Role of motor cortex in voluntary movements
Eye movement and eye gaze direction system

Prof Maja Valić
Role of motor cortex in voluntary movements

• Siegel and Sapru, chapter 19
Characteristics of voluntary movements

• Organized around the performance and purposeful task

• The effectiveness of voluntary movement improves with experience and learning

• Unlike reflexes, voluntary movements can be generated internally
Performance of the voluntary movement depends on:

1. **Motivation** – purpose of the movement (association, limbic and para-limbic system)

2. **Planning** of the movement (pre-motor area)

3. **Performance** of the movement (primary motor cortex, the cortico-spinal tract)
Main role of the motor cortex

• Movement coordination
• Improvement of the performance
• **Lower motor neurons** - innervate skeletal muscle

• **Upper motor neurons** - neurons of the brain that innervate lower motor neurons of the spinal cord and brainstem, either directly or through an interneuron

• may arise from the brainstem or cerebral cortex
The pathways of importance

• from the cerebral cortex

• The most significant of all the upper motor neurons are those that arise: corticospinal tracts and corticobulbar tracts.
The pathways of importance

• From brainstem
  • medial and lateral Vestibulospinal tracts
  • medial and lateral Reticulospinal tracts
  • Rubrospinal tract
  • Tectospinal tract
• Two other systems play important roles in the regulation of motor functions: basal ganglia and cerebellum.
The Corticospinal Tract - crucial for the expression of precise, voluntary movements.

1. From where does this tract originate?
2. What is the anatomical organization of the tract?
3. How are the fibers distributed within the spinal cord?
4. What are the important sources of inputs?
5. What are the differential contributions of the descending components?
6. What are the clinical manifestations of lesions that affect the corticospinal tract?
Origin of the Corticospinal Tract

- 30% of the fibers arise from the precentral gyrus (area 4, primary motor cortex “MI”).
- 40% arise from the postcentral gyrus (primary somatosensory cortex [S-1])
- 30% from supplemental motor area (SMA, area 6 and the premotor cortex PMC)

MI = primary motor cortex;
PMC = premotor cortex;
PPC = posterior parietal cortex;
SI = primary somatosensory receiving area;
SMA = supplementary motor area.
Primary motor cortex (MI)

- Responsible for performance of the movement
- Located in *gyrus precentralis*
- Electrical stimulation of the dorsal and medial aspect in humans produces movements associated with the lower limb
  - stimulation of more lateral aspects produces movements of the upper limb.
  - stimulation of the far lateral aspect produces movements of the face and tongue.
Somatotopical organization – motor homunculus
Non-primary motor cortex

Brodman’s field 6, in front of field 4
- MII (premotor area), dorzolateral surface
- SMA (suplemental motor area), medial surface

Responsible for planning and programing!
Brodman’s field 8

• Frontal cortex
• Frontal eye field (FEF) and Suplemental eye field (SEF)
Course of the Corticospinal Tract

1. capsula interna
2. crus cerebri
3. basis pontis
4. pyramis m.oblongate
at the lower brainstem 80-90% axons cross over to the opposite side in decussatio pyramidum and make tractus corticospinalis lateralis - lateral corticospinal tract

10-20% fibers reach the spinal cord as anterior corticospinal tract (tractus corticospinalis ventralis)
Figure 18-8 Fibers that originate in the primary motor cortex and terminate in the ventral horn of the spinal cord constitute a significant part of the corticospinal tract. The same axons are at various points in their projection part of the internal capsule, the cerebral peduncle, the medullary pyramid, and the lateral corticospinal tract.
Distribution of the Corticospinal Fibers Within the Spinal Cord

