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

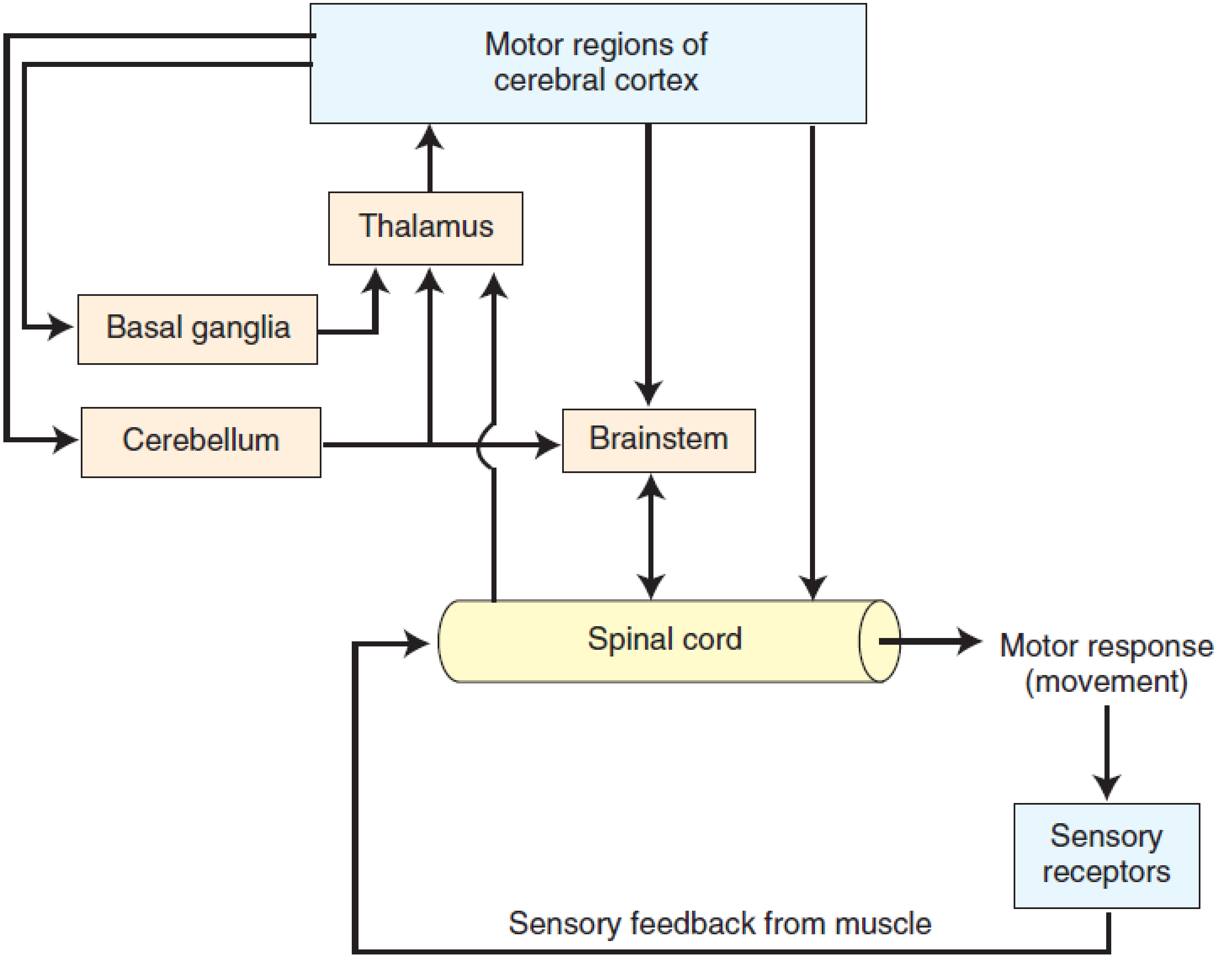
Prof. dr. sc. Maja Valić

Characteristics of voluntary movements

- Organized around the performance and purposeful task
- The effectiviness 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 (asociative, limbic and paralimbic system)
2. **Planning** of the movement (premotor area)
3. **Performance** (primary motor cortex, the corticospinal tract)


Motor regions of cerebral cortex
Thalamus
Basal ganglia
Cerebellum
Brainstem
Spinal cord
Motor response (movement)
Sensory receptors
Sensory feedback from muscle

Main role of the motor cortex

- Movement coordination, Coordination of fine motor activity
- Improvement of the performance

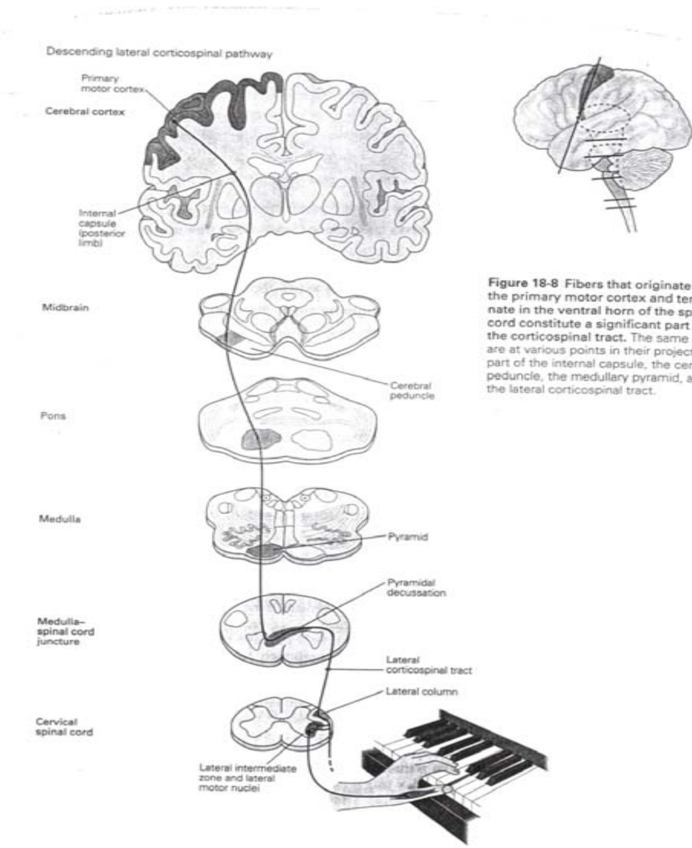


Figure 18-8 **Fibers that originate in the primary motor cortex and terminate in the ventral horn of the spinal cord constitute a significant part of the corticospinal tract.** The same axons are at various points in their projection part of the internal capsule, the cerebral peduncle, the medullary pyramid, and the lateral corticospinal tract.

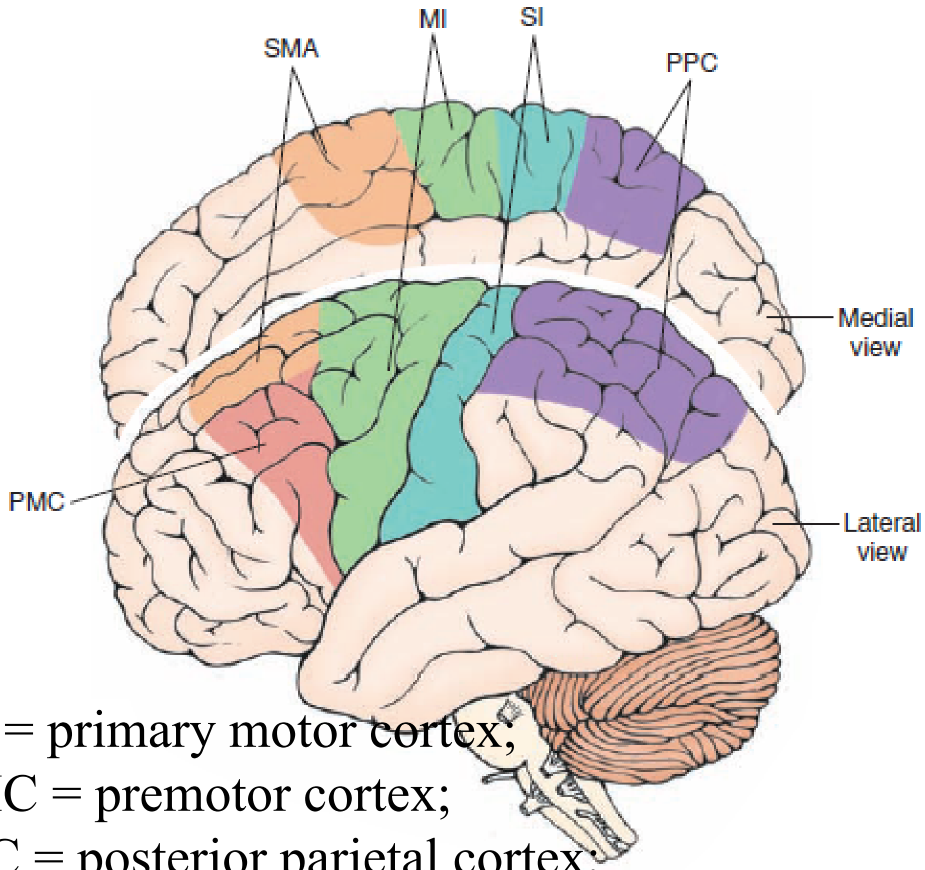
- **Lower motor neurons -** innervate skeletal muscle
- **Upper motor neurons -** neurons of the brain that innervate lower motor neurons of the spinal cord and brainstem, either directly or through an interneuron
- may arise from the brainstem or cerebral cortex
- The most significant of all the upper motor neuronsare those that arise **from the cerebral** cortex: **corticospinal tracts** and **corticobulbar tracts.**

The corticospinal tract - crucial for the expression of precise, voluntary movements.

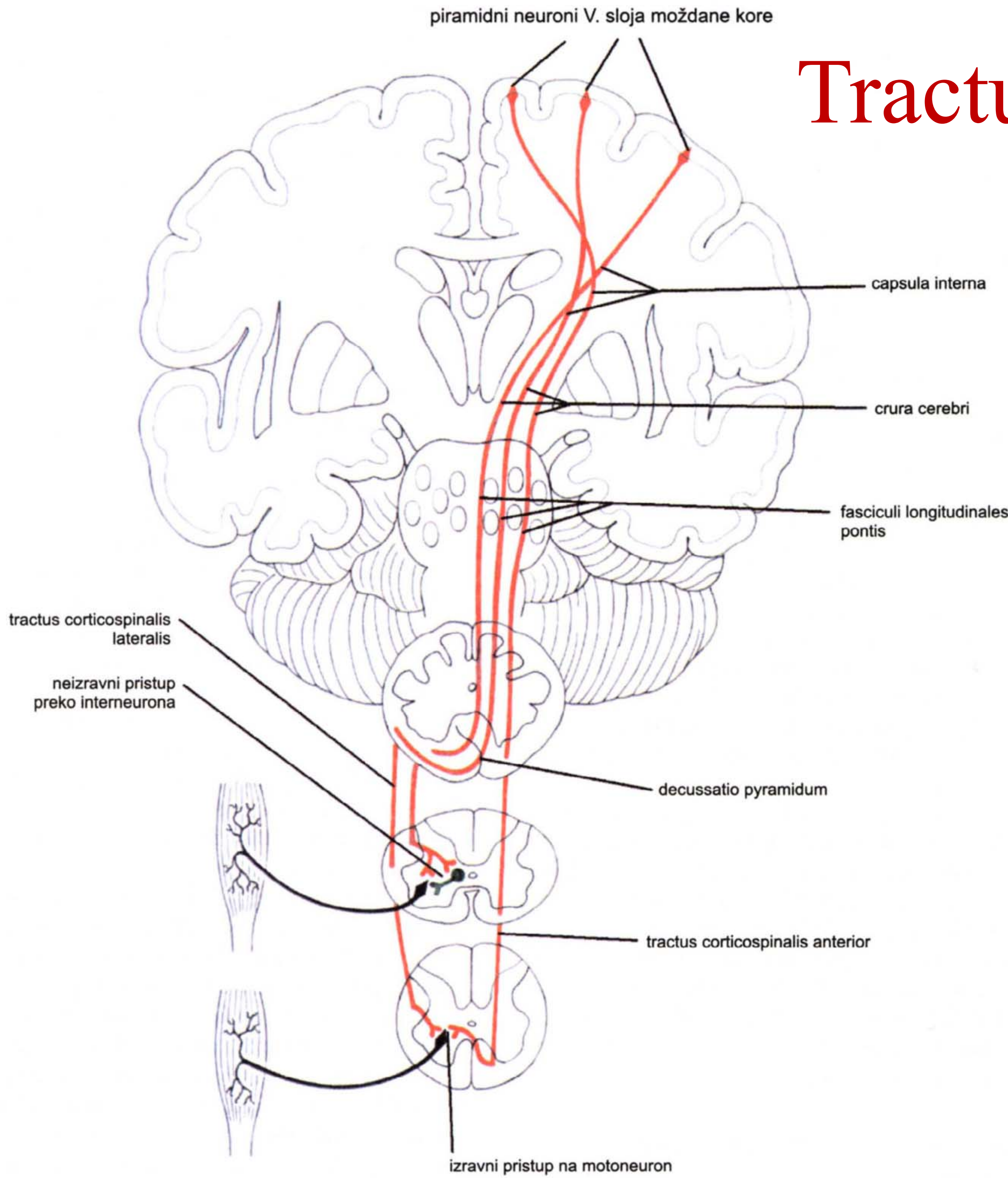
- 1. From where does this tract originate?
- 2. What is the anatomical organization of the tract?
- 3. How are the fibers distributed within the spinal cord?
- 4. What are the important sources of inputs?
- 5. What are the differential contributions of the descending components?
- 6. What are the clinical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

1. 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.



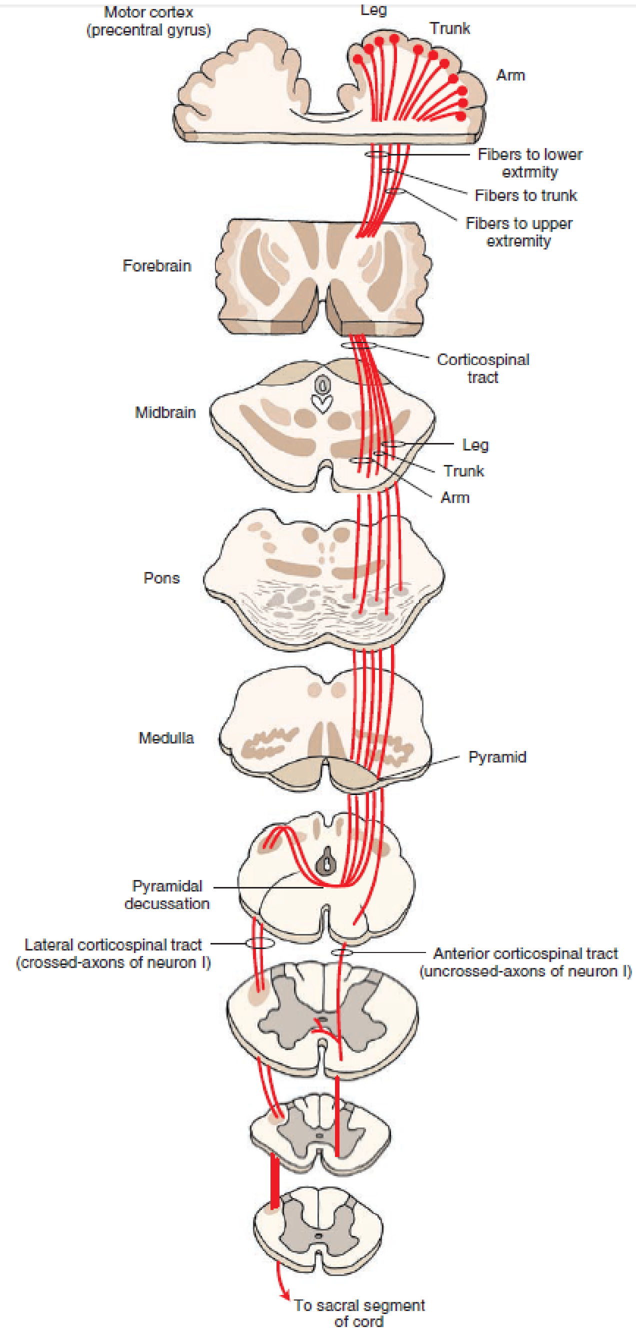
Tractus corticospinalis

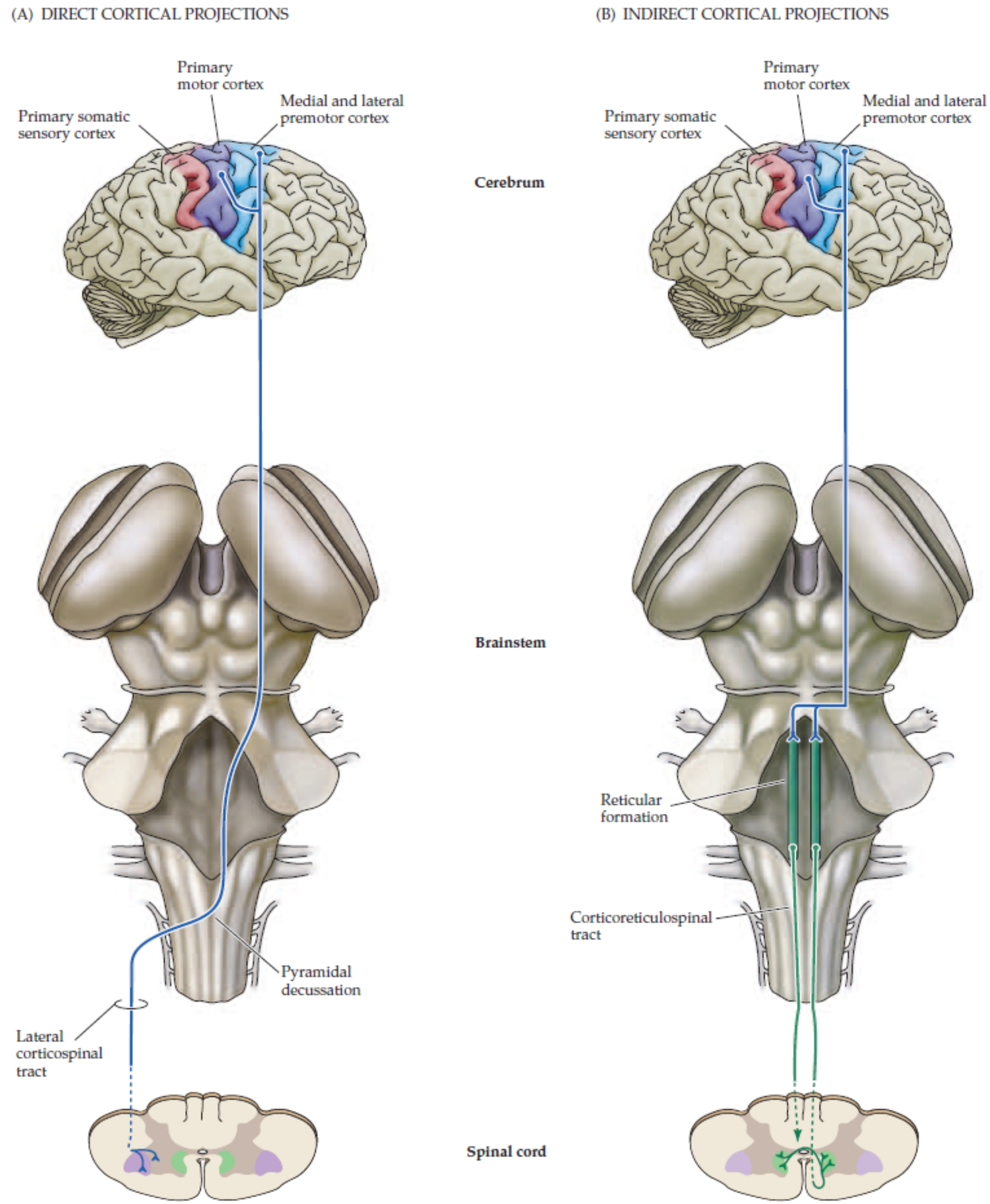
- at the lower brainstem 80-90% axons cross over to the opposite side in *decussatio pyramidum* and make ***tractus corticospinalis lateralis***
- 10-20% fibers reach the spinal cord as **anterior corticospinal tract** (***tractus corticospinalis ventralis***)

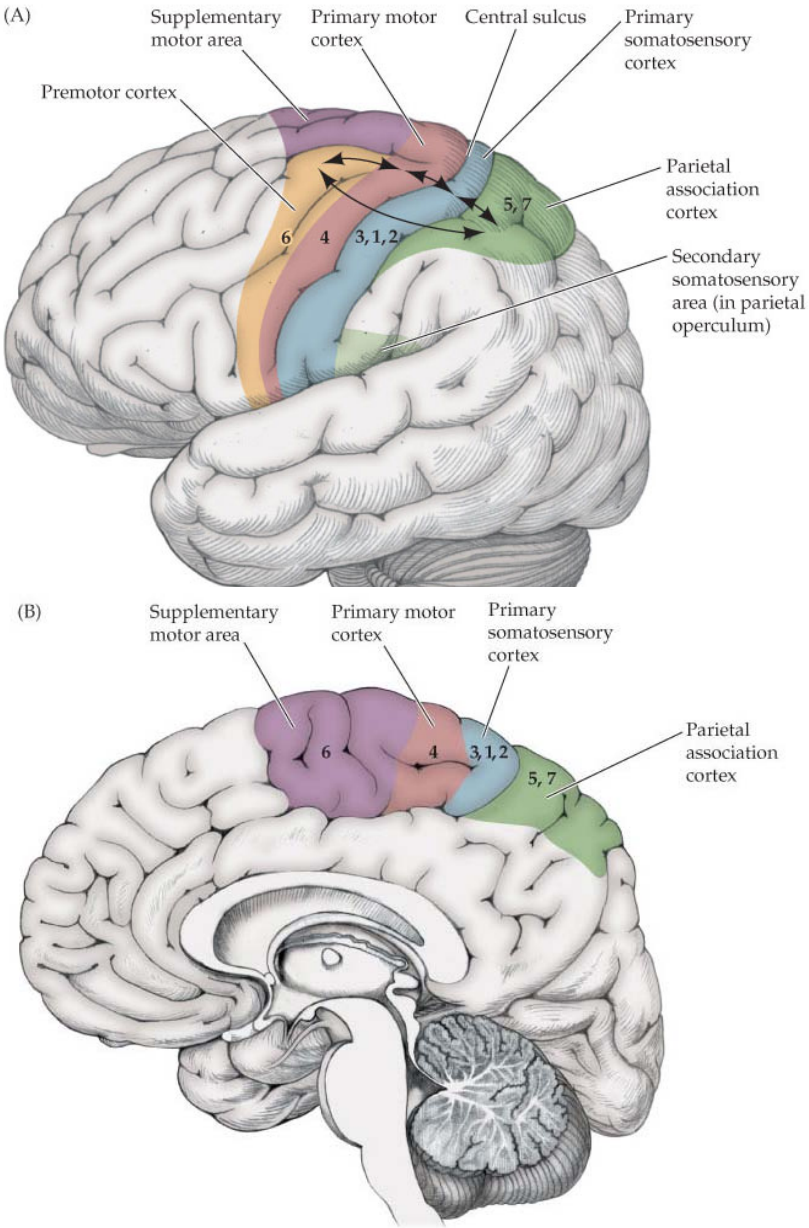
2. Anatomical organisation and Course of the Corticospinal Tract

1. capsula interna
2. crus cerebri
3. basis pontis
4. pyramis m.oblongate

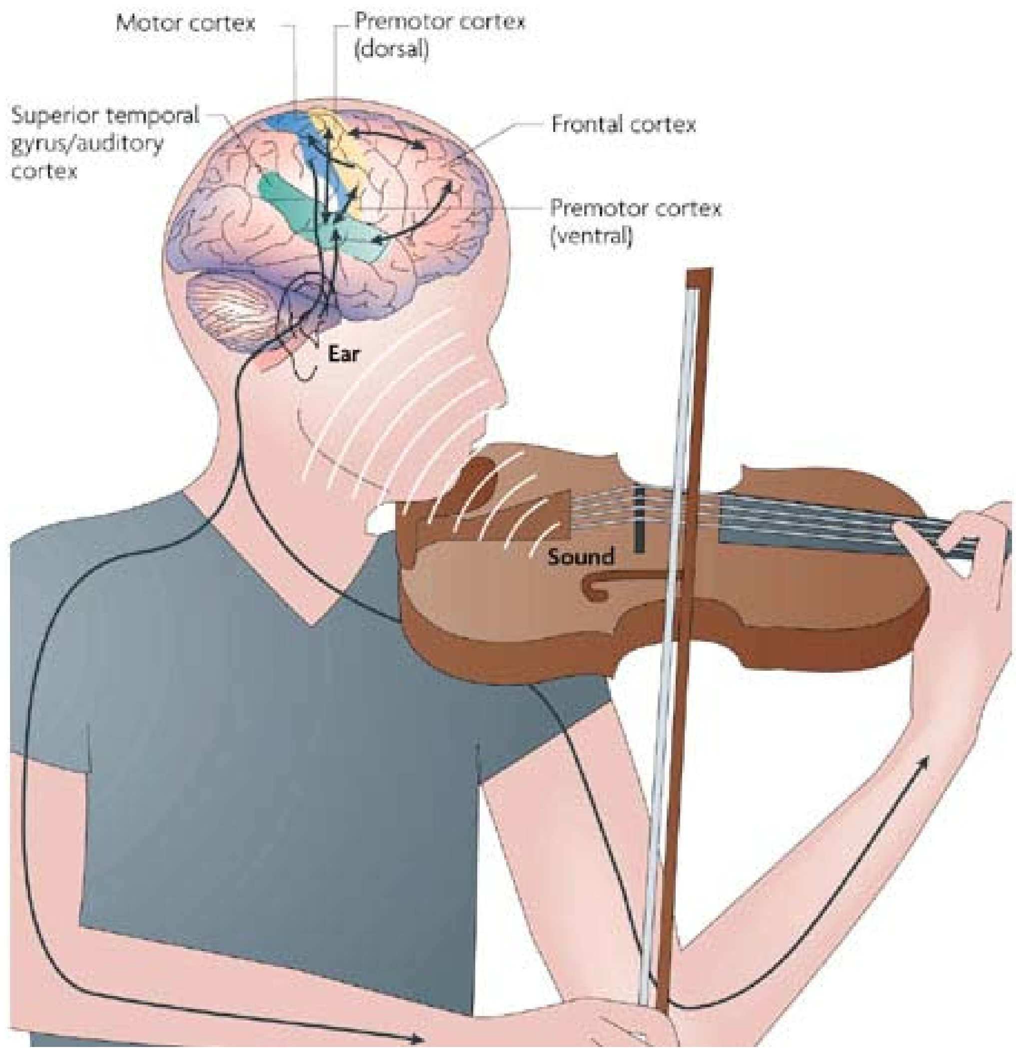
Two other systems play important roles in the regulation of motor functions: **basal ganglia** and **cerebellum.**




(A) DIRECT CORTICAL PROJECTIONS
(B) INDIRECT CORTICAL PROJECTIONS
Primary motor cortex
Medial and lateral premotor cortex
Primary somatic sensory cortex
Primary motor cortex
Medial and lateral premotor cortex
Primary somatic sensory cortex
Cerebrum
Brainstem
Reticular formation
Corticoreticulospinal tract
Pyramidal decussation
Lateral corticospinal tract
Spinal cord



(A)
Supplementary motor area
Primary motor cortex
Central sulcus
Primary somatosensory cortex
Premotor cortex
Parietal association cortex
Secondary somatosensory area (in parietal operculum)
6
4
3, 1, 2
5, 7
(B)
Supplementary motor area
Primary motor cortex
Primary somatosensory cortex
Parietal association cortex
6
4
3, 1, 2
5, 7



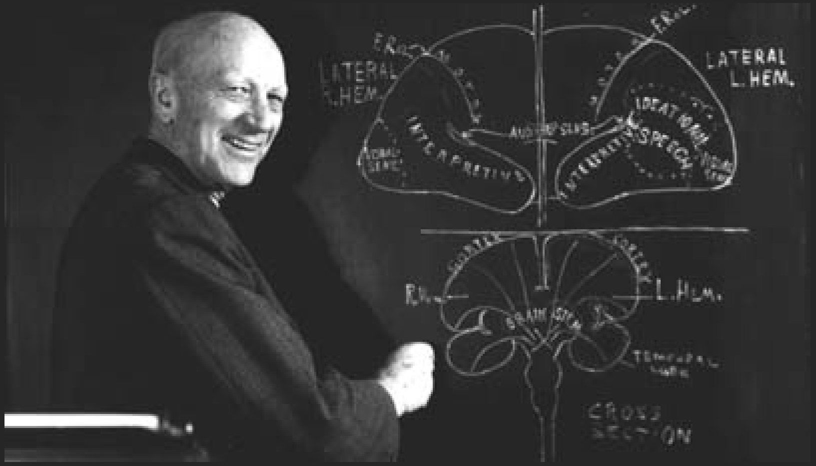
Motor cortex
Premotor cortex (dorsal)
Superior temporal gyrus/auditory cortex
Frontal cortex
Premotor cortex (ventral)
Ear
Sound

Primary motor cortex (MI)

- Responsible for performance of the movement
- Located in *gyrus precentralis*
- ***SOMATOTOPIC ORGANISATION***:
- 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.

