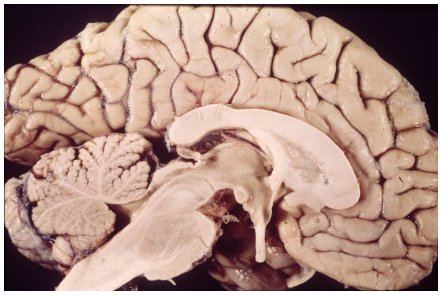
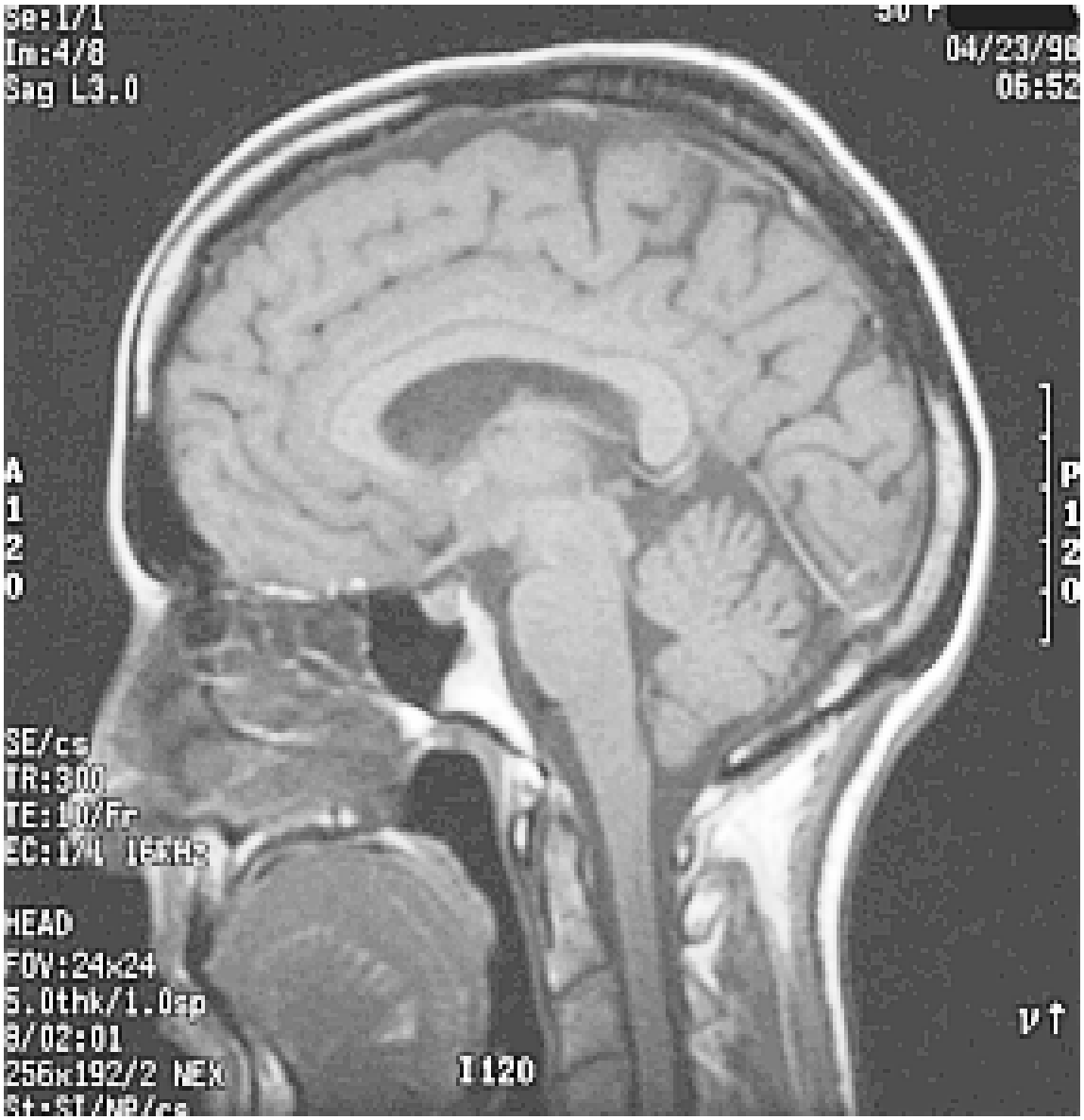
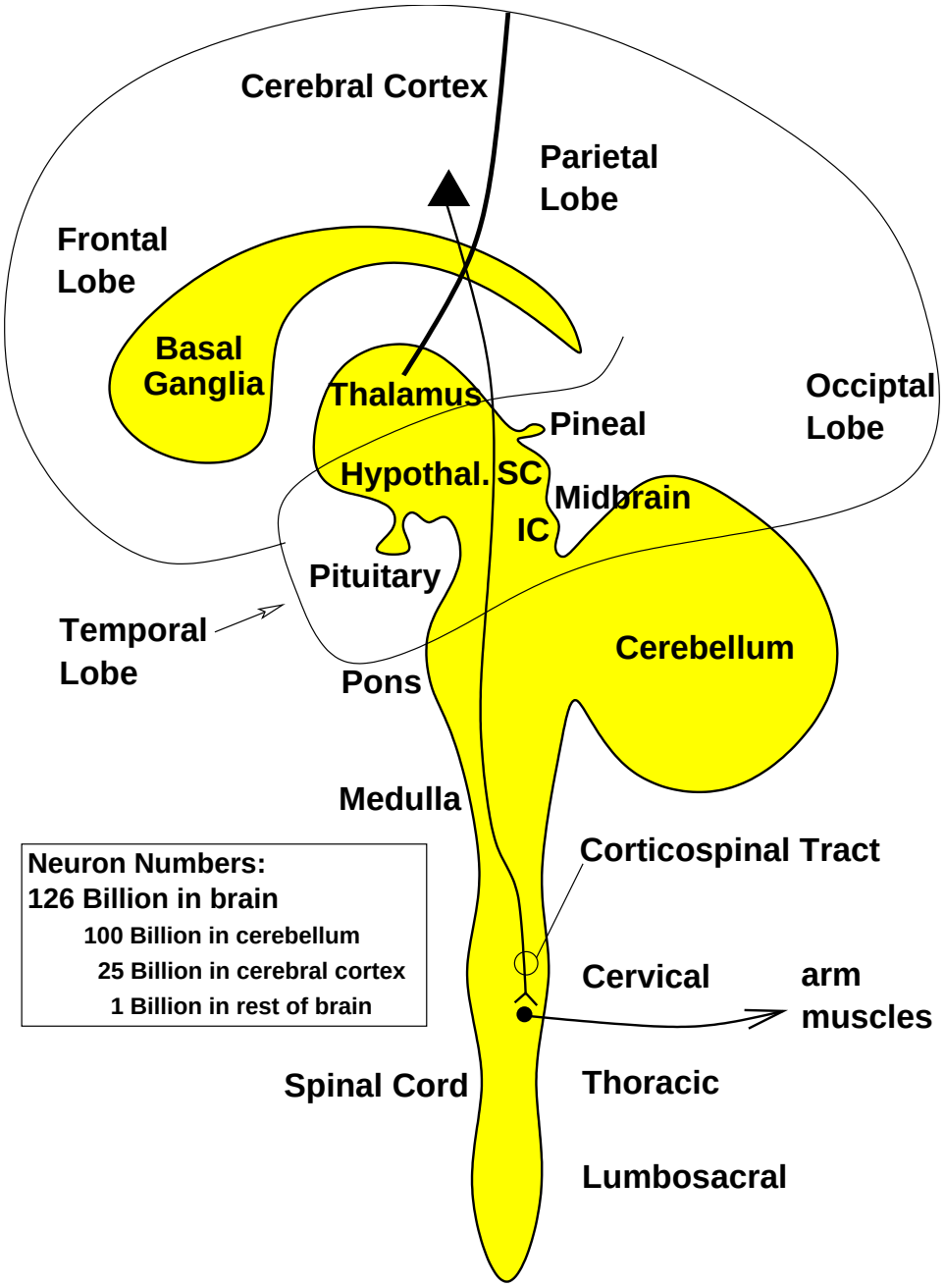
Brain Midline1