FIGURE 19-5  The distribution of axon terminals in the spinal cord of the monkey (shown as dots in spinal cord) as determined by autoradiographic tracing procedures. Depicted also are the sites of origin of the pathway in the motor and somatosensory cortices (top). Most fibers, which are uncrossed (2% of the total corticospinal tract), pass in the anterior corticospinal tract and terminate mainly in the gray matter in the medial aspect of the ventral horn, contacting neurons that innervate axial and proximal muscles, and also in the dorsal horn, contacting somatosensory neurons (bottom). Crossed fibers also supply both the dorsal and ventral horn. Fibers that issue from the postcentral gyrus (depicted in purple) supply the dorsal horn (also shown in purple), whereas those that arise from the motor cortex (depicted in red) supply the ventral horn (also depicted in red).
Characteristics of the motor fields

• Cytoarchitecture (hystology)
• Electrophysiologic characteristics
• Specific cortico-cortical projections
• Afferent-eferent neuronal projections
• Specific motor deficits after injuries of specific area
Histology of the Motor Cortex

• the cerebral cortex typically has six layers
• two layers of granule cells (an external and internal), which receive information mainly from the thalamus and other regions of the cortex.
• two layers of pyramidal cells (an external and internal), which serve as the origins of the efferent pathways of the cortex.
• The corticospinal tracts arise from the internal pyramidal cell layer situated mainly in layer V.
Histology : MI, MII i SMA

• **Agranularity and disgranularity** (no II and IV granular layer)

• **Magnocelularity** (contain big piramidal cells)

• V. layer of the MI contains gigantic Betz pyramidal neurons
Figure 3.6
Pyramidal and non-pyramidal neurons in motor cortex. The cerebral cortex is organized into six layers. These layers contain different proportions of the two main classes of cortical neurons, pyramidal and non-pyramidal cells. Pyramidal cells send long axons down the spinal cord and are the major output neurons. They are abundant in layer 5. Non-pyramidal cells have axons which terminate locally.
Afferent and efferent connections of the motor cortex
Primary Motor Cortex

• The primary motor cortex receives indirect inputs from the cerebellum and globus pallidus via the VL nucleus of the thalamus.

• The integration of all of these inputs occurs within the primary motor cortex (rather than in the thalamus).
Primary Motor Cortex

• Discharge of neurons within the basal ganglia and cerebellum occurs prior to neuronal discharges in the motor cortex.

• The neuronal discharges in the motor cortex also precede the motor response.

• Cerebellar and basal ganglia inputs provide the motor cortex with a **planning mechanism** for the **initiation** and **regulation** of a given response pattern.
Primary Motor Cortex

• The primary motor cortex also receives **somatosensory** afferents.
• These afferents enables a given region of motor cortex to receive **proprioceptive** and **tactile** inputs that relate to the specific muscle groups or body parts.
• Inputs also involve conscious proprioception, position sense, pain, and tactile information.
Brodm. area 2 sends somatosensory information into the primary motor – inhibition of neural activity in the area 2 results with the loss of coordinated movements of the fist.

Principles of Neural Science, Kandel, Schwartz, Jessell.
**Supplementary and Premotor Area Cortices**

- **SMA** and **PMC** (Area 6) make 30% of the corticospinal fibers
- **Afferent inputs**
  - 1.) Inputs from the **basal ganglia** directed mainly to the **SMA**.
  - 2.) **Cerebellar** efferent fibers send signals onto the **PMC**
  - 3.) A third source of inputs is the **posterior parietal cortex** (PPC).
    - PPC provides integrated somatosensory and visual information to area 6, which is necessary for the programming of motor sequences.
Supplemental motor area

- The most significant functional aspect of the SMA is its role in coordinating voluntary movement.
- Electrical stimulation of the supplemental (as well as premotor) cortex requires higher currents for the elicitation of motor responses.
- Motor responses are of a more complex pattern than those elicited from the primary motor cortex.
• stimulation of the SMA and PMC can elicit postural adjustments, body orientation, or closing or opening of the hands (unilateral or bilateral).

• Patients who have lesions of the SMA display **apraxia** (inability to initiate specific, purposeful movements)
Premotor Cortex

• Corticospinal fibers arising from the **PMC** innervate medial and lateral ventral horn cells, **directly** or **indirectly**, by descending fibers that supply **reticulospinal fibers**.