Somatotopic organisation

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



HOMUNCULUS!

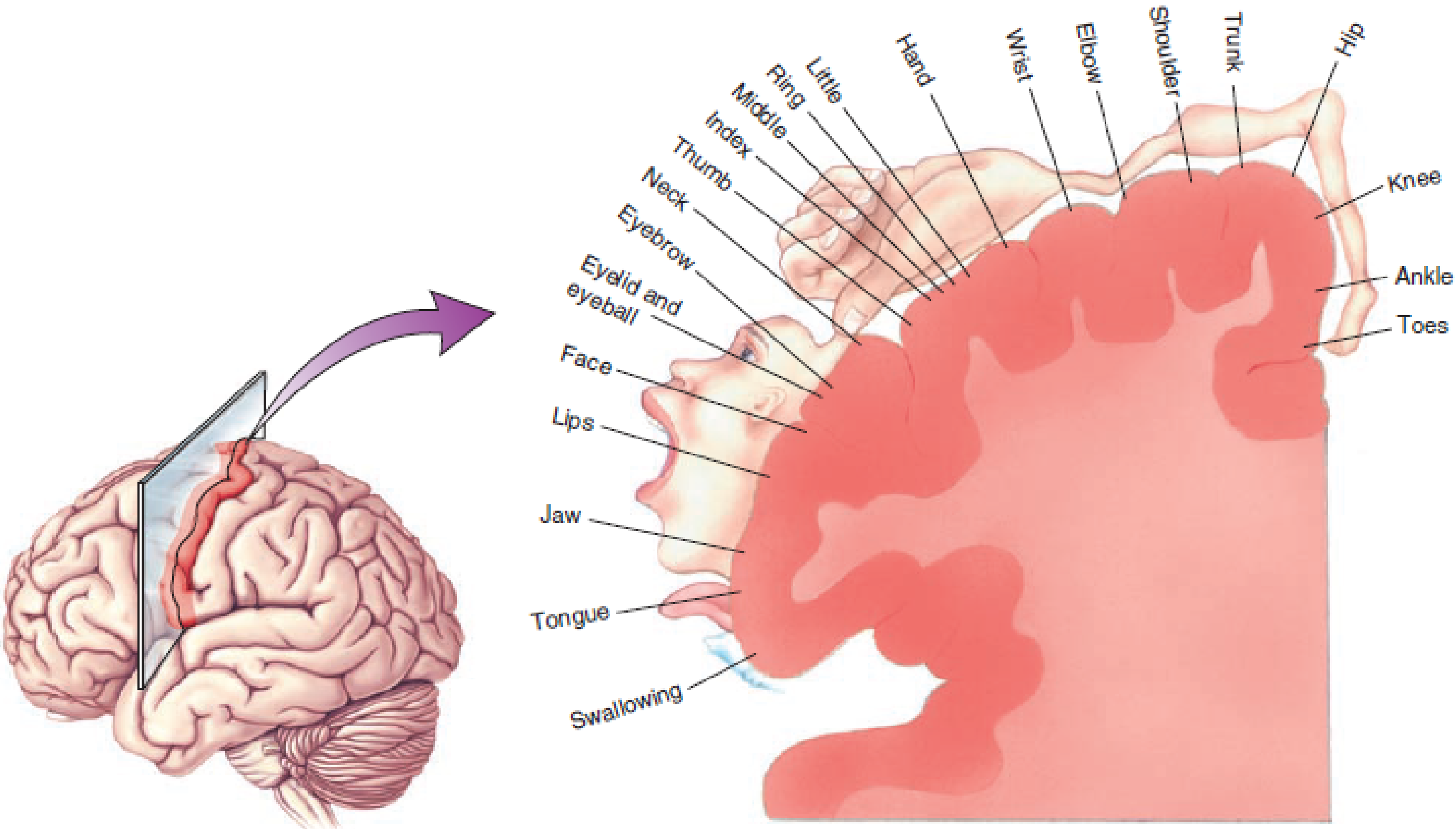
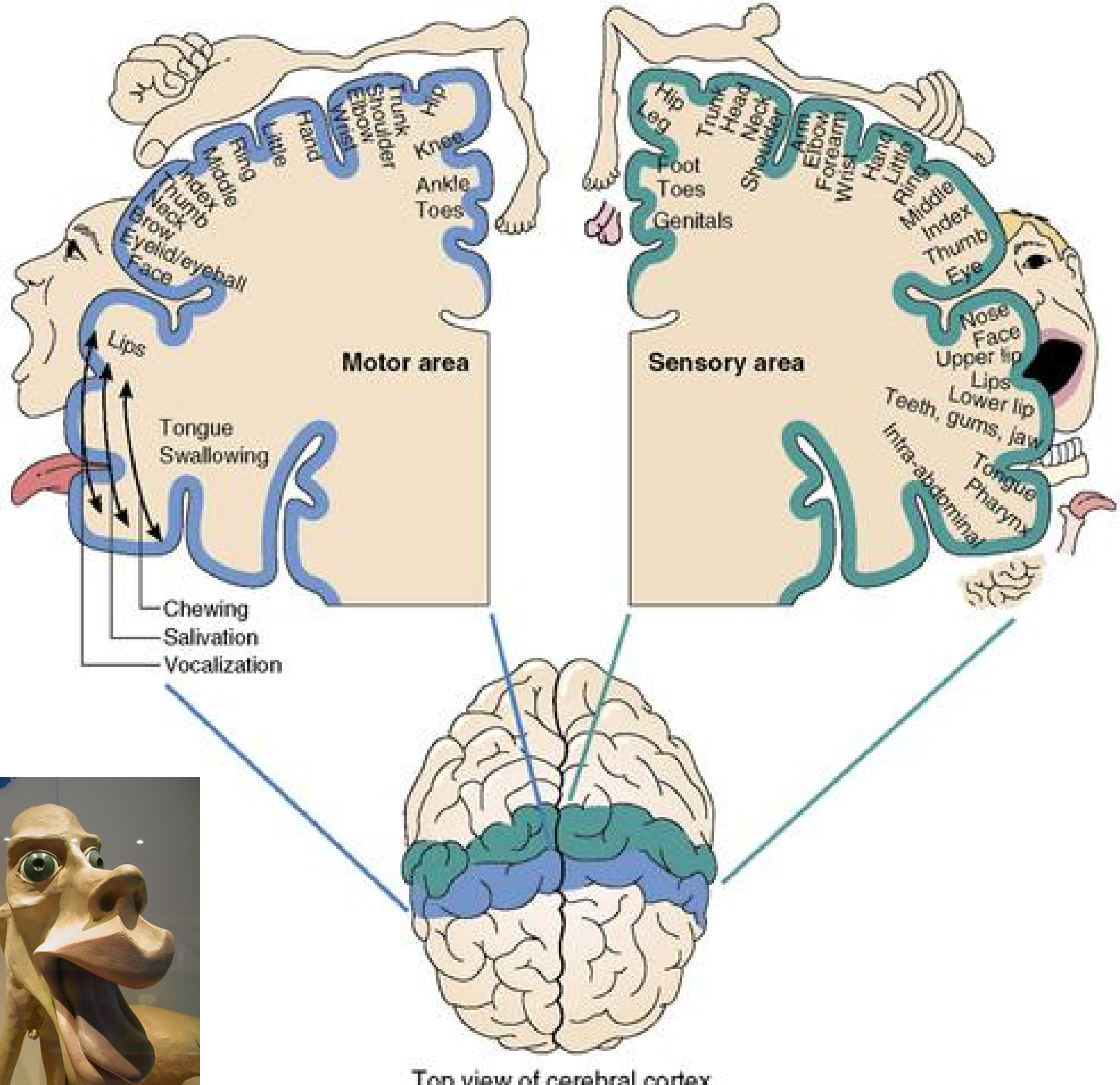
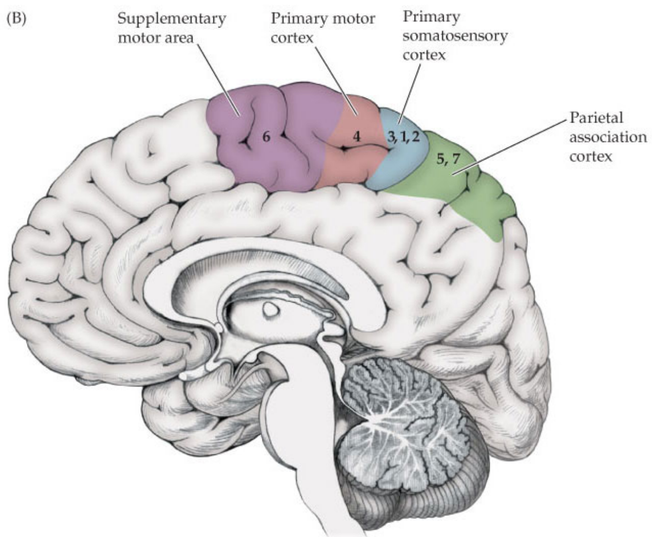
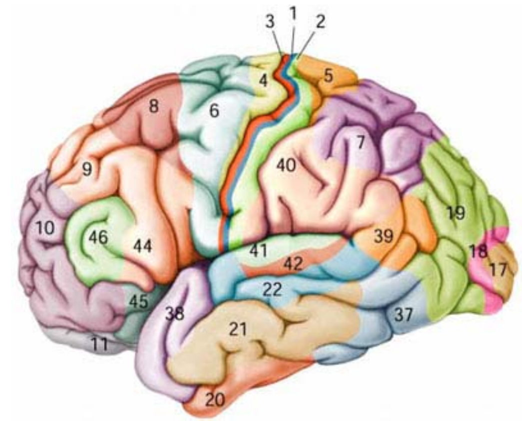


FIGURE 19–3 The relative homuncular representation of the primary motor cortex reveals the relative sizes of the regions of the primary motor cortex, which represent different parts of the body as determined by electrical stimulation experiments. (Reproduced with permission from Bear MF, et al.: Neuroscience: Exploring the Brain, 3rd ed. Baltimore: Lippincott Williams & Wilkins, 2007, p. 460.)


Motor area
Sensory area
Hip
Trunk
Shoulder
Elbow
Wrist
Hand
Little
Ring
Middle
Index
Thumb
Neck
Brow
Eyelid/eyeball
Face
Lips
Knee
Ankle
Toes
Tongue
Swallowing
Chewing
Salivation
Vocalization
Leg
Head
Arm
Forearm
Foot
Genitals
Eye
Nose
Upper lip
Lower lip
Teeth, gums, jaw
Intra-abdominal
Pharynx
Top view of cerebral cortex



Non-primary motor cortex



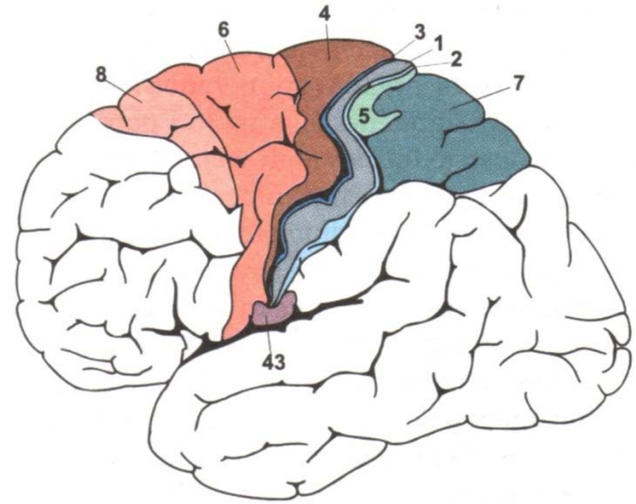
Brodman's field 6, in front of field 4

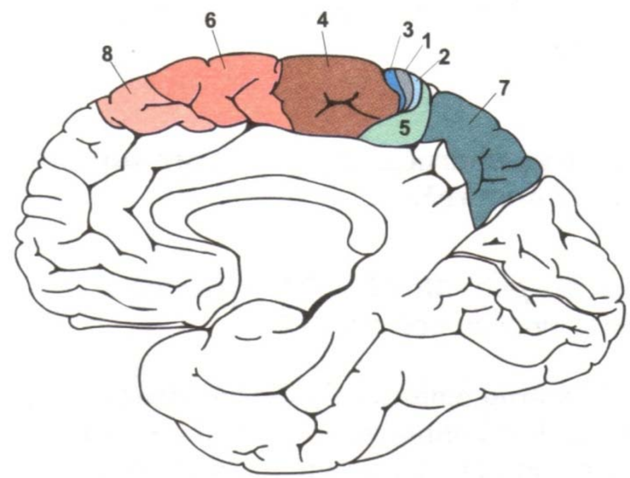
- **MII** (premotor area), dorzolateral surface
- **SMA** (suplemental motor area), medial surface

Resposible for planning and programing!

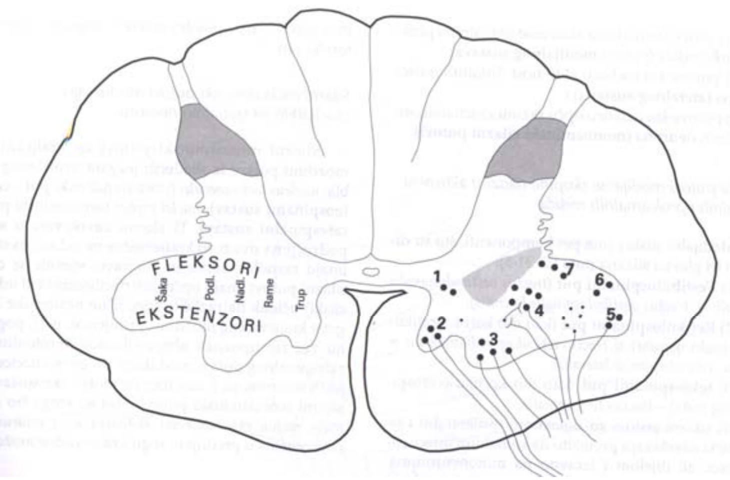
Brodman's field 8

- Frontal cortex
- Frontal eye field (FEF) and Suplemental eye field (SEF)





3. Distribution of the Corticospinal Fibers Within the Spinal Cord



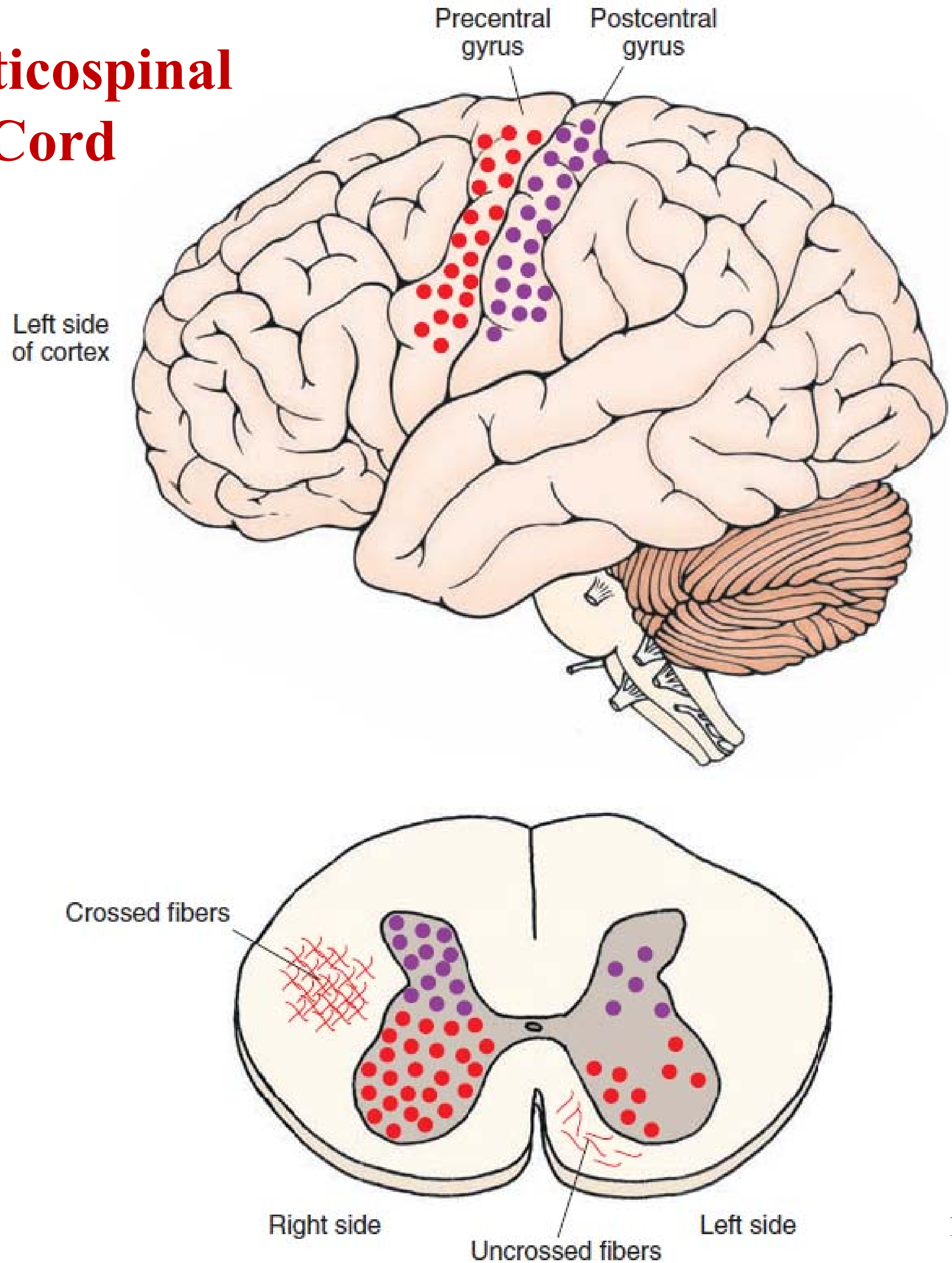
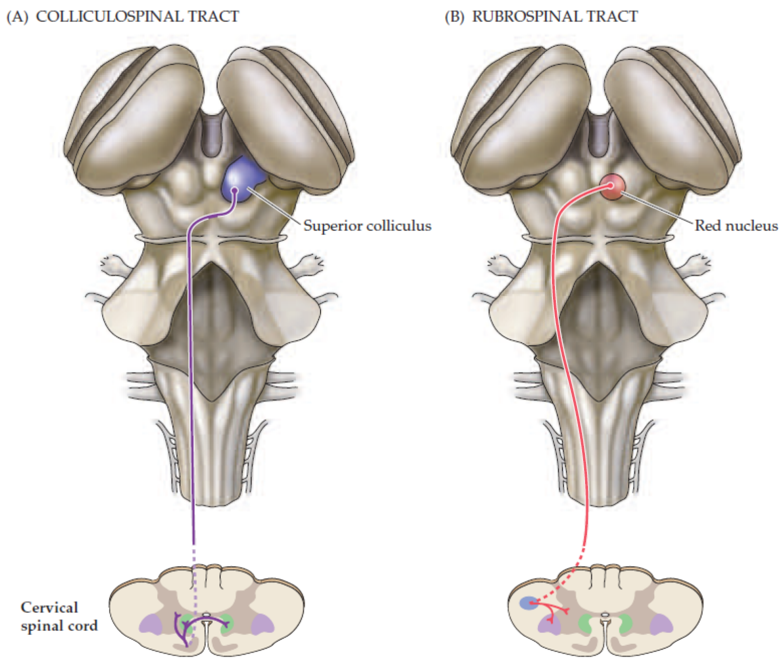


FIGURE 19–5 The distribution of axon terminals in the spinal cord of the monkey (*shown as dots in spinal cord*) as determined by autoradiographic tracing procedures. Depicted also are the sites of origin of the pathway in the motor and somatosensory cortices (top). Most fibers, which are uncrossed (2% 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*).

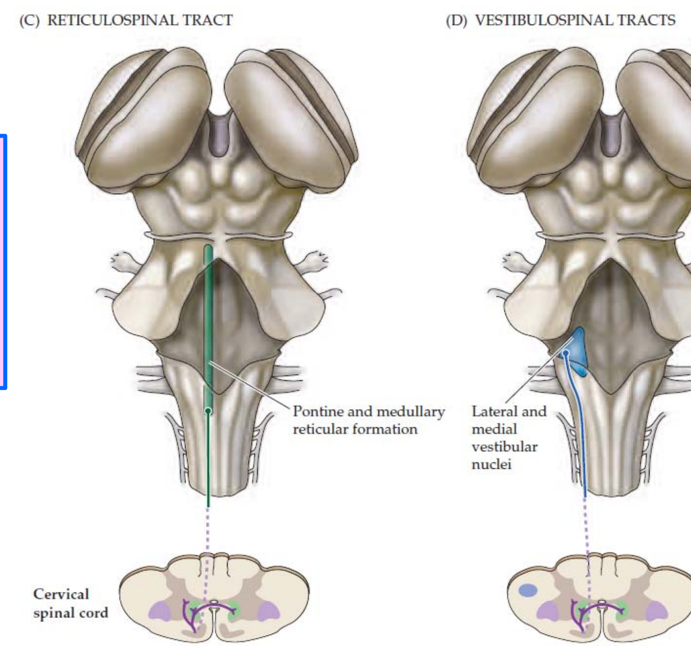
4. The pathways of importance

- **From brainstem**
- medial and lateral **Vestibulospinal tracts**
- medial and lateral **Reticulospinal tracts**
- **Rubrospinal tract**
- **Tectospinal tract**



Descending projections from the **brainstem** to the spinal cord.

Pathways that influence motor neurons in the **medial part of the ventral horn** originate in the **reticular formation**, **vestibular nucleus**, and **superior collicul**us.



- the *medial* and *lateral* ***reticulospinal tracts***
- ***the medial and lateral vestibulospinal*** **tracts**
- **the rubrospinal tract**
- **and the tectospinal tract.**

Those that influence motor neurons that control the proximal arm muscles originate in the **red nucleus** and terminate in **more lateral parts of the ventral horn**.

