Chapter 10

The Motor System

Outline

10.1 Motor Control in the Brain

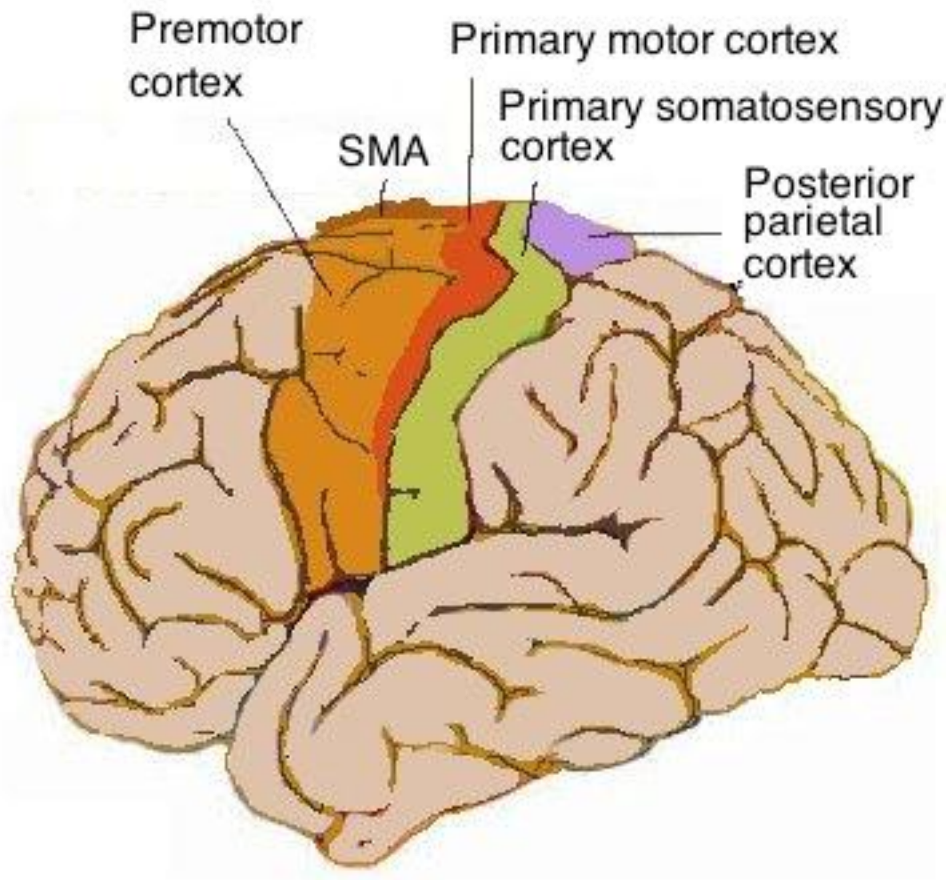
10.2 Modifiers of Descending Information

10.3 The Spinal Cord

10.4 The Muscles

Voluntary movement

- All begins in the brain
- Frontal lobe
  - Primary motor cortex (M1)
  - Premotor cortex
  - Supplemental motor areas (SMA)
  - Prefrontal cortex
- Parietal lobe
  - Posterior parietal cortex



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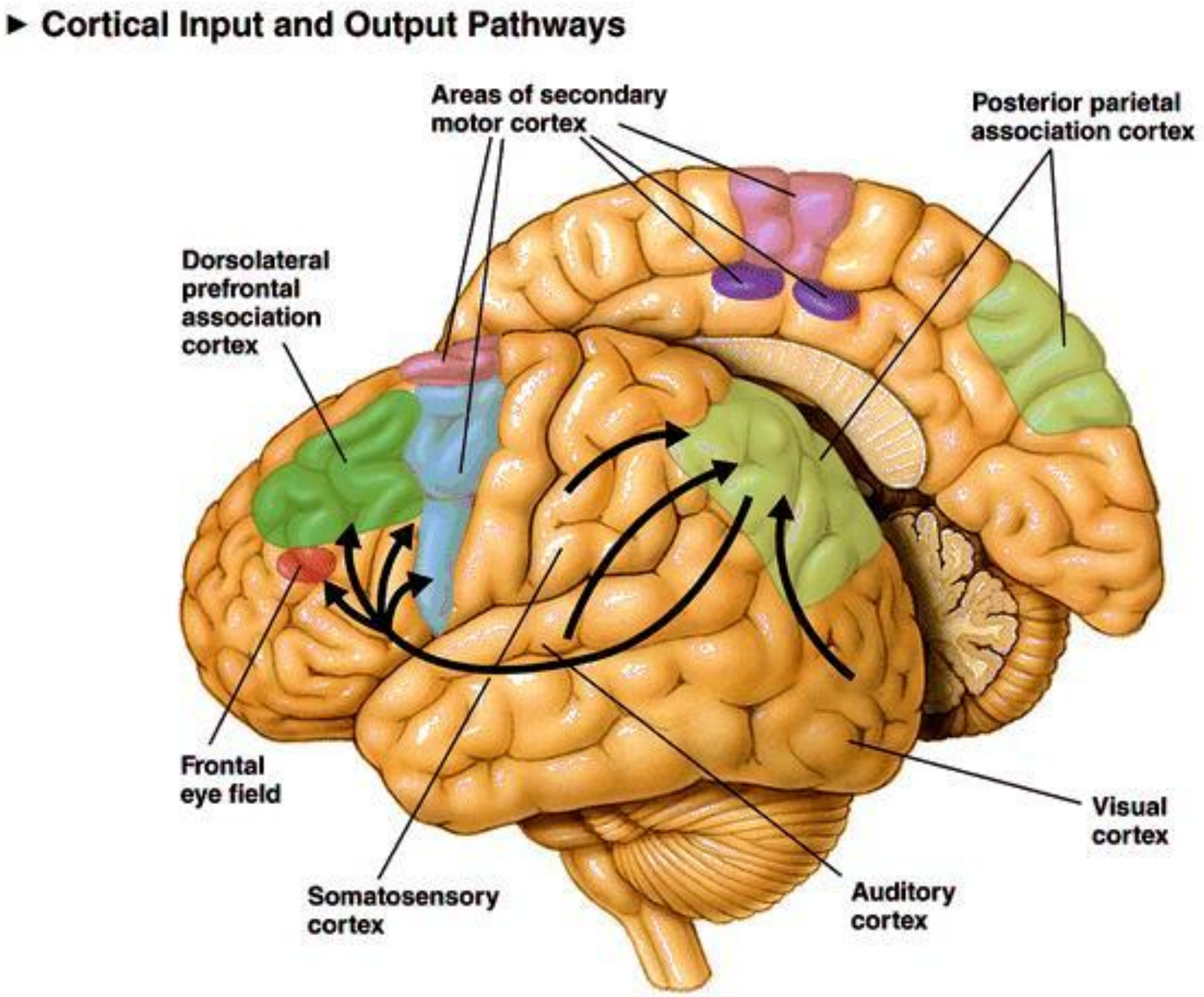

Pink = SMA

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Voluntary movement begins with high order thought processes

- Association cortices
  - Prefrontal cortex
  - Posterior parietal cortex
- Not correlated with specific muscle groups
- Activation does not necessarily cause muscle activity
- Important for initiation of motor control (ie deciding to move)

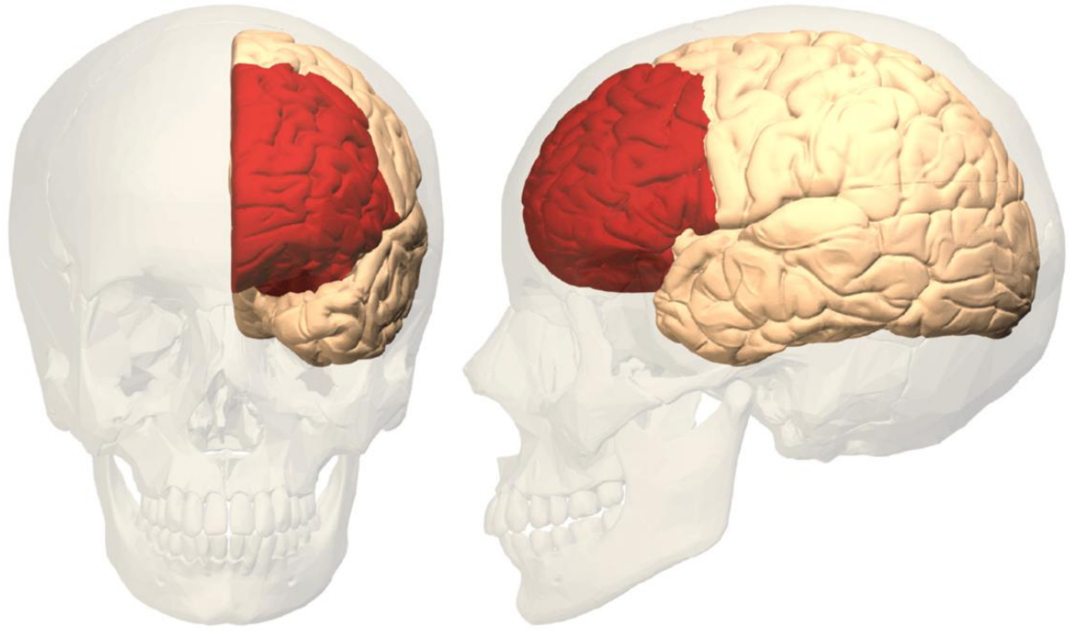


Figure 10.1 Anterior view (left) and lateral view (right) of one hemisphere showing the location of the prefrontal cortex (red).

A lot goes on in the prefrontal cortex

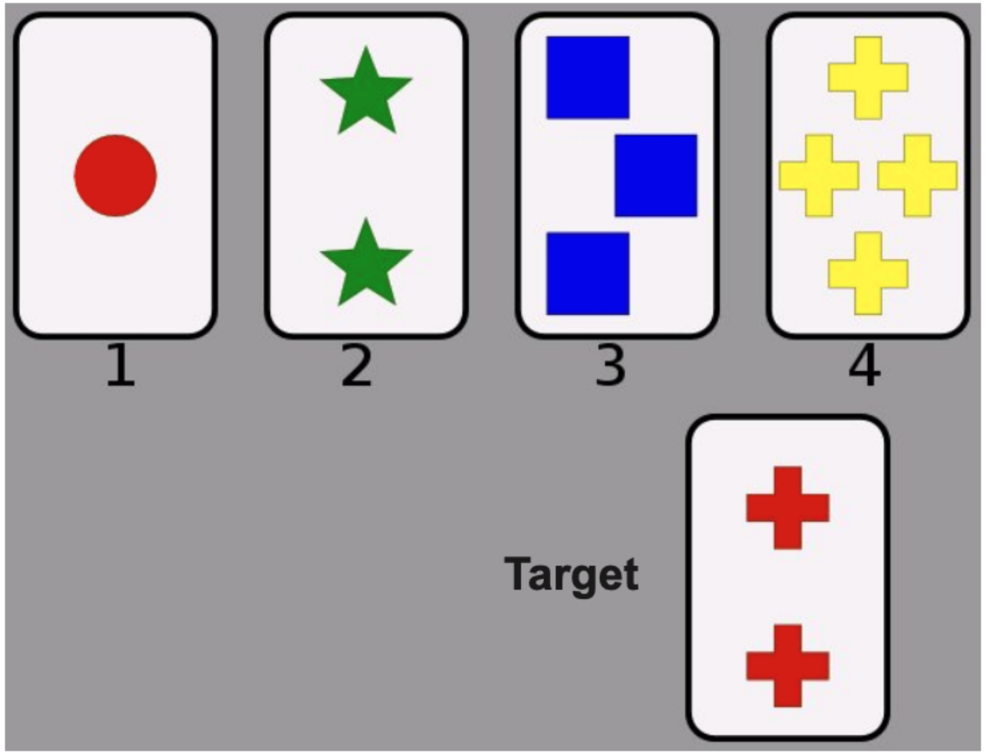


Figure 10.2 In the Wisconsin Card Sorting Task, the target card (bottom) could be placed in category 1 (matching color), category 2 (matching number of objects), or category 4 (matching shape). Ability to switch to a new set of rules uses prefrontal cortex.