• Supply the axial (medial) and distal (lateral) musculature.
Premotor Cortex

• The premotor area plays an important role in movements that require visual guidance.
• With lesions of the PMC, the patient is unable to coordinate the movement of both arms at the same time.
Role of the Posterior Parietal Cortex

- neurons discharge in response to exploratory hand movements or
- in preparation for a goal-directed response (reaching)
- spatial arrangements and
- integration of visual information with proprioceptive and other somatosensory signals
Lesion of the right Posterior Parietal Cortex

sensory neglect (or anosognosia)

**FIGURE 19-7** Drawings made by a patient with a lesion of the right posterior parietal cortex, indicating sensory neglect on the left side. The patient was asked to fill in numbers on the face of the clock (A) and to draw a flower (B).
Neurophysiological properties of the motor fields

• electrical stimulation of MI evokes movement or muscle contraction contralaterally

• electrical stimulation of MII and SMA elicits postural adjustments, body orientation, or closing or opening of the hands (unilateral or bilateral).
• http://neuroscience.uth.tmc.edu/s3/chapter03.html
Figures 3.7A, 3.7B, and 3.7C
Motor cortex encodes the force necessary to make a movement. (Evarts 1968)

Figure 3.7A. When there is little load, a motor neuron in primary motor cortex that controls an extension of the wrist fires when the wrist extends. A motor neuron that controls wrist flexion does not change its rate of activity. Note that the extension motor neuron begins to fire spikes before the onset of the movement. Text goes here.

Figure 3.7B. When a 5 lb load is placed on the left pulley, more force must be used to initially hold the weight steady and then lift it. The extension motor neuron in primary motor cortex fires more strongly to produce the greater force. Text goes here.

Figure 3.7C. When a 5 lb load is placed on the right pulley, the load is on the flexor. Thus, primary motor cortex neurons for flexion are activated to keep the weight stable. When the wrist extends, the neurons are quieter, as the force of the movement is actually produced by the weight itself. Note that motor cortex encodes the force of a movement, such as wrist extension or more complicated, multijoint movements. The force of individual muscles is encoded by alpha motor neurons in the spinal cord and brain stem. Text goes here.
Somatotopic organisation

• 1950-Wilder Penfield during neurosurgery on 1200 patients suffering EPI performed electrical stimulation of the cortex

HOMUNCULUS!
http://www.youtube.com/watch?v=68MiW2KK1us
Injuries of the motor cortex

• Due to cerebrovascular inzulta (hemorage or ischemia) damage of the cortex or corticospinal tract can develop.
THE UPPER MOTOR NEURON SYNDROME

Upper motoneuron – corticospinal tract, project to the α-motoneurona; syndrome: hypertonia, spasticity, abnormal reflexes, such as the Babinski sign also referred to as an extensor plantar response
Elektrophysiology of the CM neurons

- *Corticomotoneurons (CM neurons)* – monosinaptically excite α-motoneuron
- Speed: 70m/s, latency: 0,7 ms
- Start to fire action potentials 50-100 ms prior to the beginning of the movement.
• **Phasic CM neurons**
  – low spontaneous activity
  – Latency < 1ms
  – Fire action potentials during the movement
  – Frequency 20-80Hz
  – Delay between EPSPs 1.5 - 5 msec – suitable for summation in time

• **TONIC CM neurons:**
  – Long latency
  – Spontaneously active even when the movement doesn’t occur
Smaller CM neurons

- Majority of neurons
- Equally active during all movements,
- Responsible for slow movements of medium strength, under the somatosensory control
- Included in system “feedback loop”

Big CM neurons
Betz neurons

- 35,000 in MI
- Active in strong and fast movements
- Included in system “no feedback loop”
- **ballistic movements**
- No correction of the movement, no sensory influence.
- Can only be repeated “to hit or to miss”
CM neuron- α-motoneuron synapse

• Amplitude of one EPSP on α-motoneuron evoked by CM action potential is about 200μV – subliminal stimulus – no action potential on the α-motoneuron can be evoked after one stimulus!