Characteristics of the motor fields

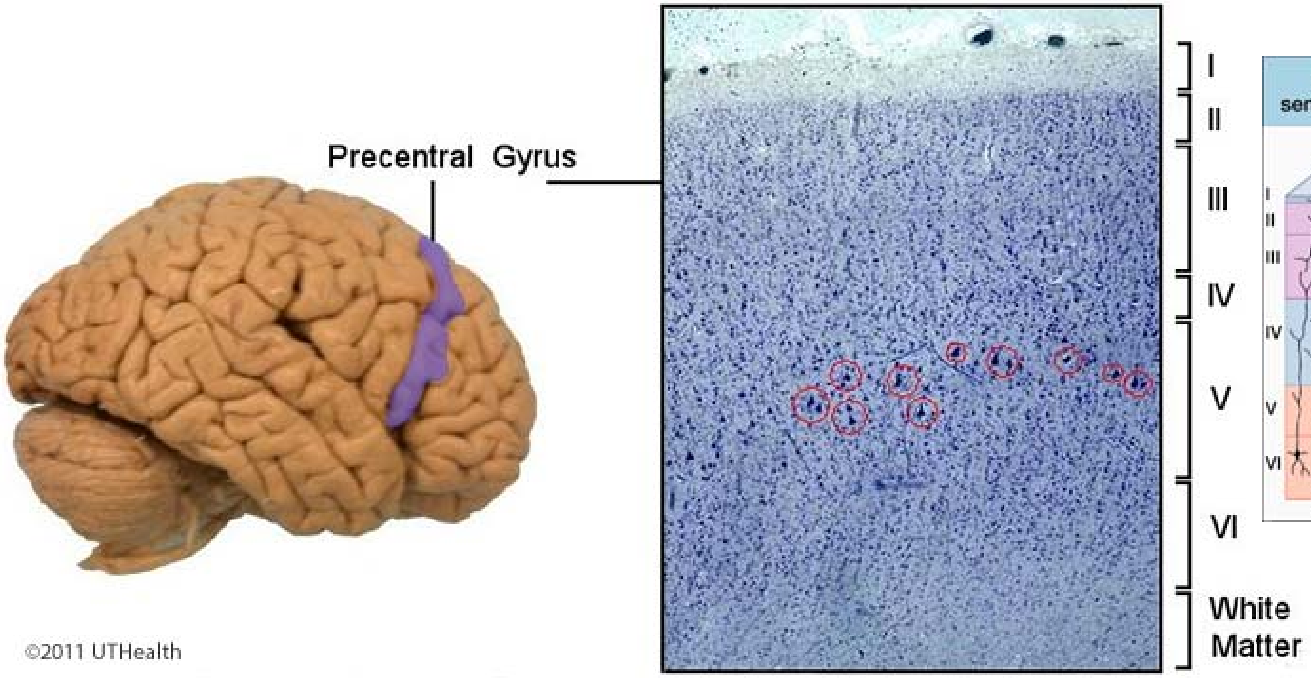
- Cytoarchitecture (hystology)
- Electrophysiologic characteristics
- Specific cortico-cortical projections
- Aferent-eferent neuronal projections
- Specific motor deficits after injuries of specific area

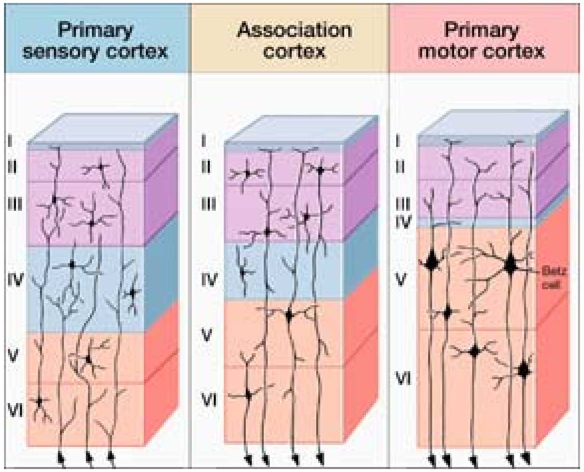
Histology of the Motor Cortex

- 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.
- Molecular layer and multiform layer
- The **corticospinal tracts** arise from the internal pyramidal cell layer situated mainly **in layer V**.

i. Molecular /plexiform layer
ii. External granular layer
iii. External pyramidal layer
iv. Internal granular layer
v. Internal pyramidal layer
vi. Multiform layer

Histology : MI, MII i SMA

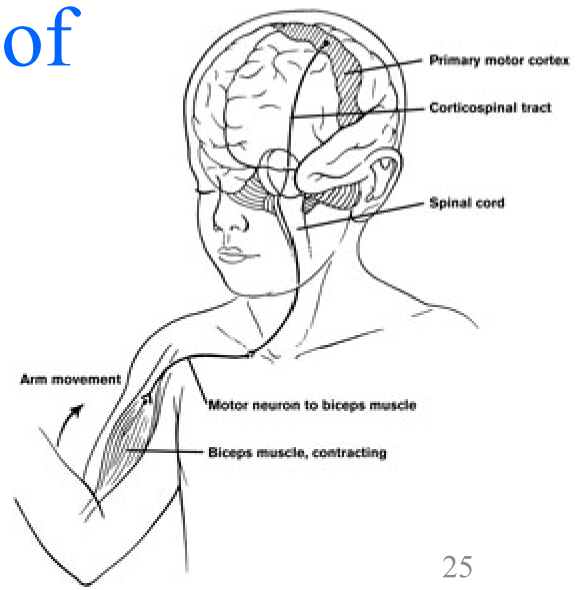




- ***Agranularity and disgranularity*** (no II and IV granular layer)
- ***magnocelularity*** (contain big piramidal cells)
- V. layer of the MI contains gigantic ***Betz pyramidal neurons***

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).

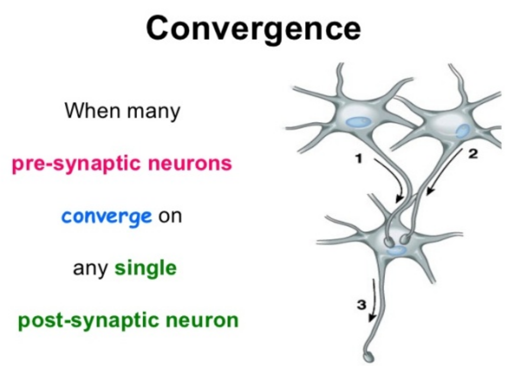


Electrophysiology of the CM neurons

- ***Corticomotoneurons (CM neurons)*** – monosinapticly 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.

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)!



- **Phasic CM neurons**
  - low spontaneous activity
  - Latency < 1ms
  - Fire action potentials during the movement
  - Frequency 20-80Hz
  - Delay between EPSPs 1.5 - 5 msec – sutable for summation in time

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

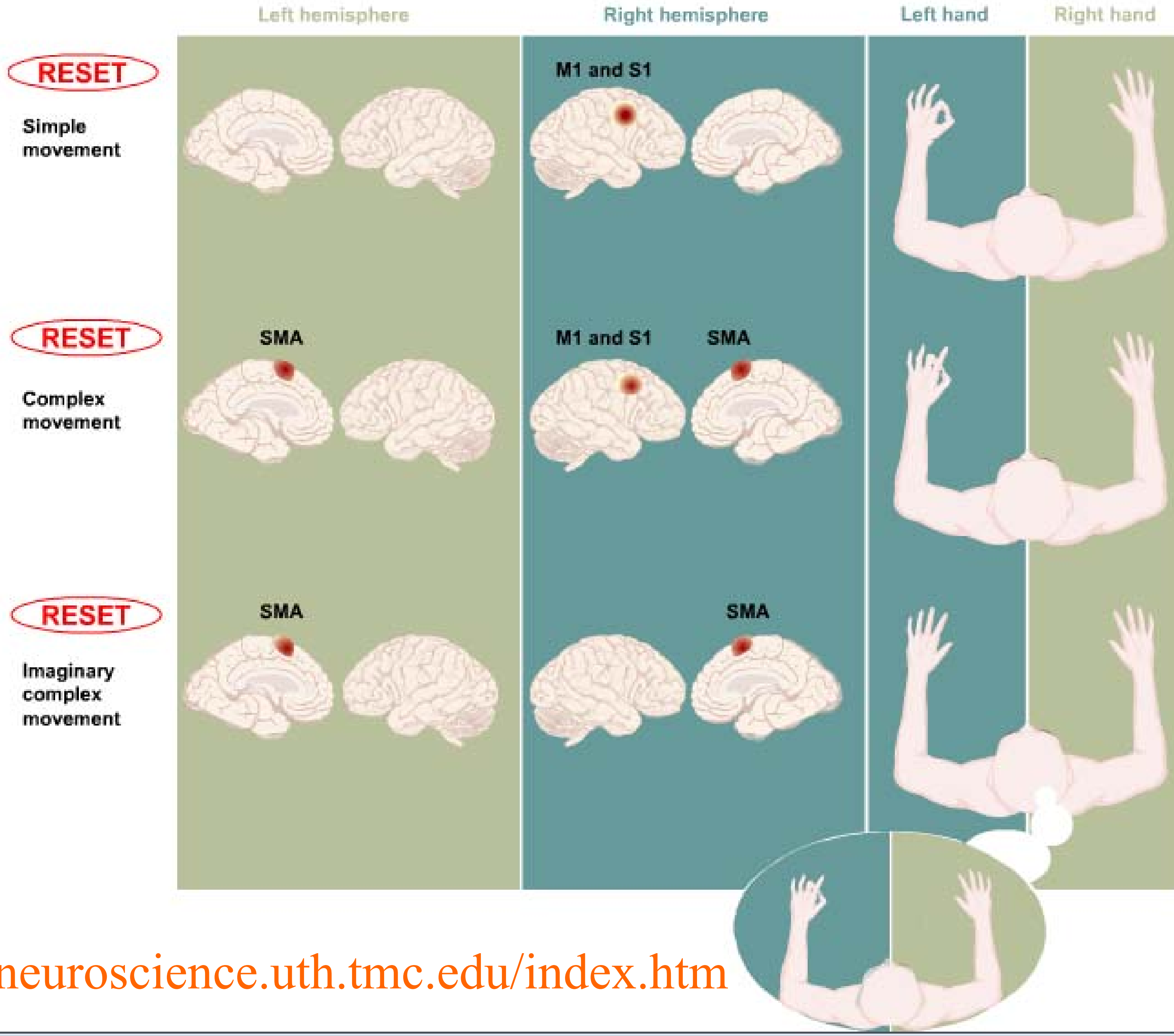
Smaller CM neurons

- Majority of neurons
- Equaly active during all movements,
- Responsible for slow movements of medium strenght, under the somatosensory control
- Included in system "closed loop"

Big CM neurons

Betz neurons

- 35.000 in MI
- active in strong and fast movements
- Included in open loop"
- *balistic movements*
- No correction of the movement, no sensory influence.
- Can only be repeated "to hit or to miss"



http://neuroscience.uth.tmc.edu/index.htm

Figures 3.7A, 3.7B, and 3.7C
Motor cortex encodes the force necessary to make a movement. (Evarts 1968)

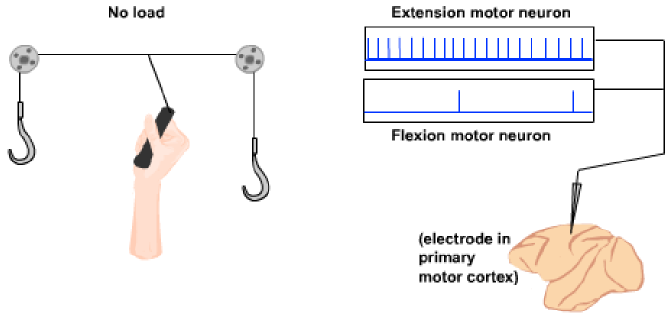


Figure 3.7A. When there is little load, a motor neuron in primary motor cortex that controls an extension of the wrist fires when the wrist extends. A motor neuron that controls wrist flexion does not change its low rate of activity. Note that the extension motor neuron begins to fire spikes before the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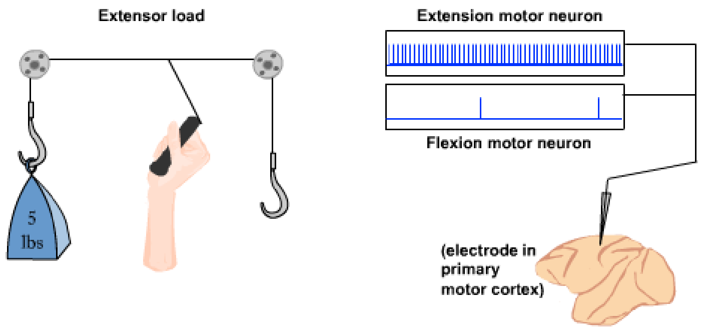

Figure 3.7B. When a 5 lb. load is placed on the left pulley, more force must be used to initially hold the weight steady and then lift it. The extension motor neuron in primary motor cortex fires more strongly to produce the greater force.
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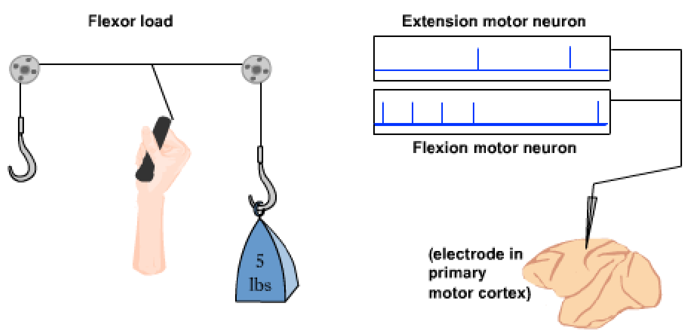

Figure 3.7C. When a 5 lb. load is placed on the right pulley, the load is on the flexor. Thus, primary motor cortex neurons for flexion are activated to keep the weight stable. When the wrist extends, the neurons are quieter, as the force of the movement is actually produced by the weight itself. (Note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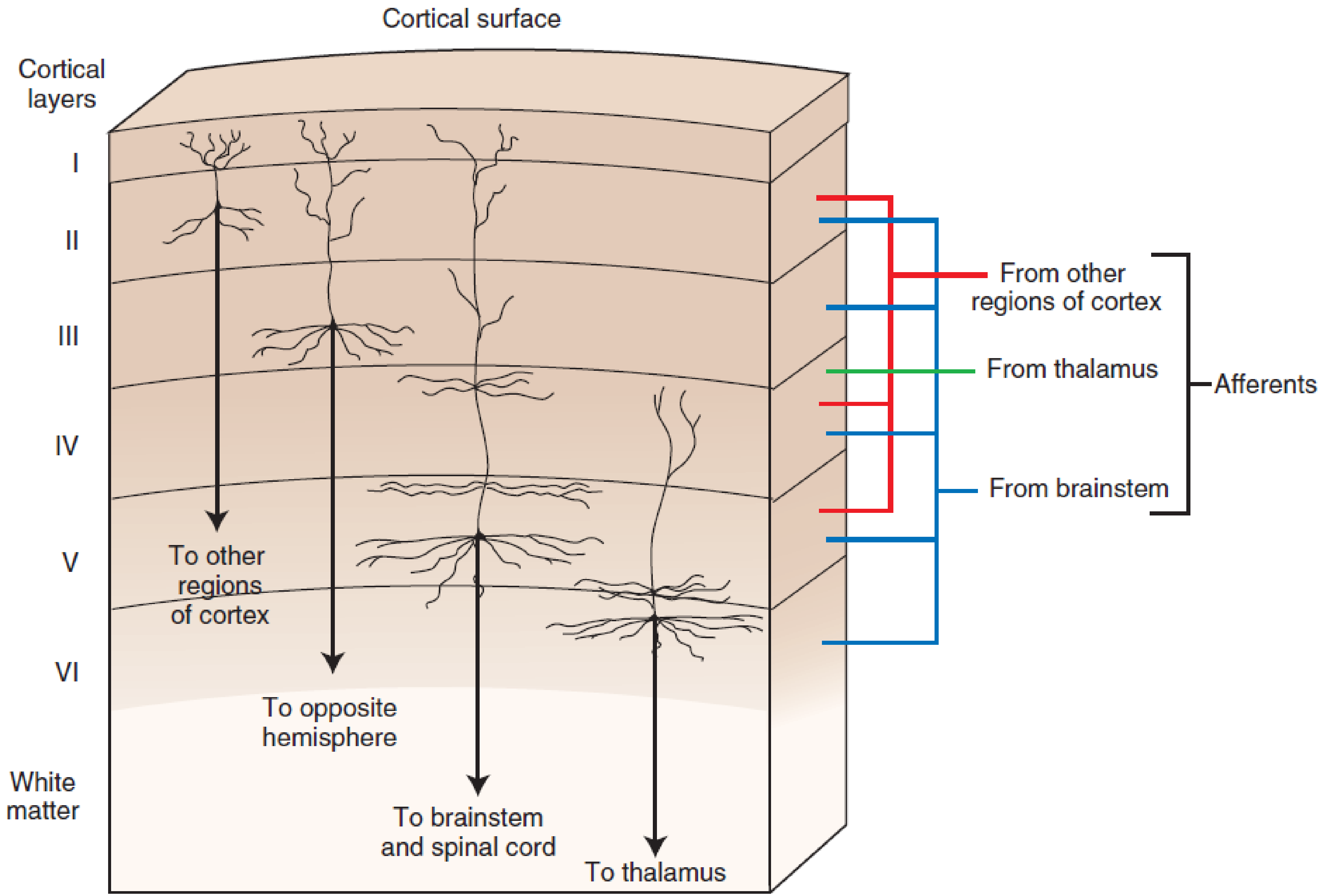
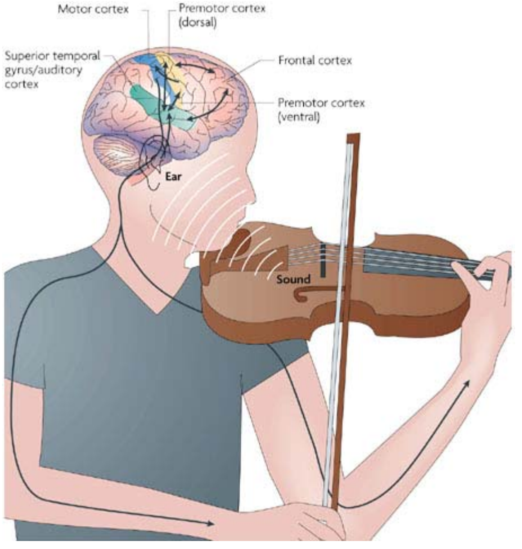
Afferent and efferent connections of the motor corte



- The **primary motor cortex** receives ***indirect inputs*** from the cerebellum and globus pallidus.
- The integration of all of these inputs occurs within the primary motor cortex rather than in the thalamus.
- 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.

- 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.



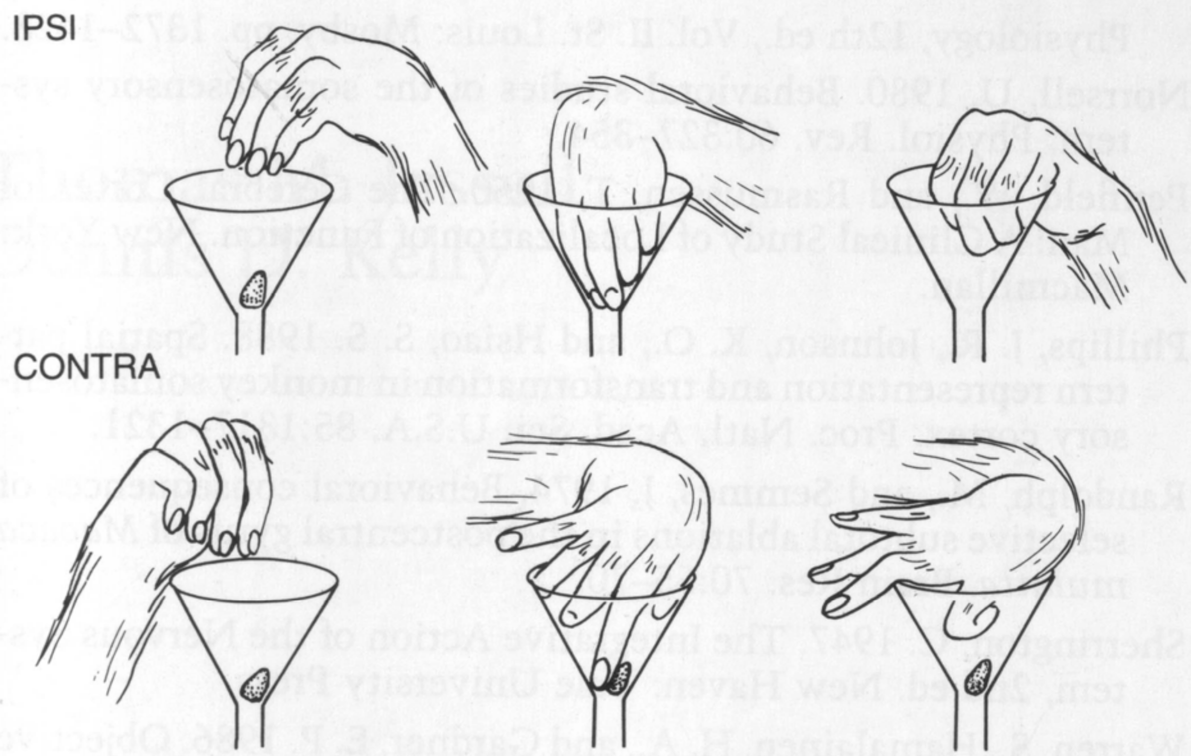


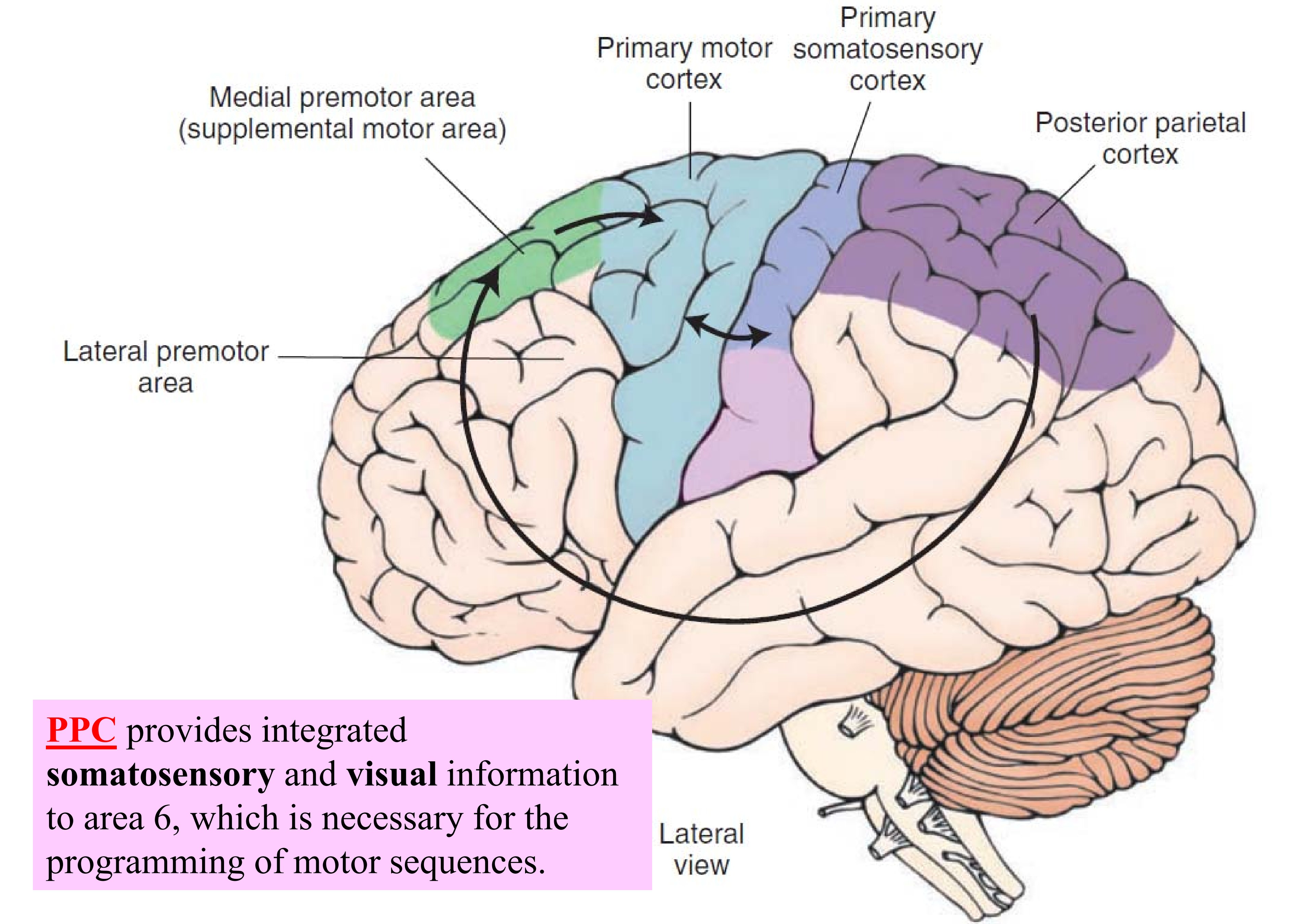
FIGURE 26–15
A monkey's finger coordination is disrupted following the injection of muscimol, a GABA agonist that inhibits synaptic transmission in the somatic sensory cortex. The left hand **(ipsi)** is able to pick up an apple piece from a funnel. Two hours following the injection of muscimol into Brodmann's area 2 on the left side, the finger coordination of the right hand **(contra)** is severely disorganized. (Adapted from Hikosaka et al., 1985.)

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**
- **<u>Afferent inputs (from basal ganglia, cerebellum and PPC)</u>**
- 1. Inputs from the basal ganglia is directed mainly to the SMA.
- 2. Cerebellar efferent fibers sends 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.