Posterior parietal cortex

- Integrating somatosensory and visual information and determining an appropriate motor action (recall dorsal stream pathway from Chap 7)
- https://pubmed.ncbi.nlm.nih.gov/31431892/

Motor cortex

- Primary motor cortex (M1), premotor area, and supplemental motor area are collectively called the **motor cortex**
- Information travels from associative cortices through the premotor area, then M1, before projecting down through brain stem and spinal cord

Premotor area

- Modulates motor output
- Activated prior to motor activity
- Activated during imagined movements

Mirror neurons

- Hypothesized population of cells in premotor cortex
- Activate during a movement OR when the same movement is observed in another individual
- Could help with learning motor behaviours and/or understanding movements in others
- Unlikely to be encoded at the level of individual neurons (too complex)

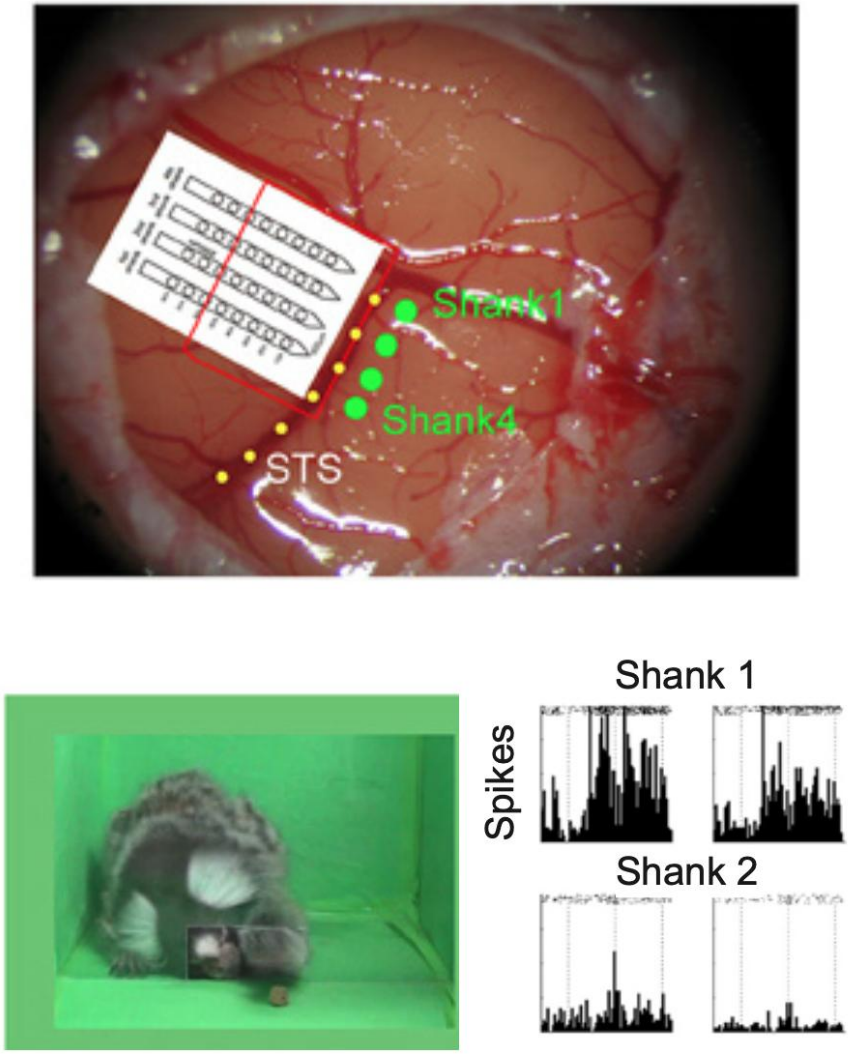


Figure 10.4 Surgically implanted electrodes (top) in a marmoset brain. While viewing a fellow marmoset reaching (bottom left), mirror neuron activity increases (bottom right).

Supplemental motor area

- Sends projects to M1 and spinal cord
- Communicates bilaterally
- Lesioning SMA causes deficits in manual coordination tasks that require both hemispheres to communicate

Primary motor cortex (M1)

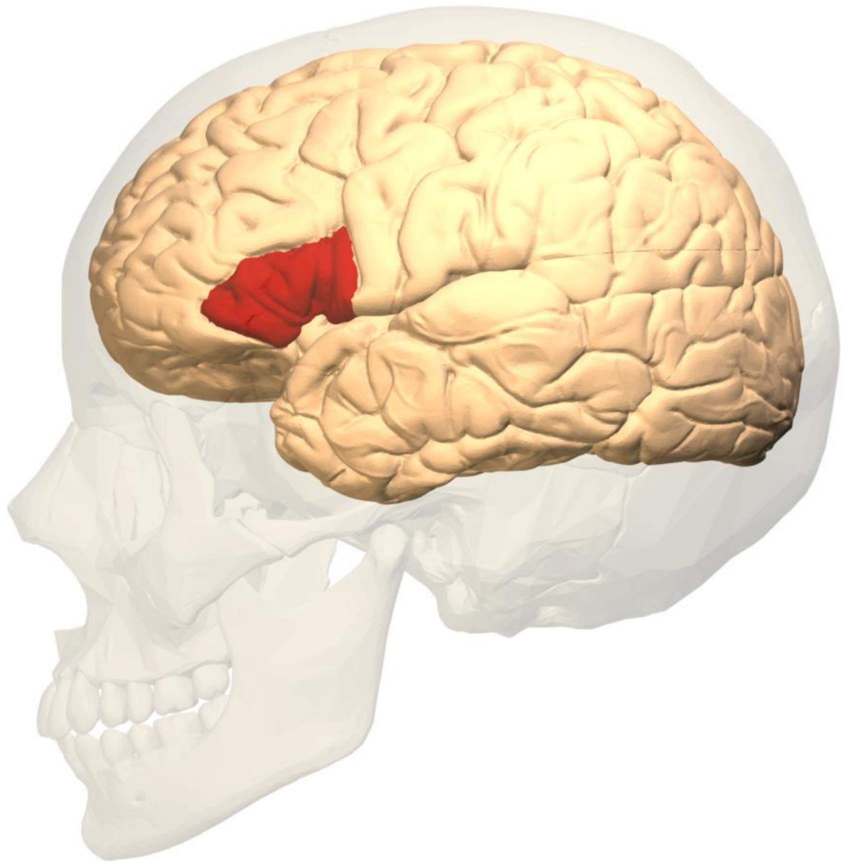
- Motor cortex cells (sometimes called **upper** motor neurons) influence **motor neurons** (sometimes called **lower** motor neurons), neurons that communicate down into the spinal cord and ultimately influence muscles or glands
  - Lower motor neurons are found at the brain stem or spinal cord and fire whenever the upper motor neuron sends a signal

Mapping M1

- Dr. Wilder Penfield
- Stimulation of M1 caused contralateral motor activity
- Different populations of M1 neurons communicate with specific muscle groups
- Dorsal M1 activates hip and trunk muscles
- Lateral M1 activates face muscles
- Similar to organization of what brain region?

Other brain areas important for movement

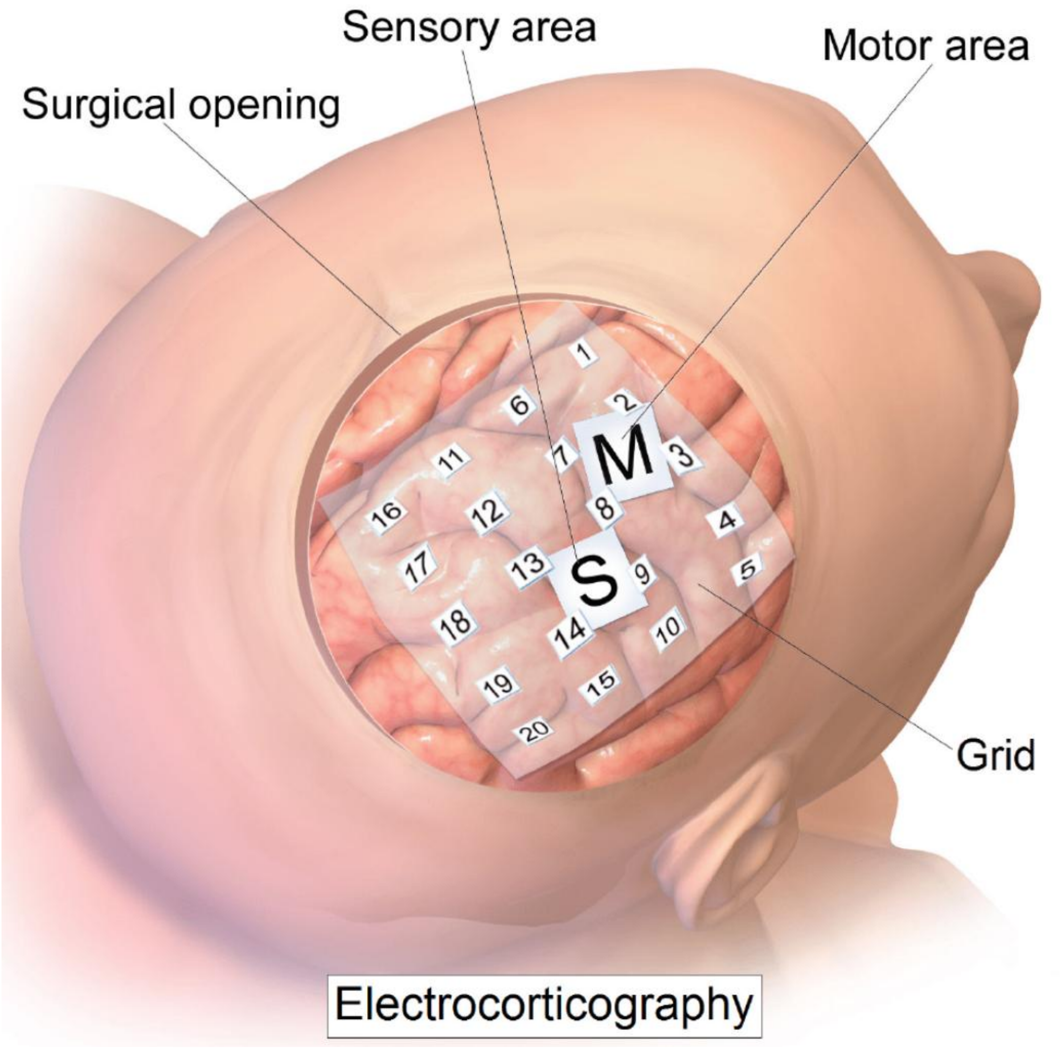
- Frontal eye field (FEF) communicates with the extraocular muscles and mediates saccade eye movement (recall from Chap 7)
- Inferior frontal gyrus (aka Broca's area) contributes to motor processes related to language (ie speech)



By Polygon data were generated by Database Center for Life Science(DBCLS)[2]. - Polygon data are from BodyParts3D[1], CC BY-SA 2.1 jp, https://commons.wikimedia.org/w/index.php?curid=32508617

Clinical connection: Prosthetic limbs (etc.)

- Brain-machine interface
- Activation of specific neurons could be used to help amputees control prosthetic limbs
- Electrocorticogram (eCoG) involve placement of high-density electrode array on the surface of M1
- Detects electrical activity at high spatial resolution (hundreds of microns) and high temporal resolution (hundreds of microseconds)
- ECoG has allowed some people with tetraplegia to control external devices
- https://www.youtube.com/watch?v=vL7yMn6kiMg