Midline MRI

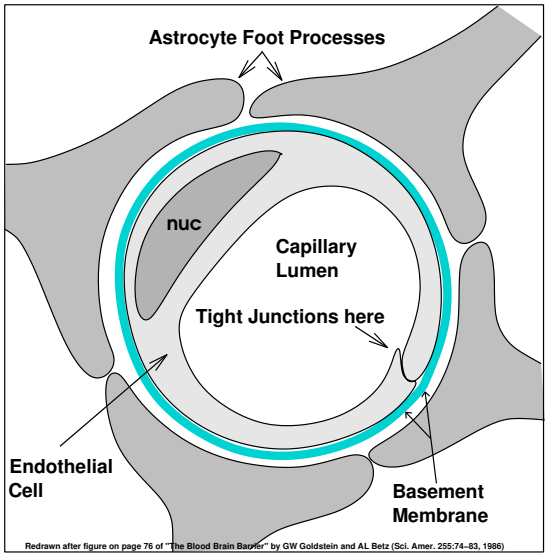

Se:1/1
Im:4/8
Sag L3.0
04/23/98
06:52
A
1
2
0
P
1
2
0
SE/cs
TR:300
TE:10/Fr
HEAD
FOV:24x24
5.0thk/1.0sp
3/02:01
256x192/2 NEX
I120

Brain Diagram

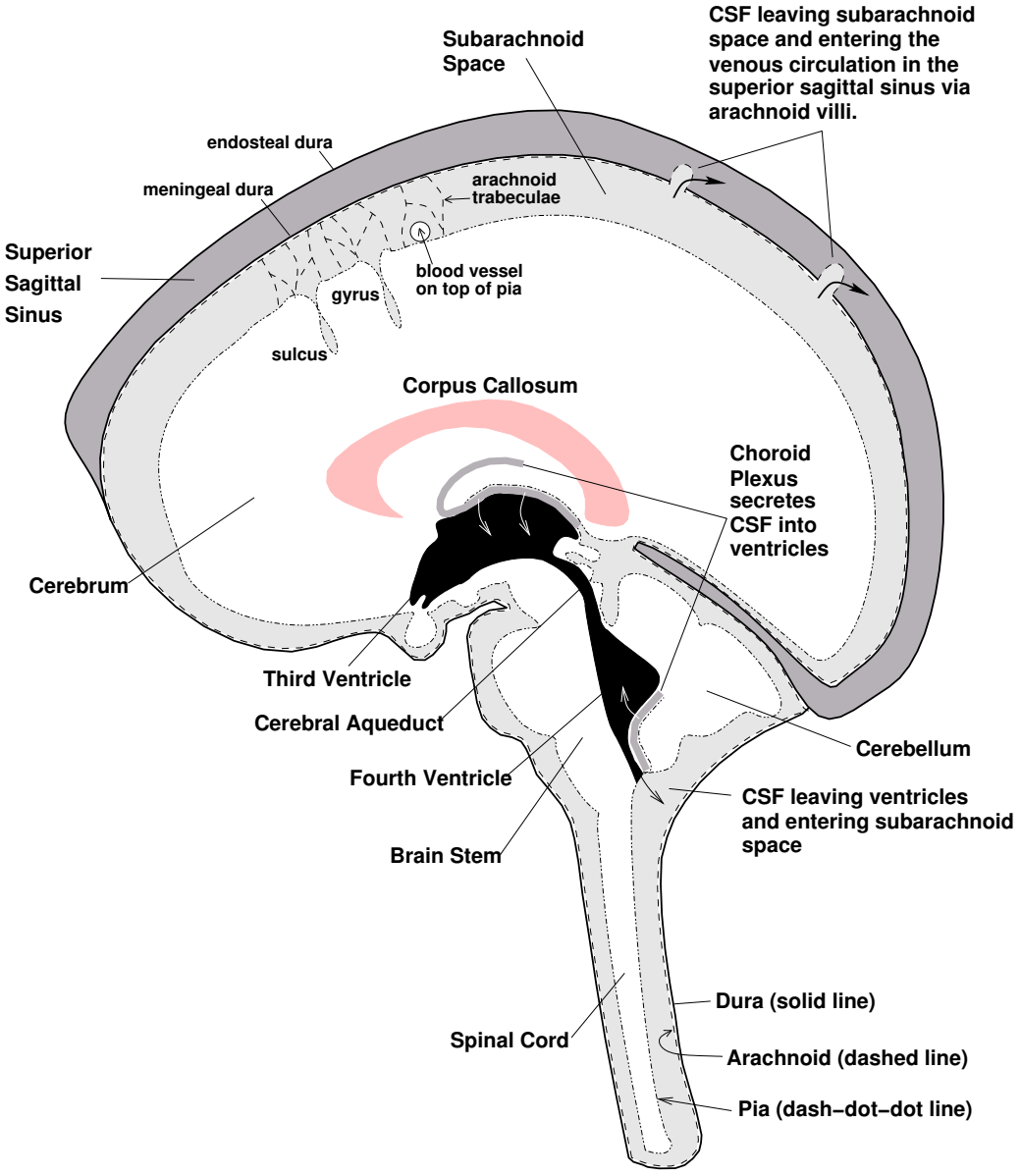


Neuron Numbers:
126 Billion in brain
- **100 Billion in cerebellum**
- **25 Billion in cerebral cortex**
- **1 Billion in rest of brain**

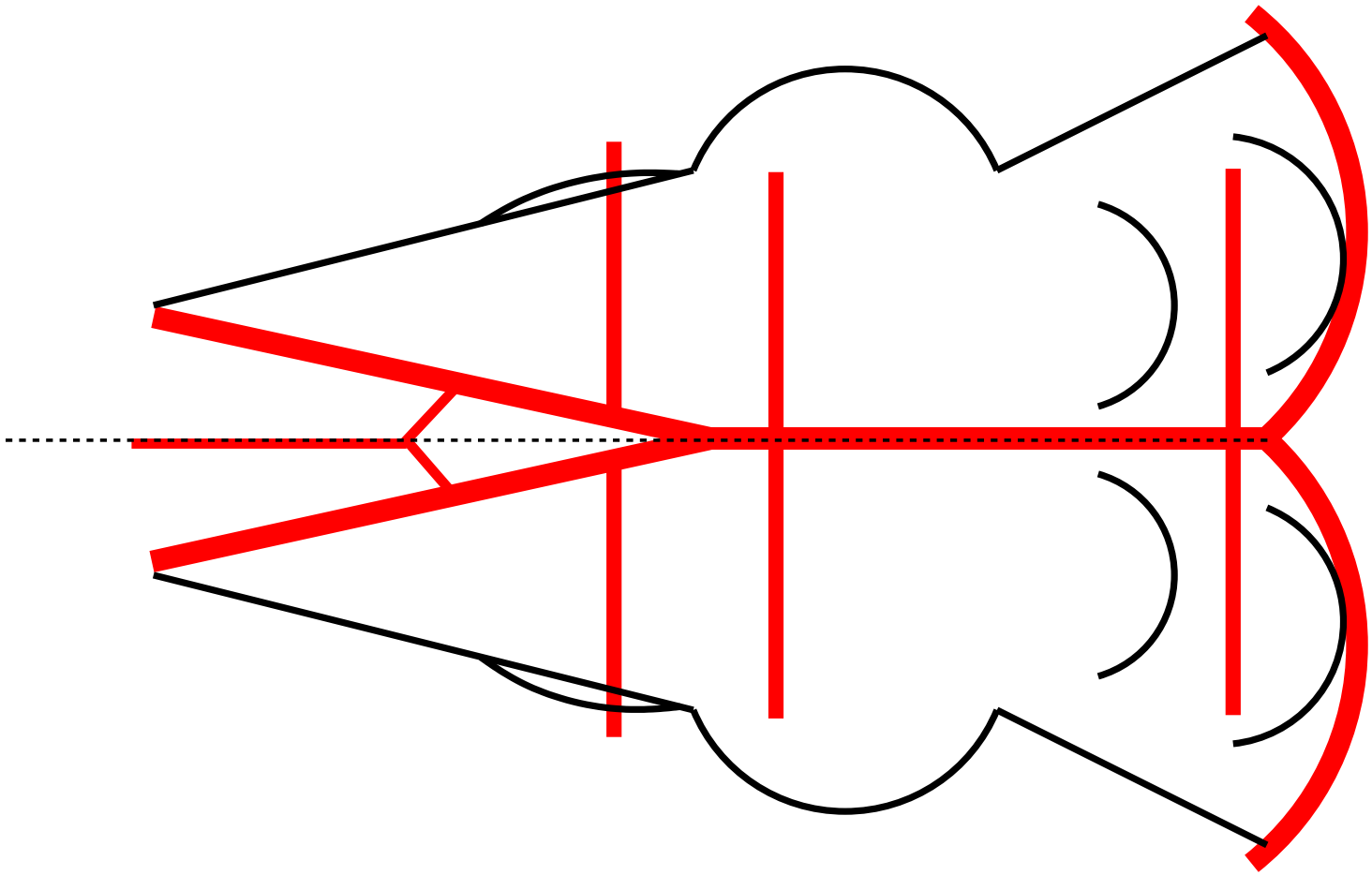
BBB1


Astrocyte Foot Processes
nuc
Capillary
Lumen
Tight Junctions here
Endothelial
Cell
Basement
Membrane
Redrawn after figure on page 76 of "The Blood Brain Barrier" by GW Goldstein and AL Betz (Sci. Amer. 255:74–83, 1986)