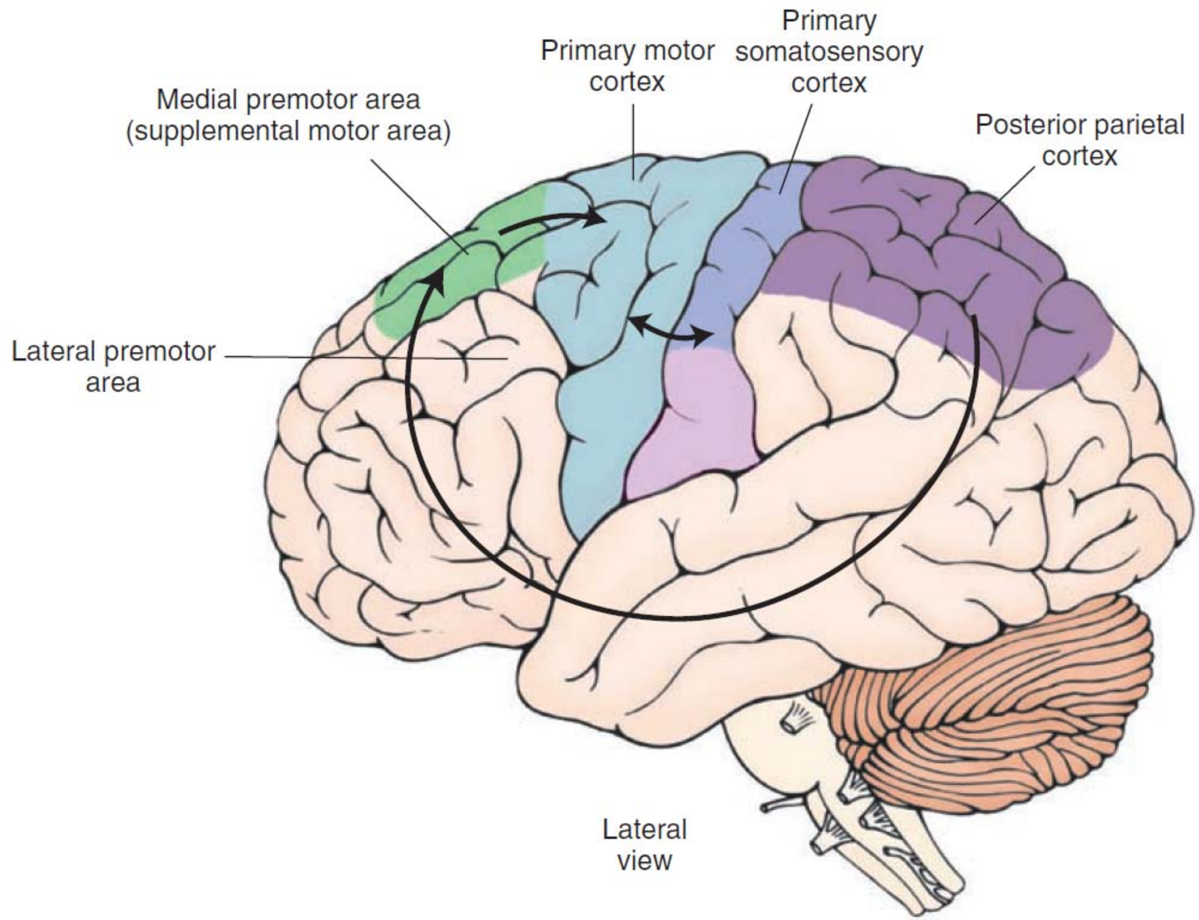
Supplemental motor area

- 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 (**inabilility 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.
- 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



sensory neglect (or anosognosia)

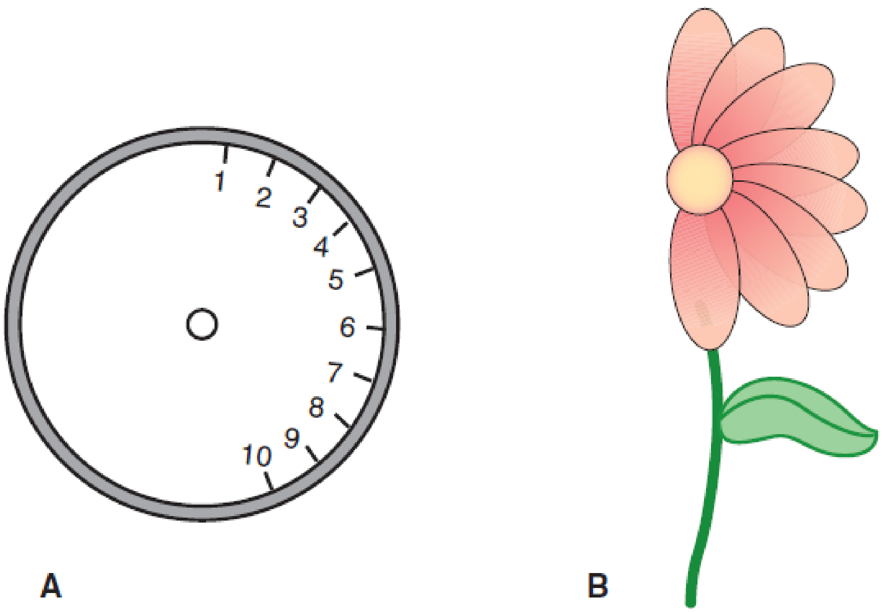
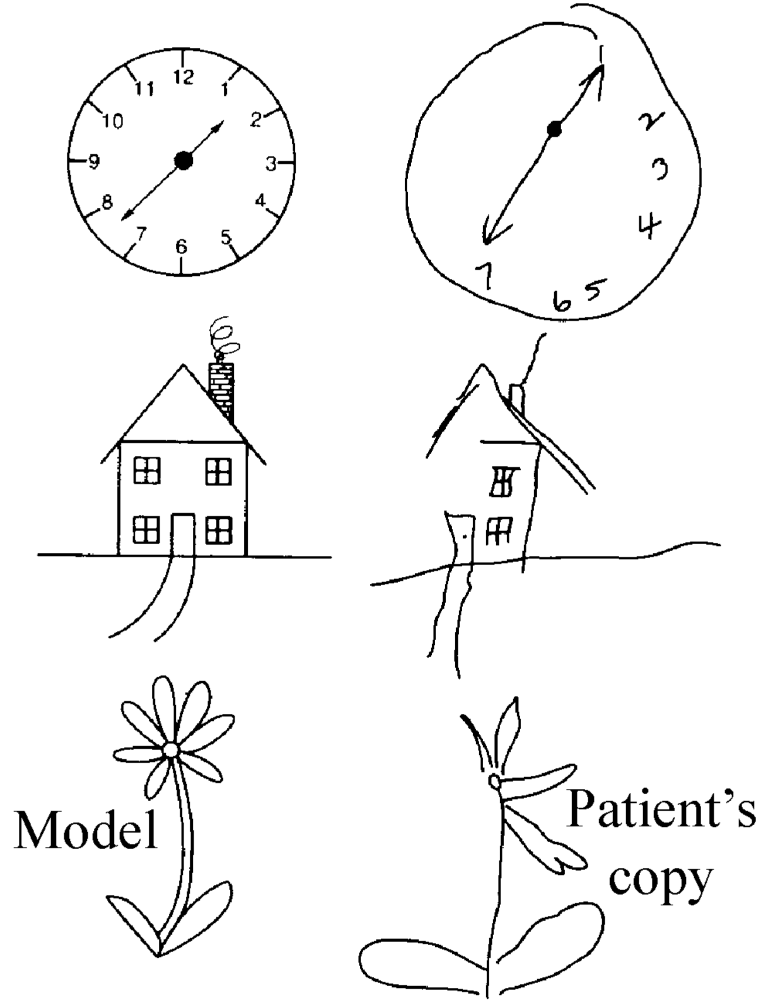


FIGURE 19–7 Drawings made by a patient with a lesion of the right posterior parietal cortex, indicating sensory neglect on the left side. The patient was asked to fill in numbers on the face of the clock (A) and to draw a flower (B).

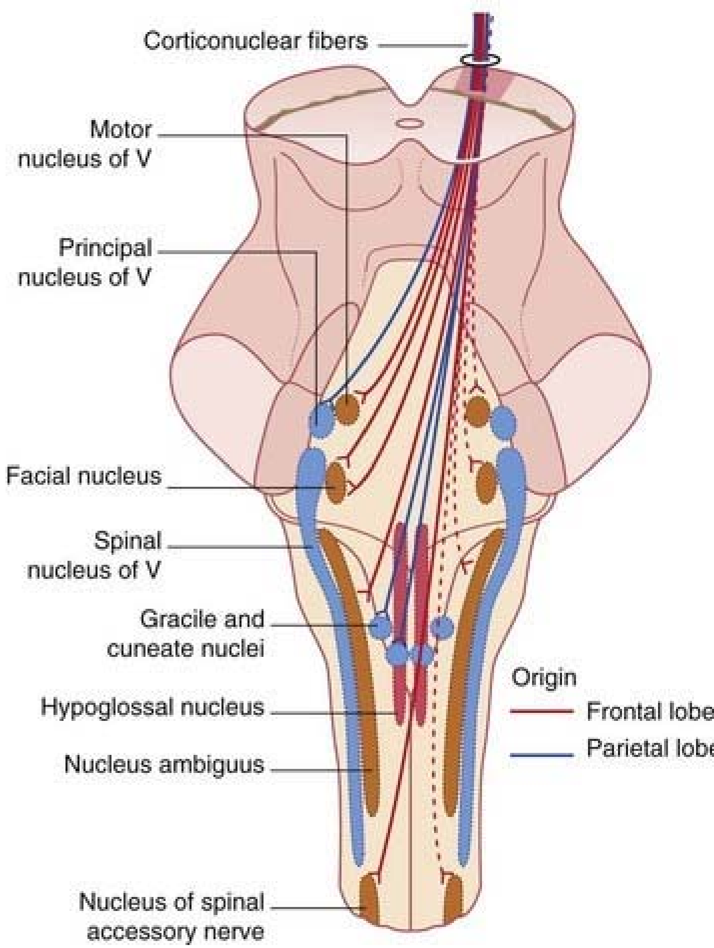


Patient denies condition

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 pathways*
- nuclues ***n. trigeminus*** **–** chewing
- nuclues ***n. facialis (vii)*****–** facial expression
- nuclues ***n. hypoglosus (xii)*** **–** tongue muscles
- nucleus ambiguus – vocal cords



Tractus corticonuclearis

- Projections are bilateral
- Exception: lower part of the facial nucleus - contralateral projections
- Corticobulbar projections to the nucleus of CN XII are completely contralateral.

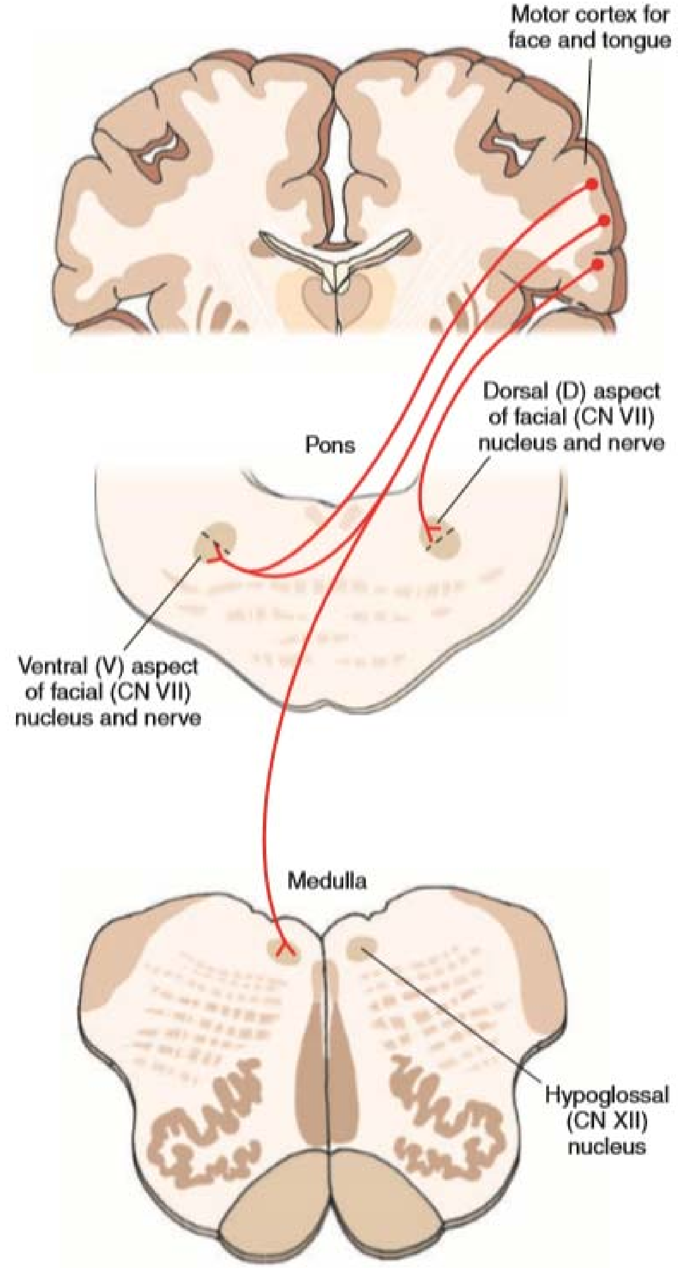
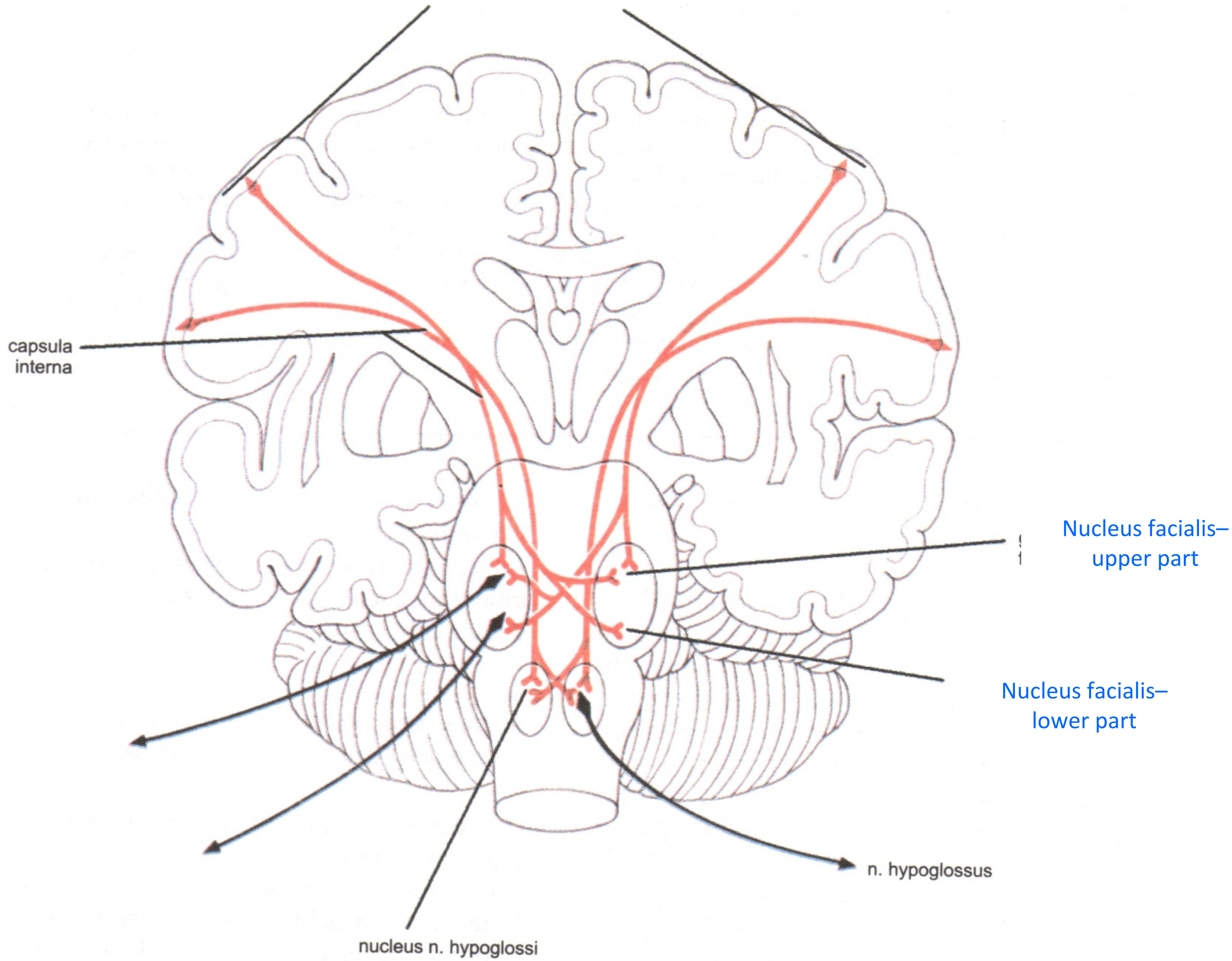


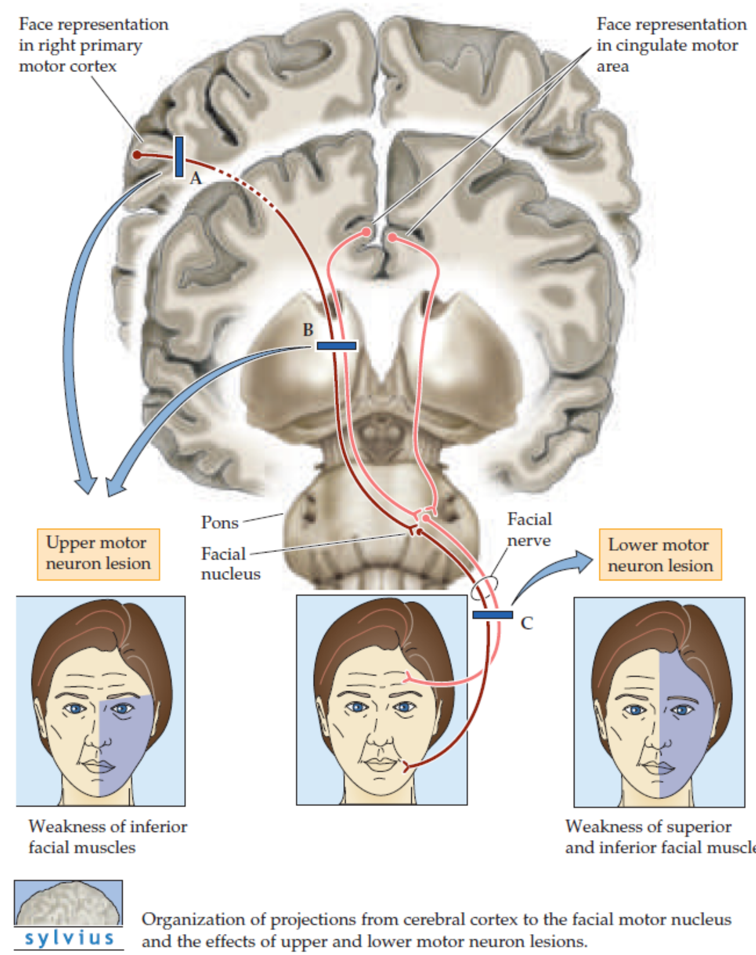
FIGURE 18.9 Projections of corticobulbar fibers to motor nuclei of cranial nerves VII and XII. The projections to CN VII, which are contralateral, innervate the ventral aspect of the nucleus of CN VII, whose axons supply the lower jaw. The projections to CN VII, which are ipsilateral, innervate the ventral aspect of this nucleus, and its projections are too much of the face region. Corticobulbar projections to the nucleus of CN XII are completely contralateral.



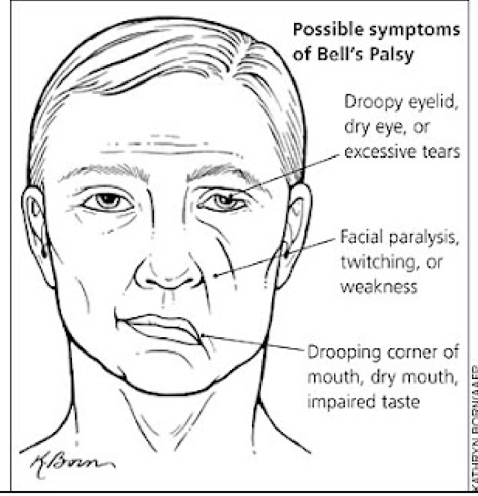
Slika 34-3. *Tractus corticonuclearis* je voljni motorički put za mišiće lica, jezika, grkljana i ždrijela. Ova slika prikazuje da su te projekcije uglavnom bilateralne – no, uočite važnu iznimku: donji dio facijalne jezgre (što inervira mišiće donjeg dijela lica) prima isključivo kontralateralne projekcije.

Lesions of Corticobulbar Fibers That Supply Nuclei of Cranial Nerves

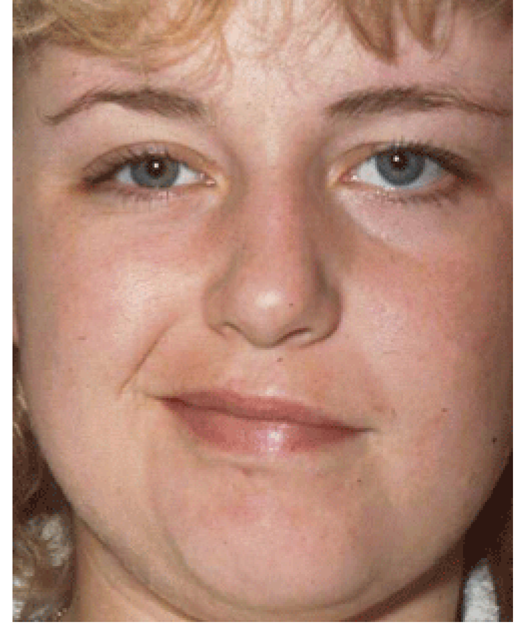
- **central facial palsy**
- **pseudobulbar palsy**

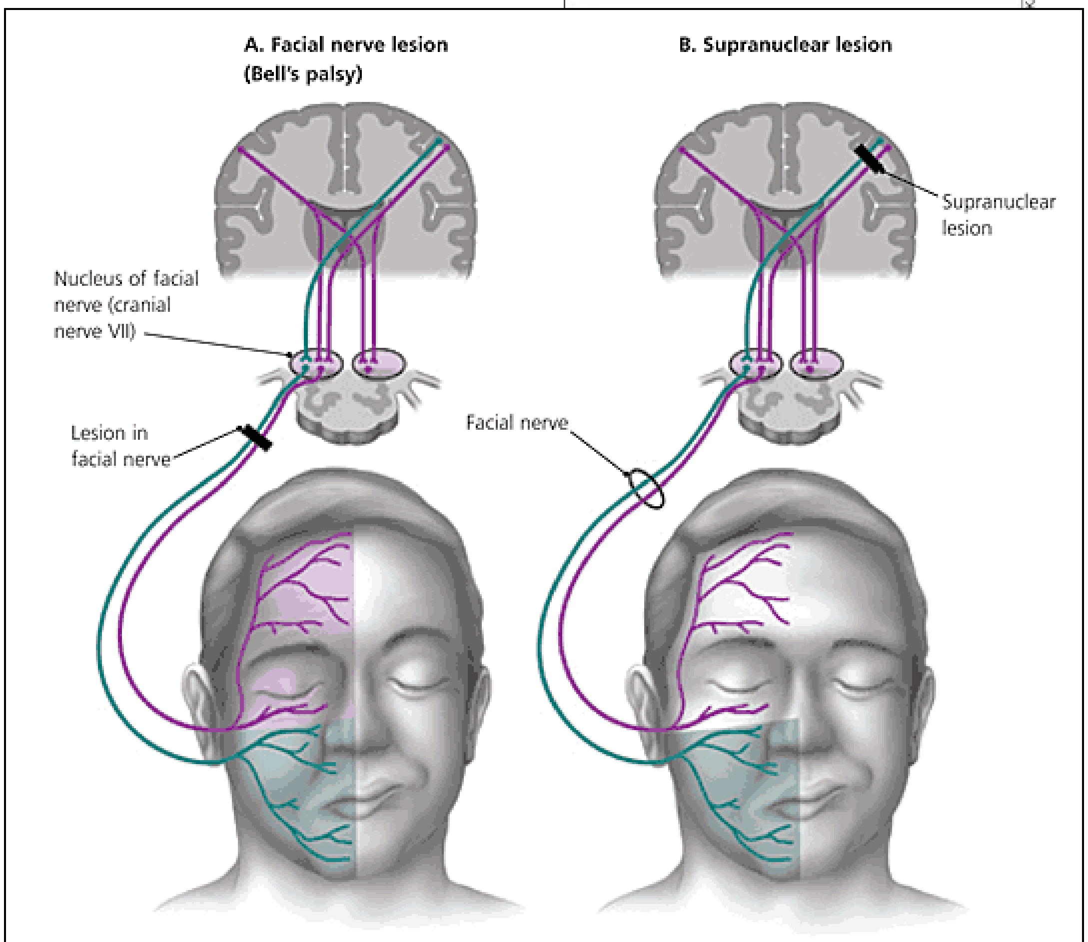


Organization of projections from cerebral cortex to the facial motor nucleus and the effects of upper and lower motor neuron lesions.

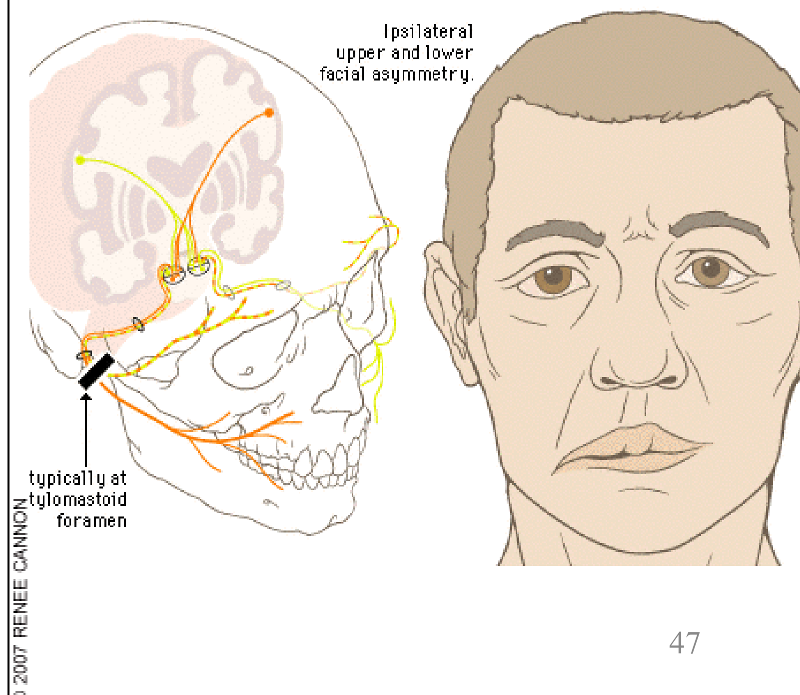


Possible symptoms of Bell's Palsy
Droopy eyelid, dry eye, or excessive tears
Facial paralysis, twitching, or weakness
Drooping corner of mouth, dry mouth, impaired taste
KATHRYN BORN/AAFP





A. Facial nerve lesion (Bell's palsy)
B. Supranuclear lesion
Supranuclear lesion
Nucleus of facial nerve (cranial nerve VII)
Lesion in facial nerve
Facial nerve



Ipsilateral upper and lower facial asymmetry.
typically at tylomastoid foramen
© 2007 RENEE CANNON

Injuries of the motor cortex

- Due to cerebrovascular insult (stroke due to hemorage or ischemia) damage of the cortex or corticospinal tract can develop.