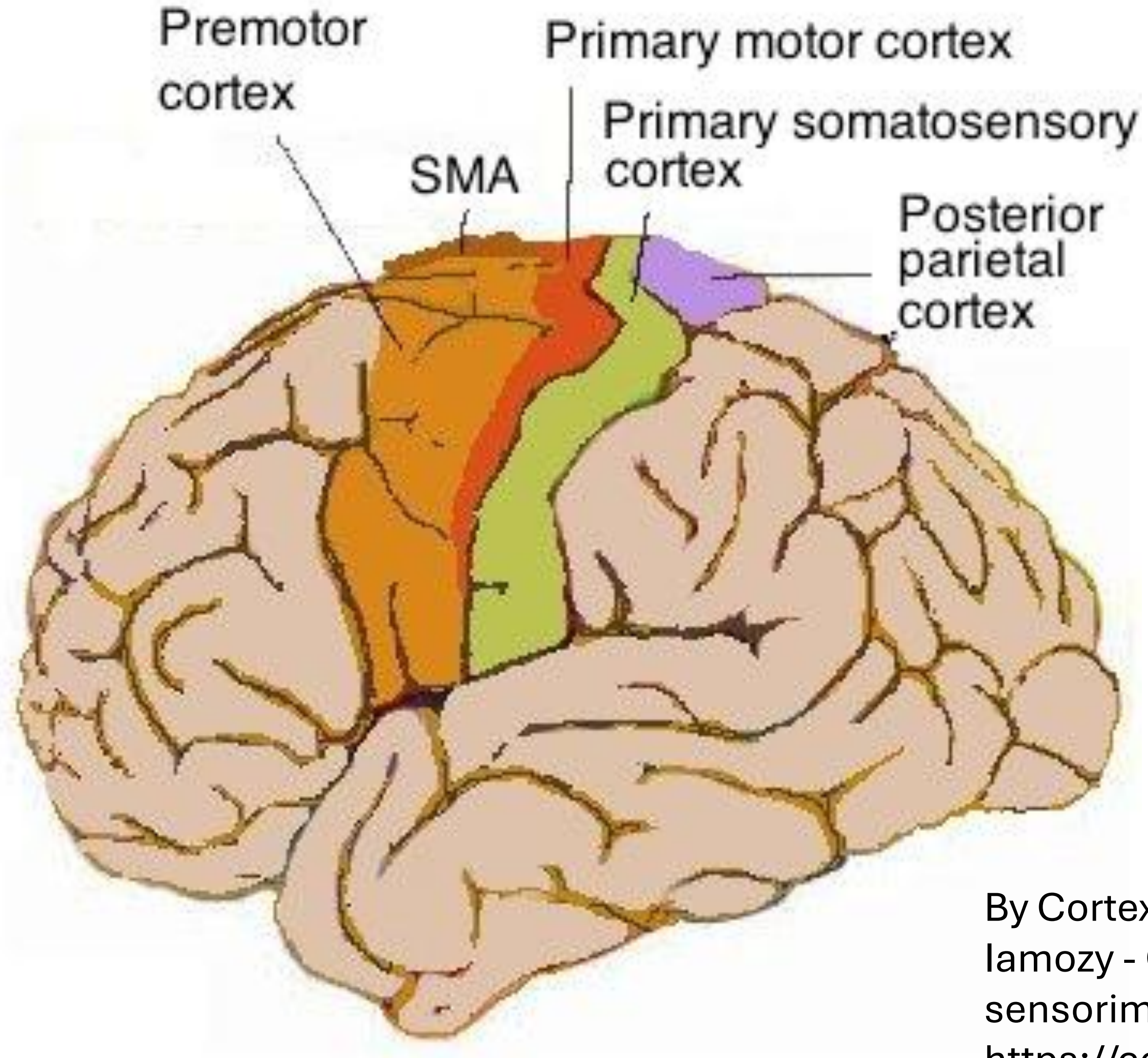


Sensory feedback

- Somatosensation: process of sensing and perceiving the body (Chap 8)
- One aspect is proprioception: perception of our body in the world
- Receptors in our muscles, tendons, joints, and skin detect contraction, stretch, and vibration
- Sensory neurons carry this information to neurons in the spinal cord, which sends information to both motor neurons and up to the brain (ie S1)
- Bidirectional connections are made onto neurons in the adjacent motor cortex
- Correct movements that go awry

Connection between S1 and M1

- Sometimes S1 and M1 are collectively referred to as the sensorimotor cortex



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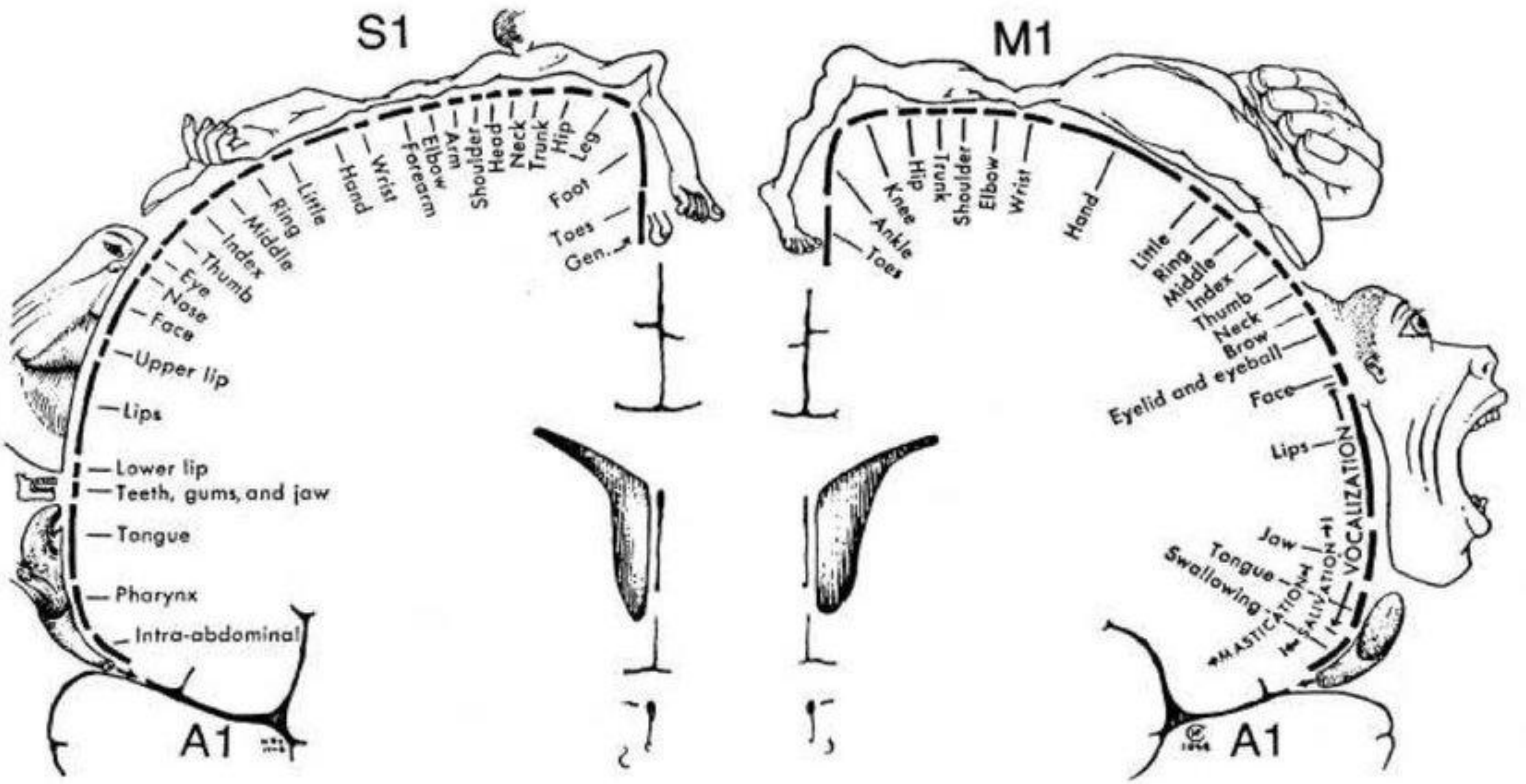
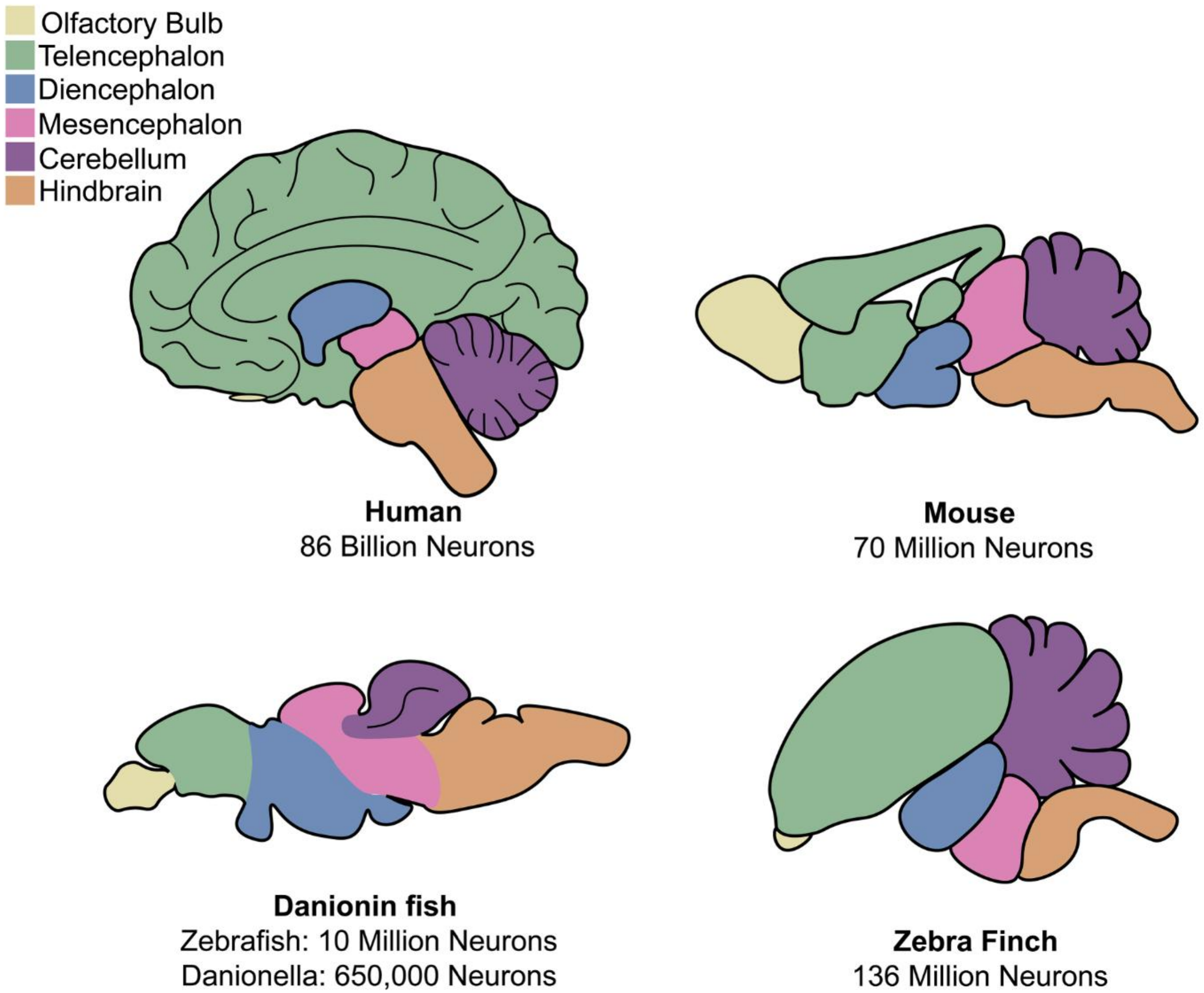


Image by Penfield

Modifiers of descending information

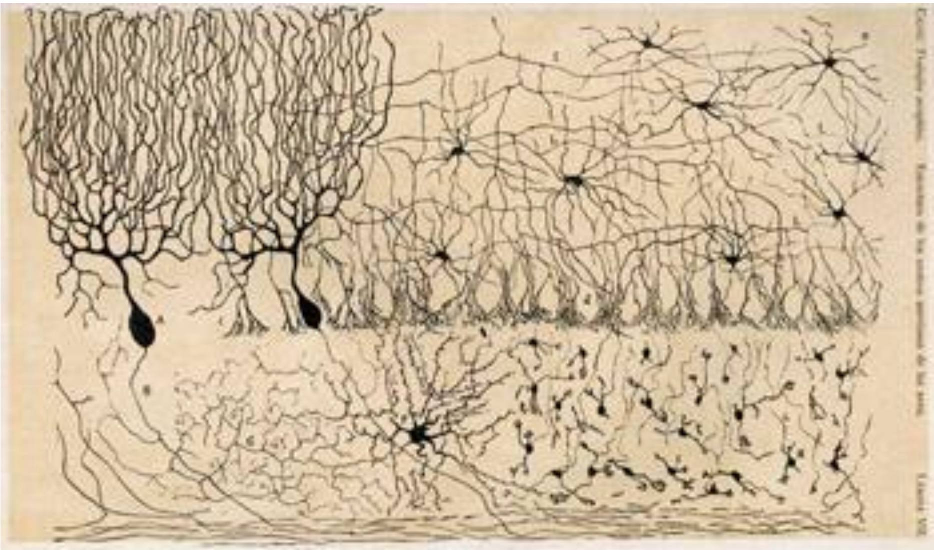
- Signals from M1 are “fine-tuned” by two major structures
  - Cerebellum
  - Basal ganglia