• Convergention and sumation is needed (in time and space)!
THE CORTICOBULBAR TRACTS

- arise from the lateral aspect of the primary motor cortex to the cranial nerve motor nuclei
- voluntary control of the muscles of facial expression, eye movements, jaw opening and closing, and movements of the tongue.
- **Monosynaptic pathway**
- Nucleus *n. trigenimus* – chewing muscles
- Nucleus *n. facialis* – facial expression
- Nucleus *n. hypoglossus* – tongue
- Nucleus ambiguus – voice
Tractus corticonuclearis

• Projections are bilateral
• Exception: lower part of the facial nucleus - contralateral projections
Lesions of Corticobulbar Fibers That Supply Nuclei of Cranial Nerves

- central facial palsy
- pseudobulbar palsy
A. Facial nerve lesion (Bell's palsy)
- Nucleus of facial nerve (cranial nerve VIII)
- Lesion in facial nerve

B. Supranuclear lesion
- Supranuclear lesion

Possible symptoms of Bell's Palsy:
- Droopy eyelid, dry eye, or excessive tears
- Facial paralysis, twitching, or weakness
- Drooping corner of mouth, dry mouth, impaired taste

Lateral view showing affected side and restoration typically at stylomastoid foramen.
DESCENDING MOTOR SYSTEMS FROM THE BRAINSTEM

• the medial and lateral reticulospinal tracts
• the medial and lateral vestibulospinal tracts
• the rubrospinal tract
• and the tectospinal tract.
Figure 33-14 Medial and lateral descending pathways from the brain stem control different groups of neurons and different groups of muscles.

A. The main components of the medial pathways are the reticulospinal, medial and lateral vestibulospinal, and tectospinal tracts that descend in the ventral column. These tracts terminate in the ventromedial area of the spinal gray matter.

B. The main lateral pathway is the rubrospinal tract, which originates in the magnocellular portion of the red nucleus. The rubrospinal tract descends in the contralateral dorsolateral column and terminates in the dorsolateral area of the spinal gray matter.
The control of Gaze
• Siegel and Sapru, chapter 14
• Kandel, chapter 39
Eye movement and eye gaze direction system

eyes
muscles
nerves
CNS

FIGURE 14–10 Origin and distribution of cranial nerves (CN) VI, IV, and III, which innervate extraocular eye muscles. The focus of the upper part of this figure includes the abducens nerve (CN VI) and the general somatic efferent component of the oculomotor nerve (CN III), which are essential for horizontal gaze. The lower part of this figure depicts the muscles of the eye and their relationship with CN III, IV, and VI.
Gaze system perform its function through:

- 1.) Oculomotor system moves the eyes in the orbit.
- 2.) Head movement system moves the orbits in space.
- The gaze system keeps the eye still when the image is still and stabilizes the image when the object moves.
- In 1890 Edwin Landott discovered that, when we read, the eyes do not move smoothly along a line of text but make little jerky movements-saccades-each followed by a short pause.
Six Neuronal Control Systems Keep the Fovea on Target

- three that keep the fovea on a visual target in the environment
- two that stabilize the eye during head movement
- one holds the eyes still during intent gaze
Six Neuronal Control Systems Keep the Fovea on Target

• 1. *Saccadic eye movements* shift the fovea rapidly to a visual target in the periphery.

• 2. *Smooth pursuit movements* keep the image of a moving target on the fovea.

• 3. *Vergence movements* move the eyes in opposite directions so that the image is positioned on both foveae.
• **4. Vestibulo-ocular movements** hold images still on the retina during brief head movements and are driven by signals from the vestibular system.

• **5. Optokinetic movements** hold images during sustained head rotation and are driven by visual stimuli.

• **6. Fixation system** holds the eye still during intent gaze
• All eye movements but *vergence movements* are **conjugate**: Each eye moves the same amount in the same direction.

