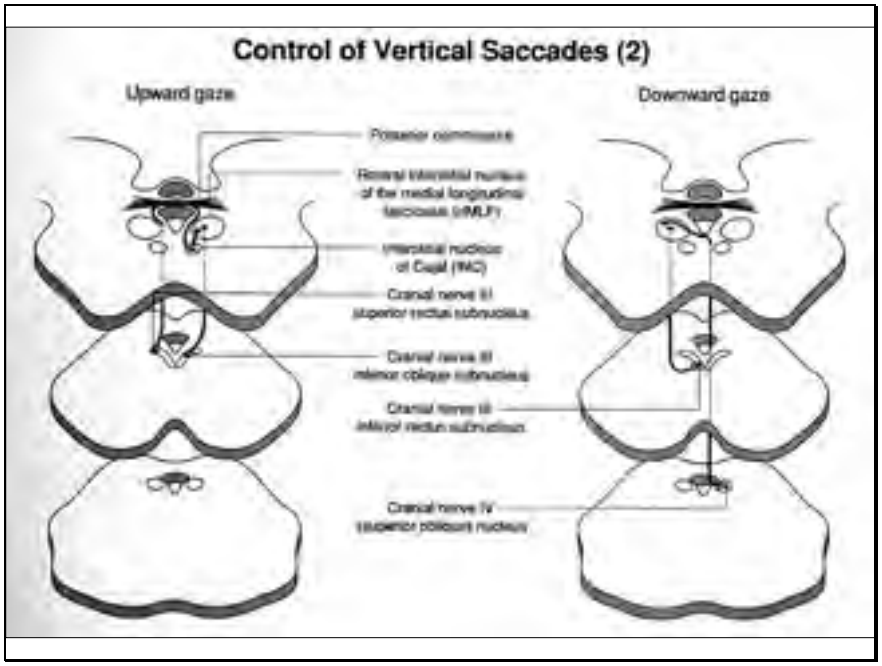
For vertical voluntary saccades, the cortical signal descends via the same pathways as for horizontal voluntary saccades, but its destination is the rostral interstitial nucleus of the medial longitudinal fasciculus (riMLF) in the midbrain. There are separate pathways for upward and downward gaze.



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For upward gaze, burst cells in riMLF send signals through interstitial nucleus of Cajal (INC) and posterior commissure (PC) to end on the superior rectus subnucleus; by means of a separate, more ventral, pathway, signals travel to the inferior oblique subnucleus. For downward gaze, signals travel by means of a pathway that does not go through PC and end on the inferior rectus subnucleus and superior oblique nucleus (cranial nerve 4).

Saccadic Disorders

1. Absent (gaze palsy)
2. Reduced amplitude ("hypometric")
3. Slow
4. Inaccurate ("dysmetric")
5. Intrusive

Saccadic disorders show up as completely absent saccades, saccades of reduced amplitude ("hypometric"), saccades that are not accurate in moving the eyes to a desired new position ("dysmetric"), or occurring when you don't want them, when the eyes are supposed to be still ("saccadic intrusions").

Vergence

1. Move eyes closer to one another ("convergence")

2. Move eyes farther apart from one another ("divergence")

The job of the vergence pathway is to move the eyes from a distant to a near target and vice versa.

Vergence Pathway

1. Generated in both parieto-occipital regions
2. Travels to midbrain
3. Exact pathway not known

The pathway serving vergence is not well known, but is believed to start in the parieto-occipital regions bilaterally and descend to the midbrain. There may be special brain stem nuclei for convergence and divergence.

Vergence Disorders

- Excessive convergence
- Insufficient convergence
- Excessive divergence
- Insufficient divergence

Vergence disorders can produce too much or too little convergence or divergence (see below).

Vergence Disorders

1. Excessive convergence
    a. Congenital defect
    b. Too much accommodation
    c. Loss of vision
    d. Nonspecific brain insult
2. Insufficient convergence
    a. Idiopathic
    b. Nonspecific brain insult

Excessive convergence, the most common vergence abnormality, shows up as esotropia. It may be a congenital defect, occur in certain conditions that provoke excessive accommodation, and after many types of brain insults. Insufficient convergence is a poorly understood disorder that occurs in many otherwise normal subjects. It may also occur in many types of brain insults.

Vergence Disorders

1. Excessive Divergence
    a. Congenital defect
    b. Loss of vision

2. Insufficient Divergence
    a. Nonspecific brain insult

Excessive divergence is usually a congenital or early childhood disorder showing up as exotropia. Many acquired brain disorders can cause insufficient divergence, showing up as esotropia .