Haynes et al., 2022; CC-BY 4.0

The cerebellum

- Evolutionarily ancient
- In humans: 10% of brain mass, 80% of neurons



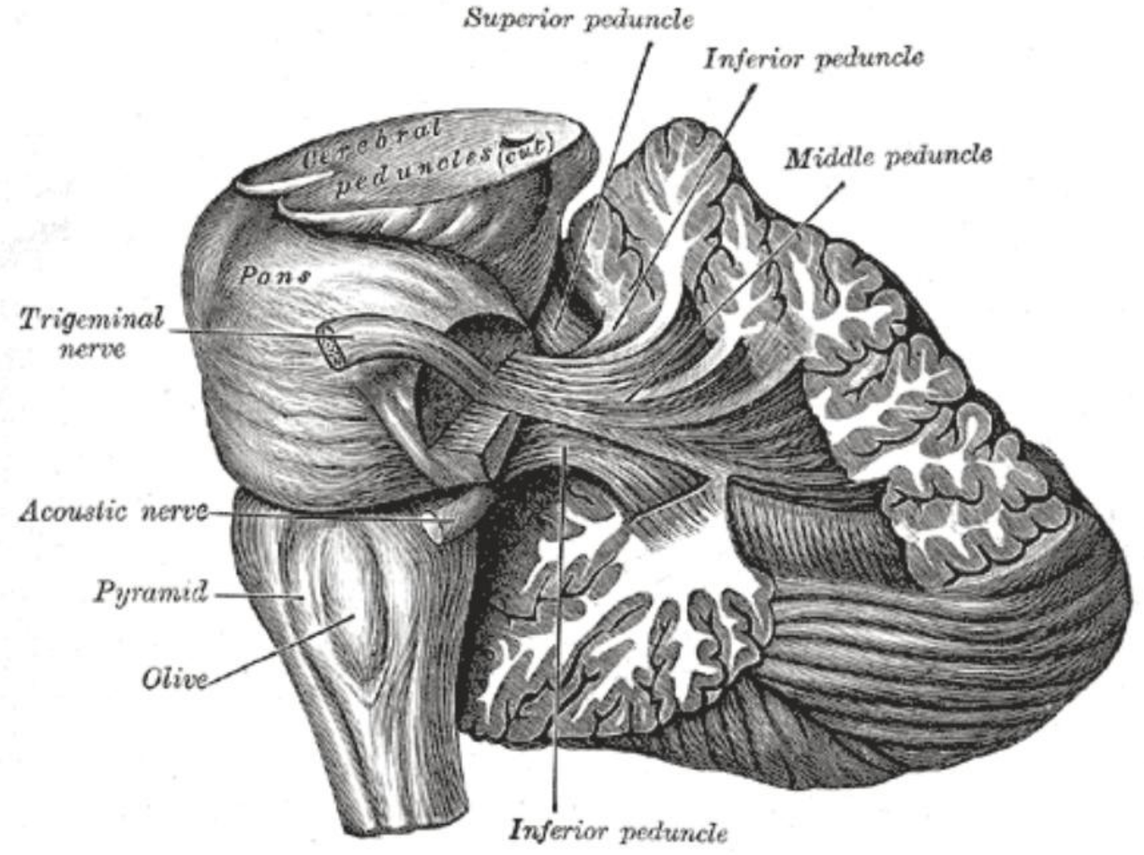
Drawing of the cells of the chick cerebellum by Santiago Ramón y Cajal, 1905

Role of the cerebellum

- Coordinated movement
- Integrating sensory information (to produce coordinated movement)
- Refining motor-related outputs to learn motor tasks (motor learning)
- Processing cognitive and executive functions
- Autonomic functions
- Associative learning
- Cerebellar cognitive affective syndrome: Characterized by dysfunction in executive tasks, impaired visual-spatial memory, changes to personality and emotional control, and problems with language production

Structure and function of the cerebellum

- External layer of gray matter (cerebellar cortex)
- Internal core of white matter
- 3 pairs of deep nuclei (fastigial nucleus, interposed nucleus, dentate nucleus)
- Connected to dorsal brain stem by 3 pairs of peduncles (stalks of tissue): inferior cerebellar peduncle, middle cerebellar peduncle, superior cerebellar peduncle



Lobes and fissures

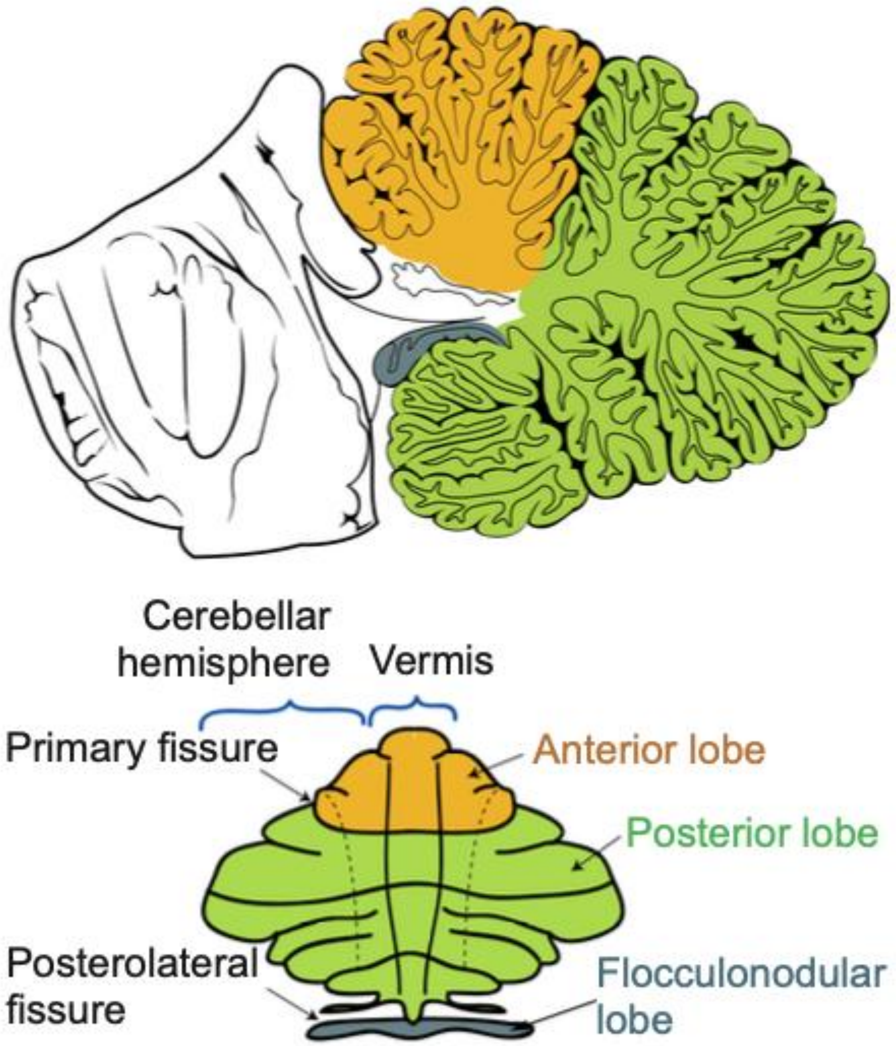
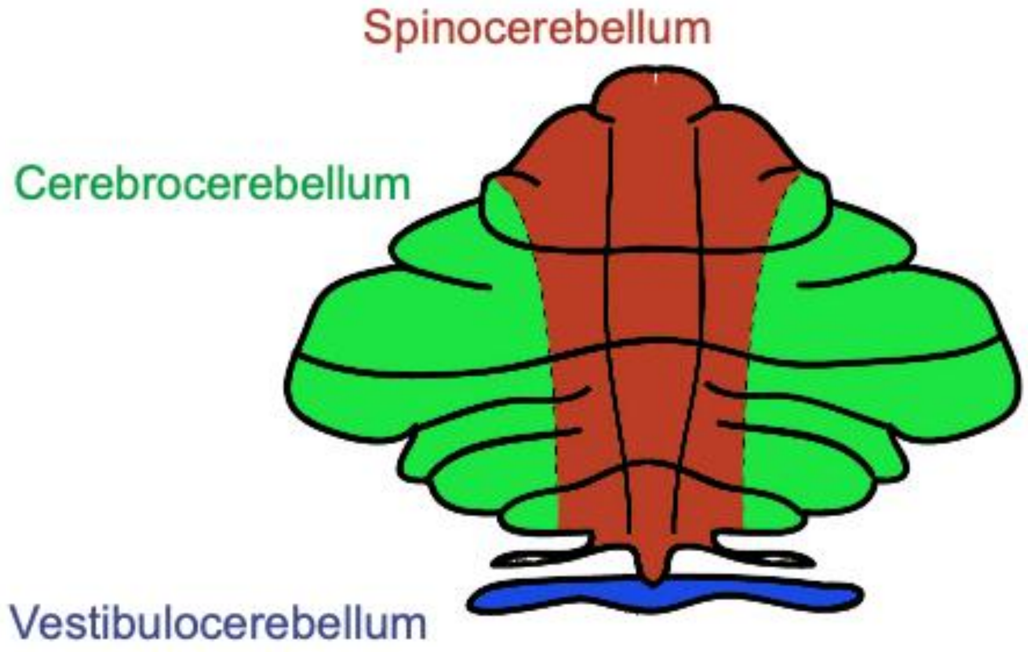


Figure 10.11 A midsagittal view of the cerebellum (top) and an "unrolled" view (bottom) labeling the major anatomical structures.

Functional regions

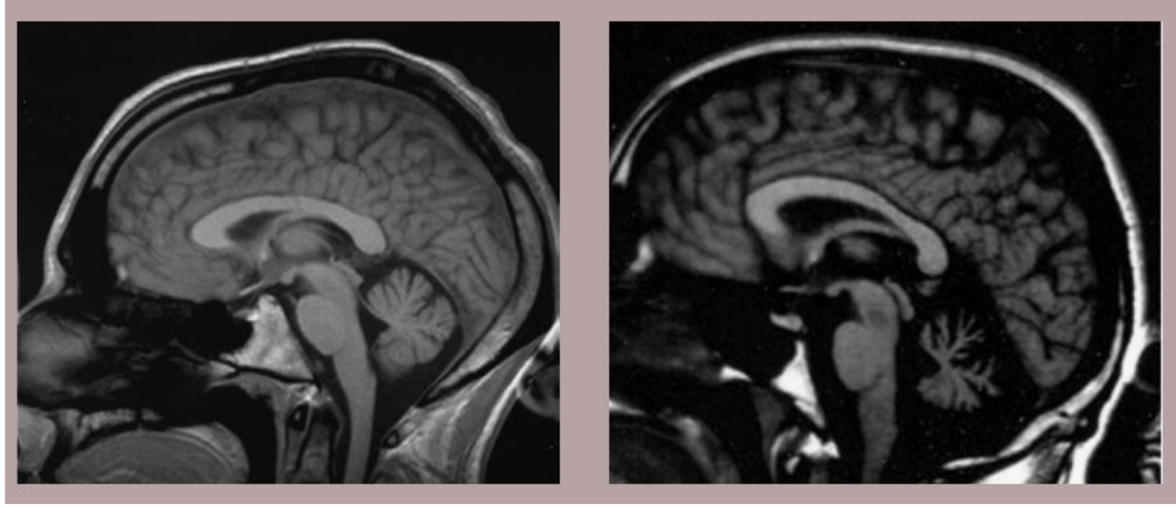


- Vestibulocerebellum
  - Location: Flocculonodular lobe
  - Integrates visual and vestibular inputs
  - Most evolutionarily conserved
    - Medial – head and trunk (posture), limb extensors (balance)
    - Lateral – eye movements
- Spinocerebellum
  - Location: Vermis and intermediate regions of the hemispheres
  - Locomotion and extremity movement
- Cerebrocerebellum
  - Location: Lateral regions of the posterior lobe
  - Most evolutionarily recent
  - Outputs travel to motor and premotor cortex (+ nonmotor associative cortices)
  - Planning and executing movement (+ cognitive functions)

Somatotopic organization of cerebellum

- See **A century of cerebellar somatotopy: a debated representation**

Ataxia



- Poor coordination of voluntary movements
- Atypical eye movements
- Poor balance
- Changes in gait
- Dysarthria (difficulty articulating speech)
- Often associated with cerebellar atrophy
- Many causes (stroke, tumor, toxin, radiation, hereditary disease, diet-induced autoimmune mediated cell death [gluten])

How might we improve this figure legend?