• Vergence movements are **disconjugate**: The eyes move in different directions and sometimes by different amounts.
Control of Eye Movements:

- Cranial Nerves III (Oculomotor), IV (Trochlear), and VI (Abducens) are essential for eye movements.
- The control of eye movements is under supranuclear control and includes:
  - A) cerebral cortex
  - B) region adjacent to the abducens nucleus called the pontine gaze center
  - C) vestibular nuclei
  - D) vertical gaze center (rostral midbrain-PAG)
Role of the Pontine Gaze Center

- Motor nuclei of CN III, IV, and VI **do not** receive direct inputs from the cortex.
- The major structure for the integration and control of horizontal gaze is the **pontine gaze center**.
- It receives inputs from the contralateral cerebral cortex (i.e., **frontal eye field**).
• The pontine gaze center projects its axons to the nucleus of CN VI (abducens) on the ipsilateral side and the nucleus of CN III (oculomotorius) on the contralateral side.

• Thus, stimulation of the right pontine gaze center will result in activation of the ipsilateral CN VI and the contralateral CN III.

• This will cause the right eye to be abducted and the left eye to adduct (i.e., the eyes are directed to the right).
• if there is a lesion of the right pontine gaze center, then the eyes cannot be moved to the right.

• **doll’s eye (oculocephalic) maneuver:**

• Testing of the horizontal eye movement reflex

• A lesion affecting the brainstem in the region between the midbrain and pons where vestibular and oculomotor pathways (the MLF) are affected would cause the eyes to move in the same direction as the head.
Head turns to side, but eyes are still facing forward, or move back to midline - INTACT BRAINSTEM

Head turns to side, eyes turn with the head if do not correct - BRAINSTEM AFFECTED
Cortical and Vestibular Control of Extraocular Eye Muscles

• Voluntary Control of Eye Movements.
  • the region of the frontal cortex called the frontal eye fields projects to the contralateral pontine gaze center.
  • the basis for voluntary control of horizontal eye movements,
  • if there is a lesion of the projection from the left cortex to the right pontine gaze center, the ability to gaze to the right will be impaired.
FIGURE 14–16  Diagram of the anatomical substrates for lateral gaze (left side of figure) and the deficits that occur following lesions at different sites along this pathway (right side of figure). For purposes of illustration, the diagram depicts the mechanisms involved in right conjugate gaze. Voluntary right conjugate gaze is initiated from pathways arising from the left frontal lobe that project to the pontine (lateral) gaze center. Involuntary pathways mediating conjugate gaze are associated with the occipital cortex. Note that the pontine gaze center projects to the ipsilateral abducens (cranial nerve [CN VI] nucleus and contralateral oculomotor nerve (CN III). The loci of the lesions (1–4) are shown on the left illustration. The corresponding deficits are depicted on the illustration on the right side.
• **Horizontal movements:**
  • saccadic movements,
  • smooth pursuit movements.
1. Saccadic eye movements

- The purpose of the saccade is to move the eyes as quickly as possible
- Saccades are highly stereotyped
- Extremely fast, within a fraction of a second, at speeds up to 900°/s
- There is no time for visual feedback to modify the course of the saccade
- Corrections to the direction are made in successive saccades
1. Saccadic eye movements

![Diagram of saccadic eye movements with labels for eye position and eye velocity, showing a target movement and specified angles and velocities.](image)
• Pulse is based on fast activation of **BN cells (burst neurons)**

• BN control horizontal saccades and are located in **pontine gaze center (paramedian pontine reticular formation (PPRF))**

• **BN cells** can be excitatory (**EBN**), inhibitory (**IBN**), or tonic (**TN**)
OPN cells

• “omnipause neurons”
• located in *nucleus raphe interpositus (RIP)*
• project on the BN cells of the opposite side
• tonically active EXCEPT during saccade (“shut down” 16 msec prior the beginning of the saccade)
Eye movement

Beginning of saccade
Figure 39-10 The motor circuit for horizontal saccades in the brain stem. Excitatory neurons are orange and inhibitory neurons are gray. The dotted line represents the midline of the brain stem.

