Eye Movements:

Neurology, Abnormalities, Testing

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Session Objectives

The acquired knowledge and skills directly relate to the general objectives for the Ophthalmology rotation. The student shall be able to:

- Identify different types of eye movements and differentiate between normal and abnormal eye movement findings. [General Objectives, c)]
- Perform an eye movement exam. [Essential Objectives, Skills, d)]
- Describe the nature of strabismus and amblyopia and be able to explain these to a patient or parent of a patient. [Essential Objectives, Knowledge, j)]

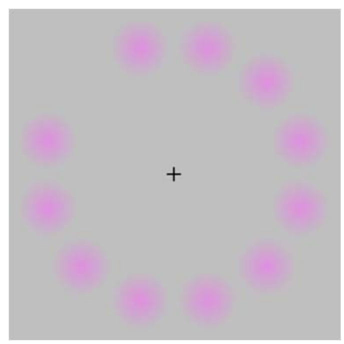
More specifically, the student shall:

- Know the characteristic features of saccades, pursuit, vergence, fixational eye movements, VOR and OKN.
- Be able to name the extraocular muscles and their primary and secondary functions.
- Know the basic brainstem mechanisms driving saccadic eye movements.
- Know the unilateral or alternating cover test, its general principles, and be able to perform it on a patient.
- Be able to identify different types of ocular deviation (tropias and phorias), possible perceptual consequences and clinical management.

Content

- **Human eye movement repertoire**
- Neurology of eye movements
- Common eye movement abnormalities
  - *Clinical skills I: conducting an eye movement exam*
- Strabismus and amblyopia
  - *Clinical skills II: detecting strabismus, measuring acuity and stereovision*

Why do we move our eyes?



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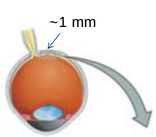
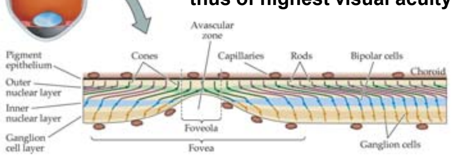
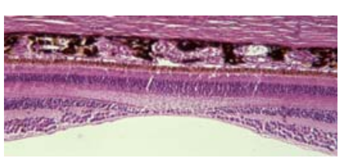


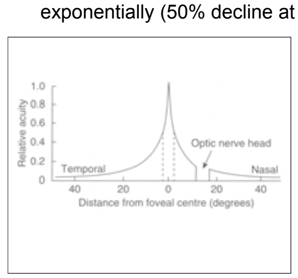
Fovea is the area of greatest photoreceptor (cone) density and thus of highest visual acuity



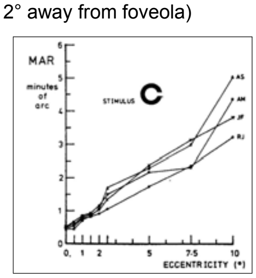


Visual acuity outside the fovea

- as we move away from the fovea, visual acuity drops exponentially (50% decline at 2° away from foveola)



Land & Tatler, "Active vision", 2009



Jacobs, Vision Res 1979

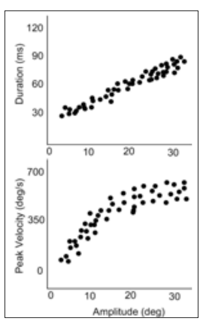
Horizontal saccades, smooth pursuit, VOR, VOR cancellation, convergence.

Leigh & Zee, Neurology of Eye Movements, 2006

Saccades

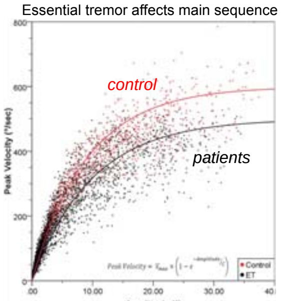
- Fast movements that redirect gaze
- Main types:
  - **Volitional** saccades
  - Predictive / anticipatory saccades
  - Memory-guided saccades
  - Antisaccades
  - **Reflexive** saccades