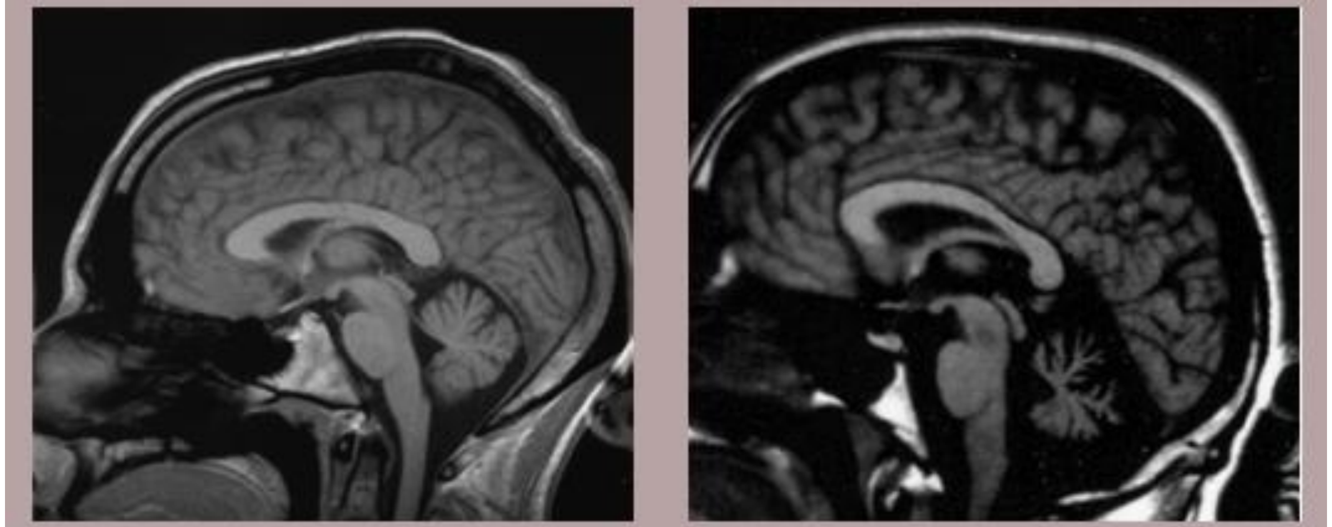
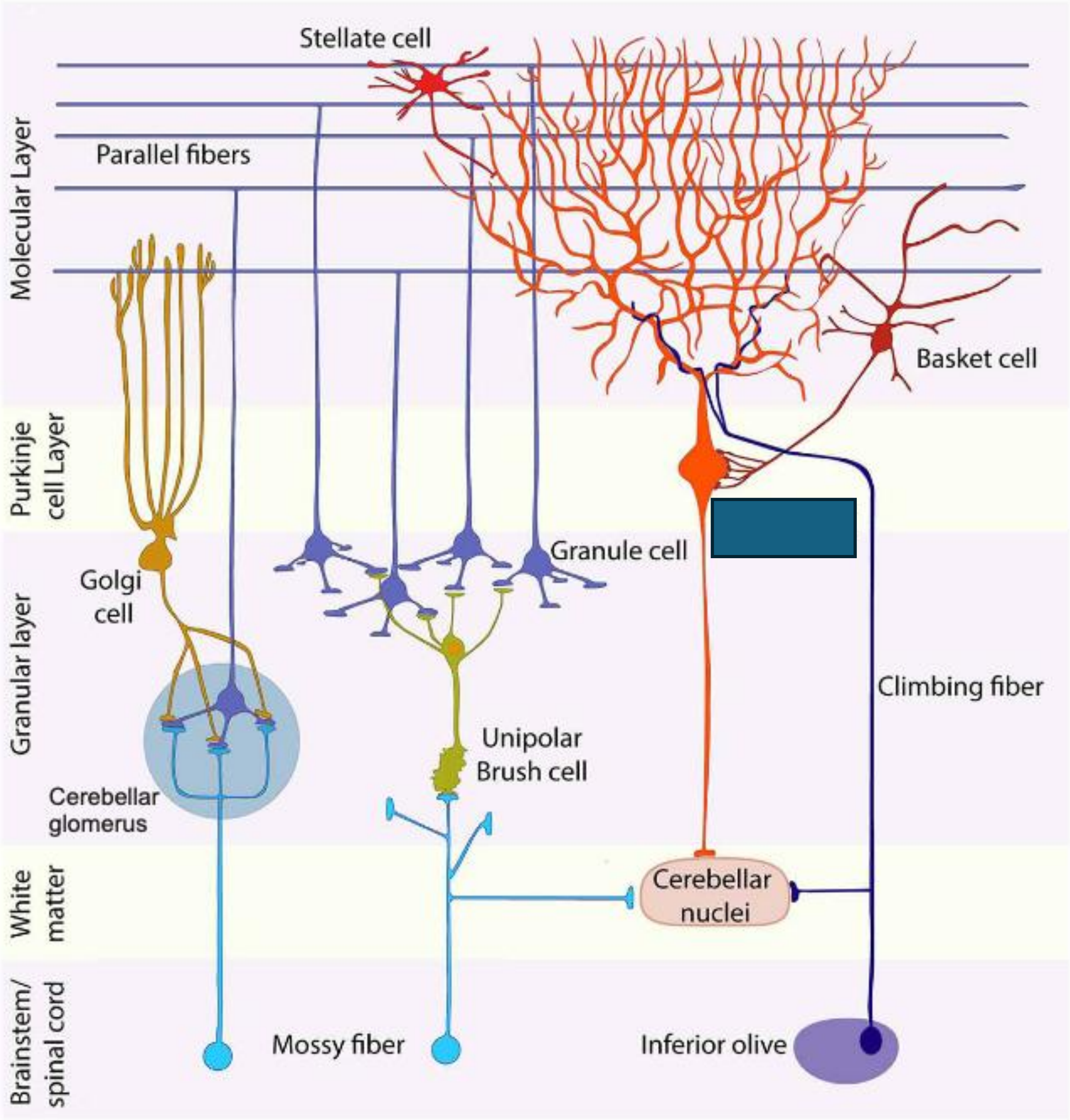
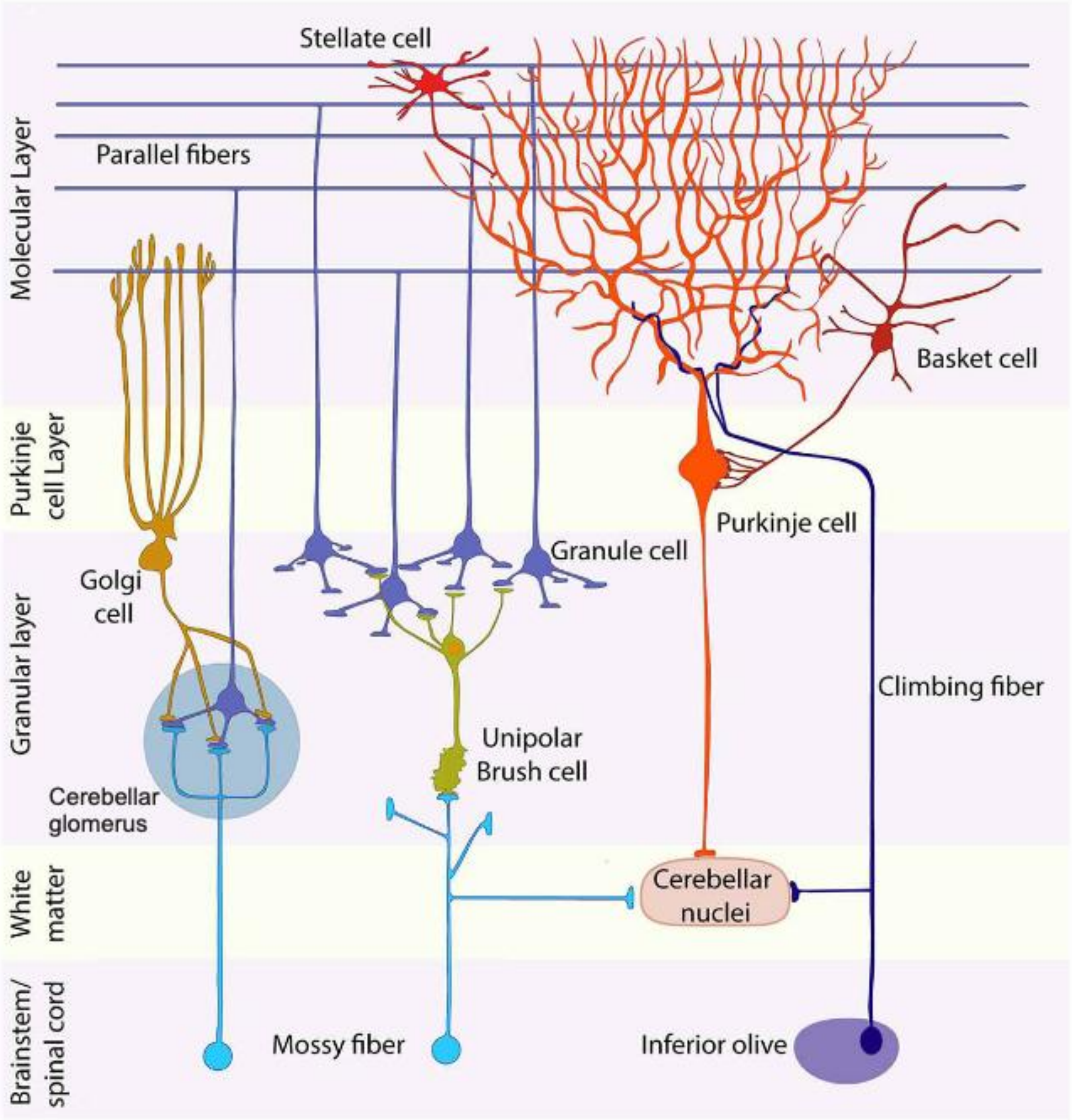


Figure 10.12 MRI showing a healthy cerebellum (left) and one experiencing degeneration from ataxia (right).