A. Long-head burst neurons relay signals from higher centers to the excitatory burst neurons. The eye velocity component of the motor signal arises from excitatory burst neurons in the paramedian pontine reticular formation that synapse on motor neurons and interneurons in the abducens nucleus. The abducens motor neurons project to the ipsilateral lateral rectus muscles while the interneurons project to the contralateral medial rectus muscle via fibers that cross the midline and ascend in the medial longitudinal fasciculus. Excitatory burst neurons also drive ipsilateral inhibitory burst neurons that inhibit contralateral abducens and excitatory burst neurons. The medial vestibular nucleus also inhibits contralateral abducens neurons. Omnipause neurons inhibit excitatory burst neurons and abducens neurons, preventing unwilled eye movements. The eye position component of the motor signal arises from a “neural integrator” comprised of neurons distributed throughout the medial vestibular nuclei and nucleus prepositus hypoglossi on both sides of the brain stem. These neurons receive velocity signals from excitatory burst neurons and integrate this velocity signal to a position signal. The position signal is transmitted to the ipsilateral abducens neurons.

B. Different neurons provide different information for a horizontal saccade (above). The motor neuron has both position and velocity signals. The tonic neuron in the nucleus prepositus hypoglossi has only an eye position signal. The excitatory burst neuron in the paramedian pontine reticular neuron has only eye velocity information. The omnipause neuron discharges at a high rate except before, during, and after the saccade.
Cortical pathways for saccadic eye movements in the monkey

Cortical areas controlling saccades

The intermediate supra-nuclear structures

the brain-stem reticular formation
2. Smooth pursuit movements

• Keeps the image of a moving target on the fovea
• It calculates how fast the target is moving and moving the eyes accordingly
• The system requires a moving stimulus
• Verbal command or an imagined stimulus cannot produce smooth pursuit
• A maximum velocity of about 100°, much slower than saccades
• Drugs, fatigue, alcohol, and even distraction degrade the quality of these movements.
2. Smooth pursuit movements

1. The eye briefly moves away from the target
2. The saccade enables the eye to adjust its position to catch the target.
3. From then on the smooth pursuit keeps the eye on the target.
• http://www.youtube.com/watch?v=JdqlV7Sb8S0
• http://www.youtube.com/watch?v=YsU1Yb6wkJ0
3. Vergence movements

- disconjugate movements of the eyes
- When we look at an object that is close to us our eyes rotate toward each other, or **converge**
- when we look at an object further away they rotate away from each other, or **diverge**
3. Vergence movements

• These disconjugate movements ensure that the object of interest is on the same place in both retinas.
• The visual system uses slight differences of retinal position, or *retinal disparity*, to create a sense of *depth*.
• The vergence system uses retinal disparity to drive disconjugate movements.
• Accommodation and vergence are linked.
• Whenever accommodation occurs, the eyes also converge!
3. Vergence movements

- At the same time the pupils transiently constrict to increase the depth of field of the focus.
- The linked systems of accommodation, vergence, and pupillary constriction comprise the *near response*. 
4. Vestibulo-ocular movements

• When an individual’s head is rotated to the right, the eyes will turn toward the left.
• Vestibular nuclei receive direct inputs from the semicircular canals of the vestibular apparatus.
• The vestibular nuclei transmit these inputs to both the pontine gaze center and nuclei of CN VI, IV, and III.
**The normal state**
Head movement towards a canal (yellow in figure) will cause activation of that canal, and reflex movement of the eyes in the opposite direction - that is, away from the canal.

**The pathological state and the basis of the head thrust test**
Head movement towards a defunct canal (blue in figure) will result in failure of activation of the vestibular ocular reflex and thus the visual target will be lost from fixation during sudden head movements. In the head thrust test, the examiner turns the patient’s head with a high acceleration but low amplitude head thrust, in this case to the patient’s left. The test is positive when the patient makes a catch-up saccade to refixate the visual target (usually towards the examiner’s nose).
• This is because the inertia of the endolymph in the semicircular canals generates a force across the cupula, moving it in the opposite direction to movement.