CSF Circulation

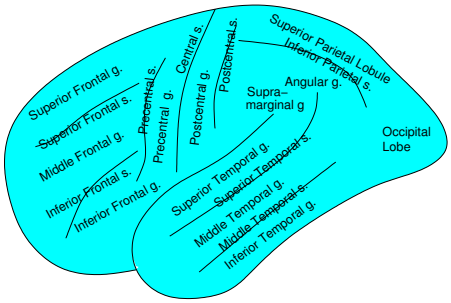


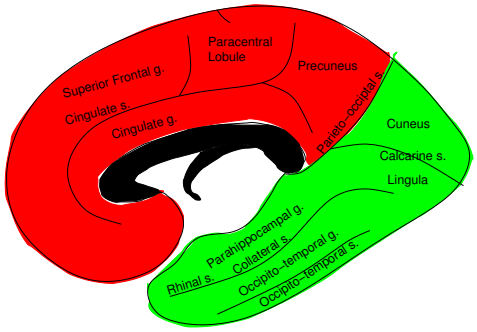
Vertebrobasilar Circulation

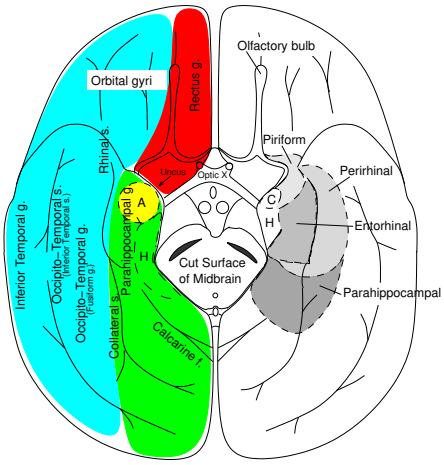


Cerebral Circulation

Cerebral Circulation Territories





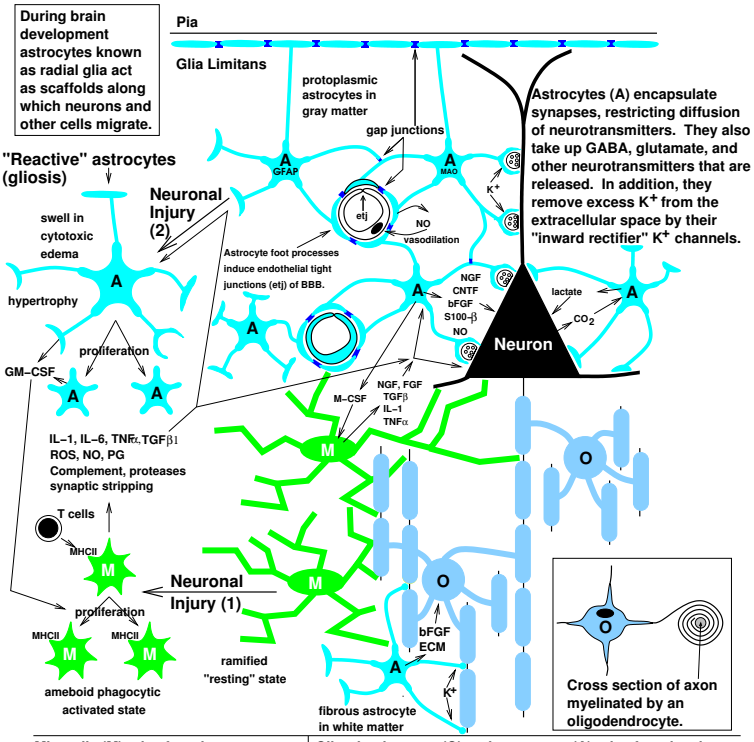


Middle cerebral a.

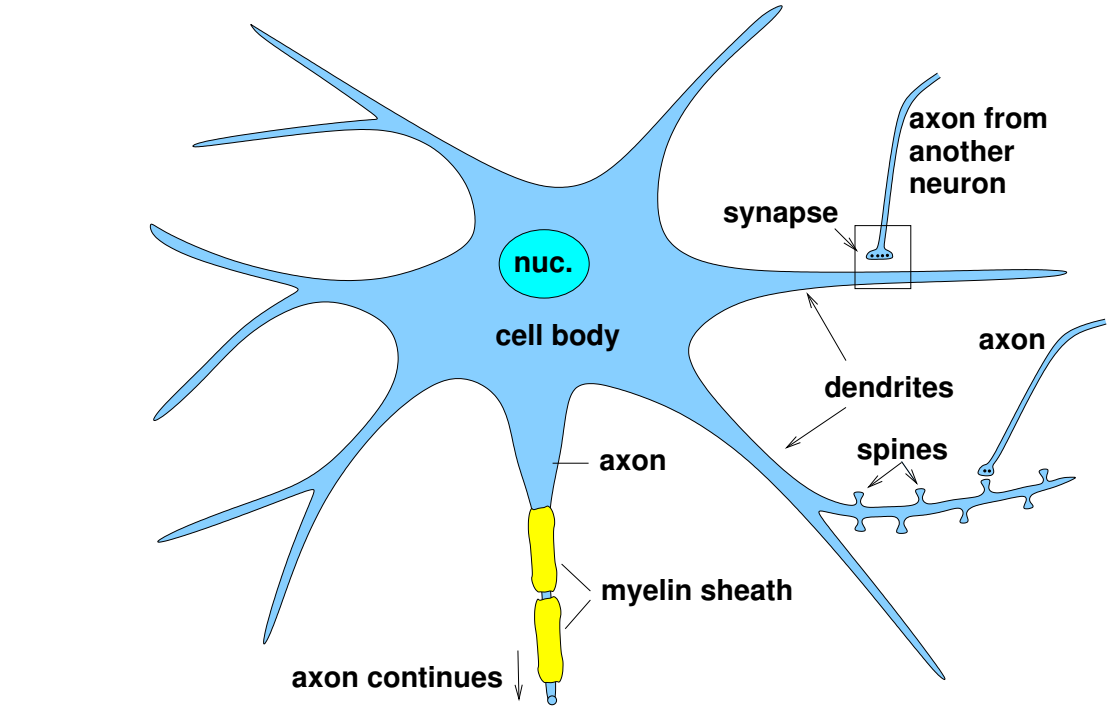
Anterior cerebral a.

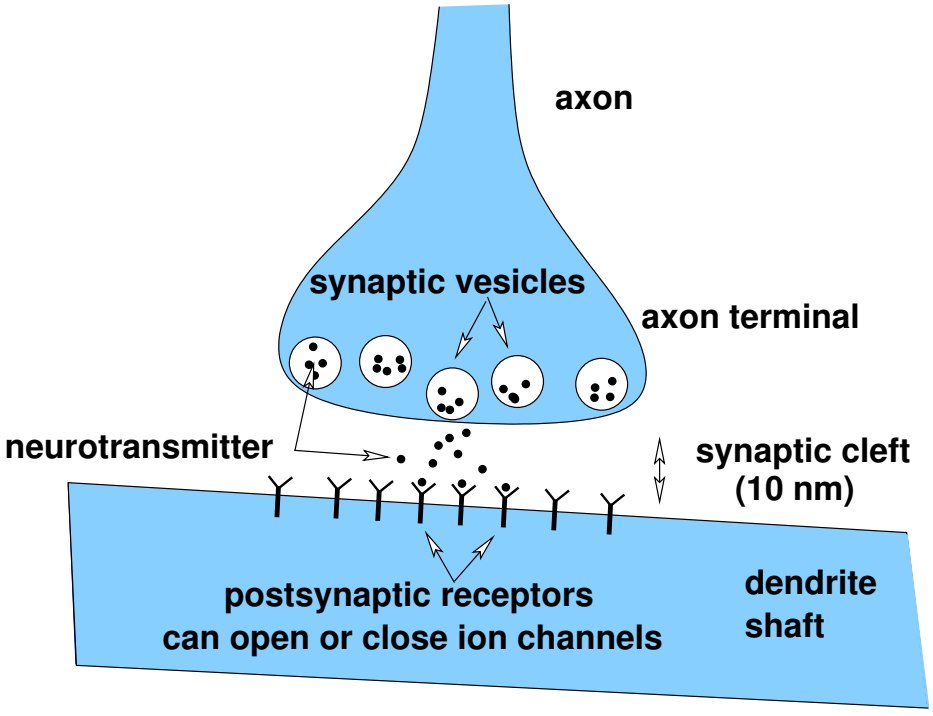
Posterior cerebral a.