Saccade main sequence



Carpenter, Eye Movements, 1981

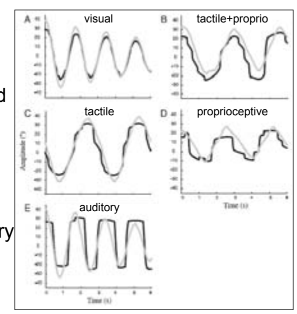
Essential tremor affects main sequence



Gitchel et al. 2013

Pursuit

- Pursuit requires the perception of a *visual* target; imaginary targets can usually not be tracked (but we can make a saccade to an imaginary target)
- Pursuit in response to nonvisual stimuli – auditory, proprioceptive, tactile – is poor



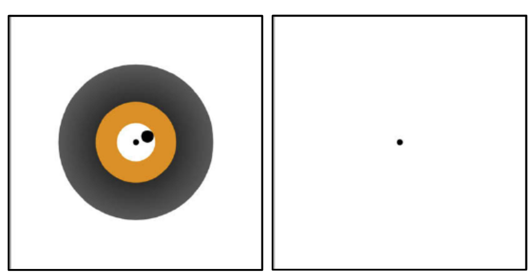
Berryhill et al., J Neurophysiol 2006

Vergence

- Vergence: movements that rotate the eyes simultaneously in opposite direction, for example, to look at targets at different distances (i.e., in depth)
- Binocular movements: 2 eyes are separated by several centimeters; rotation of each eye has to be controlled separately
- Stimulus for vergence: disparity (difference in depth) and blur (accommodation)

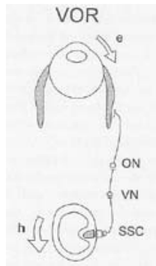
Fixation

- Microsaccades



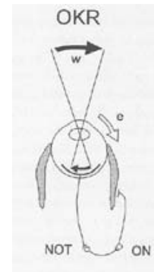
Vestibulo-ocular reflex (VOR)

- Compensation for head movements
- Head movements are detected by the vestibular organ in the inner ear; this organ is specialized to detect movement along all axes
- Acceleration in any given plane will activate extraocular muscles to counteract this movement



Optokinetic reflex (OKR)

- Fast movements that compensate for retinal image motion from large-field motion



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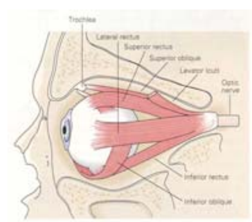
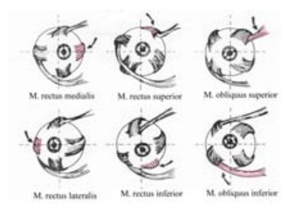
Orbital mechanics – how does the eye move?

- 3 pairs of extraocular muscles move the eye and hold it in place at eccentric positions





horizontal / vertical

oblique (torsion)

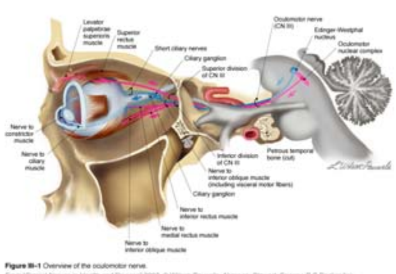
Oculomotor nerve function and third nerve palsy

- CN III (oculomotor nerve) controls:

medial rectus
superior rectus
inferior rectus
inferior oblique

levator muscle of the eyelid

pupillary sphincter muscles



Oculomotor nerve function and third nerve palsy

- impaired CN III function can result in:

droopy eyelids (ptosis)

dilated and poorly reactive pupil

horizontal and vertical diplopia

impaired ability to move the eye up (elevate), down (depress) and in (nasal); eye will turn out and down

Trochlear nerve function and fourth nerve palsy

- CN IV (trochlear nerve) controls:

superior oblique

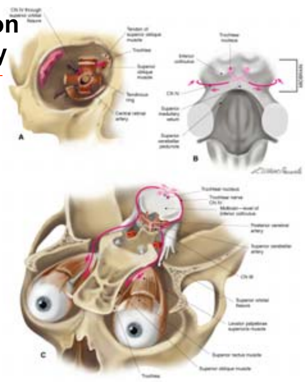


Figure IV-1 A. Apex of the right orbit illustrating the tendinous ring. B. Dorsal aspect of the brain stem. C. Somatic motor tract from the trochlear nucleus in the brain stem to the superior oblique muscle.