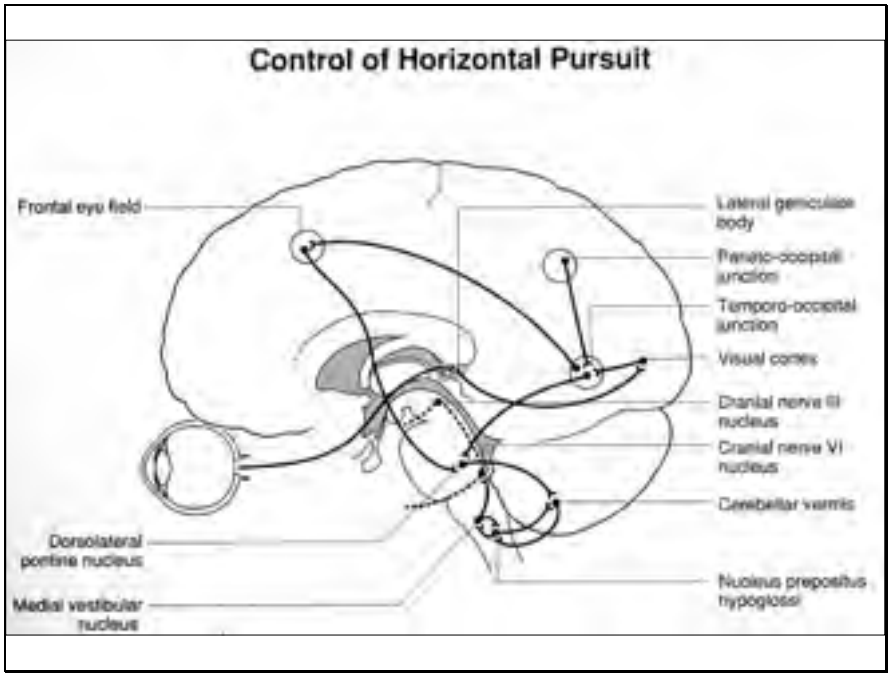
Pursuit

Test by having patient follow
a moving light or finger at about 30 degrees/second

Note smoothness of motion, amplitude of excursion, presence of oscillations

Pursuit is tested by having the patient follow your finger or light at no more than 30 degrees/second.

Check for smoothness of movement, how far the eyes moves, and whether there are any oscillations (nystagmus).



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Pursuit eye movements are initiated exclusively in a center located at the parieto-occipital junction. Signals go through the pulvinar to the ipsilateral dorsolateral pontine nucleus (DLPN). They then travel to cerebellar vermis, to nucleus prepositus hypoglossi (NPH), to medial vestibular nucleus (MVN), and then to cranial nerve 6 nucleus for horizontal pursuit, and to the midbrain for vertical pursuit by uncertain routes.

Pursuit Disorders

1. Cogwheel ("saccadic")
2. Absent

The most common clinical abnormality of pursuit is "cogwheel" or "saccadic pursuit." As a damaged pursuit system fails to keep up with a moving target, the more robust saccadic subsystem takes over.

Vestibulo-ocular

In awake patients with intact voluntary eye movements, can test only with special techniques

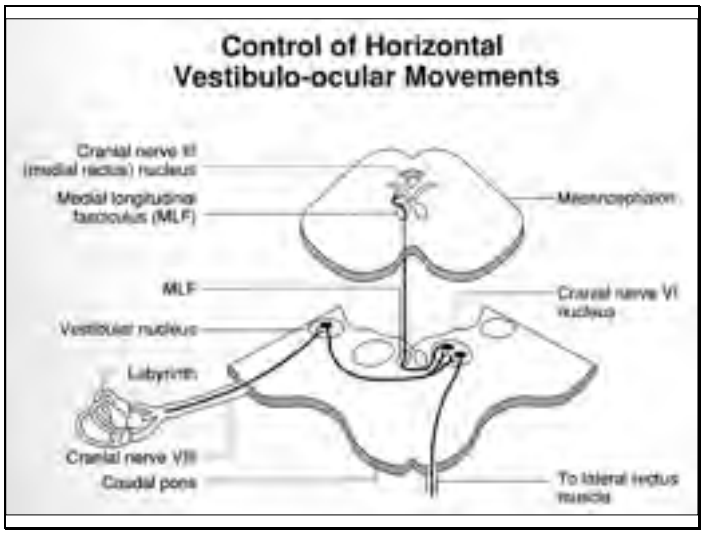
The vestibulo-ocular reflex can be assessed in awake patients only with special techniques such as chair rotation with videonystagmography.

Vestibulo-ocular

In comatose patients and in awake patients with poor voluntary eye movements, two tests:

1. Doll's Head Maneuver. Move head rapidly and look for slow contraversive conjugate eye movements
2. Cold Water Calorics. Look for ipsiversive slow conjugate eye movements and perhaps contraversive involuntary saccades ("nystagmus")

In comatose patients and those without voluntary eye movements, you can assess for an intact vestibulo-ocular reflex by performing the Doll's Eye Maneuver or the Cold Calorics Maneuver.



The signal is generated by semi-circular canals and travels to the medial vestibular nuclei. From there, the signal gets to cranial nerve 6 for horizontal eye movements, and to cranial nerves 3 and 4 for vertical eye movements through uncertain pathways.

Vestibulo-ocular Disorders

- General hypofunction (creates oscillopsia)
- Imbalance (creates nystagmus)

Vestibulo-ocular disorders may be bilateral or unilateral. Bilateral disorders cause jiggly, blurred vision ("oscillopsia") with head movement. Unilateral disorders cause nystagmus. Can you figure out why?

(Here's why. If the lesion is bilateral, as the head moves, the damaged VOR cannot keep the eyes still in space. Viewed objects seem to move. If the lesion destroys only one side, intact side constantly forces the eyes to one side and the nervous system provides a recovery movement in the opposite direction, or *nystagmus,* a rhythmic oscillation of the eyes.)

Supranuclear Gaze Palsy

- Absent voluntary gaze (saccades and pursuit)
- Intact reflex gaze (vestibulo-ocular), elicited by Doll's Head Maneuver or Cold Water Calorics
- Means that brain stem gaze pathways are intact but cerebral gaze pathways are not intact

Here is a final concept. When saccades and pursuit (voluntary) eye movements are absent, yet vestibulo-ocular (reflex) eye movements are intact, the patient has a "supranuclear gaze palsy." Where would the responsible lesion(s) be located to produce this?

Additional Source Information

for more information see: http://open.umich.edu/wiki/CitationPolicy

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