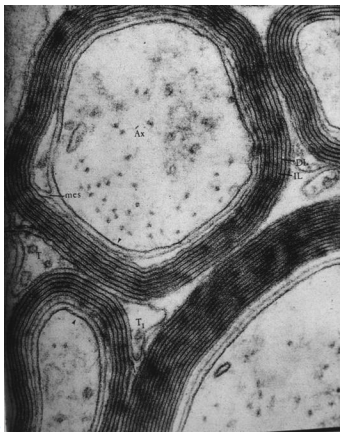
Glia


During brain development astrocytes known as radial glia act as scaffolds along which neurons and other cells migrate.
Pia
Glia Limitans
protoplasmic astrocytes in gray matter
gap junctions
Astrocytes (A) encapsulate synapses, restricting diffusion of neurotransmitters. They also take up GABA, glutamate, and other neurotransmitters that are released. In addition, they remove excess K+ from the extracellular space by their "inward rectifier" K+ channels.
"Reactive" astrocytes (gliosis)
swell in cytotoxic edema
hypertrophy
proliferation
GM–CSF
Neuronal Injury (2)
Astrocyte foot processes induce endothelial tight junctions (etj) of BBB.
etj
NO vasodilation
NGF
CNTF
bFGF
S100-β
NO
lactate
CO2
Neuron
M–CSF
NGF, FGF
TGFβ
IL–1
TNFα
IL–1, IL–6, TNFα, TGFβ1
ROS, NO, PG
Complement, proteases
synaptic stripping
T cells
MHCII
Neuronal Injury (1)
proliferation
ramified "resting" state
ameboid phagocytic activated state
bFGF
ECM
K+
fibrous astrocyte in white matter
Cross section of axon myelinated by an oligodendrocyte.
Microglia (M) arise from bone marrow derived monocytes (mesoderm) that take up residence in CNS where they serve as "macrophage sentries" that monitor state of brain and mediate interactions with immune system.
Oligodendrocytes (O) and astrocytes (A) arise from local proliferation of migratory, common precursor cells from neuroepithelium (ectoderm) of ventricles and central canal. Oligodendrocytes can myelinate as many as 50 CNS axons over the long (+5 yr) and regionally varied period of brain development.

Neuron

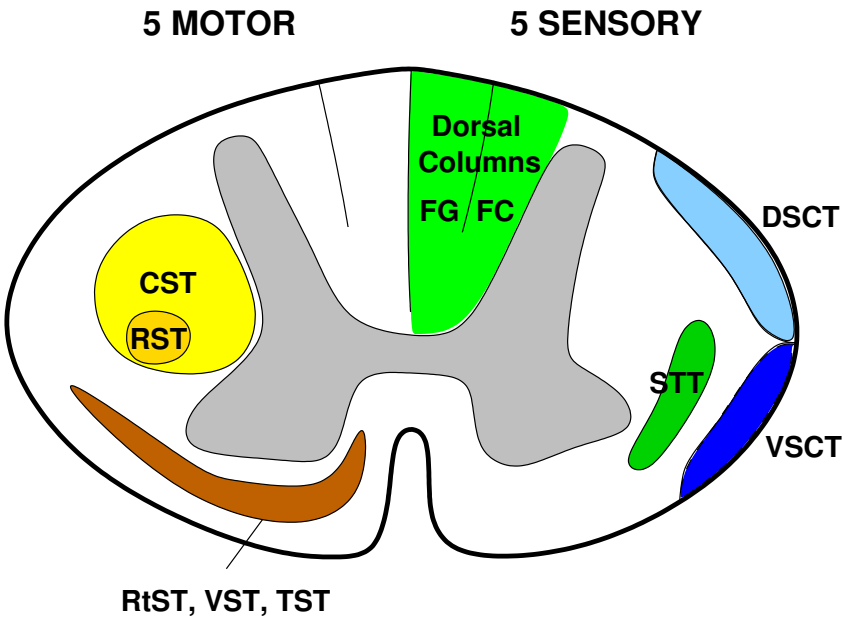

axon from another neuron
synapse
nuc.
cell body
axon
dendrites
spines
axon
myelin sheath
axon continues


axon
synaptic vesicles
axon terminal
neurotransmitter
synaptic cleft
(10 nm)
postsynaptic receptors
can open or close ion channels
dendrite
shaft



Spinal Cord Tracts

10 Important Tracts



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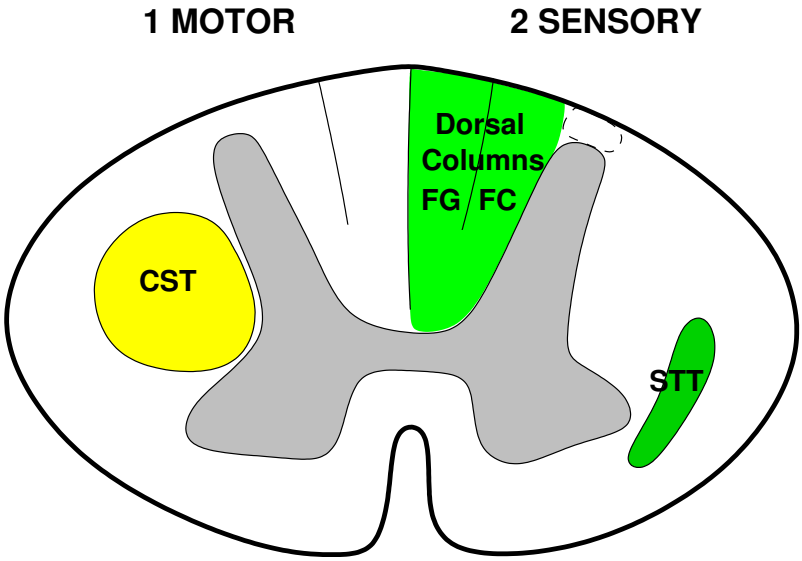
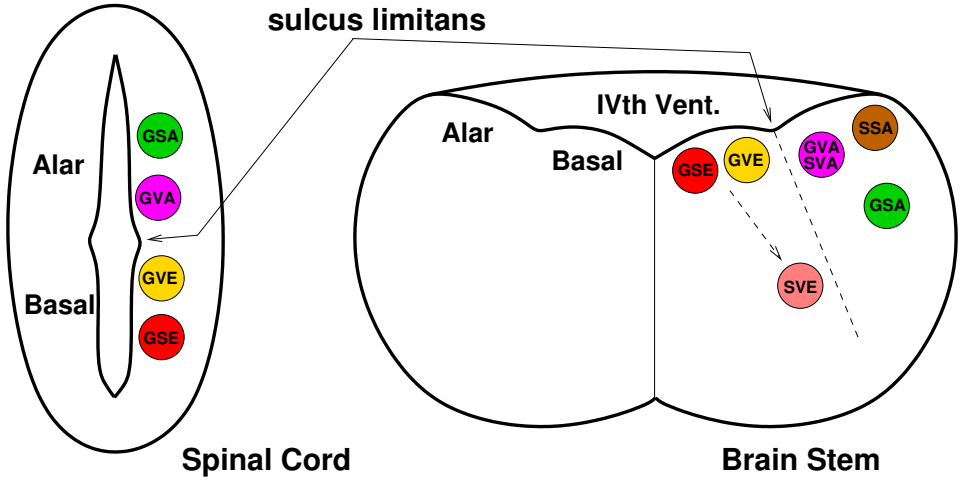
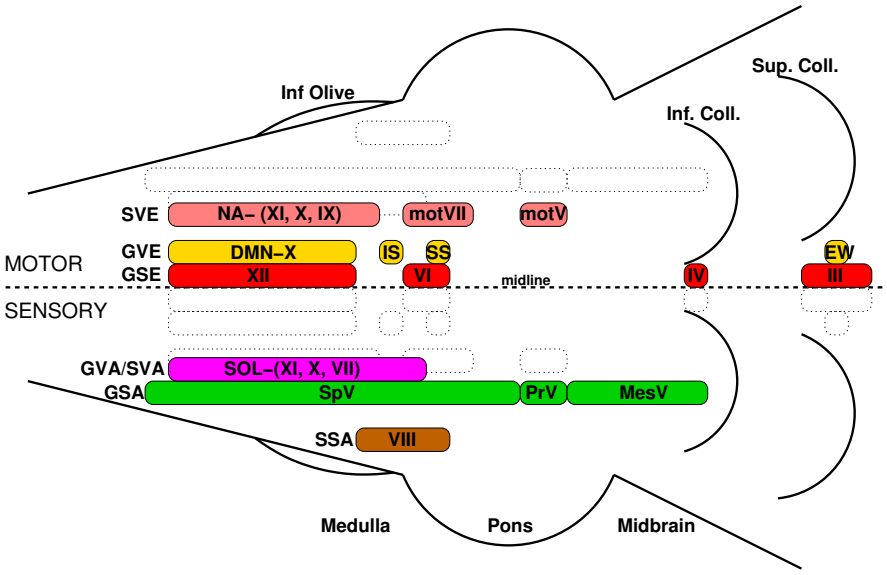
3 REALLY IMPORTANT Tracts