From "Cranial Nerves in Health and Disease" 2002, © Wilson-Pauwels, Akesson, Stewart, Spacey, B C Decker Inc.

Trochlear nerve function and fourth nerve palsy

- impaired CN IV function can result in:

vertical diplopia *(following complete paralysis); most pronounced in downgaze; patient may tilt head toward opposite shoulder to minimize double vision*

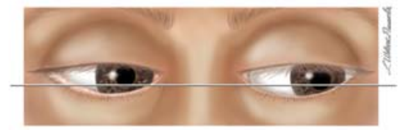


Figure IV-6 Harpinder is unable to move her right eye downward.
From "Cranial Nerves in Health and Disease" 2002,
© Wilson-Pauwels, Akesson, Stewart, Spacey, B C Decker Inc.

Abducens nerve function and sixth nerve palsy

- CN VI (abducens nerve) controls

lateral rectus

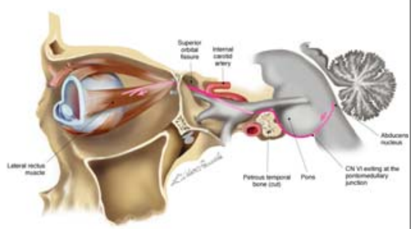


Figure VI-1 Overview of the abducens nerve.
From "Cranial Nerves in Health and Disease" 2002, © Wilson-Pauwels, Akesson, Stewart, Spacey, B C Decker Inc.

Abducens nerve function and sixth nerve palsy

- impaired CN IV function can result in:

horizontal diplopia *(following complete paralysis); most pronounced when gaze is directed towards affected side*

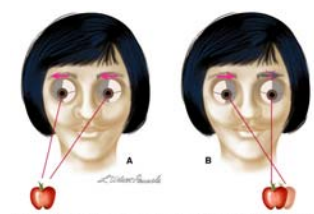
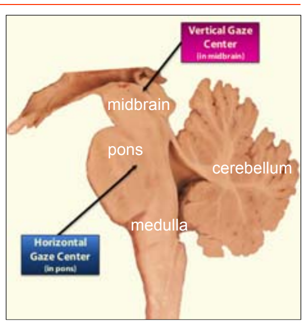


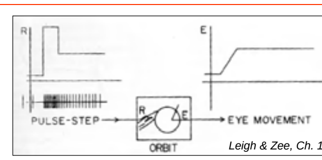
Figure VI-6 A. When looking to the right, Grace was able to direct both eyes toward the same object. **B.** On attempted left lateral gaze, Grace was unable to abduct her left eye due to paralysis of her left lateral rectus muscle; therefore, she experienced double vision.
From "Cranial Nerves in Health and Disease" 2002, © Wilson-Pauwels, Akesson, Stewart, Spacey, B C Decker Inc.

Brainstem centres for saccade generation

- Most eye movements are either horizontal or vertical
- Vertical gaze control is in the midbrain (midbrain reticular formation)
- Horizontal gaze control is in the pons (paramedian pontine reticular formation)



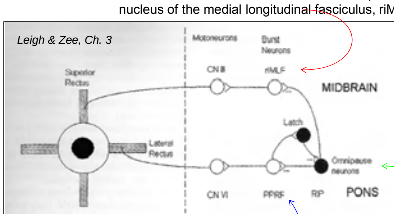
Brainstem saccade control: model



- Saccades are generated by a pulse-step innervation
- Pulse (**velocity command**) invokes phasic muscle contraction that moves the eye rapidly against viscous drag imposed by orbital tissues
- Step (**position command**) invokes tonic muscle contraction that opposes elastic forces of the orbit and holds eye steadily at new position
- Normal saccades require that both the pulse and the step are accurately programmed and matched to each other

Brainstem burst generators

Motoneurons innervating vertically acting extraocular muscles receive saccadic commands (pulses of innervation) from burst neurons in the midbrain reticular formation (rostral interstitial nucleus of the medial longitudinal fasciculus, riMLF)