• This triggers action potentials in the first-order vestibular neurons on the left side that project to the left vestibular nuclei.

• The left vestibular nuclei, via the MLF, excite the lateral gaze center and motor nucleus of CN VI on the right.
• Thus it excites the lateral rectus muscle
• Also excites, through interneurons, motor neurons of CN III on the left side that supply the medial rectus muscle.
• As the head continues to be rotated, the eyes show a smooth pursuit movement in the opposite direction to continue to fixate upon the object.
• Eventaly, individual will attempt to fixate on another object.
• This phenomenon is called the vestibulo-ocular reflex or nystagmus
• nystagmus

• http://www.youtube.com/watch?v=68mOqjl4N1I
Vestibulo-ocular Reflex

Cold water:

Warm water:
• Nystagmus can also occur clinically, usually in association with lesions of the MLF at levels rostral to the pontine gaze center.

• Internuclear ophtalmoplegia
1. Gaze to right impaired
2. Impaired adduction of left eye
3. Impaired adduction of left eye and ptosis
4. Eyes cannot be moved to right. Intact left pontine center moves eyes to left.
5. Optokinetic movements

- The optocinetic system supplements the vestibulo-ocular reflexes.
- It drives the eyes in the direction of the image motion to stabilize the image on retina.
- It responds to very slow visual image motion.
- The combination of vestibular and optokinetic reflexes enables rotatory nystagmus in light environment to continue for as long as the head moves.
6. Fixation system

- When we look at an object of interest a neural system of fixation actively prevents the eyes from moving!
- Some patients with disorders of the fixation system—for example, those with congenital nystagmus have poor vision not because their eyes are abnormal but because they cannot hold their eyes still enough for the visual system to work accurately.
The Eye Is Moved by Six Muscles

• Three axes of rotation:
  • Y: torsional
    • *Intorsion* (rotates the top of cornea toward the nose)
    • *Extorsion* (rotates it away from the nose)
  • Z: horizontal
    • *Adduction* (towards the nose)
    • *Abduction* (away the nose)
  • X: okomita ravnina
    • *Elevatio* (up)
    • *Depressio* (down)
Figure 39-5 The origins and insertions of the extraocular muscles.

A. Lateral view of a left eye with the orbital wall cut away. The recti insert in front of the equator of the globe, so that contraction rotates the cornea toward the muscle. The obliques insert behind the equator, and contraction rotates the cornea away from the insertion. The superior oblique muscle passes through a pulley of bone, the trochlea, before it inserts.

B. Superior view of the left eye with the roof of the orbit cut away. The superior rectus passes over the superior oblique and inserts in front of it.
The Six Extraocular Muscles Form Three Complementary Pairs

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<thead>
<tr>
<th>Muscle</th>
<th>Adduction</th>
<th>Abduction</th>
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<tbody>
<tr>
<td>Superior rectus</td>
<td>Intorsion</td>
<td>Elevation</td>
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<tr>
<td>Inferior rectus</td>
<td>Extorsion</td>
<td>Depression</td>
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<td>Superior oblique</td>
<td>Depression</td>
<td>Intorsion</td>
</tr>
<tr>
<td>Inferior oblique</td>
<td>Elevation</td>
<td>Extorsion</td>
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Cranial Nerves of the Pons and Midbrain Associated With the Control of Eye Movements