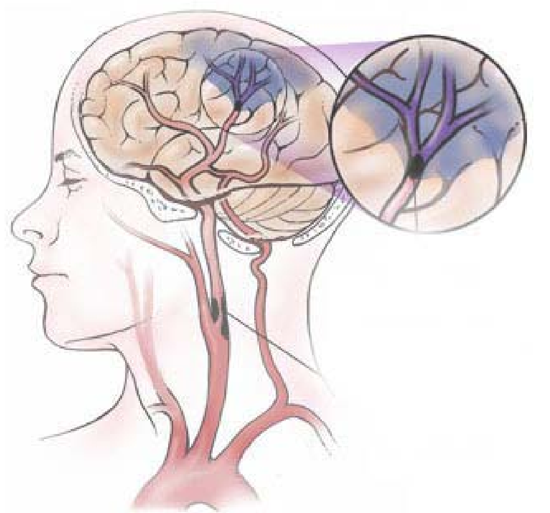


TABLE 16.1
Signs and Symptoms of Upper and Lower Motor Neuron Lesions

Upper Motor Neuron Syndrome	*Lower Motor Neuron Syndrome*
Weakness	Weakness or paralysis
Spasticity	Decreased superficial reflexes
Increased tone	Hypoactive deep reflexes
Hyperactive deep reflexes	Decreased tone
Clonus	Fasciculations and fibrillations
Babinski's sign	Severe muscle atrophy
Loss of fine voluntary movements	

THE UPPER MOTOR NEURON SYNDROME

Upper motoneuron – corticospinal tract, project to the α-motoneurona; syndrome: **hypertonia, spasticity,** abnormal ref exes, such as the **Babinski sign** also referred to as an **extensor plantar response**

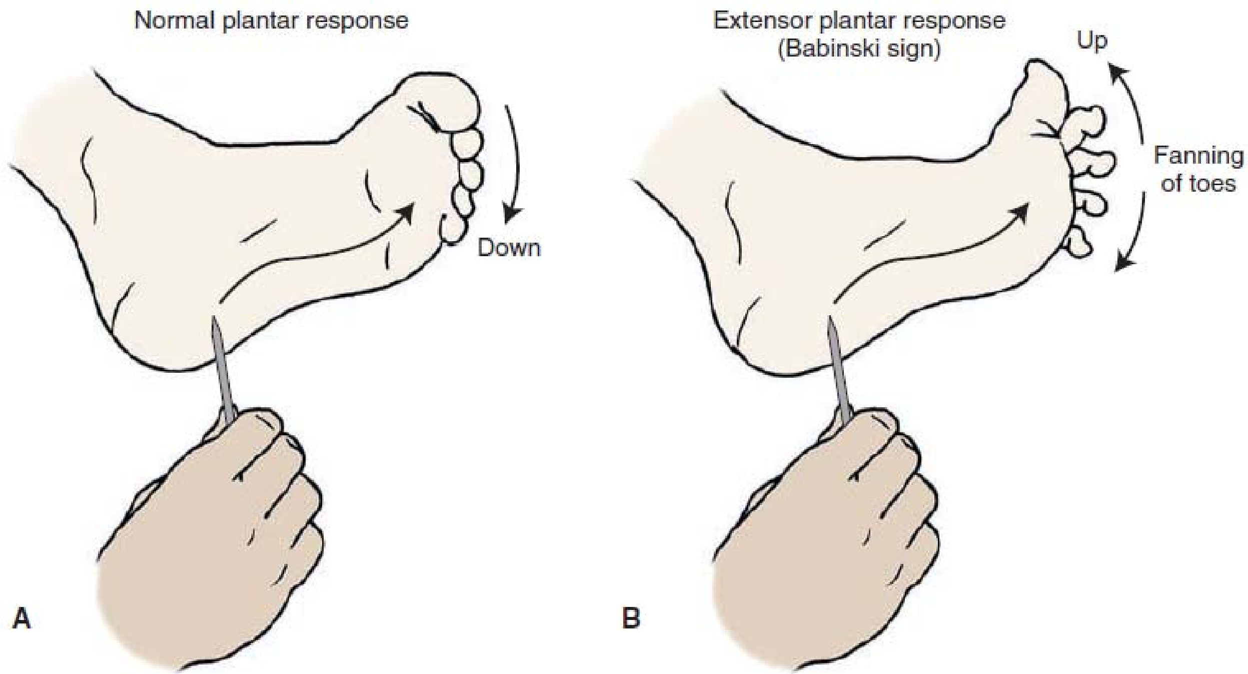
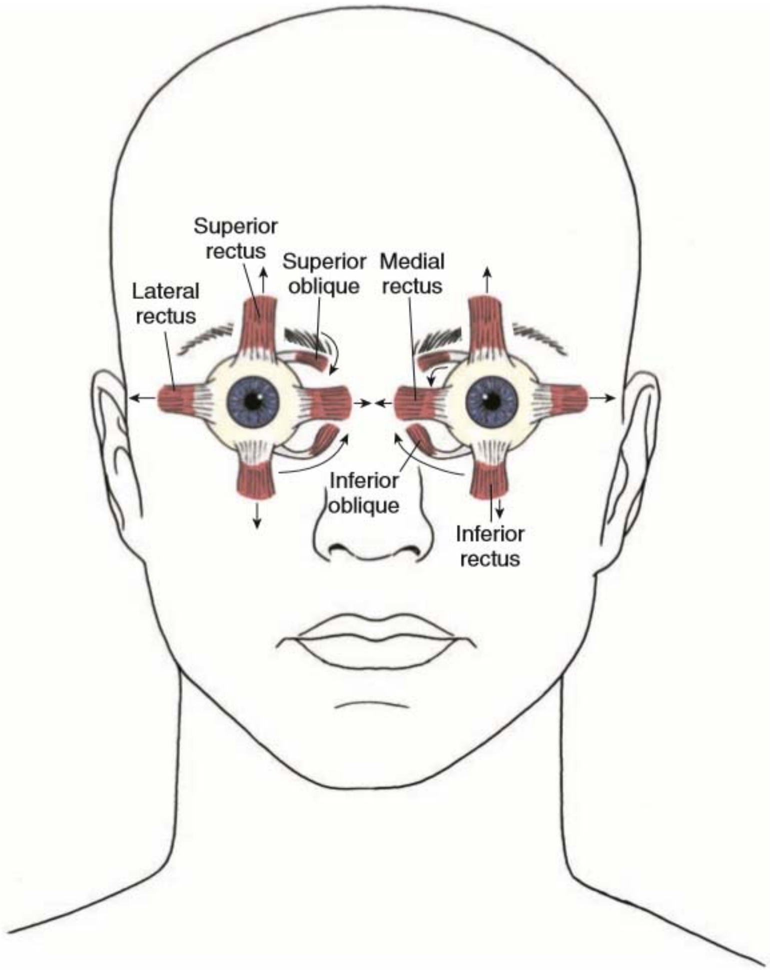


FIGURE 19–9 Illustration of a Babinski test. The sole of the foot is stroked with a sharp object from front to back. This kind of stimulation typically produce flexion of the foot (A). However, a patient who has an upper motor neuron paralysis will show extension of the large toe coupled with a fanning out of the other toes (B).

The control of Gaze

Eye movement and eye gaze direction system

- eyes
- muscles
- Nerves
- CNS



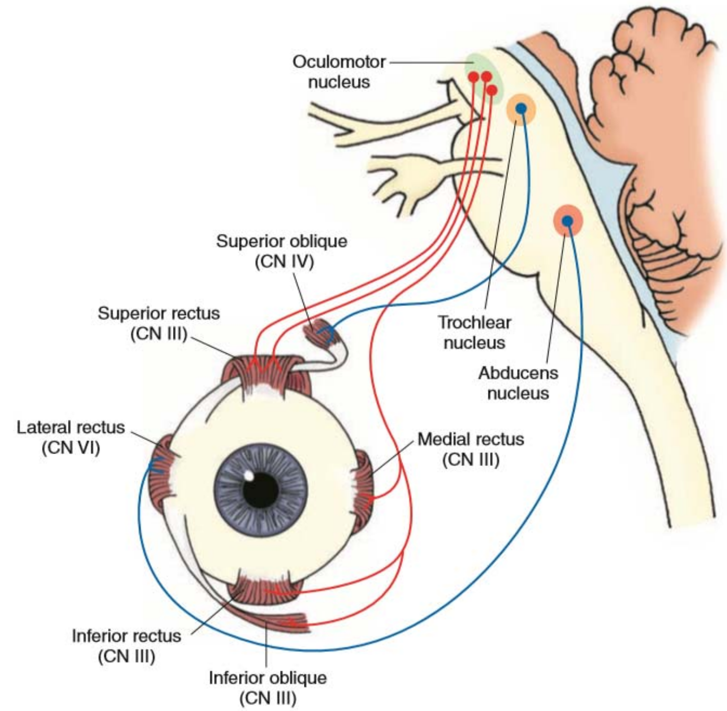
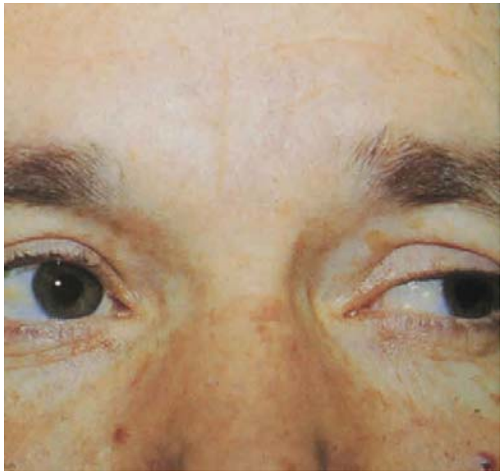


FIGURE 13.12 Diagram illustrating the direction of actions of the extraocular muscles of the eye (indicated by *arrows*). The lateral rectus muscle is innervated by the abducens nerve, the superior oblique muscle is innervated by the trochlear nerve, and the remaining muscles are innervated by the oculomotor nerve.

N. oculomotorius (III)

- Supply to: ***m. rectus superior, inferior, medialis*** ***and*** ***m. obliquus inferior***
- *m. levator palpebrae superioris*
- *m. ciliaris and m. constrictor pupillae*

<u>Lesion of *n. occulomotorius*</u>



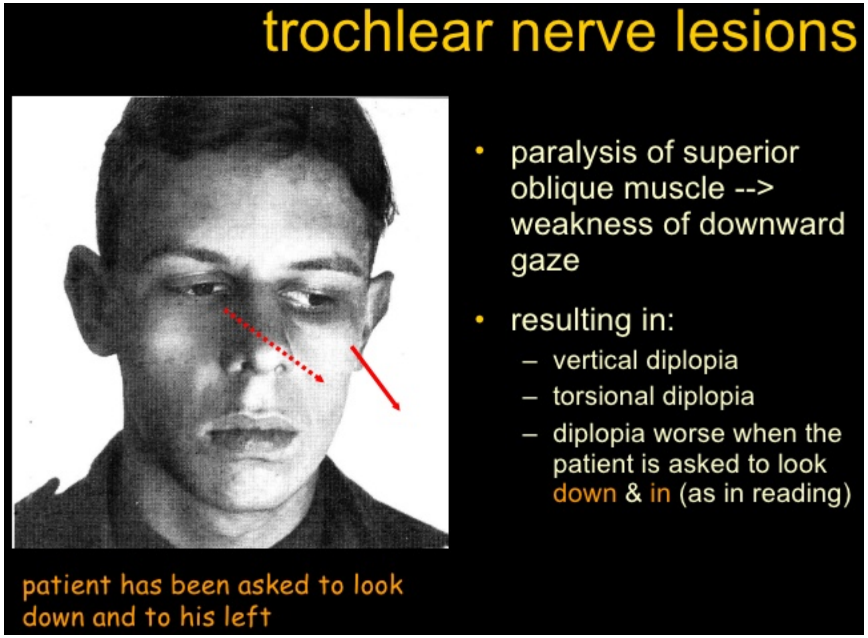
- Activity of the *m. rectus lat i m. obliquus superior* overrides the activity of other muscles – eye is rotated lateral and down
- *diplopia*
- No adduction
- Pupil is wide (midriasis), no constriction during lightening or accomodation
- *ptosis*
- No accomodation

N. trochlearis (IV)

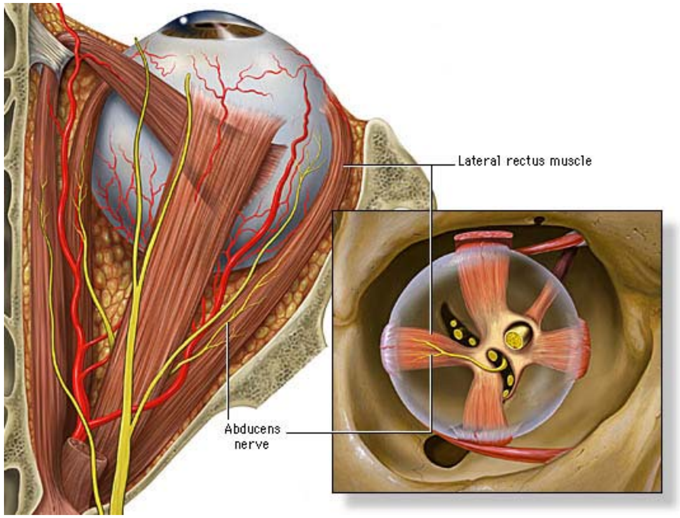
- supplies ***m. obliquus superior***

Lesion of the *n. trochlearis*

- Activity of the *m. obliquus inferior overrides other muscles*
- Eye is elevated *(elevatio) and rotated* out *(extorsio)*
- *diplopia*



N. abducens (VI)



- supplies *m. rectus lateralis*
- **Lesion of the *n. abducens***
  - Activity of the *m. rectus medialis overrides other muscles*
  - Eye is *adducted*
  - Can't look on the side
  - *diplopia*

- Occulomotor system moves the eyes in the orbit.
- 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

- Movements that keep the fovea on a visual target in the environment
- Movements that stabilize the eye during head movement

- There are four basic types of eye movements: ***saccades***, ***smooth pursuit movements***, ***vergence movements***, and ***vestibulo-ocular movements***.

- ***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 except *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.

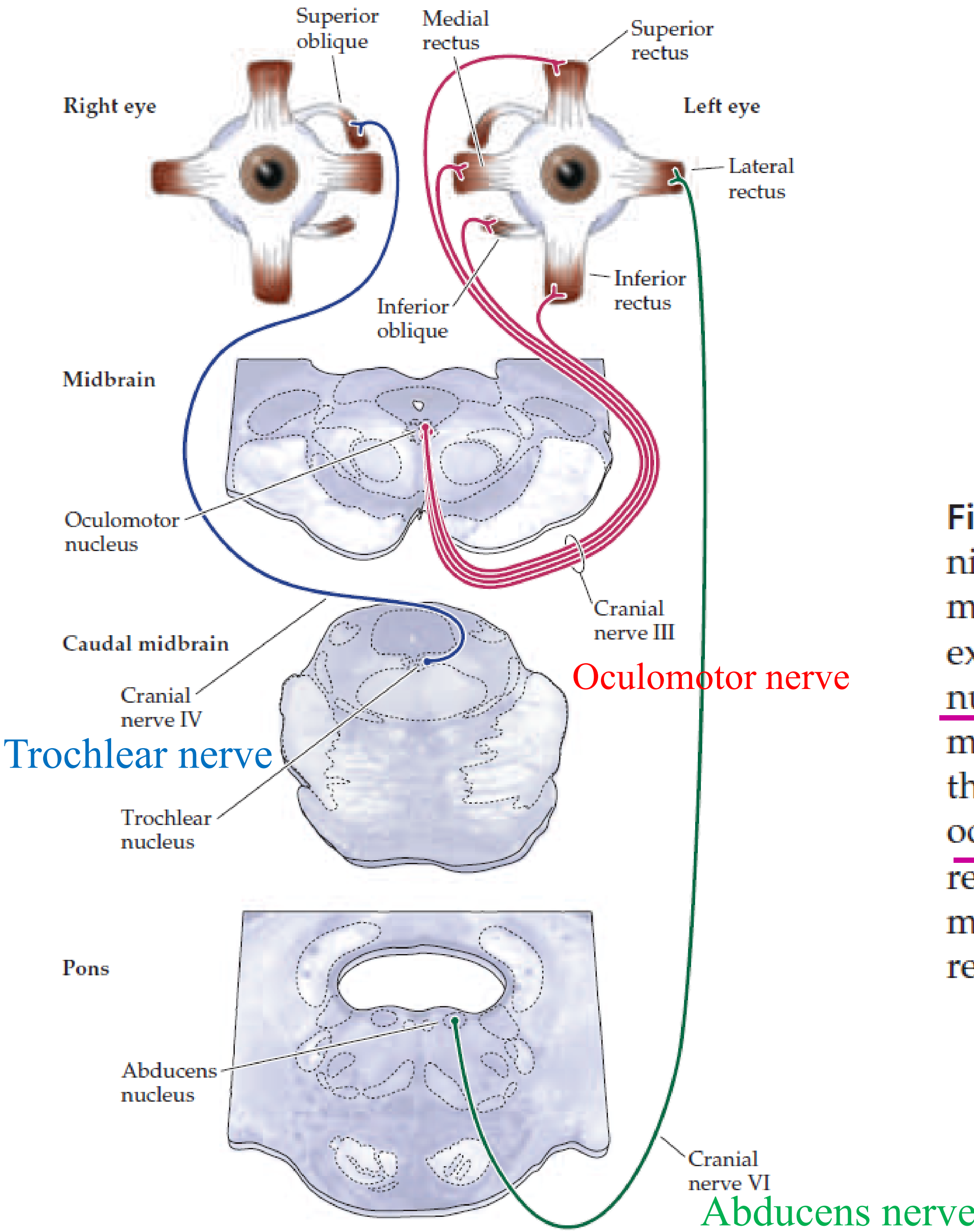


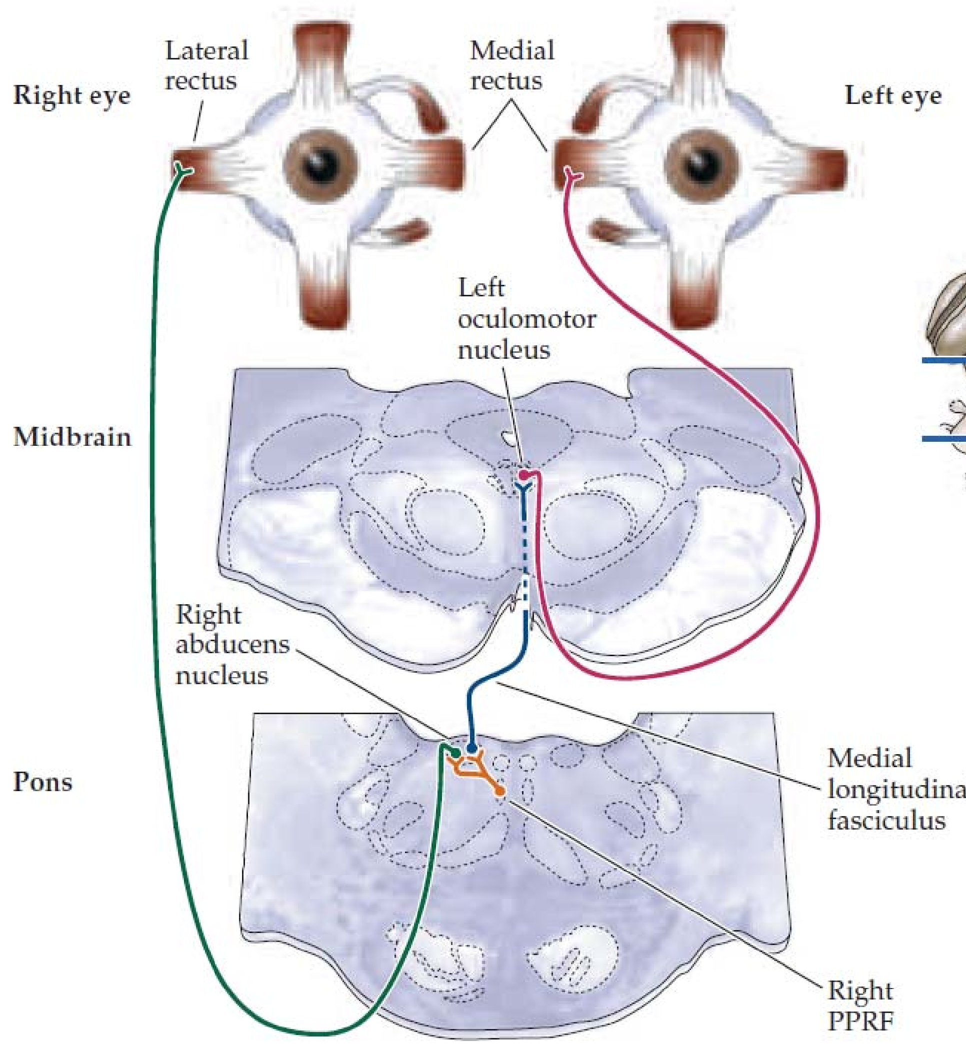
Figure 19.3 Organization of the cranial nerve nuclei that govern eye movements, showing their innervation of the extraocular muscles. The abducens nucleus innervates the lateral rectus muscle; the trochlear nucleus innervates the superior oblique muscle; and the oculomotor nucleus innervates all the rest of the extraocular muscles (the medial rectus, inferior rectus, superior rectus, and inferior oblique).

Control of Eye Movements: Role of the Pontine Gaze Center

- 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 horizontal or **pontine gaze center** or **parapontine reticular formation (PPRF)**
- **C) vestibular nuclei**
- **D) vertical gaze center (**rostral midbrain-PAG**)**

Eyes to the right

Figure 19.7 Simplified diagram of synaptic circuitry responsible for horizontal movements of the eyes to the right. Activation of local circuit neurons in the right horizontal gaze center (the PPRF; orange) leads to increased activity of lower motor neurons (red and green) and internuclear neurons (blue) in the right abducens nucleus. The lower motor neurons innervate the lateral rectus muscle of the right eye. The internuclear neurons innervate lower motor neurons in the contralateral oculomotor nucleus, which in turn innervate the medial rectus muscle of the left eye.



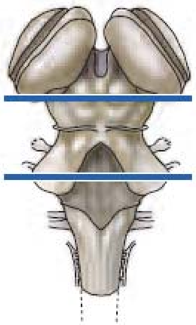
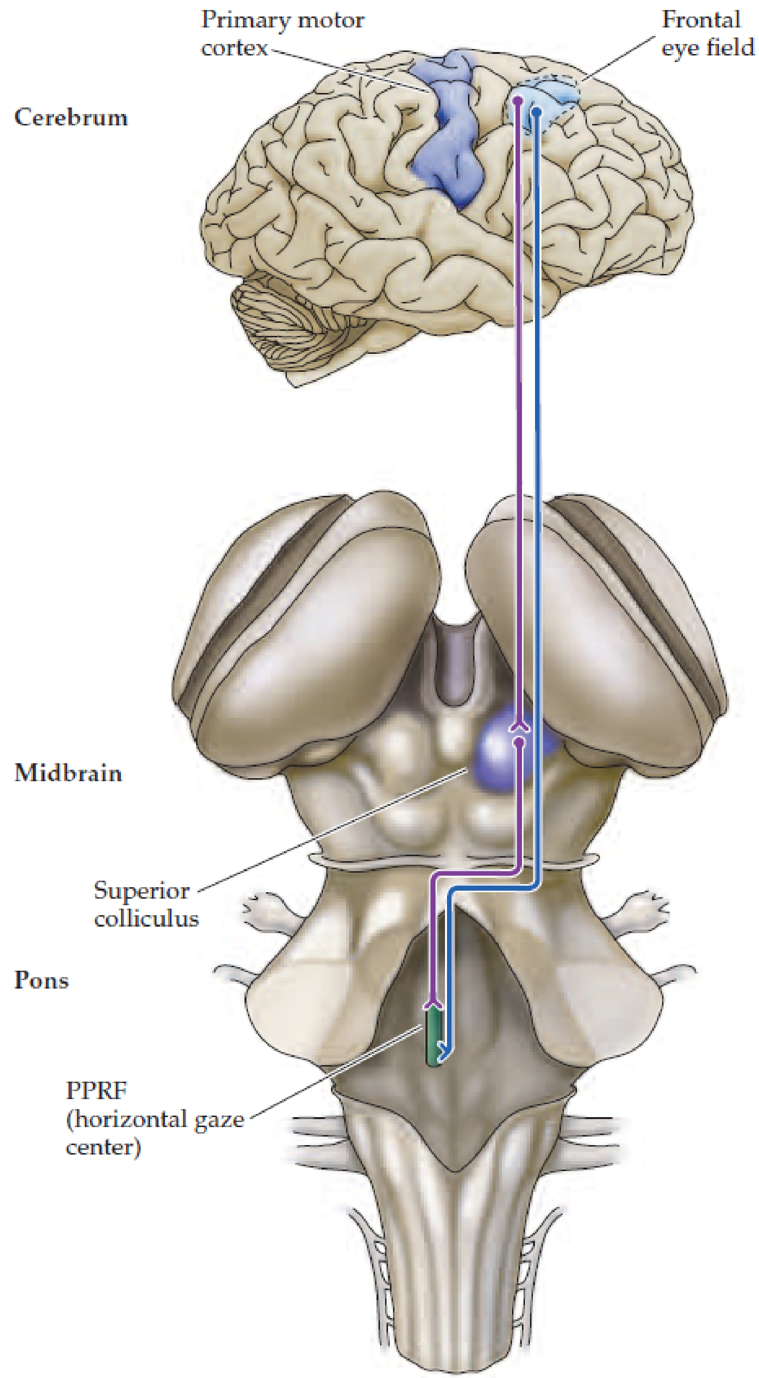
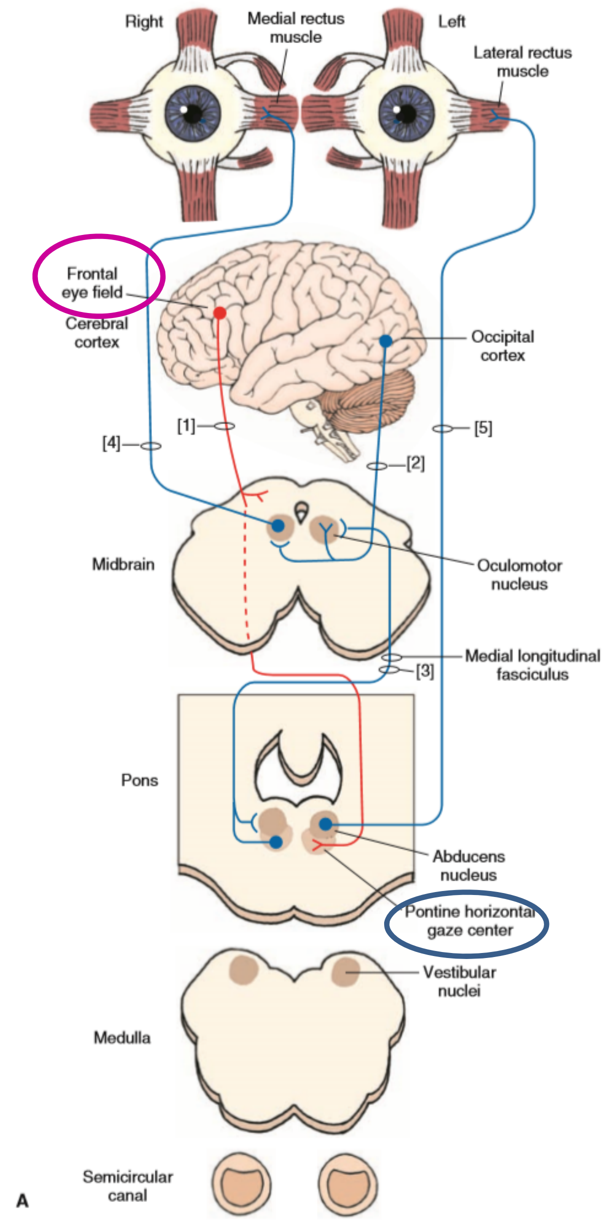


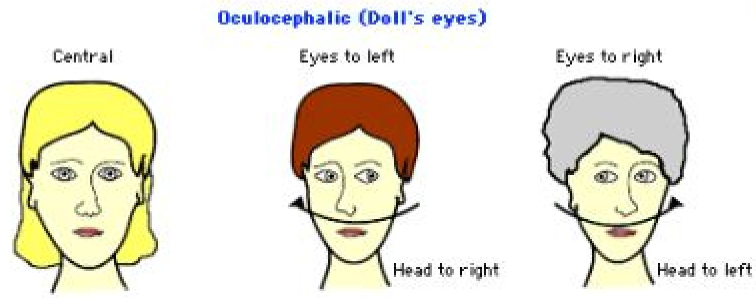
Figure 19.9 The relationship of the frontal eye field in the right cerebral hemisphere (Brodmann's area 8) to the superior colliculus and the horizontal gaze center (PPRF). There are two routes by which the frontal eye field can influence eye movements in humans: indirectly by projections to the superior colliculus, which in turn projects to the contralateral PPRF, and directly by projections to the contralateral PPRF.