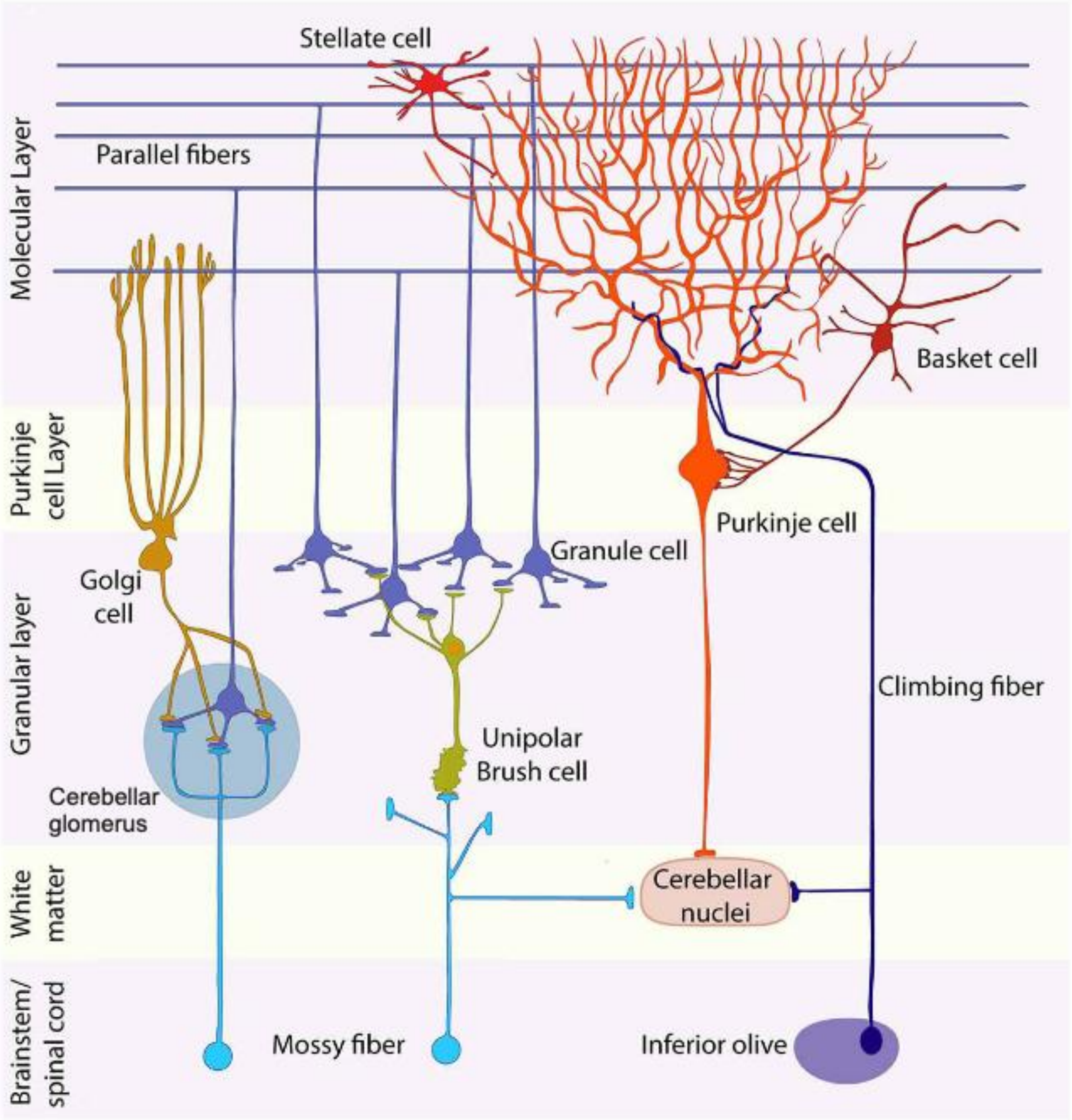
Cellular organization of the cerebellum



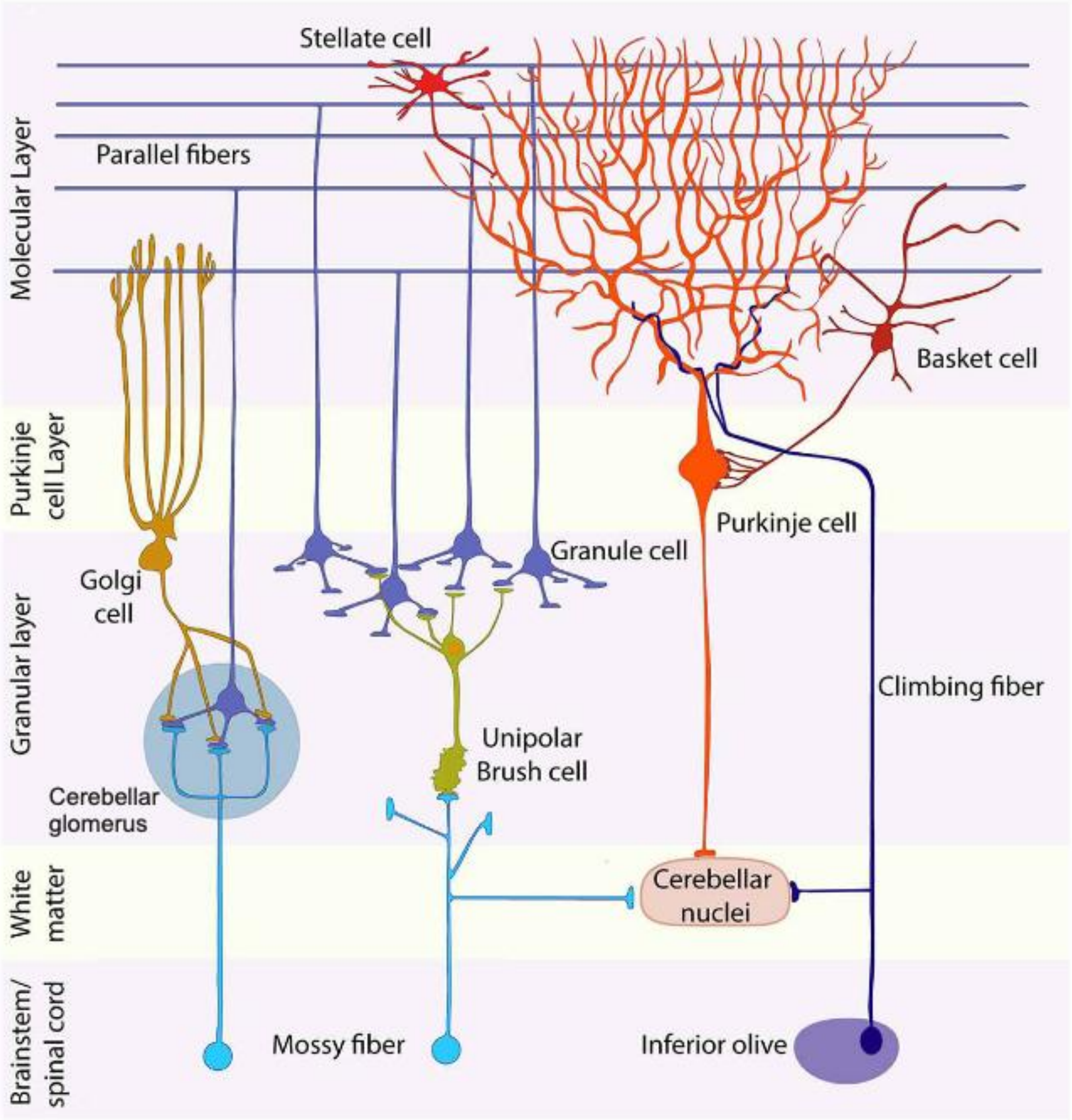
Stellate cell
Parallel fibers
Molecular Layer
Basket cell
Purkinje cell Layer
[redacted]
Granule cell
Golgi cell
Granular layer
Climbing fiber
Unipolar Brush cell
Cerebellar glomerus
Cerebellar nuclei
White matter
Brainstem/ spinal cord
Mossy fiber
Inferior olive



- Deepest layer
- Input layer
- Granule and unipolar brush cells are excitatory
- Neurotransmitter spillover and crosstalk in the glomeruli



- Output layer
- Project to deep cerebellar nuclei (in cerebellar white matter) or vestibular nuclei (in brain stem) where GABA is released

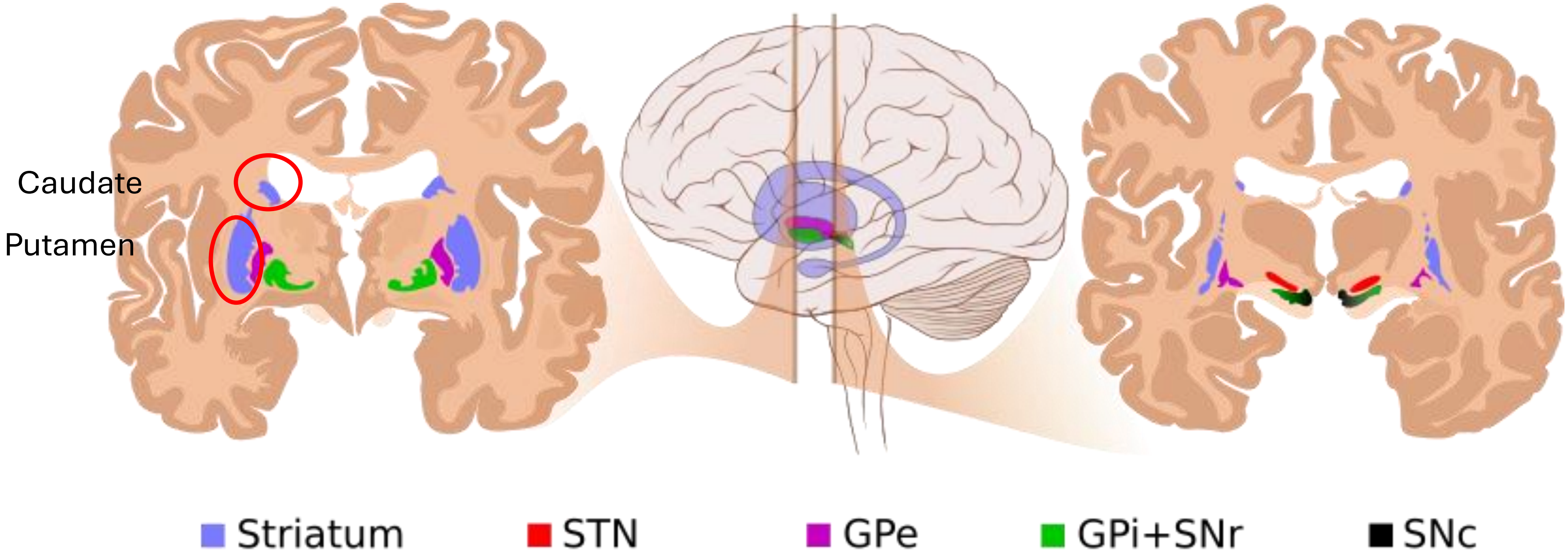


- Apical dendrites of Purkinje cells
- Parallel fibres (axons of granule cells) – horizontal bifurcations
- Inhibitory interneurons (basket and stellate cells): provide feedforward inhibition to Purkinje cells

Basal ganglia – link to lab

Basal ganglia

Series of subcortical brain structures



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- SNpc to striatum: Nigrostriatal pathway

- Thalamus and cortex are not part of basal ganglia

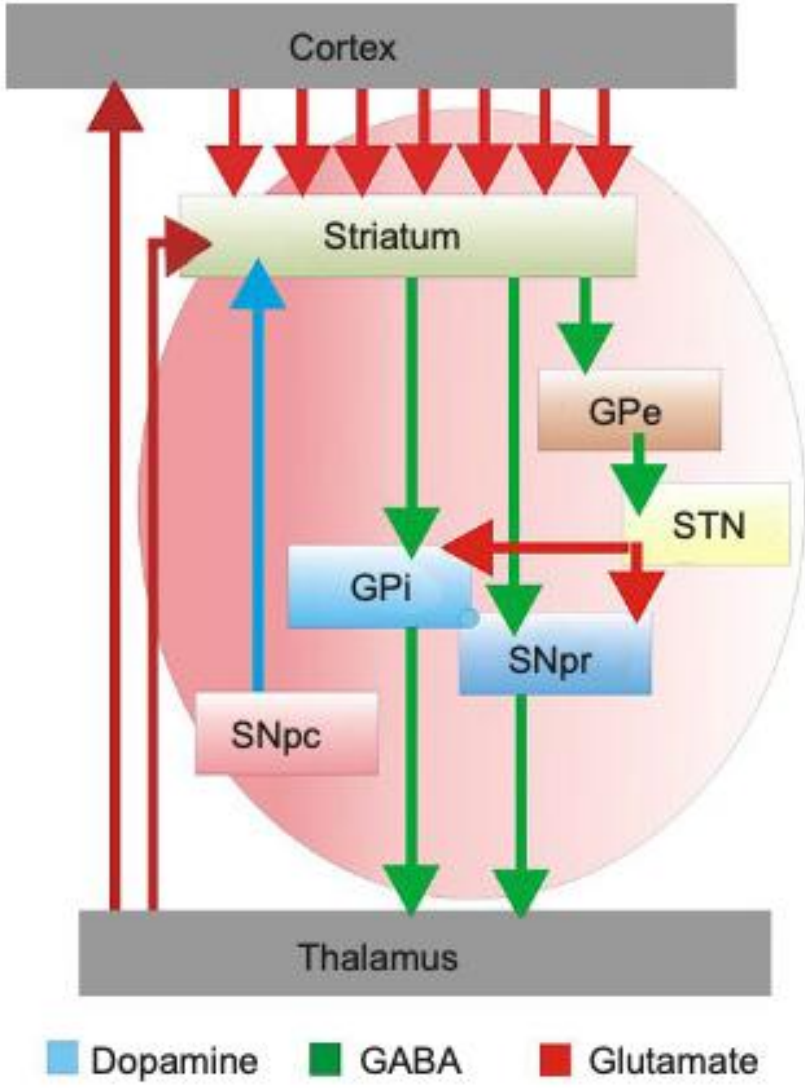
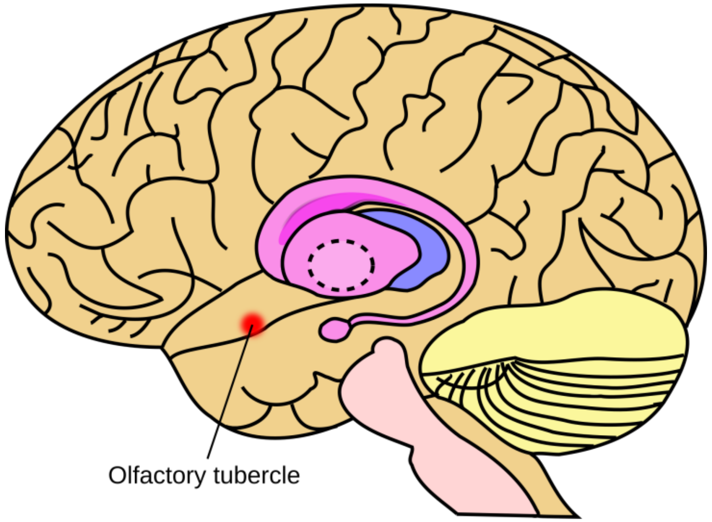


Figure 10.15 Diagram showing the connectivity and the nature of the signaling between different nuclei of the basal ganglia. (in pink circle)

Striatum

- Dorsal striatum
  - Caudate + putamen (not distinguishable in rodent brain)
  - Habit learning (e.g., serial reaction time test)
  - Caudate: goal directed actions (e.g., Skinner box)
  - Putamen: motor-associated procedural learning (e.g., mirror tracing)
- Ventral striatum
  - Olfactory tubercule + nucleus accumbens (red outline)
  - Reward, motivation, and aversion
  - Increased dopamine in ventral striatum is a "learning signal" that encourages animal to repeat behaviour



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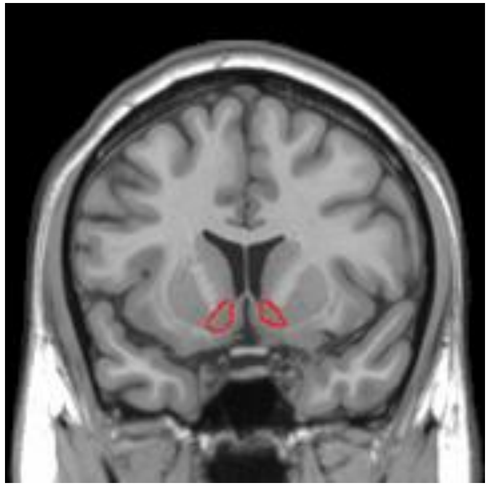