Cranial Nerve Organization into Functional Groups

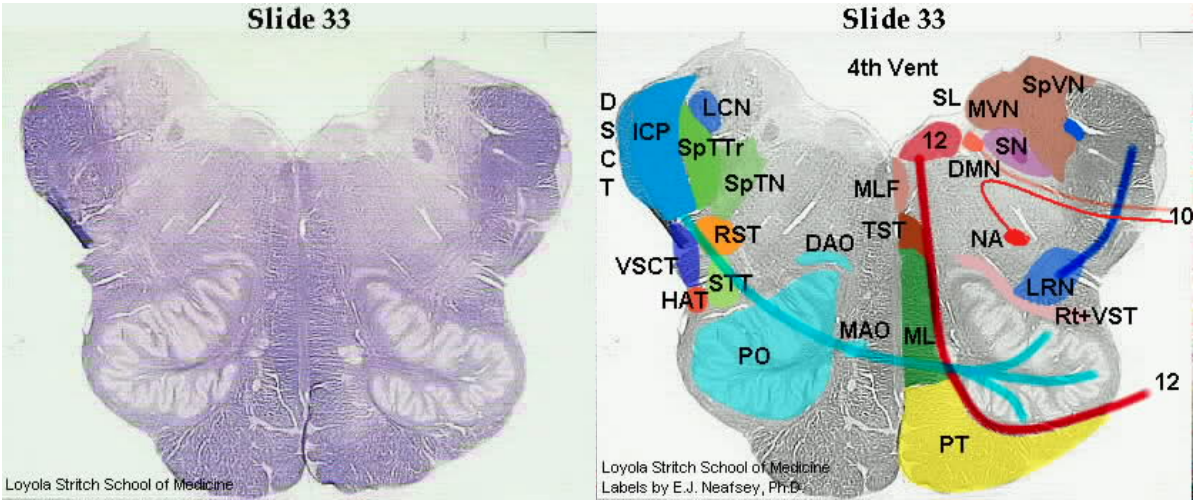
Efferents (E)			Afferents (A)		
GSE	General Somatic	→ myotome skeletal muscle	GVA	General Visceral	← heart, stomach, etc.
SVE	Special Visceral	→ arch skeletal muscle	SVA	Special Visceral	← taste
GVE	General Visceral	→ smooth & cardiac muscle, glands	GSA	General Somatic	← face
			SSA	Special Somatic	← cochlea, semicirc. canals





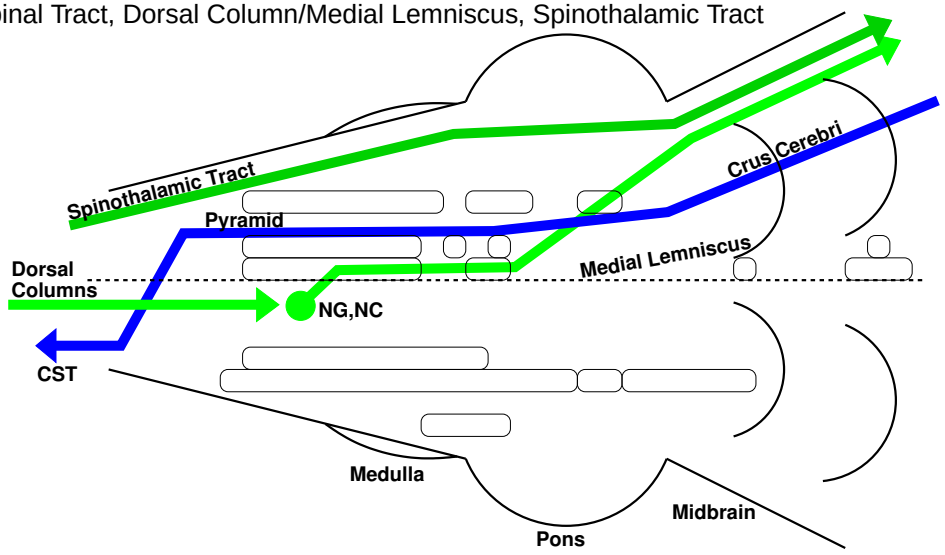
Cranial Nerve	Name	GSE	SVE	GVE	GSA	GVA	SVA	SSA
III	Oculomotor	x		x				
IV	Trochlear	x						
V	Trigeminal		x		x			
VI	Abducens	x						
VII	Facial		x	x	x	x	x	
VIII	Vestibulocochlear							x
IX	Glossopharyngeal		x	x	x	x	x	
X	Vagus		x	x	x	x	x	
XI	Accessory	x	x					
XII	Hypoglossal	x						

Lateral Medullary Syndrome (Wallenberg)