- 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 (PPRF).**
- 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.

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.

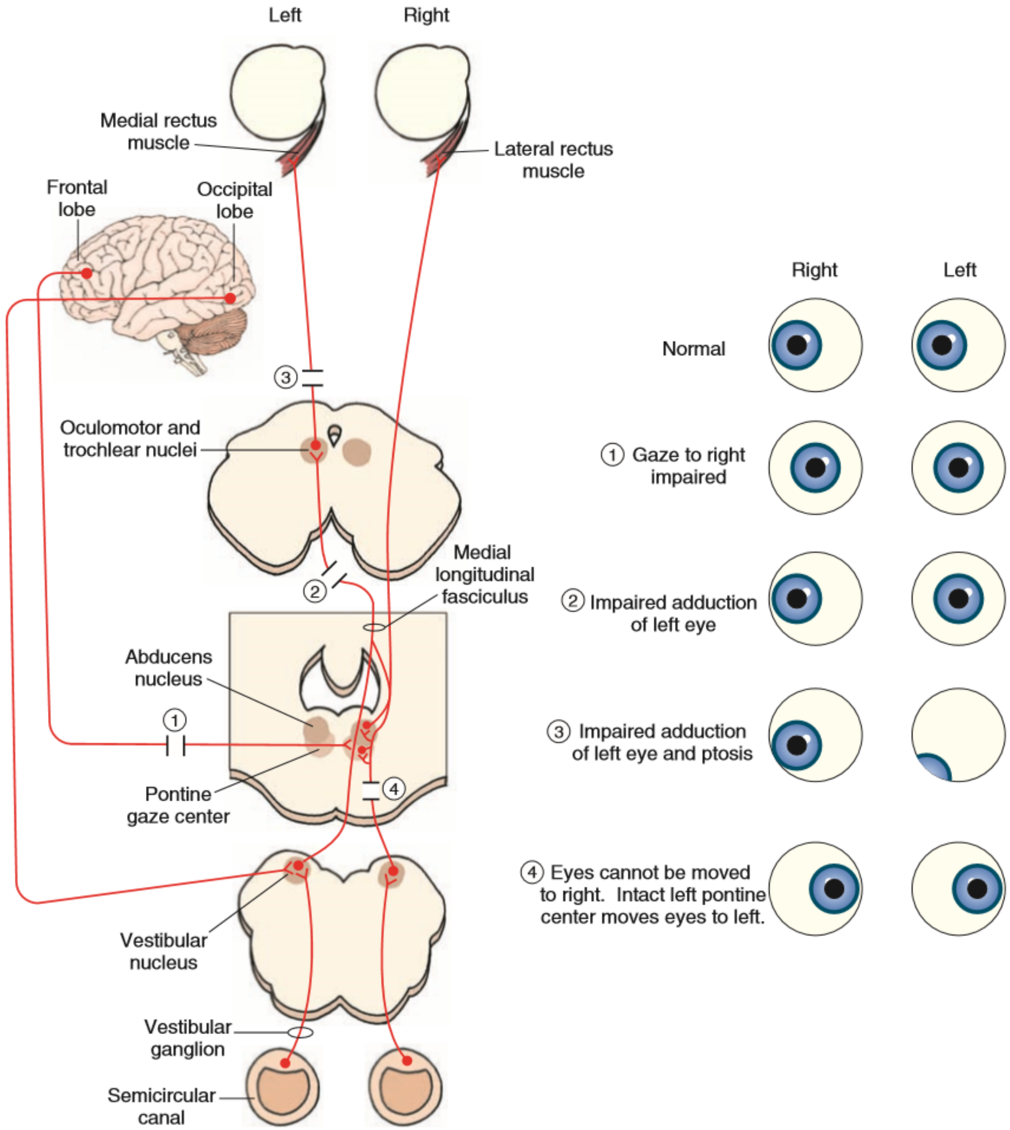
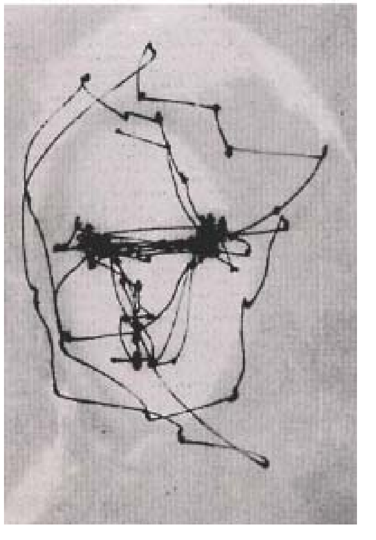


FIGURE 13.17 Diagram of the anatomical substrates for lateral gaze (left side of figure) and the deficits that occur following lesions at different sites along this pathway (right side of figure). For purposes of illustration, the diagram depicts the mechanisms involved in right conjugate gaze. Voluntary 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.

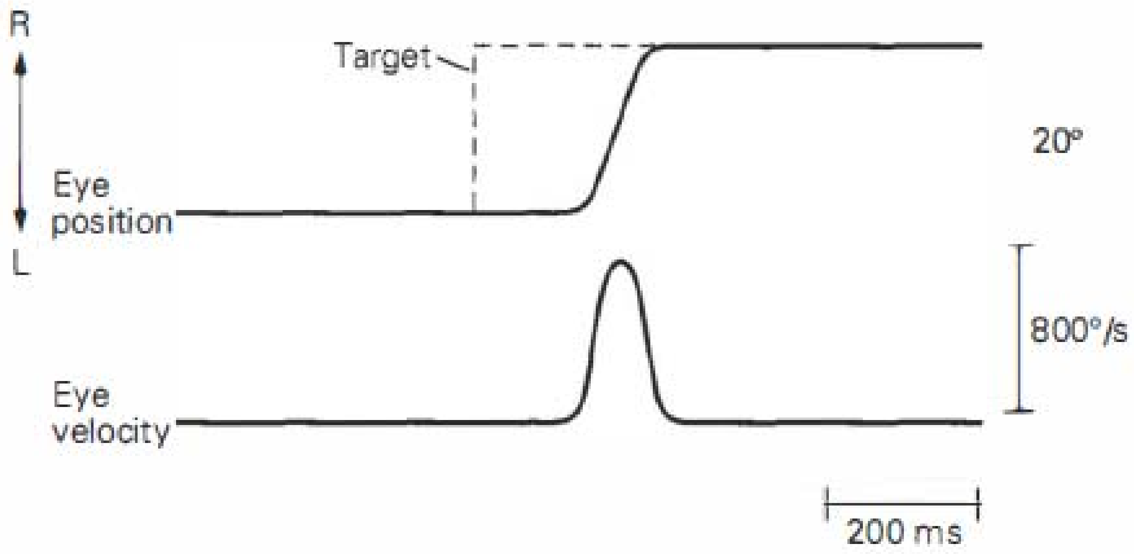
- **<u>Horizontal movements:</u>**
- **saccadic movements,**
- **smooth pursuit movements.**

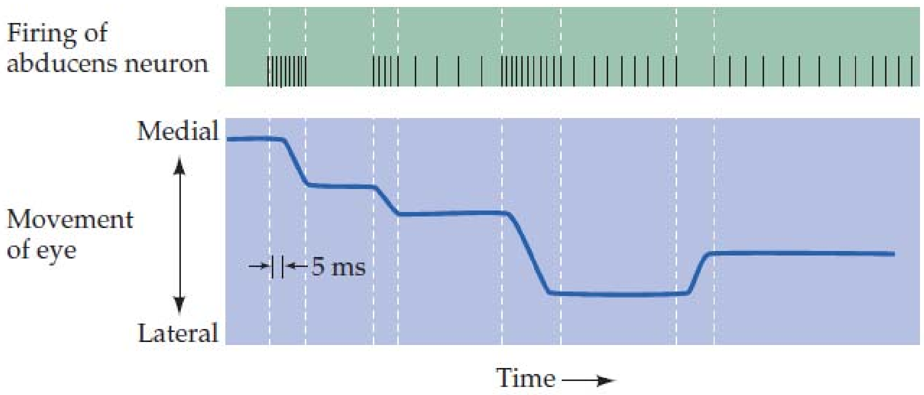
1. Saccadic eye movements

- 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





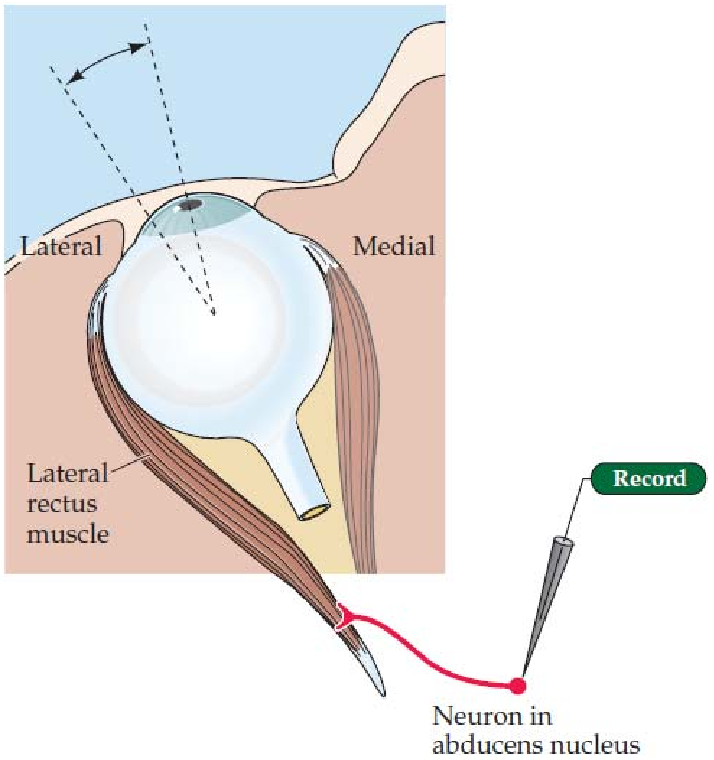
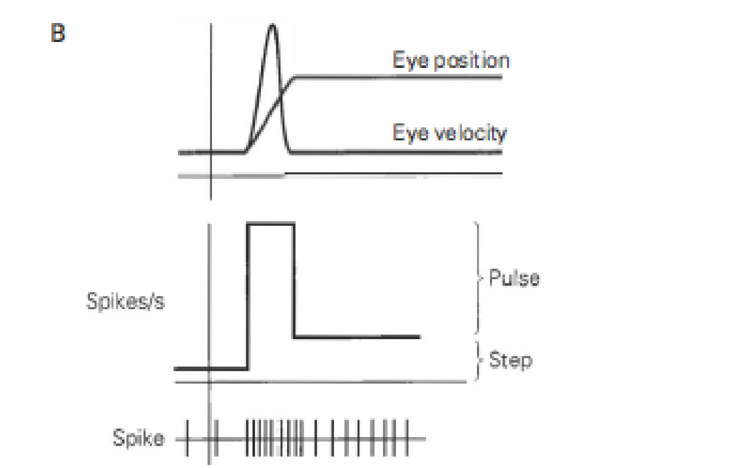


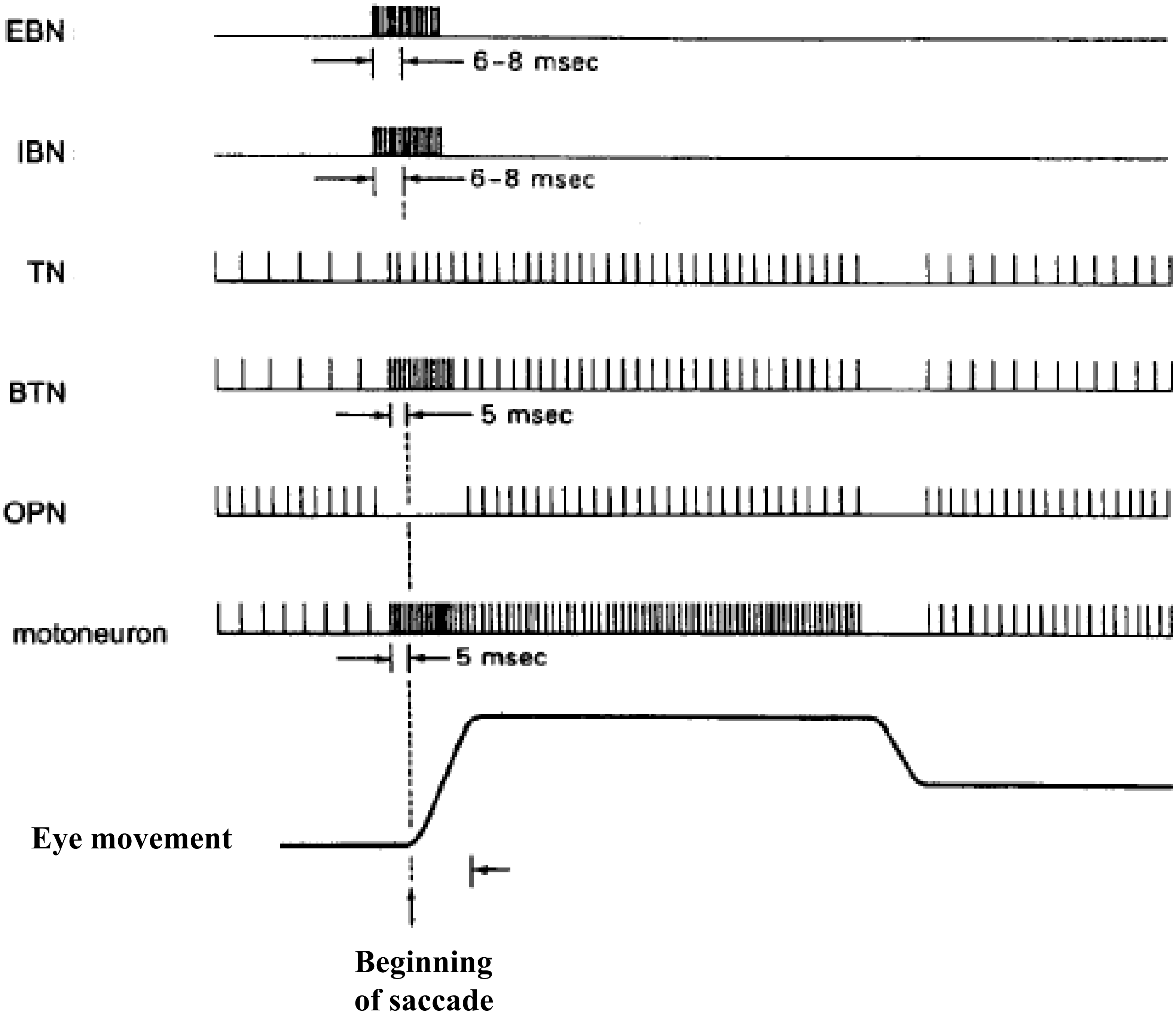
Figure 19.6 Motor neuron activity in relation to saccadic eye movements. The experimental setup is shown on the right. In this example, an abducens lower motor neuron fires a burst of activity (upper trace) that precedes and extends throughout the movement (solid line). An increase in the tonic level of firing is associated with more lateral displacement of the eye. Note also the decline in firing rate during a saccade in the opposite direction. (After Fuchs and Luschei, 1970.)



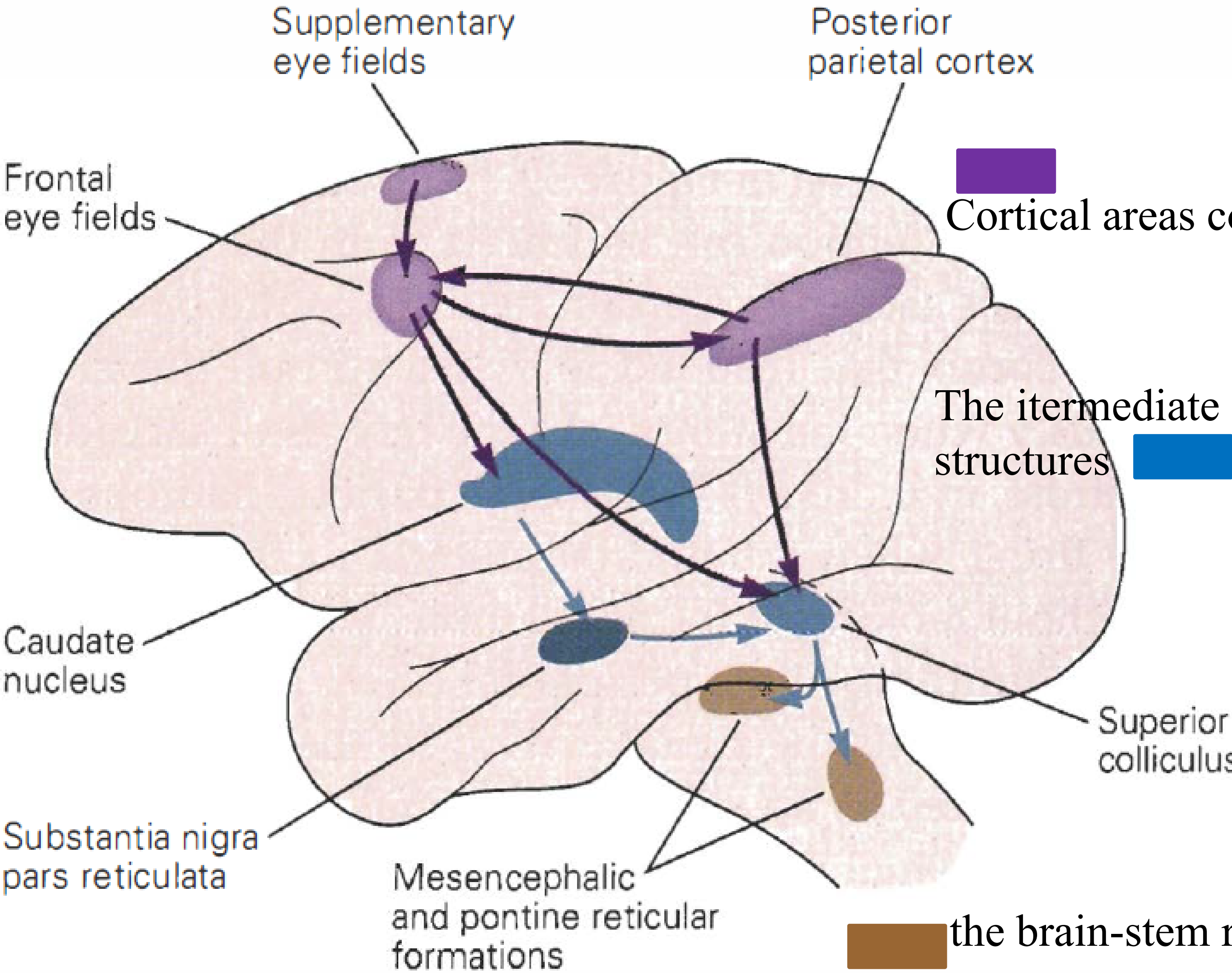
- Pulse is based on fast activation of ***BN cells (burst neurons)***
- BN control horizontal saccades and are located in *pontine gaze center* ***(paramedian pontine reticular formation (PPRF)***
- ***BN cells*** can be exscitatory ***(EBN),*** inhibitory ***(IBN)***, or tonic ***(TN)***

OPN cells

- “omnipause neurons”
- Located in *nucleus raphe interpositus (RIP)*
- Project on BN cells on the opposide side
- Continuously active EXCEPT durring sacades (“shut down” 16 msec before)


EBN
6-8 msec
IBN
6-8 msec
TN
BTN
5 msec
OPN
motoneuron
5 msec
Eye movement
Beginning
of saccade

Cortical pathways for saccadic eye movements in the monkey



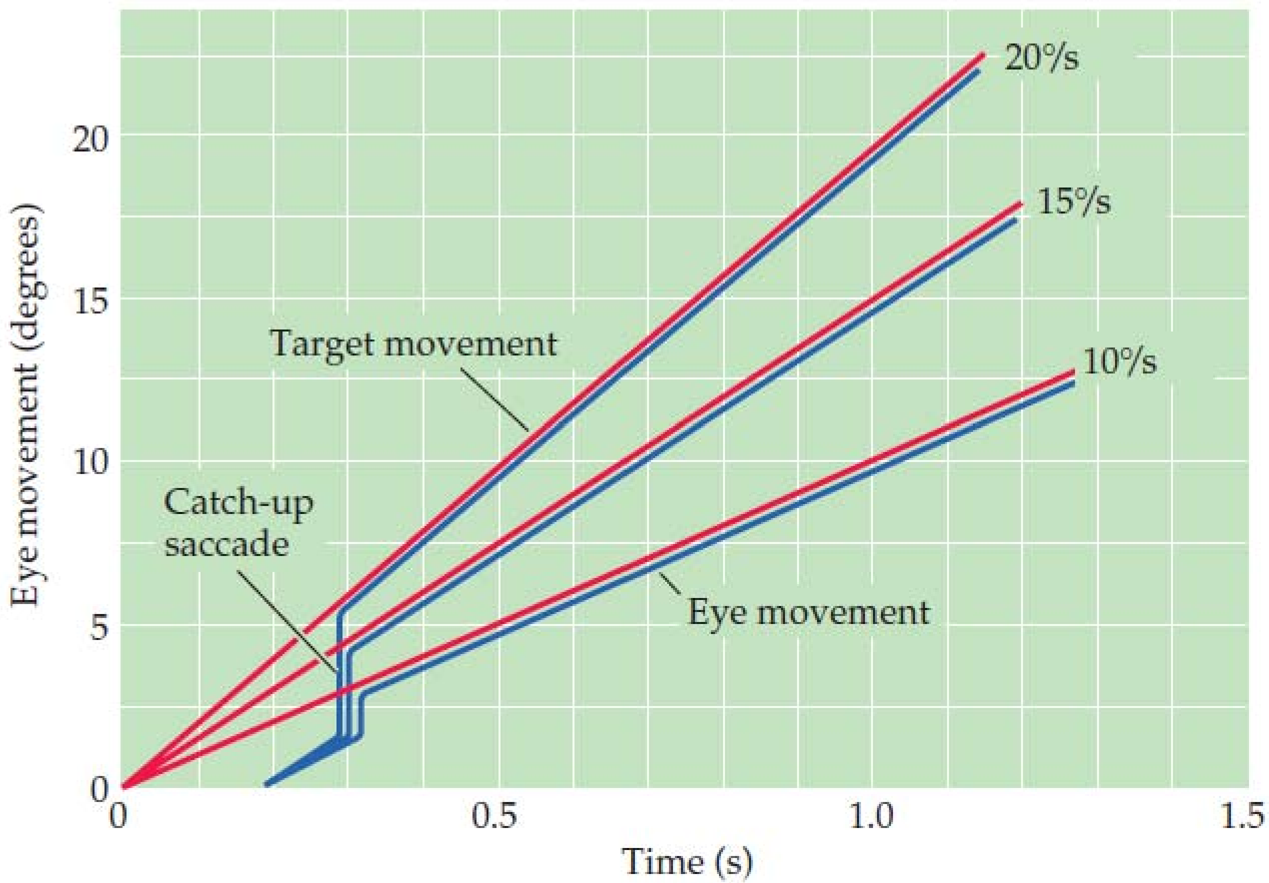
Cortical areas controlling saccades

The itermediate supra-nuclear structures

the brain-stem reticular formation

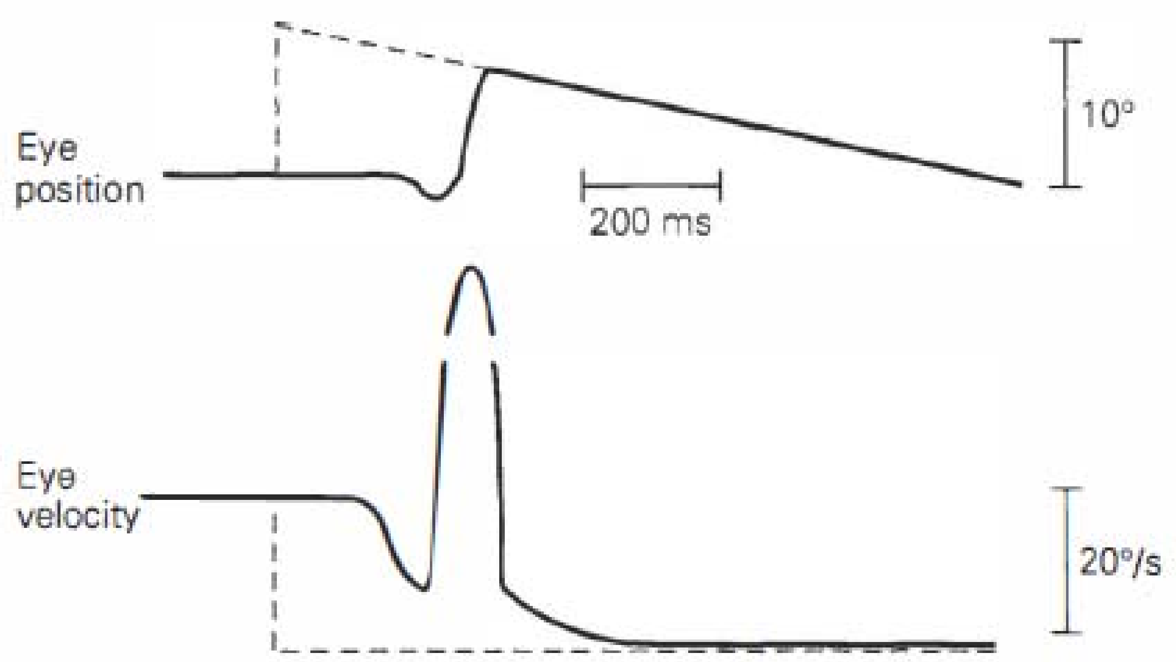
2. Smooth pursuit movements

- 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.