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Globus pallidus (GP)

- Internal (GPi): movement promoting
- External (GPe): movement inhibiting

Subthalamic nucleus (STN)

- Target of some DBS procedures to alleviate symptoms of PD

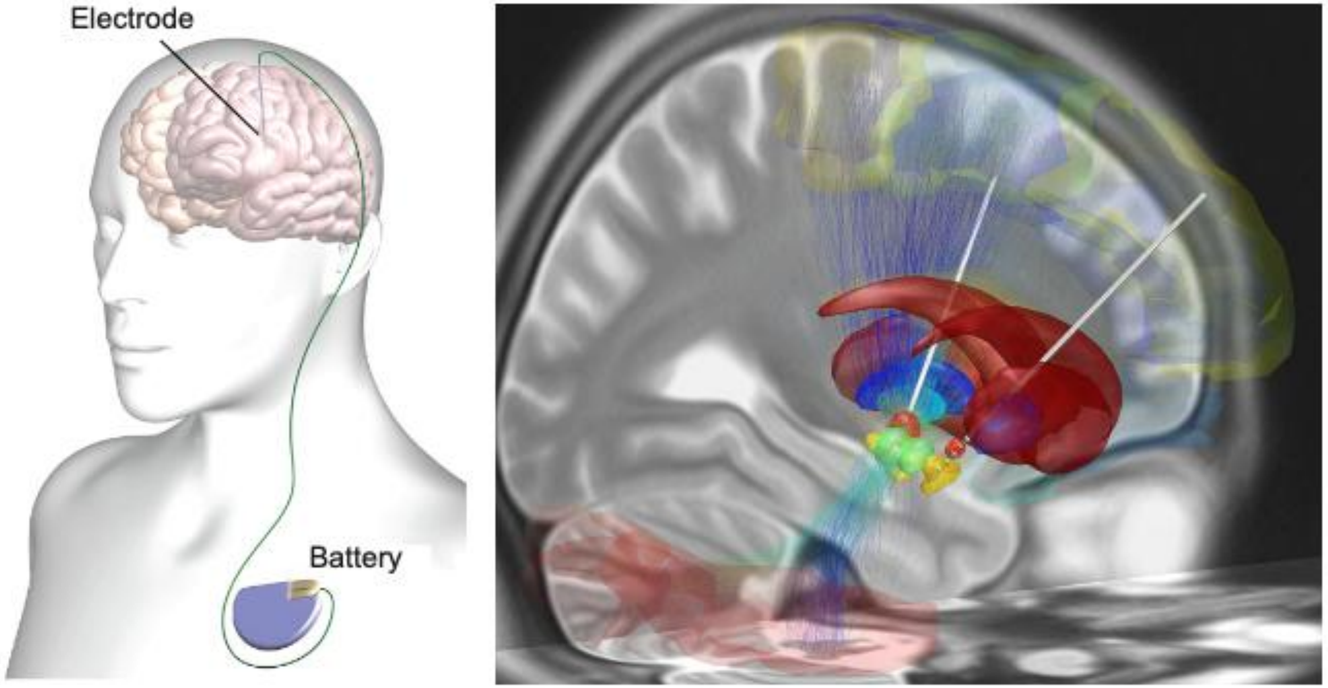


Figure 10.19 A deep brain stimulator (left) is controlled by an external battery. During surgery, the STN (right) can be targeted as a treatment for Parkinson's disease.

Substantia nigra (SN)

- Midbrain structure
- Appears darker due to expression of neuromelanin
- SN pars compacta (SNpc or SNc)
  - Neurons degenerate in PD
- SN pars reticulata (SNpr or SNr)
  - Important for saccades

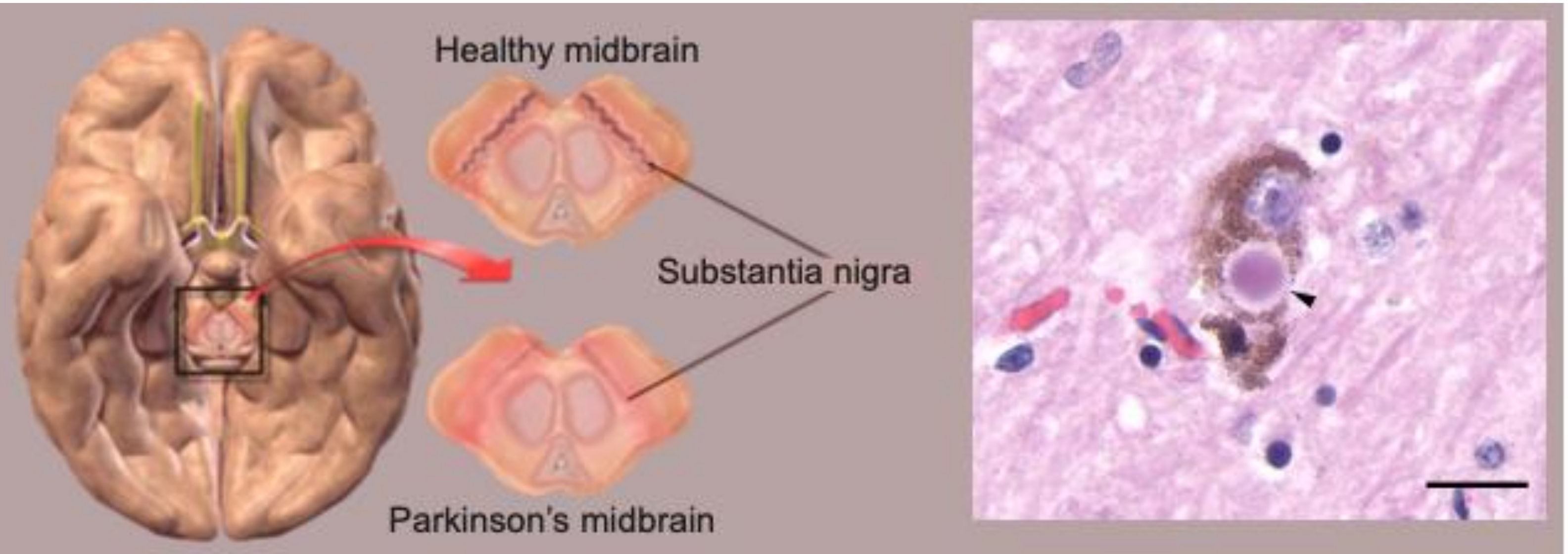


Figure 10.20 The substantia nigra selectively degrades in PD (left), possibly due to accumulation of Lewy bodies inside the neurons (right, arrow head).

Clinical connection: Huntington's Disease

Huntington's disease is a rare neurodegenerative movement disorder resulting from various dysfunctional signaling pathways of the basal ganglia. Symptoms include **hyperkinesia** (uncontrolled movement), poor coordination, and cognitive and psychiatric changes eventually leading to dementia. The onset of symptoms happens when a person is in their 30s-50s, and prognosis is generally fatal within 15 years after diagnosis. There is currently no cure for the disease.

Huntington's disease passed through families in an autosomal dominant manner. A protein called **huntingtin (htt)** is implicated, and mutant huntingtin has several repeats of the amino acid glutamine. The greater the number of repeats, the more severe the symptoms and the earlier the onset of the disease. A leading theory suggests that mutant htt accumulates inside neurons, leading to neurodegeneration.

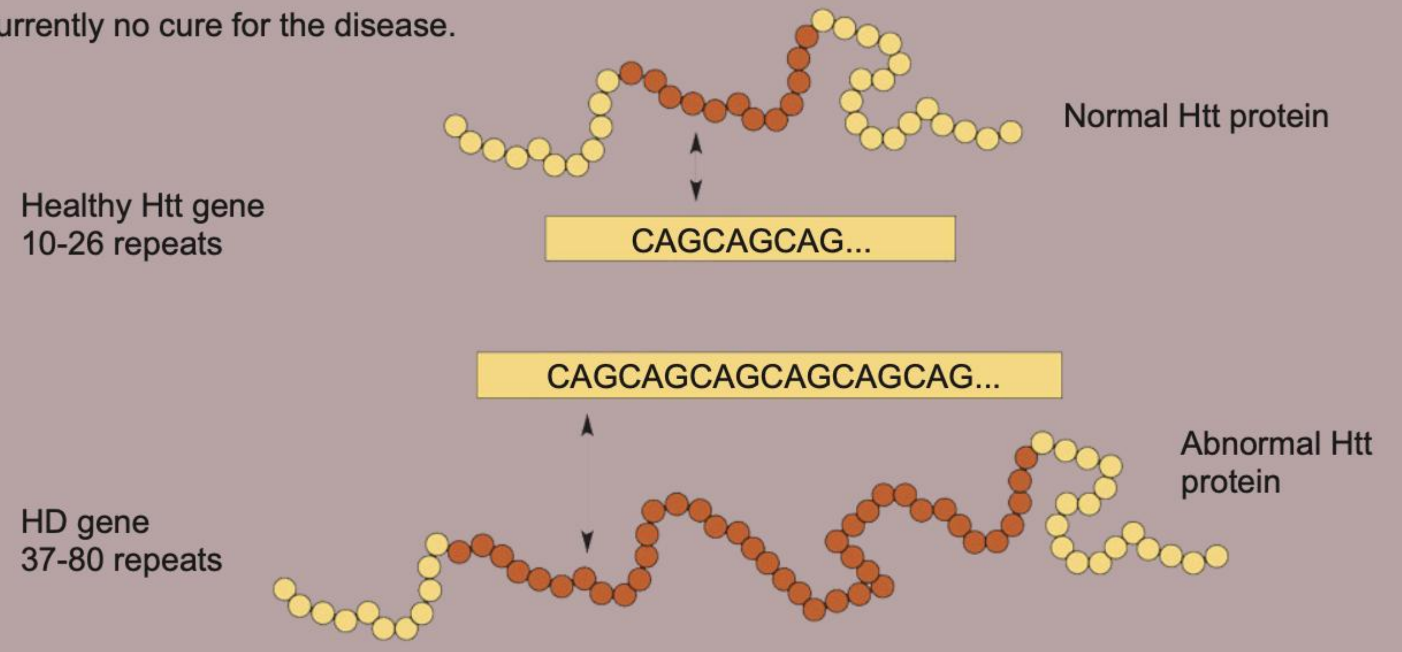
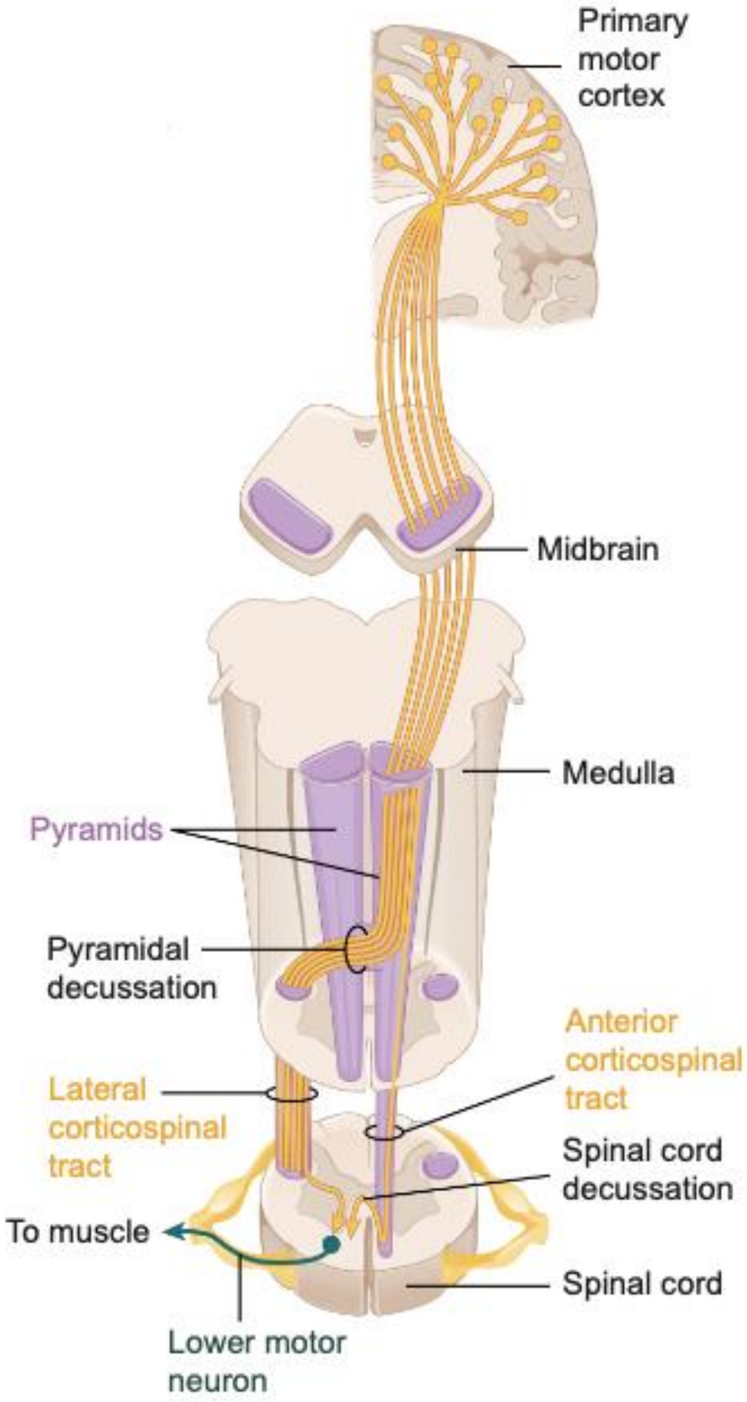


Figure 10.18 Longer CAG repeats in the Htt gene code for abnormal proteins. Greater number of repeats are associated with earlier age of onset of symptom presentation.