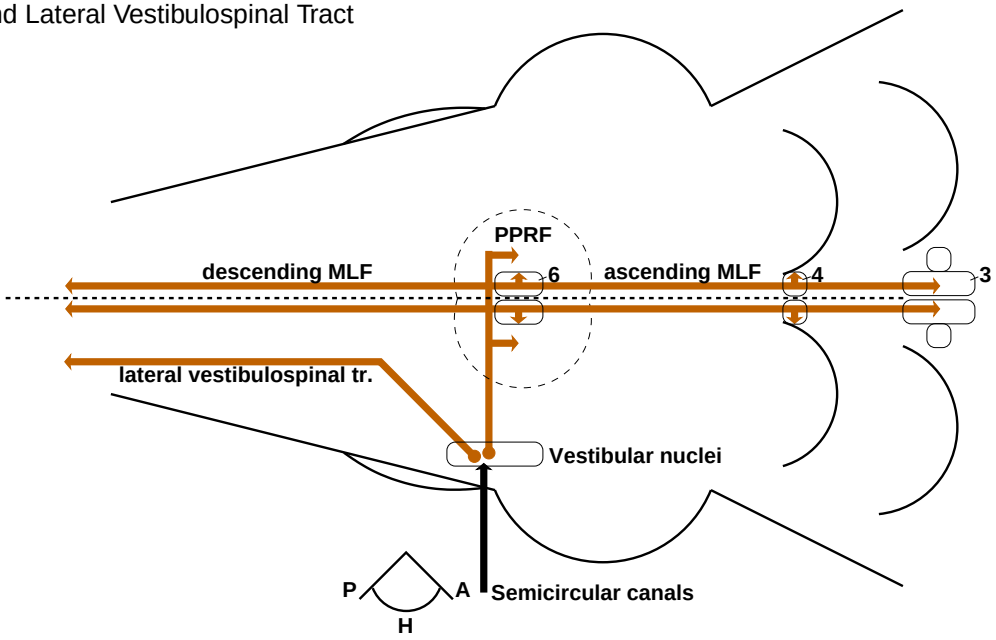


Pathways in the Brain Stem

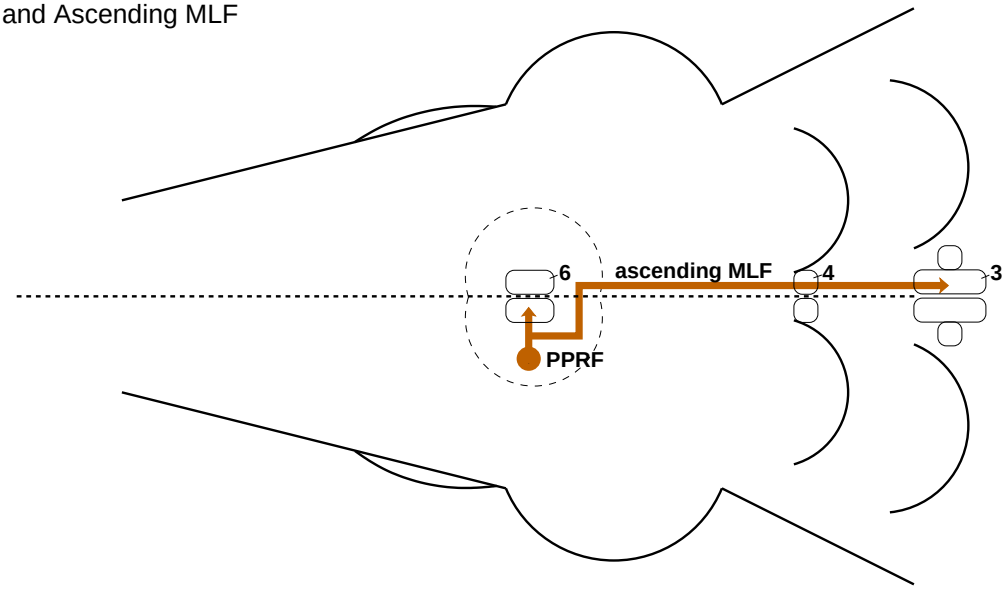
Corticospinal Tract, Dorsal Column/Medial Lemniscus, Spinothalamic Tract