20°/s
15°/s
10°/s
Target movement
Catch-up saccade
Eye movement
Eye movement (degrees)
0
5
10
15
20
0
0.5
1.0
1.5
Time (s)

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.

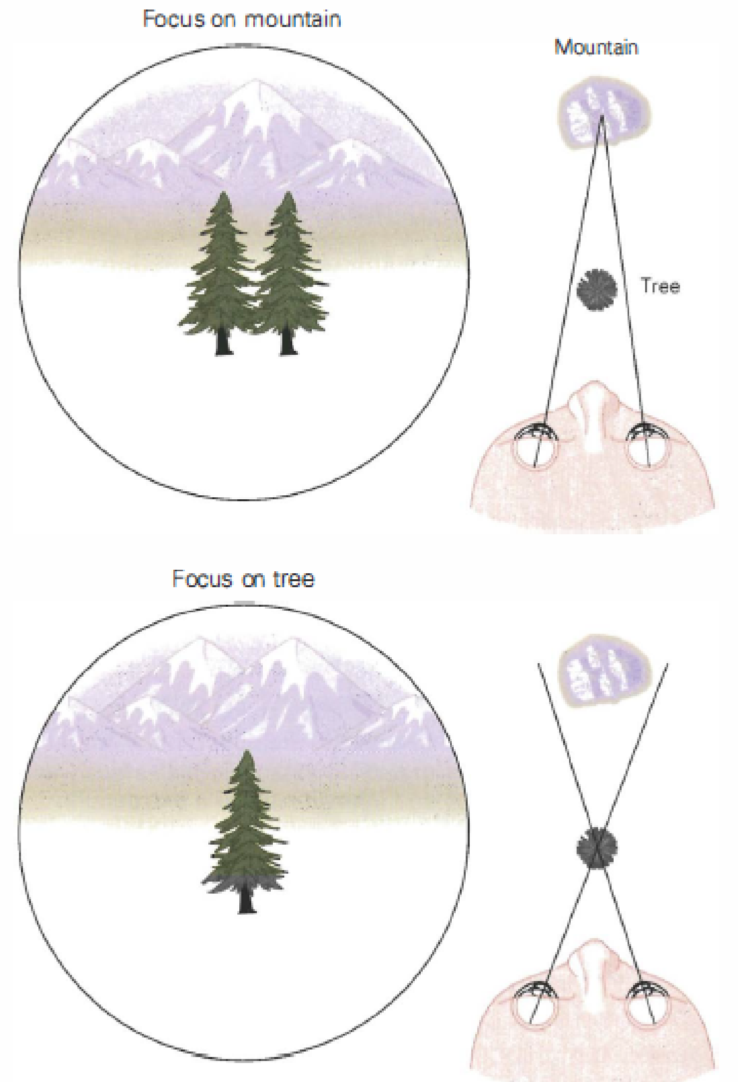
2. Smooth pursuit movements

Neural Control of Smooth Pursuit Movements

Smooth pursuit movements are also mediated by neurons in the PPRF, but are under the influence of motor control centers other than the superior colliculus and frontal eye field. (The superior colliculus and frontal eye field are exclusively involved in the generation of saccades.) The exact route by which visual information reaches the PPRF to generate smooth pursuit movements is not known (a pathway through the cerebellum has been sug-

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***



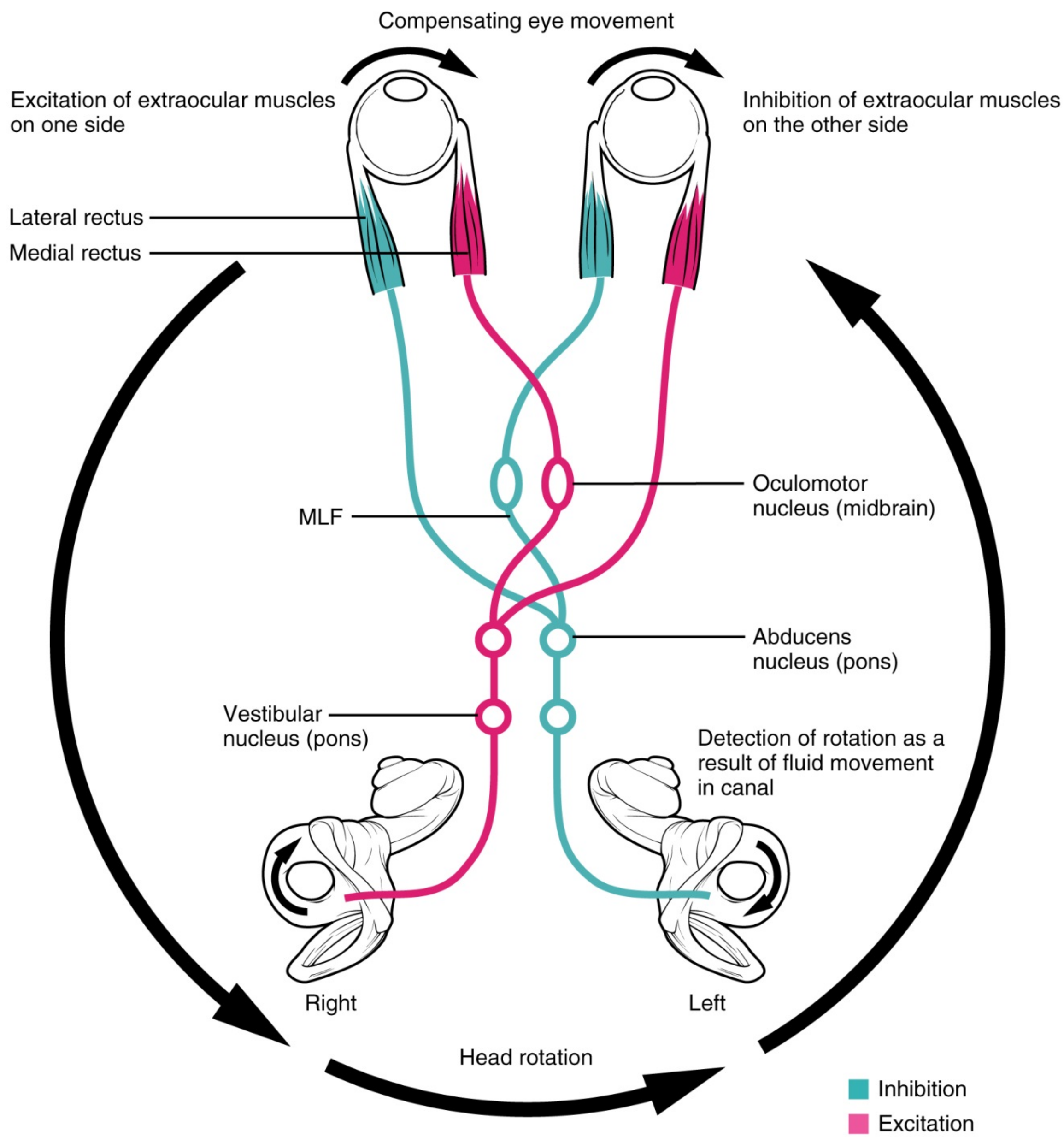
3. Vergence movements

- 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.

- 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

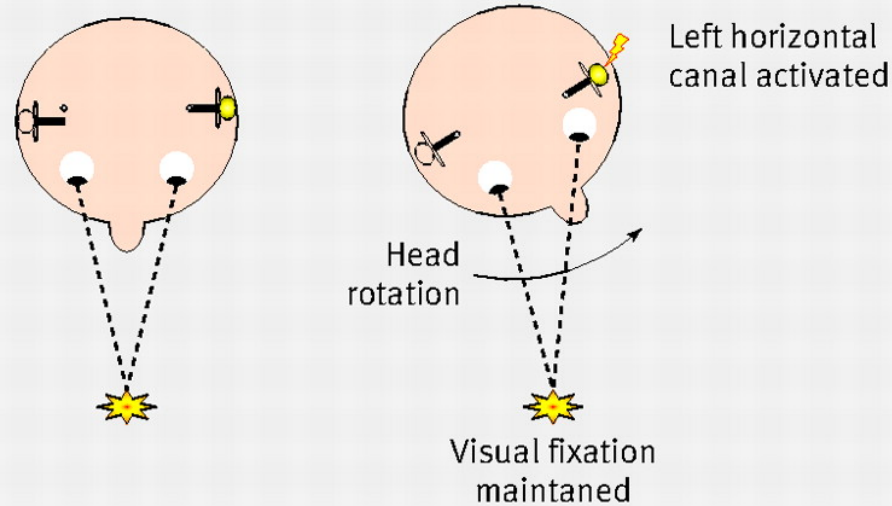
- When an individual's head is rotated to the right, the eyes will turn toward the left.
- Vestibular nuclei receive direct inputs from the semicircular canals of the vestibular apparatus.
- The vestibular nuclei transmit these inputs to both the **pontine g**aze center and nuclei of **CN VI, IV, and I**II.



Compensating eye movement
Excitation of extraocular muscles on one side
Inhibition of extraocular muscles on the other side
Lateral rectus
Medial rectus
Oculomotor nucleus (midbrain)
MLF
Abducens nucleus (pons)
Vestibular nucleus (pons)
Detection of rotation as a result of fluid movement in canal
Right
Left
Head rotation
Inhibition
Excitation

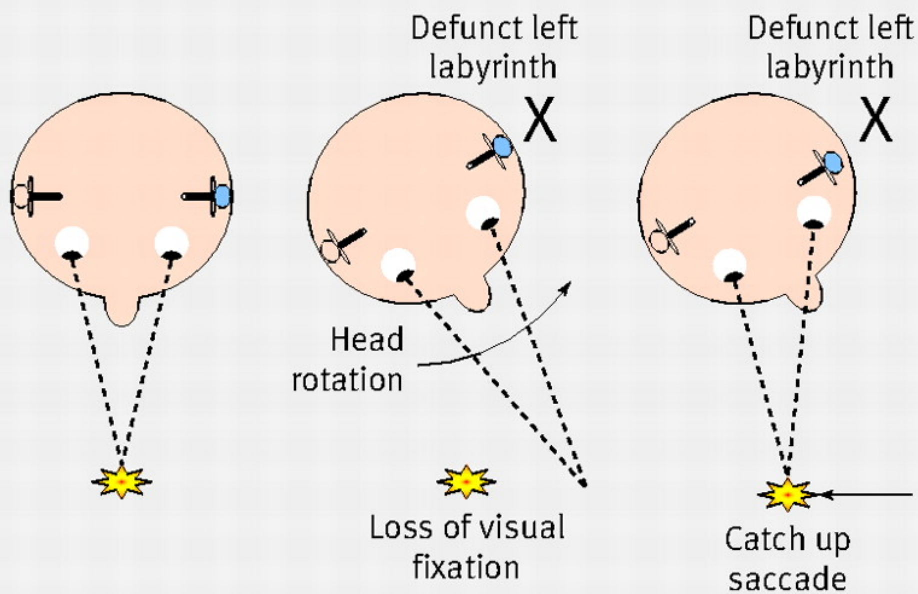
The normal state

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)



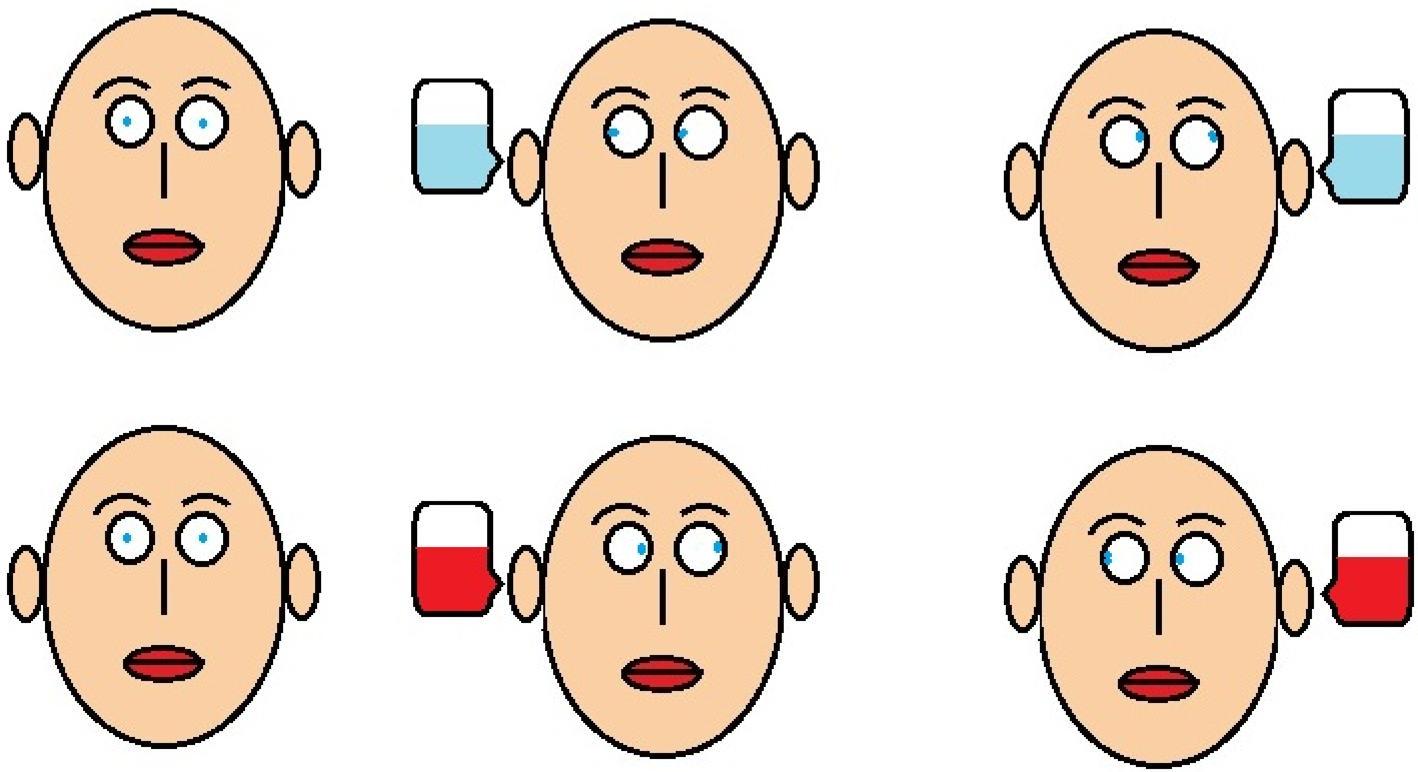
- This is because the inertia of the **endolymph** in the **semicircular canals** generates a force across the cupula, moving it in the **opposite direction** to movement.
- This triggers **action potentials** in the first-order **vestibular ne**urons on the left side that project to the left vestibular 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 <u>lateral rectus muscle</u>
- Also excits, through interneurons, motor neurons of <u>CN III on the left side </u>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**
- https://www.youtube.com/watch?v=YntJiBCz3pA

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

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 optocinetic reflexes enables rotatory nystagmus in light enviroment to continue for as long as the head moves.

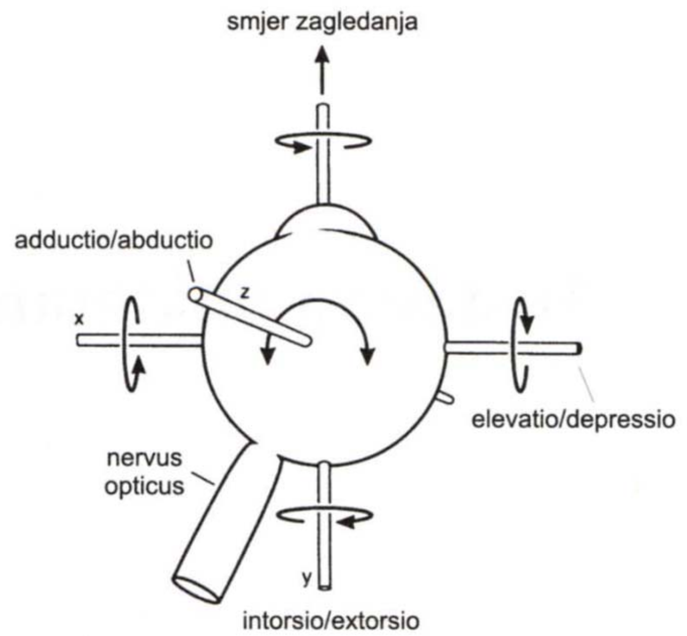
6. Fixation system

- When we look at an object of interest a neural system of fixation actively <u>prevents the eyes from moving</u>
- 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.

THANK YOU!

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
- Z: horizontal
    - ***Adduction (***towards the nose)
    - ***Abduction*** (away the nose)
- X: okomita ravnina
    - ***Elevatio*** (up)
    - ***Depressio*** (down)



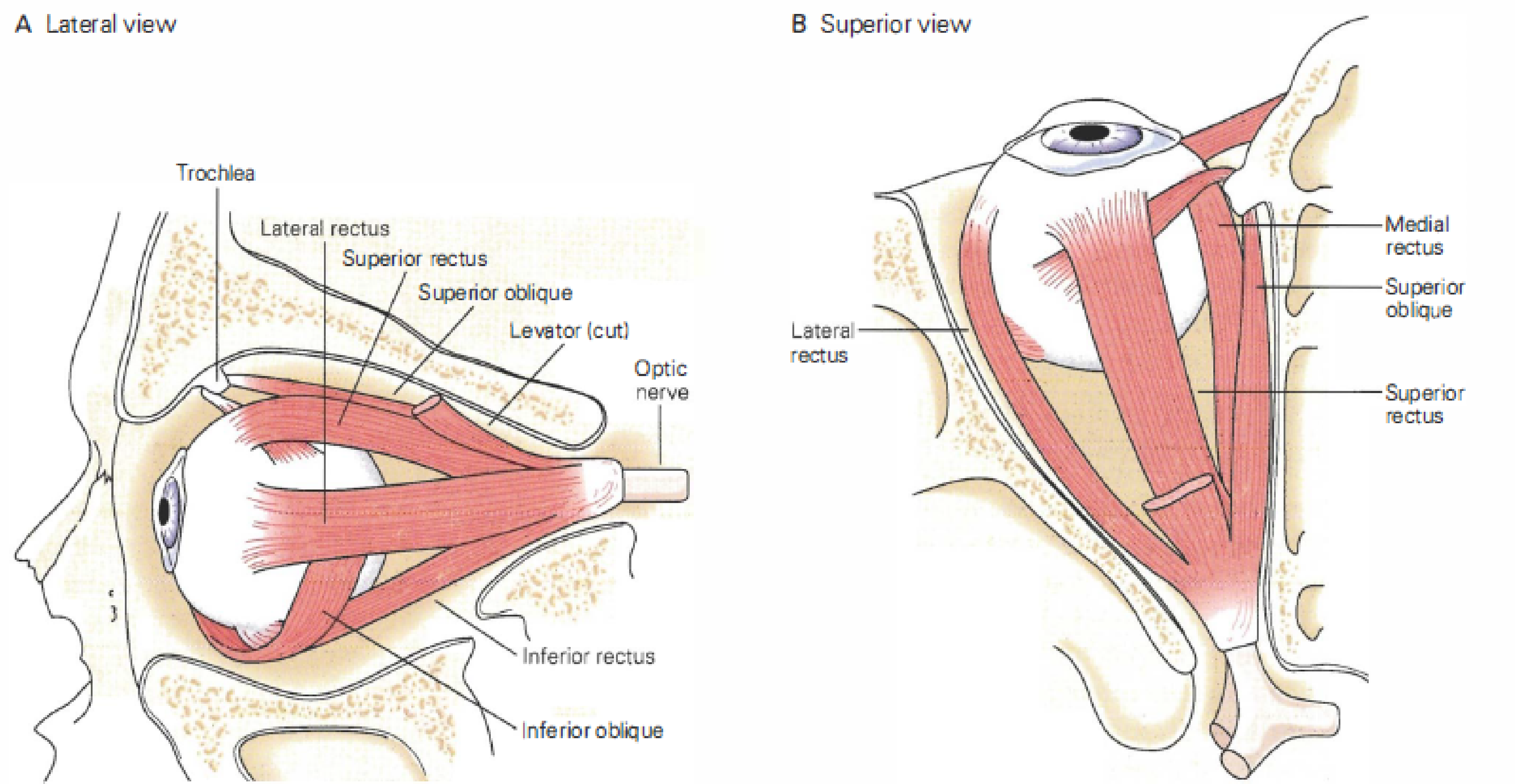


Figure 39-5 The origins and insertions of the extraocular muscles.

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

Table 39-1 Vertical Muscle Action in Adduction and Abduction

Muscle	Adduction	Abduction
Superior rectus	Intorsion	Elevation
Inferior rectus	Extorsion	Depression
Superior oblique	Depression	Intorsion
Inferior oblique	Elevation	Extorsion

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)*

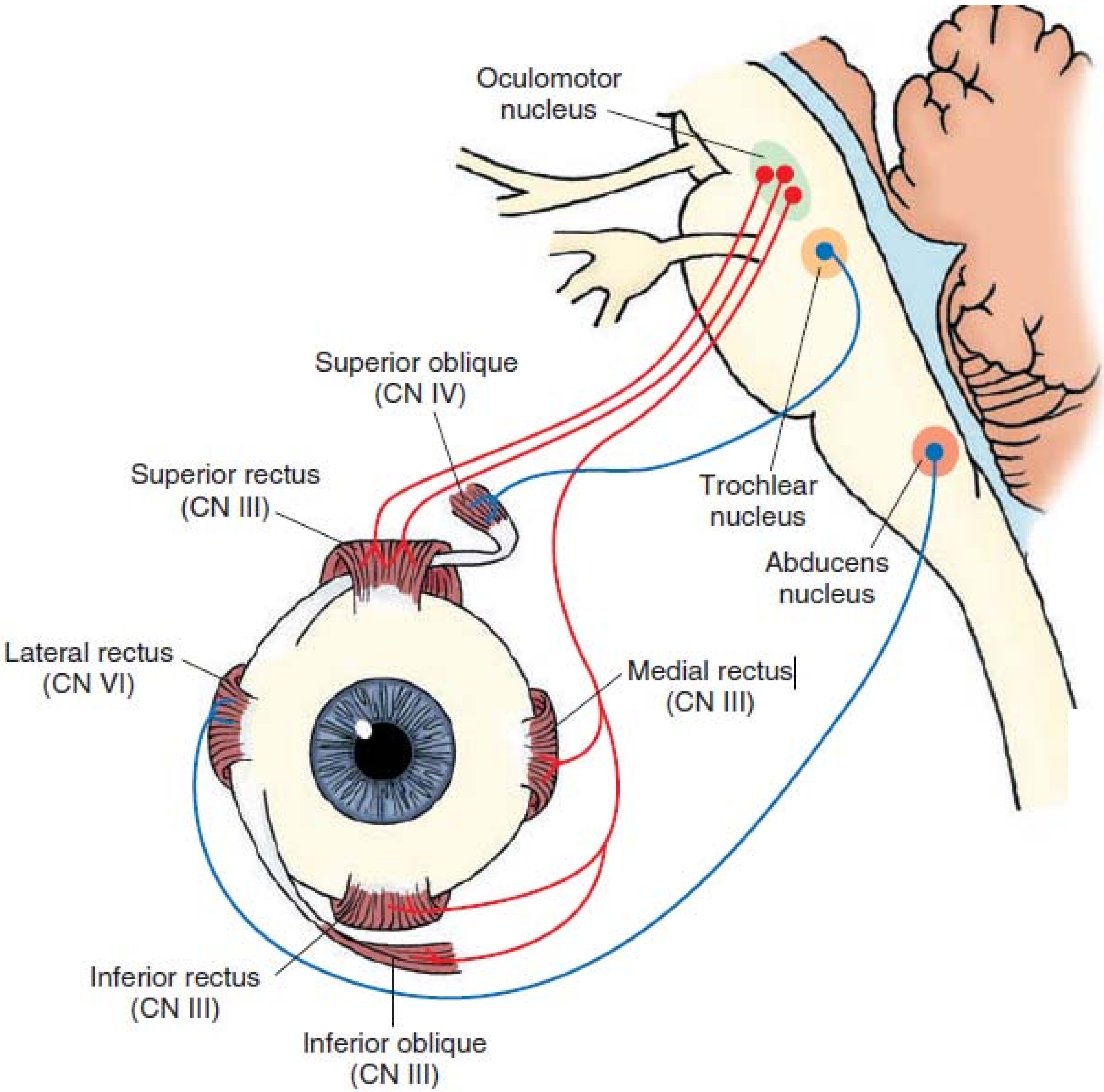


FIGURE 14–10 Origin and distribution of cranial nerves (CN) VI, IV, and III, which innervate extraocular eye muscles. The focus of the upper part of this figure includes the abducens nerve (CN VI) and the general somatic efferent component of the oculomotor nerve (CN III), which are essential for horizontal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

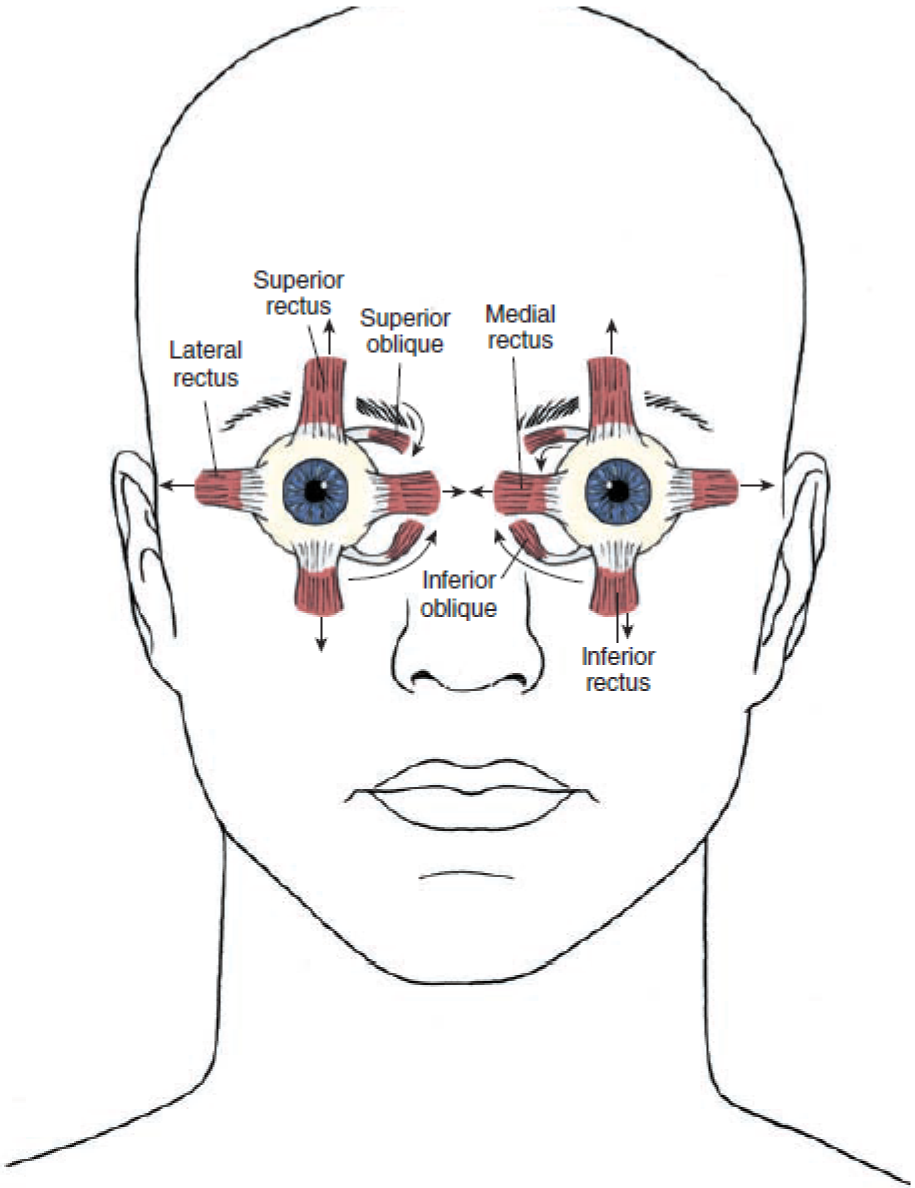


FIGURE 14–12 Diagram illustrating the direction of actions of the extraocular muscles of the eye (indicated by arrows). The lateral rectus muscle is innervated by the abducens nerve, the superior oblique muscle is innervated by the trochlear nerve, and the remaining muscles are innervated by the oculomotor nerve.