• *Abducens Nerve (Cranial Nerve VI)*
• *Trochlear Nerve (Cranial Nerve IV)*
• *Oculomotor Nerve (Cranial Nerve III)*
FIGURE 14-10 Origin and distribution of cranial nerves (CN) VI, IV, and III, which innervate extraocular eye muscles. The focus of the upper part of this figure includes the abducens nerve (CN VI) and the general somatic efferent component of the oculomotor nerve (CN III), which are essential for horizontal gaze. The lower part of this figure depicts the muscles of the eye and their relationship with CN III, IV, and VI.
FIGURE 14–12 Diagram illustrating the direction of actions of the extraocular muscles of the eye (indicated by arrows). The lateral rectus muscle is innervated by the abducens nerve, the superior oblique muscle is innervated by the trochlear nerve, and the remaining muscles are innervated by the oculomotor nerve.
FIGURE 14-11 Origin and distribution of the trochlear nerve (cranial nerve IV) to the superior oblique muscle. As indicated in the cross section of the brainstem, note that this nerve exits the brain from the dorsal aspect, and it is the only nerve that is crossed. Arrow indicates direction of movement of the bulb downward and inward.
FIGURE 14–13  Origin and distribution of the oculomotor nerve (cranial nerve [CN] III). The anatomical organization of the general somatic efferent (GSE) cell columns of the oculomotor nerve (CN III) complex, whose axons innervate all of the extraocular eye muscles except the lateral rectus and superior oblique muscles, is shown; the Edinger-Westphal nucleus, whose axons (general visceral efferent) serve as preganglionic parasympathetic neurons, innervate the ciliary ganglia. The postganglionic parasympathetic neurons from the ciliary ganglia (not shown in figure) innervate the constrictor muscles of the pupil and the ciliary muscle.
Lesions of the extraocular muscles or their nerves

- complain of double vision (diplopia)
• An isolated lesion of the abducens nerve (VI): results in loss of abduction beyond the midline diplopia when patients attempt to look in the direction of the paralyzed lateral rectus muscle
• paralysis of the lateral rectus muscle
• medial strabismus
• the patient moves his or her head so that the affected eye is facing the object directly
• An isolated lesion of the trochlear nerve
• there is an outward rotation of the eye due to the unopposed action of the inferior oblique muscle
• when walking down a staircase, they experience double vision and will tend to fall down
• Sign: Tilting of the head upon downward gaze
• A lesion of the oculomotor nerve results in loss of eye movement medially or upward from the mid position.
• (1) the inability to move the eye inward or vertically (loss of all of the recti muscles, except the lateral rectus muscle, and the loss of the inferior oblique muscle)
• (2) lateral strabismus, in which the eye on one side is now not coordinated with the opposite eye whose extraocular eye muscles are intact, causing diplopia (double vision);
• (3) drooping of the eyelid (called **ptosis**), which **results** from damage to the nerves innervating the levator palpebrae superior muscle.
Figure 39-8  A patient with a deficit of the left superior oblique muscle. When the patient looks straight ahead, the left eye is mildly elevated relative to the right (A). This elevation occurs because there is no superior oblique tension to counteract the left superior oblique. When the patient looks to the right, the eye becomes even more elevated as more of the superior oblique force is dedicated to elevation (B). When the patient attempts to look down, the left eye cannot be depressed below the midline (C). When the head tilts to the right, the vertical deviation is lessened (D). Patients with a lesion of the trochlear nerve frequently adopt this posture to eliminate diplopia. (Adapted from Leigh and Zee 1991.)
Thank you!
Figure 44-6 Cranial nerve nuclei are organized into functional columns.

A. This dorsal view of the brain stem shows the organization of the cranial nerve sensory columns (right) and motor columns (left).

B. This simplified schematic view of the same structures as in panel A shows more clearly the organization of the motor and sensory columns.

C. The locations of the cranial nuclei with respect to one another and the sulcus limitans (see Figure 44-7) as seen in a cross section at the level of the medulla.
FIGURE 43–2
The optokinetic reflex. A human’s horizontal eye position as he sits still inside a vertically striped drum rotating slowly to his right. Eye position is plotted against time. Note that during the slow phase the eyes move in the same direction as the striped drum so as to keep the drum still on the retina.
FIGURE 43–3

The smooth pursuit system. A monkey’s eye position (solid line) plotted against time as he follows a target (dotted line) that begins to move at time 0. Note that the monkey makes a rapid movement (saccade) to catch up to the target and then follows it with an eye movement that has the same speed as the target. Pursuit is shown for three different target speeds. (Adapted from Fuchs, 1967.)