MLF and Lateral Vestibulospinal Tract

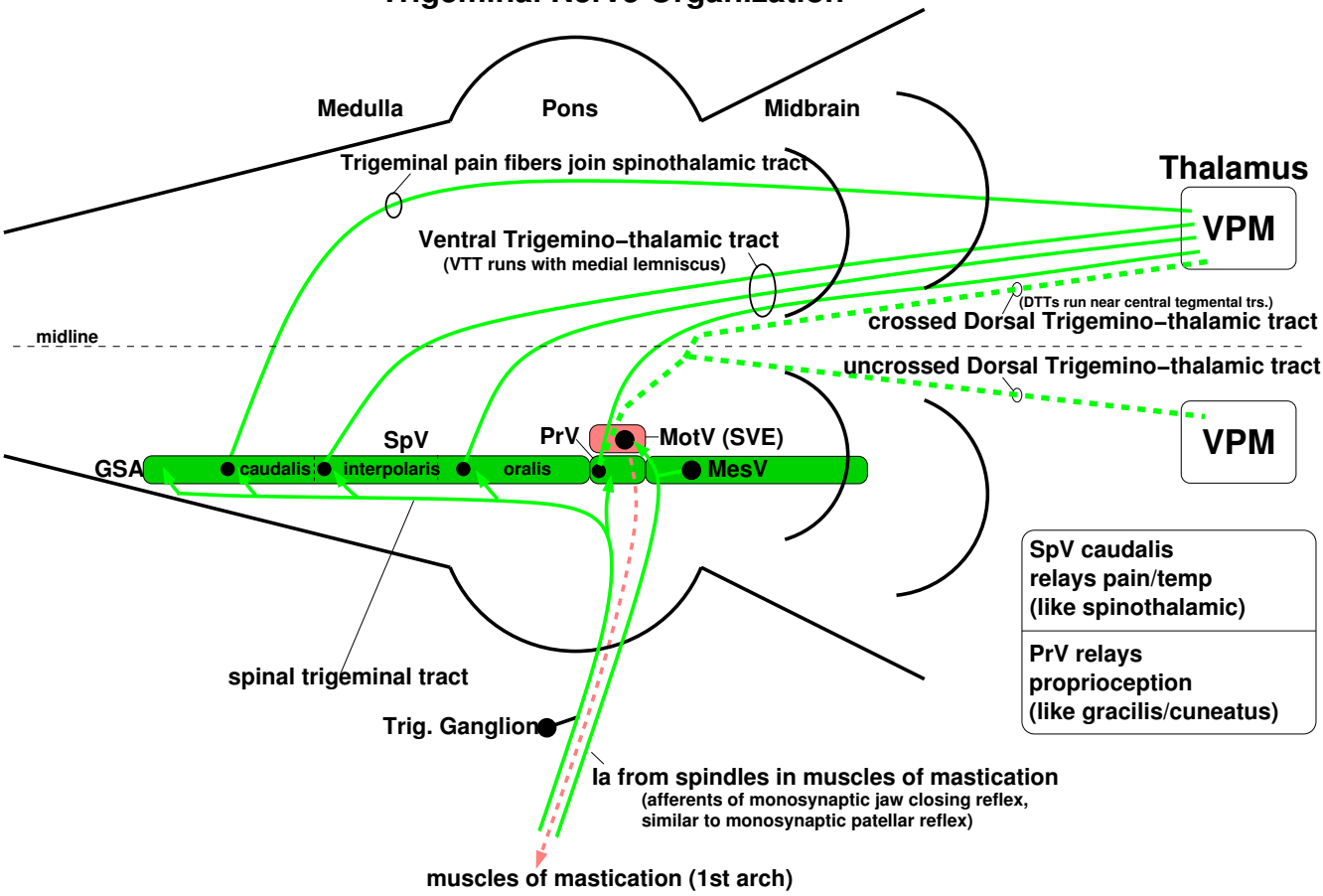


PPRF and Ascending MLF

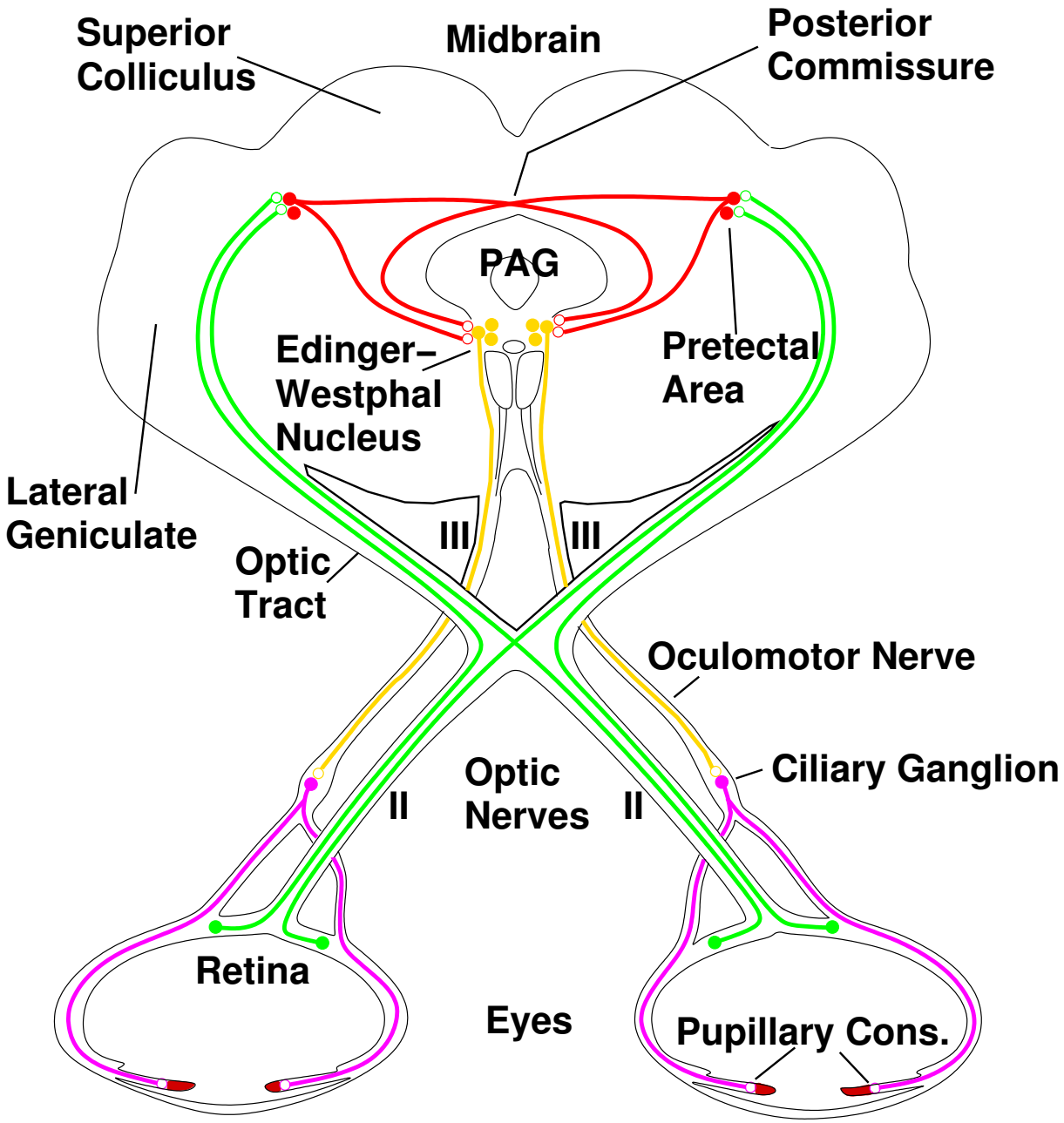


Trigeminal

Trigeminal Nerve Organization

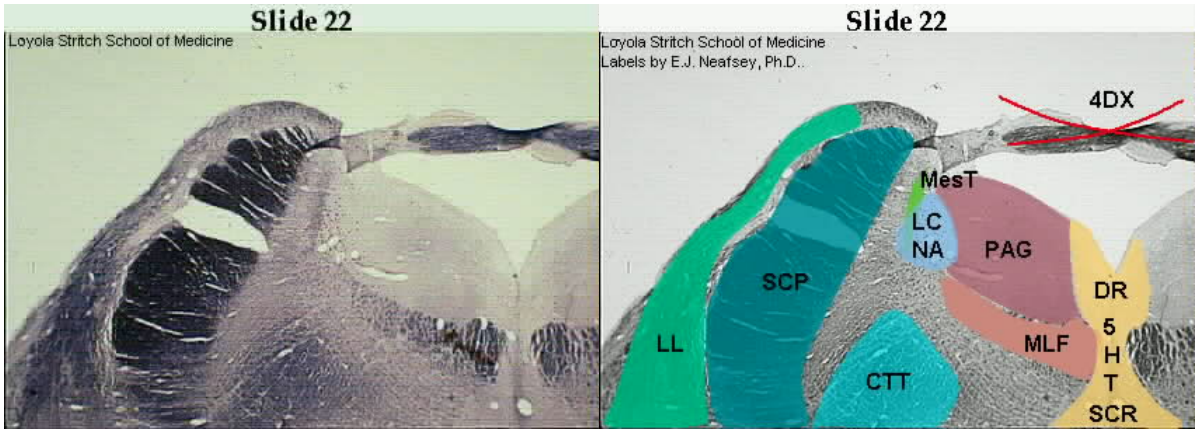


Pupillary Light Reflex Circuitry

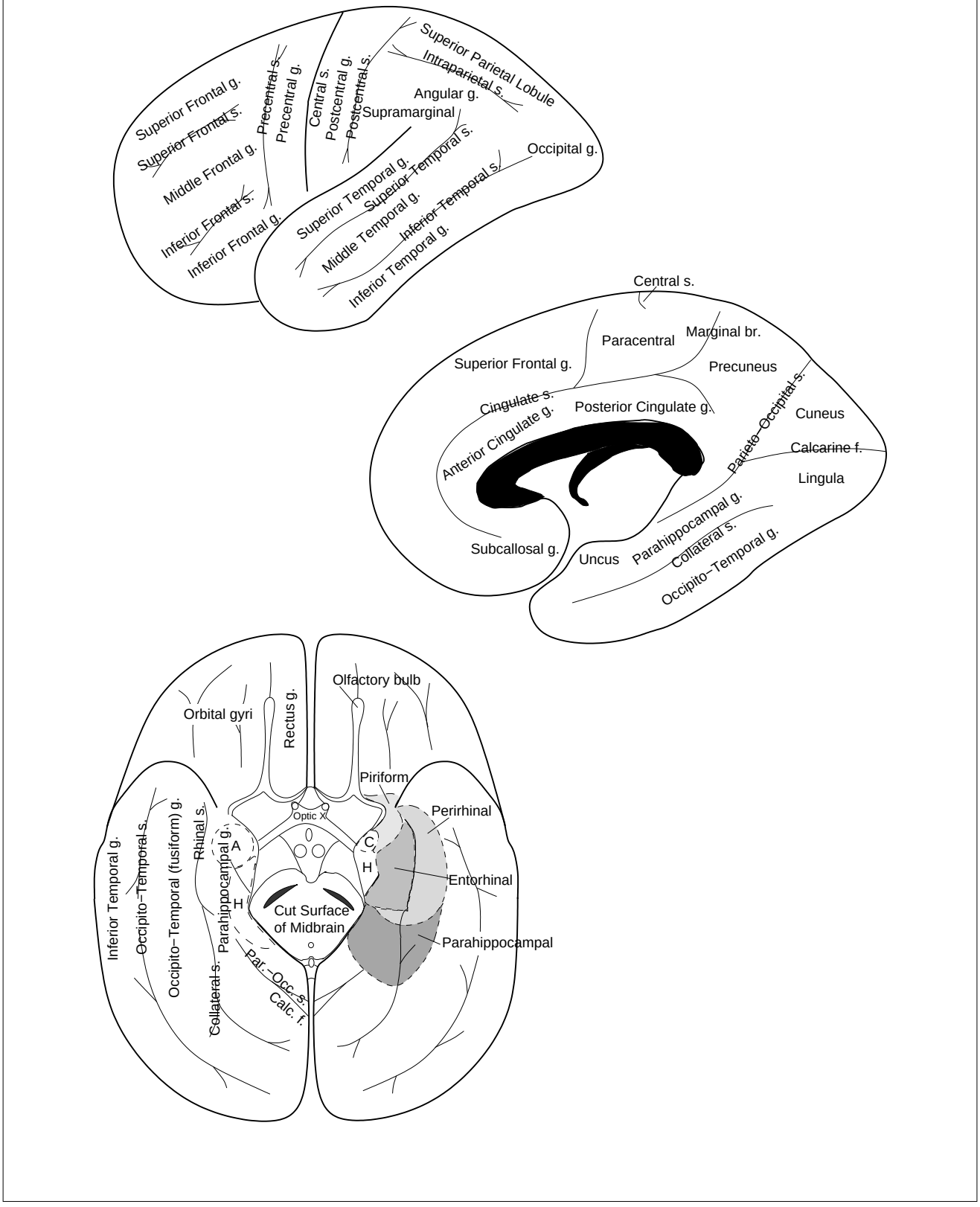


Adapted from Kourouyan, H. D.. and Horton, J. C., 1997, Transneuronal retinal input to the primate Edinger–Westphal nucleus. J. Comp. Neurol. 381:68–80.