Corticospinal tract (from M1 to spinal cord)

- Lateral corticospinal tract
  - 90% of axons in CST
  - Crosses at pyramidal decussation
  - Forms synapses with lower motor neurons in the ventral horn of spinal cord
- Anterior corticospinal tract
  - Carries information to the muscles of the trunk
  - Crosses in spinal cord
  - Forms synapses with lower motor neurons in the ventral horn of spinal cord

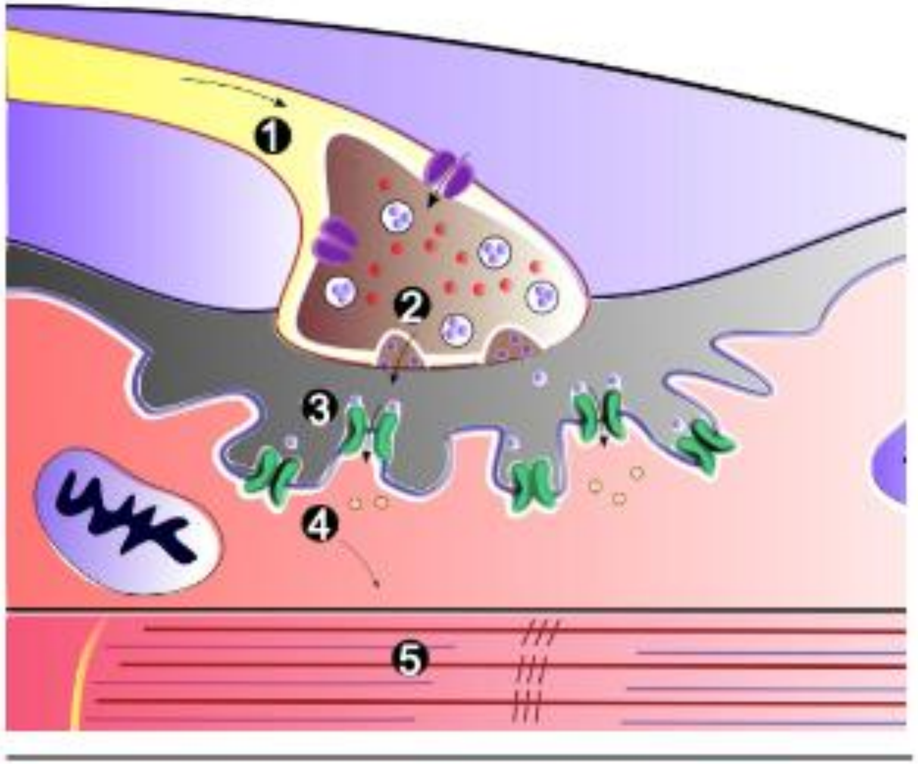


Central pattern generators

- https://www.youtube.com/watch?v=MRMMdW6wz64
- https://www.youtube.com/watch?v=wPiLLplofYw

Muscles

- Neuromuscular junction (NMJ) review
    - Chemical synapse (neurotransmitter: Ach)
    - Postsynaptic cell is a muscle cell
    - Presynaptic cell is a lower motor neuron
- Sarcolemma
    - Postsynaptic site with several folds to increase surface area
    - Contains nicotinic ACh receptors (ionotropic)
- Depolarization of muscle cell causes contraction (shorter, wider shape)



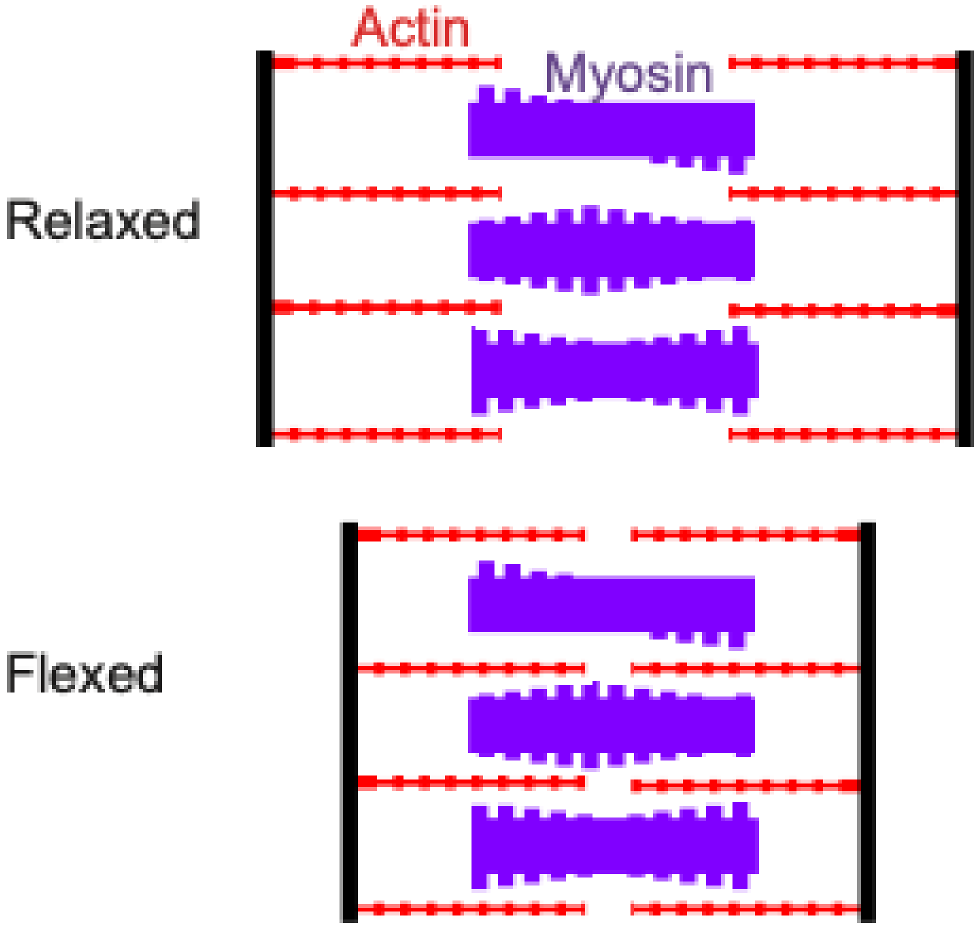
What can you guess about the type of ACh ionotropic receptors are found at the NMJ?

Clinical connection: Myasthenia gravis (MG)

- Autoimmune disorder characterized by muscle weakness, resulting in difficulty with speech, trouble with movement and swallowing, drooping eyelids, and double vision
- Immune system-mediated destruction of the nAChRs at NMJs
- Treat with acetylcholinesterase inhibitors (allow ACh to be in the synapse longer) or immunosuppresant therapy

Muscle anatomy and function

- Sarcomeres
  - 100000s in each muscle fibre, each one 1.5-3.5 microns
  - Functional units of muscle contraction
- Actin and myosin
  - Proteins in sarcomeres
  - Actin: Thin filament, forms scaffolding
  - Myosin: Thick filament, pulls actin together



Muscle types

	Striated	Smooth
Voluntary	**Skeletal**	
Involuntary	**Cardiac**	**Smooth**

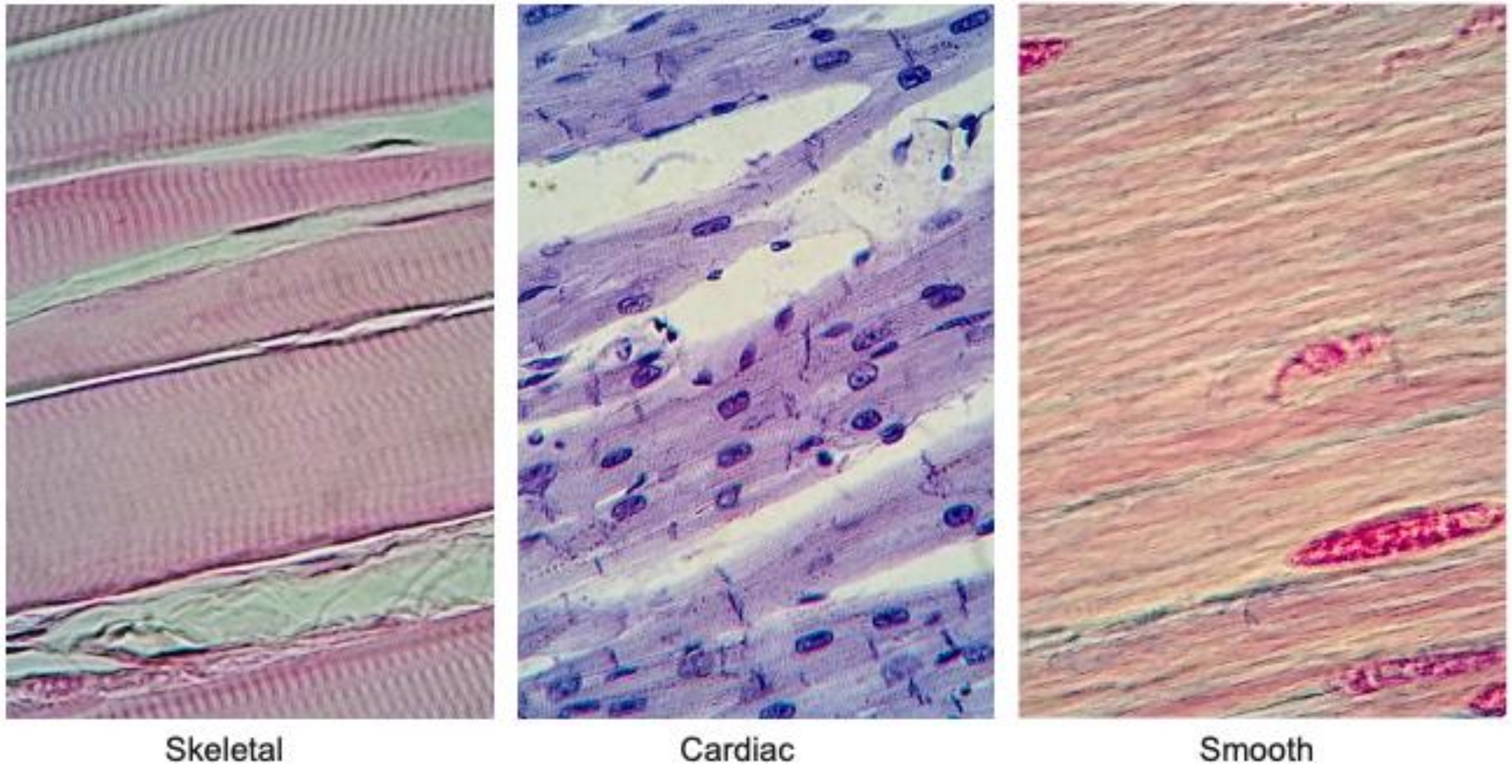


Figure 10.29 Different types of muscle as seen at 400x magnification. Muscle fibers are seen running from left to right. In skeletal and cardiac muscles, striations can be seen perpendicular to the muscle fibers.

- Smooth muscles
  - Embedded in organs
  - Involuntary
- Cardiac muscles
  - Striated but involuntary

Cooperation and opposition of skeletal muscles

- Agonist muscle
  - Main mover
  - Supported by synergistic muscles (e.g., provide stability)
- Antagonist muscles
  - Move in opposite direction
  - Lengthens while the agonist contracts

Types of skeletal muscle

- Fast twitch
  - E.g., sprints
- Slow twitch
  - E.g., jogging