Motoneurons innervating horizontally acting extraocular muscles receive saccadic commands from burst neurons in the paramedian pontine reticular formation (PPRF)

Omnipause neurons in the nucleus raphe interpositus (rip) inhibit both sets of burst neurons

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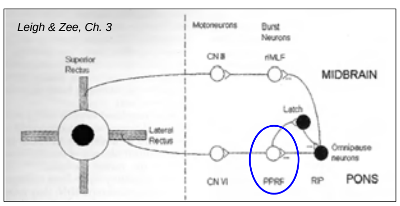
Effect of PPRF lesions



Slow or absent horizontal saccades

esp. in hereditary degenerative disorders involving the pons

(e.g., spinocerebellar ataxia, Huntington's disease, Parkinson's disease, PSP, Whipple's disease, Wilson's disease, Tetanus, dementia, MS, ALS, peripheral nerve palsies, etc.)

Slow horizontal eye movements in SCA Type 2

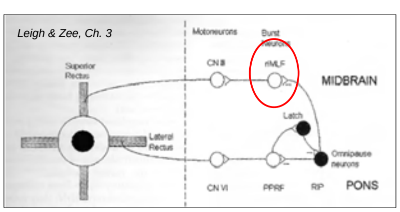
horizontal gaze palsy: lesion in pons

Leigh & Zee, 2006

Note:

- Slowed horizontal saccades with normal amplitude *(full ocular motor range)*
- Vertical saccades are faster than horizontal saccades
- Curved (L-shaped) trajectories of diagonal saccades

Effect of riMLF lesions



Slow or absent vertical and torsional saccades

in disorders involving midbrain lesions

(e.g., lipid storage diseases such as Tay-Sachs and Niemann-Pick Type C)

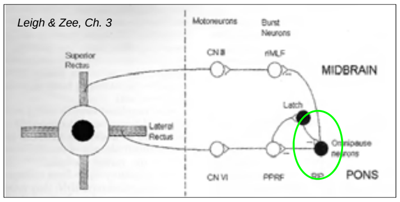
Slow vertical saccades in Niemann-Pick Type C disease

vertical gaze palsy: lesion in midbrain

Note:

- Curved and slow vertical saccades, esp. downward
- Vertical gaze holding preserved

Effect of omnipause neuron lesions



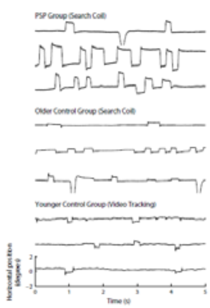
Saccadic oscillations or slow saccades

Loss of OPN inhibition may lead to flutter and opsoclonus

Note:

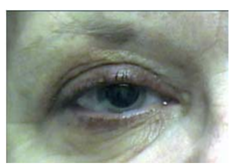
- Back to back oscillations without an intersaccadic interval with horizontal, vertical and torsional components
- Oscillations occur in all directions (opsoclonus), not just along the horizontal (ocular flutter)

Saccadic intrusions: square-wave jerks



Otero-Millan et al. J. Neurosci. 2011

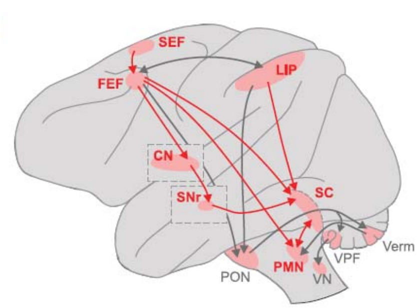
- square-wave jerks consist of horizontal saccade pairs
- most common form of saccadic intrusion in neurodegenerative diseases



Clinical disorders of saccades

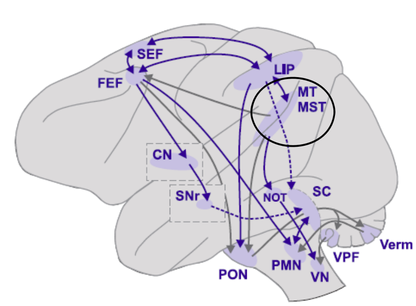
- Disorders of speed
  - Slow saccades
  - Transient decelerations
- Disorders of accuracy (amplitude)
  - Hypometric saccades
  - Hypermetric saccades
- Disorders of initiation
- Saccadic intrusions / saccadic oscillations