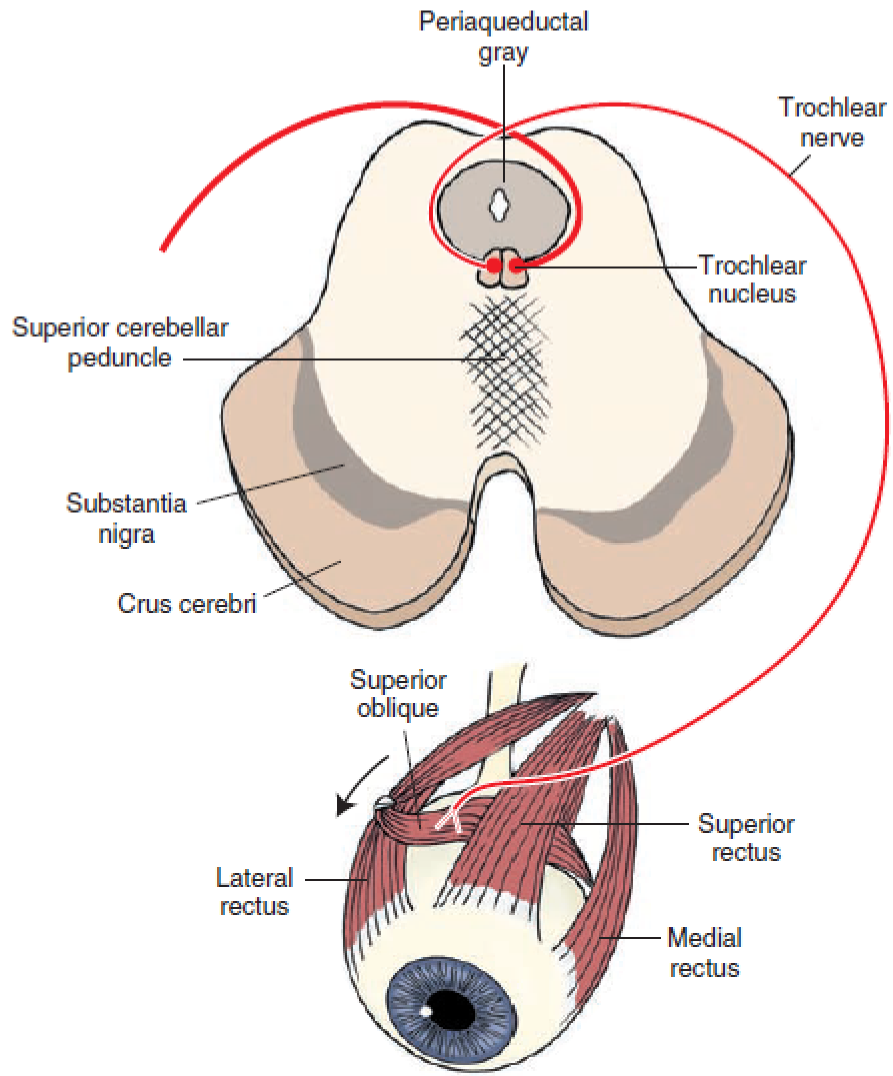


FIGURE 14–11 Origin and distribution of the trochlear nerve (cranial nerve IV) to the superior oblique muscle. As indicated in the cross section of the brainstem, note that this nerve exits the brain from the dorsal aspect, and it is the only nerve that is crossed. Arrow indicates direction of movement of the bulb downward and inward.

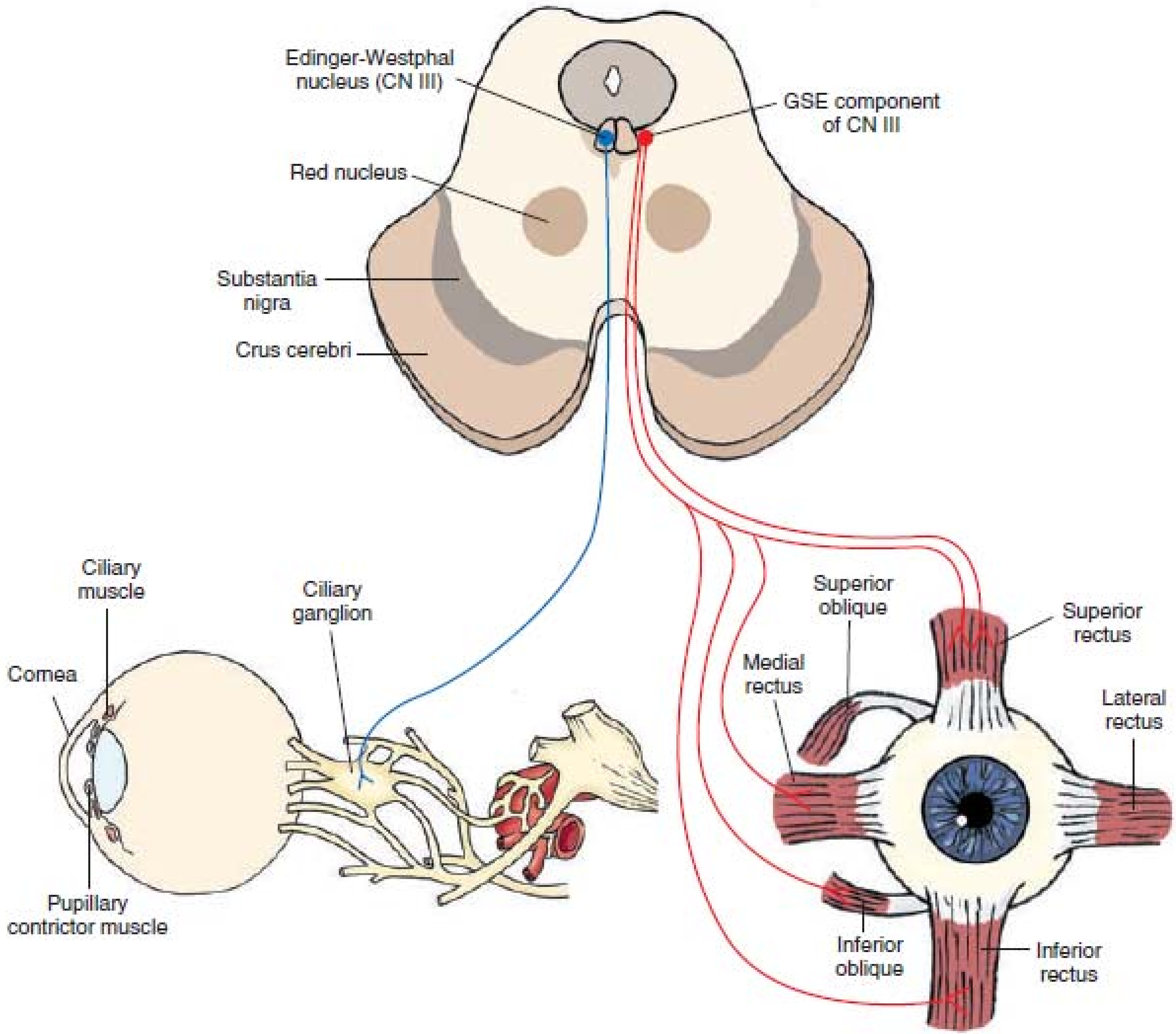


FIGURE 14–13 Origin and distribution of the oculomotor nerve (cranial nerve [CN] III). The anatomical organization of the general somatic efferent (GSE) cell columns of the oculomotor nerve (CN III) complex, whose axons innervate all of the extraocular eye muscles except the lateral rectus and superior 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

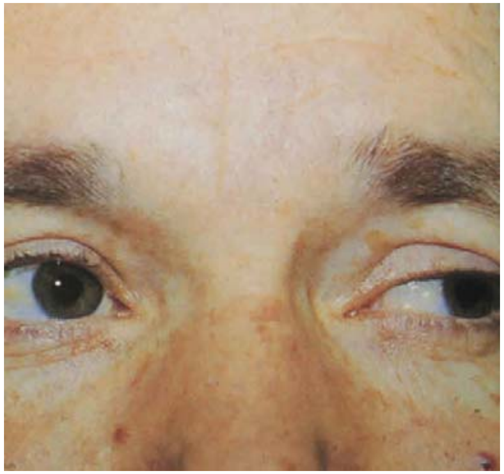
- complain of double vision (diplopia)

- 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 the stairs, they experience double vision and will tend to fall down
- Sign: Tilting of the head upon downward gaze

- 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.



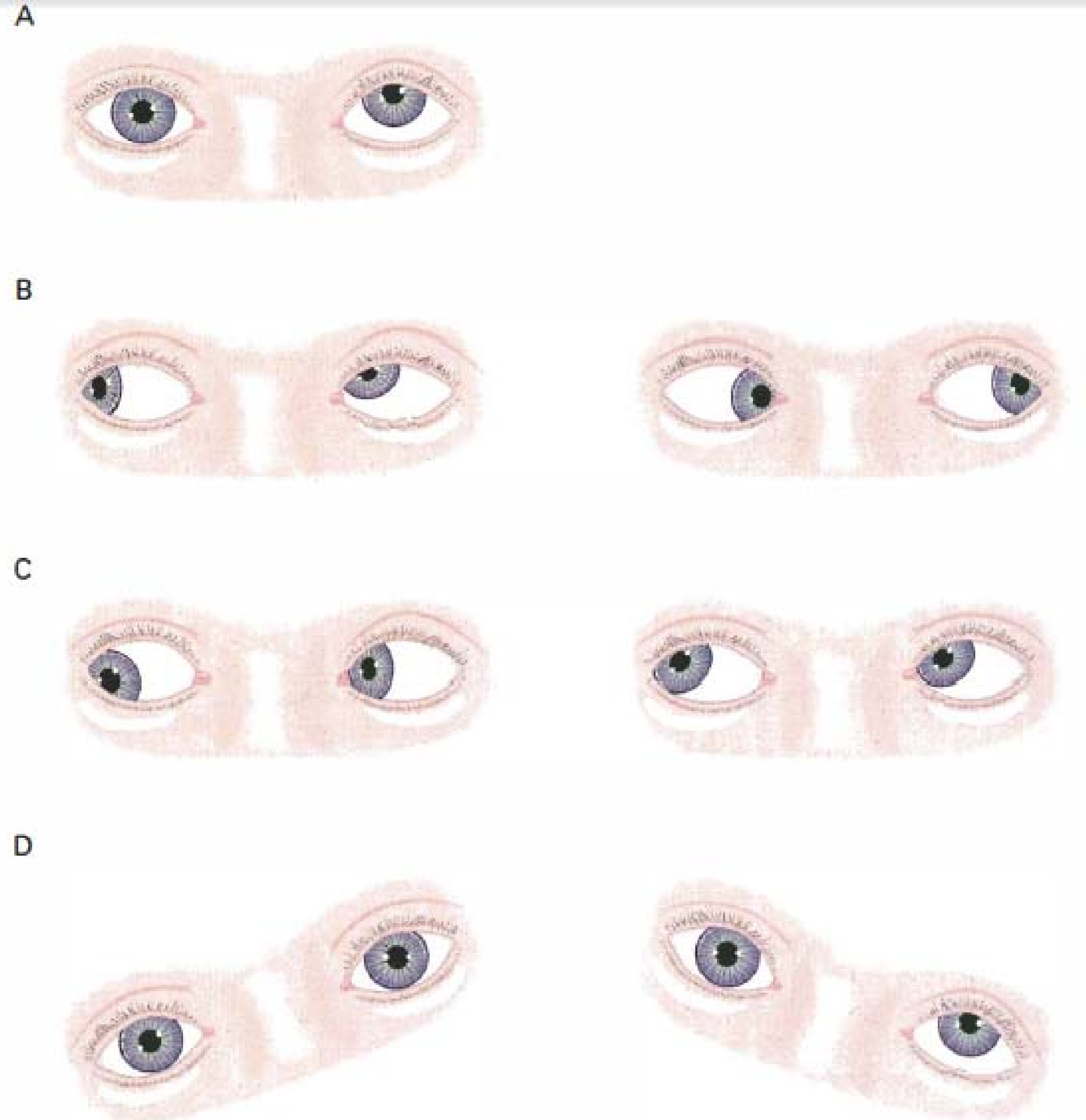
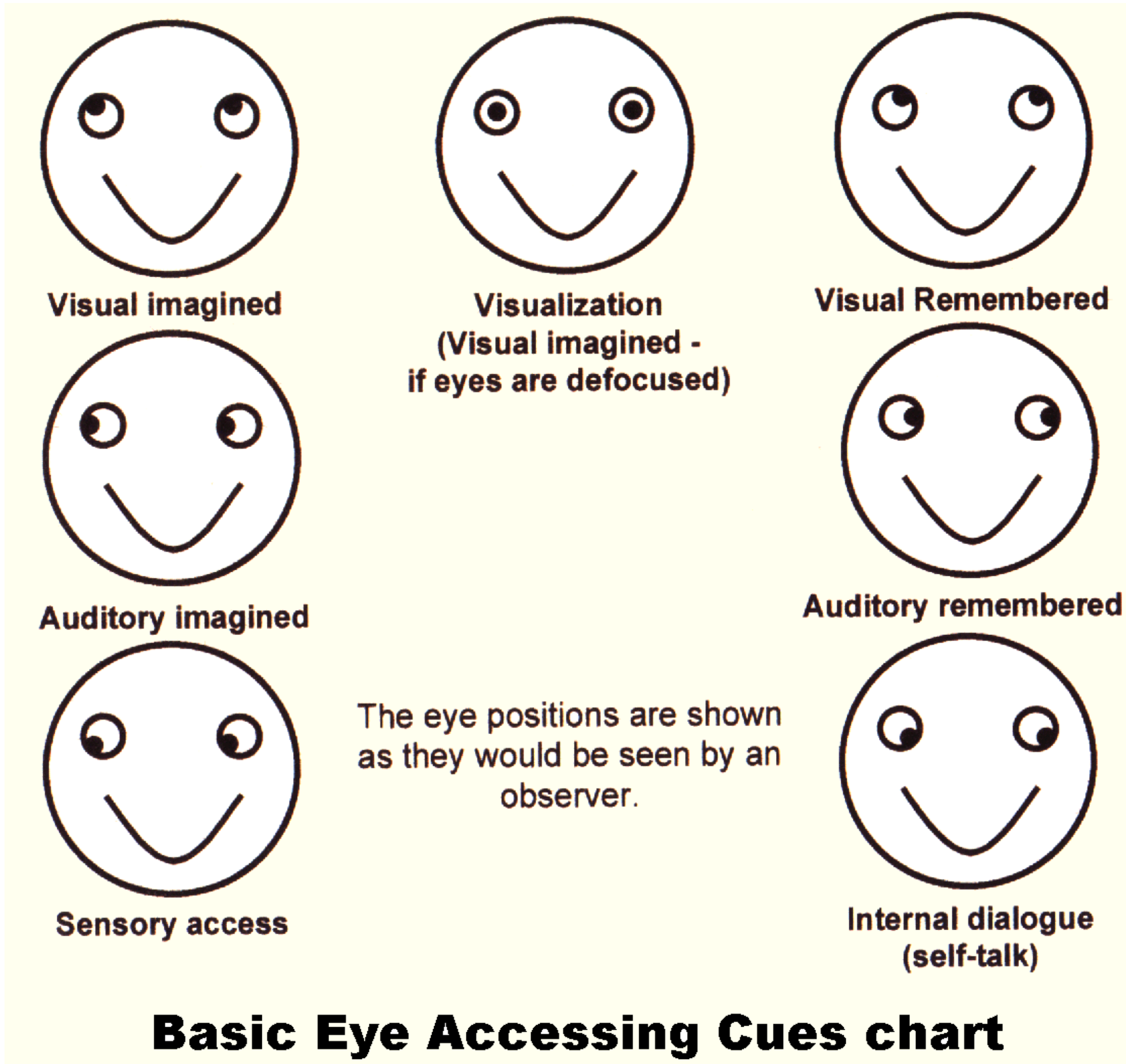
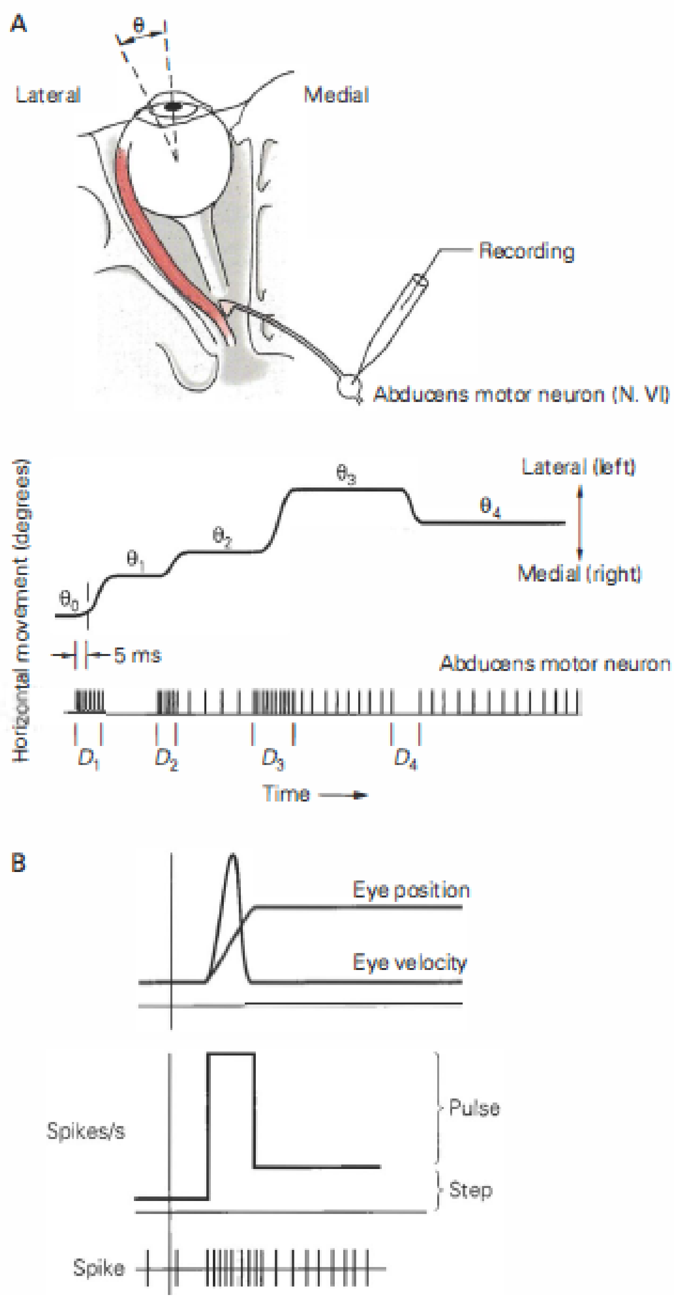
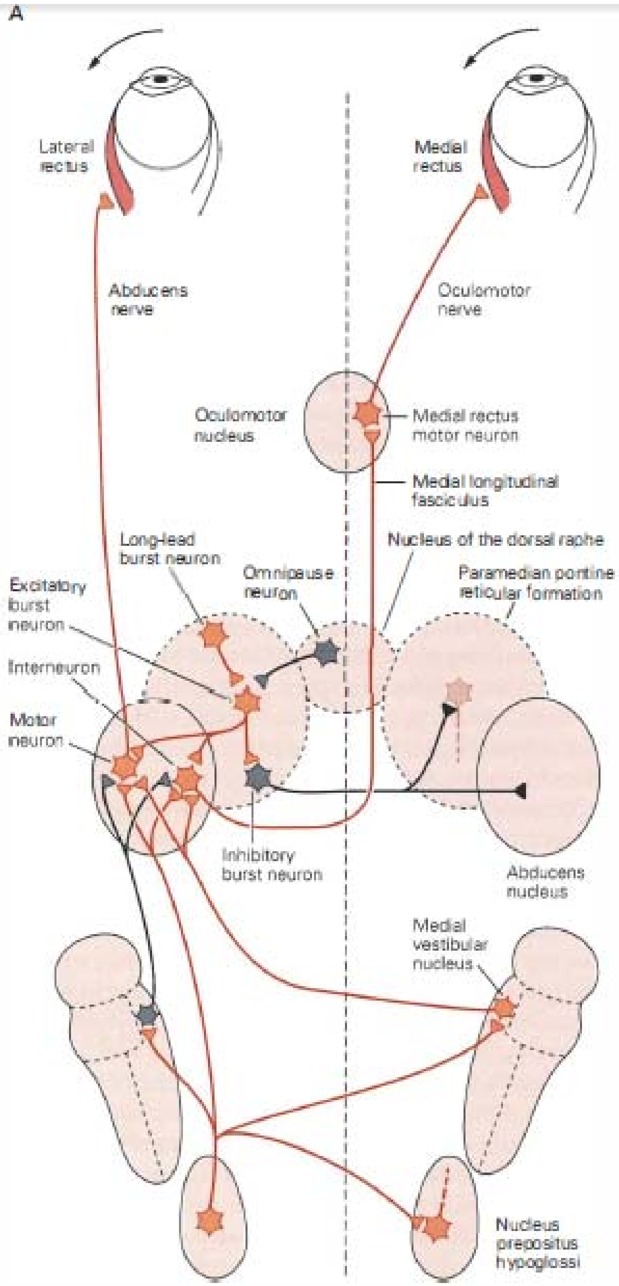


Figure 39-8 A patient with a deficit of the left superior oblique muscle. When the patient looks straight ahead, the left eye is mildly elevated relative to the right (**A**). This elevation occurs because there is no superior oblique tension to counteract the left superior oblique. When the patient 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.)


Visual imagined
Visualization
(Visual imagined -
if eyes are defocused)
Visual Remembered
Auditory imagined
Auditory remembered
The eye positions are shown
as they would be seen by an
observer.
Sensory access
Internal dialogue
(self-talk)
Basic Eye Accessing Cues chart

Thank you!


A
θ
Lateral
Medial
Recording
Abducens motor neuron (N. VI)
Horizontal movement (degrees)
θ0
θ1
θ2
θ3
θ4
Lateral (left)
Medial (right)
5 ms
Abducens motor neuron
D1
D2
D3
D4
Time
B
Eye position
Eye velocity
Spikes/s
Pulse
Step
Spike



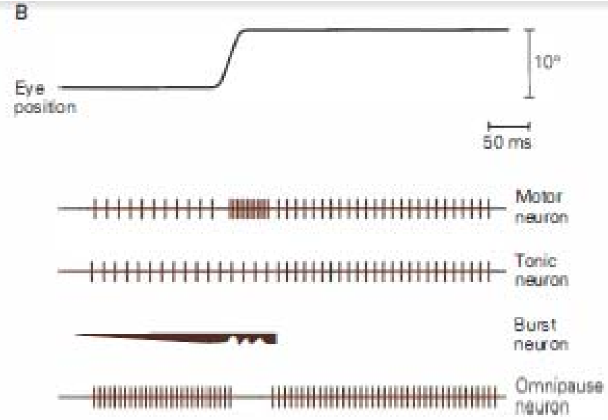


Figure 39-10 The motor circuit for horizontal saccades in the brain stem. Excitatory neurons are **orange** and inhibitory neurons are **gray**. The **dotted line** represents the midline of the brain stem.

A. Long-lead 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 unwanted 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.