Saccade control in the brain



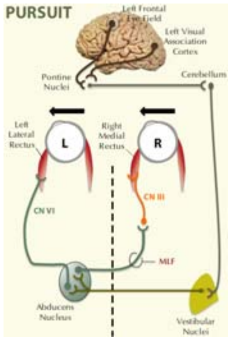
Krauzlis, J Neurophysiol 2004

Pursuit control in the brain



Krauzlis, J Neurophysiol 2004

Pursuit control



- ipsilateral FEF and MT/MST to ipsilateral pontine nuclei (brainstem)
- pontine nuclei to contralateral cerebellum
- cerebellum to ipsilateral vestibular nuclei (brainstem)
- vestibular nuclei to contralateral abducens nucleus
- rest is the same as for saccades

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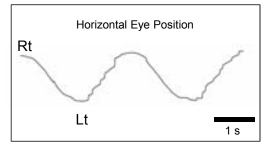
Impaired pursuit and VOR cancellation following cerebral hemispheric lesion

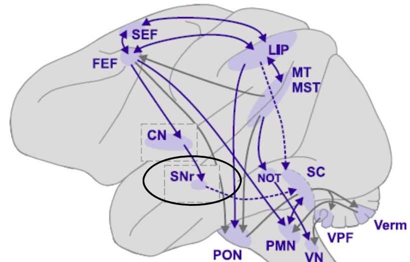


- impaired pursuit (jerky) and VOR cancellation (note saccades) to right (**ipsilateral to side of lesion**)
- same patient also showed hypometric saccades to left (contralesional; not shown)

Impaired pursuit and VOR cancellation following cerebral hemispheric lesion

- same patient showed hypometric saccades to left (contralesional)

Pursuit control in the brain



Krauzlis, J Neurophysiol 2004

Ocular motor abnormalities in disease of the basal ganglia

Note:

- Normal horizontal gaze, but difficulty making self-paced saccades (second half of the movie); saccades are hypometric

Conducting an Eye Movement Exam

- Ocular alignment: normal?
- Range of movement of either eye: limited?
- Range of conjugate gaze: able to look in all directions with either eye?
- Nystagmus: present?

Harper, 2010; Ch. 7 (Neuro-Ophthalmology)

Conducting an Eye Movement Exam

- Ocular alignment: normal?
- Fixation: stable?
- Range of movement of either eye: limited?
- Range of conjugate gaze: able to look in all directions with either eye?
- Saccades / pursuit / vergence / VOR
- Nystagmus: present?

Harper, 2010; Ch. 7 (Neuro-Ophthalmology)

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Strabismus

List the clinical features of strabismus:

eye misalignment: *both eyes cannot be directed toward an object of regard*

other features are: loss of binocular sensitivity, diplopia (in adult-onset strabismus), unstable gaze, impaired eye movements

Types of strabismus

- Concomitant strabismus (non-paralytic): angle / degree of misalignment is equal in all directions; normal extraocular muscle functioning
- Incomitant strabismus (paralytic): degree of misalignment varies with direction of gaze; usually indicates a neurological disorder (such as third nerve paresis)
- Other subtypes
  - Horizontal: exotropia (outwards, away from midline) and esotropia (inwards, towards the nose)
  - Vertical (less common): hypertropia (upward) and hypotropia (downward)

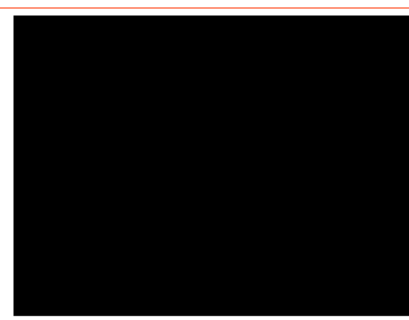
Conditions associated with strabismus: esophoria & exophoria

- phoria: latent tendency for eye misalignment that becomes manifest only if binocular vision is interrupted (e.g., by **alternating cover test**)

esophoria: *latent* convergent strabismus, tendency to drift inwards (esodeviation)

exophoria: *latent* divergent strabismus (exodeviation)

Infantile strabismus – esotropia



Courtesy of Dr. Larry Tychsen, St. Louis Children's Hospital, Washington University Medical Center

Amblyopia

What is amblyopia?

reduced visual acuity in one eye *that cannot immediately be corrected by lenses and occurs in the absence of a detectable organic disease*

What causes amblyopia?

deprivation of normal vision for a prolonged period during development before the age of 8 years

- strabismic amblyopia (40%)
- refractive amblyopia
- a combination of both
- form-deprivation amblyopia due to congenital cataract, corneal scarring, or ptosis

How is amblyopia treated?

- remove amblyogenic factors
  - anisometropic: refraction–initially for 4 weeks
  - strabismic amblyopia: patching, surgery
    spared stereopsis may predict successful surgery
    Kim et al. 2014
- occlude clinically unaffected fellow eye to improve visual acuity (TRADITIONALLY before the age of 8 years)
  - no definitive guidelines for occlusion therapy (2 hrs to all day)
  - patch until visual acuity is equal OR until reverse amblyopia in the fellow eye
- topical atropine is an option when patching compliance is poor: higher acceptability, lower cost, slightly slower rate of improvement

http://pedig.jaeb.org/

Pedig 2002; 2003

Clinical management

eye misalignment

- surgery (shortening, lengthening or repositioning extraocular muscles)
- glasses
- vision therapy

amblyopia

- patching (for more information see also http://pedig.jaeb.org/ - webpage of the Pediatric Eye Disease Investigator Group)
- vision therapy

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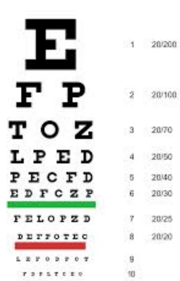
Testing Visual Acuity

- visual acuity – measurement of the smallest object a person can identify at a given distance from the eye
- visual field



How to test visual acuity in children

- newborns

  general ocular status (light reflex, pupillary testing, fundus)

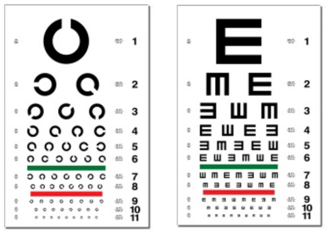
- infants – 2 y/o

  assess basic visual function

  ocular motility

How to test visual acuity in children

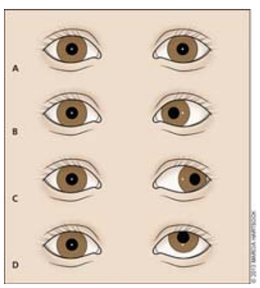
- children (pre-literate)
  - picture cards
  - tumbling E
  - Landolt C
  - HOTV test
- children (5 and older)
  - Snellen



Color and stereovision

- color vision, e.g., Ishihara plates (an online example can be found here: http://www.color-blindness.com/ishihara-38-plates-cvd-test)
- stereo vision, e.g., Randot test or Stereo Fly test for fine, local stereopsis

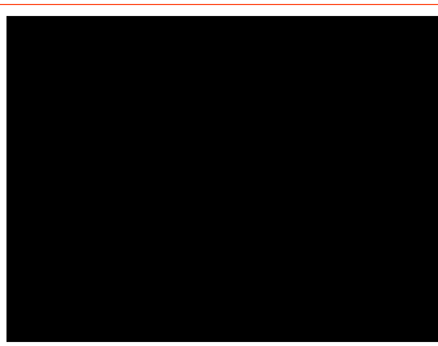
Corneal light reflex test



Cover tests

- Single cover (cover-uncover) test:
  - usually performed first
  - the presence of any movement in a single cover test (in the uncovered eye!) indicates a ***tropia***; i.e., contralateral eye will move to pick up fixation when the fixing eye is occluded
  - in the case of ***phoria*** the uncovered eye does not move but the covered eye moves under the occluder and returns to straight position when occluder is removed
- Alternating cover test (ACT)
  - performed after single cover test
  - switch between eyes to break fusion, cover each eye for several seconds to allow non-occluded eye to p/u fixation

Alternating cover test reveals exophoria



Thank you!

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