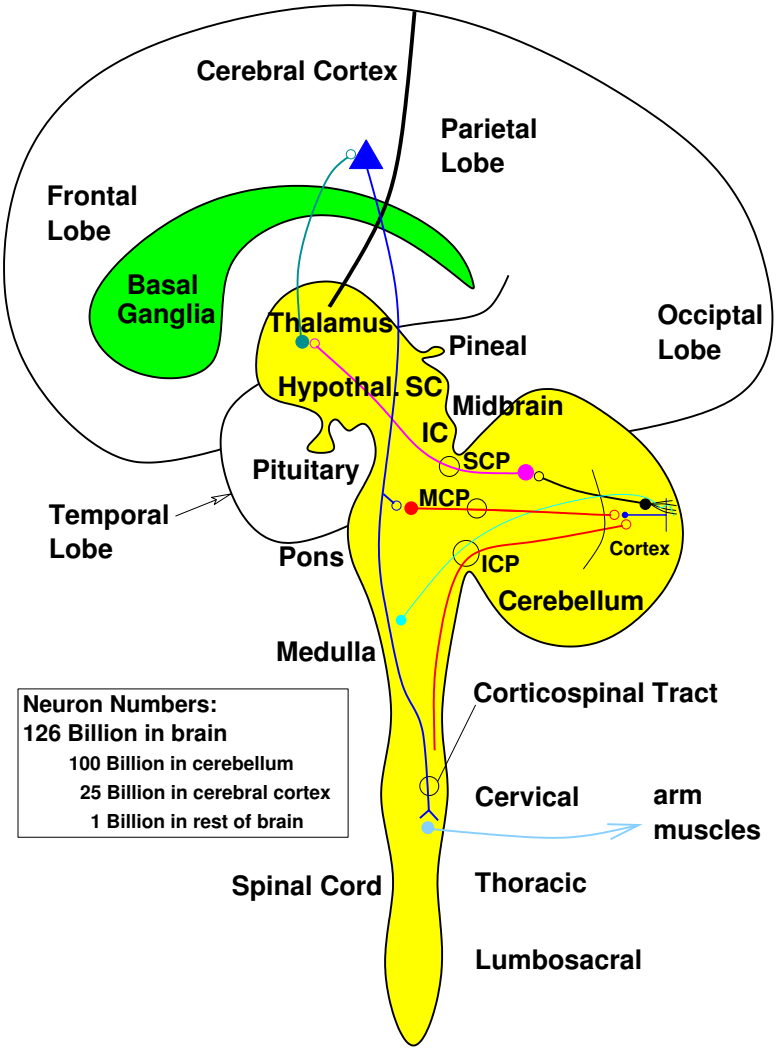
Locus Ceruleus/Raphe



Cortex

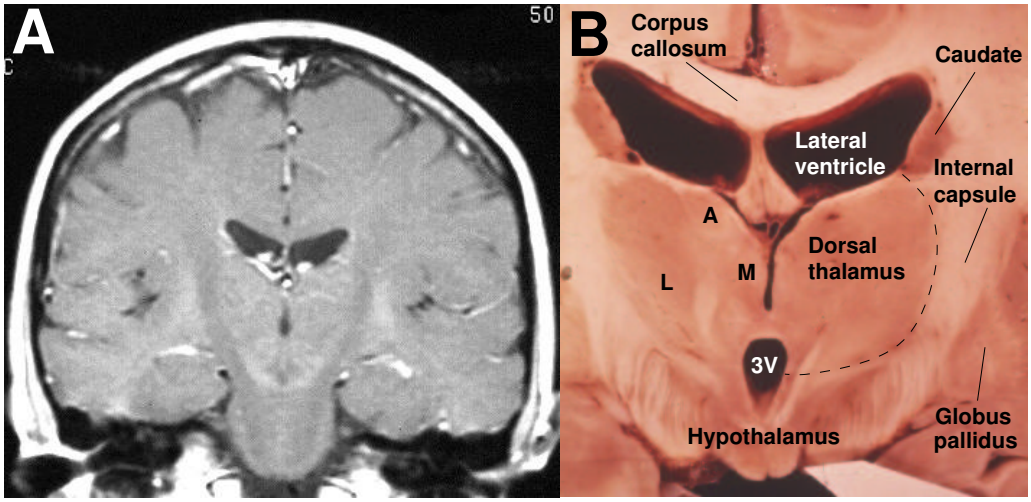

Superior Frontal g.
Superior Frontal s.
Middle Frontal g.
Inferior Frontal s.
Inferior Frontal g.
Precentral s.
Precentral g.
Central s.
Postcentral g.
Postcentral s.
Superior Parietal Lobule
Intraparietal s.
Angular g.
Supramarginal
Occipital g.
Superior Temporal g.
Superior Temporal s.
Middle Temporal g.
Inferior Temporal s.
Inferior Temporal g.
Central s.
Paracentral
Marginal br.
Superior Frontal g.
Precuneus
Cingulate s.
Anterior Cingulate g.
Posterior Cingulate g.
Parieto−Occipital s.
Cuneus
Calcarine f.
Lingula
Subcallosal g.
Uncus
Parahippocampal g.
Collateral s.
Occipito−Temporal g.
Olfactory bulb
Orbital gyri
Rectus g.
Piriform
Optic
Perirhinal
A
C
H
Entorhinal
H
Cut Surface
of Midbrain
Parahippocampal
Inferior Temporal g.
Occipito−Temporal s.
Occipito−Temporal (fusiform) g.
Rhinal s.
Parahippocampal g.
Collateral s.
Pat.−Occ. s.
Calc. f.

Cerebellum

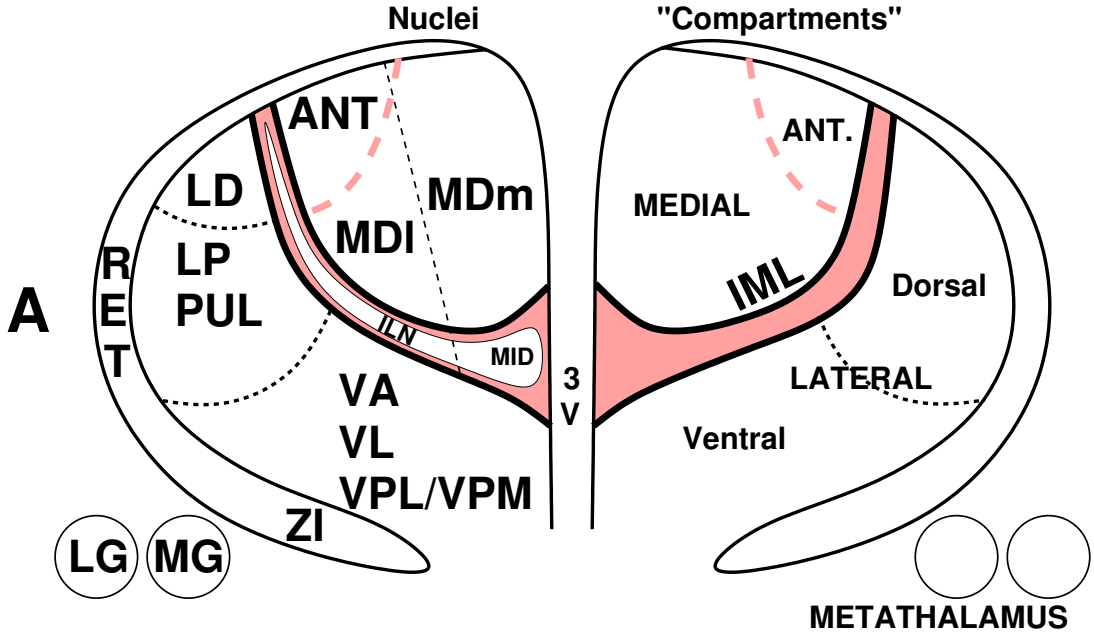


Neuron Numbers:
126 Billion in brain
- **100 Billion in cerebellum**
- **25 Billion in cerebral cortex**
- **1 Billion in rest of brain**

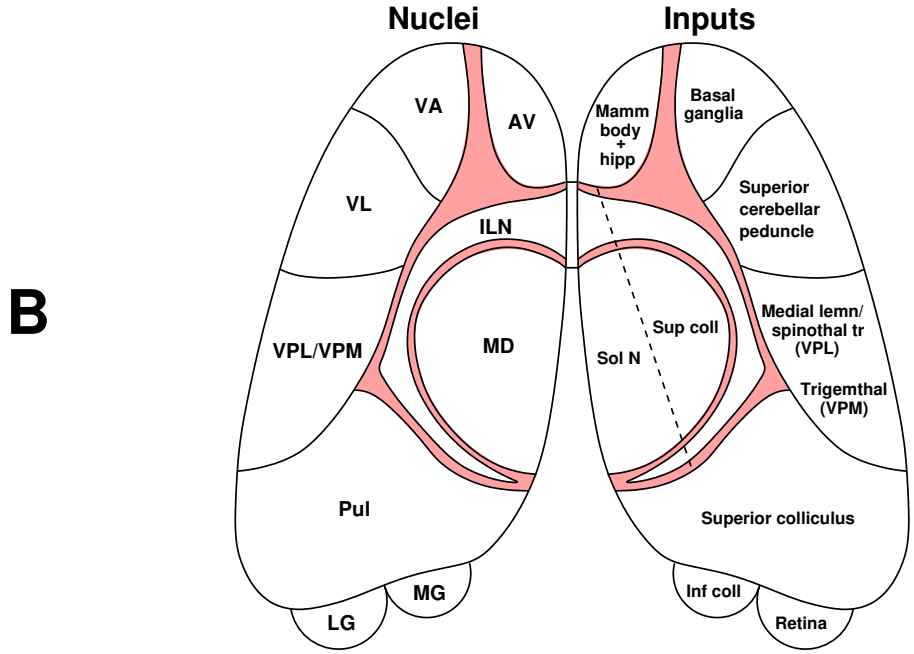
Thalamus


A
50
B
Corpus
callosum
Caudate
Lateral
ventricle
Internal
capsule
A
Dorsal
thalamus
M
L
3V
Hypothalamus
Globus
pallidus

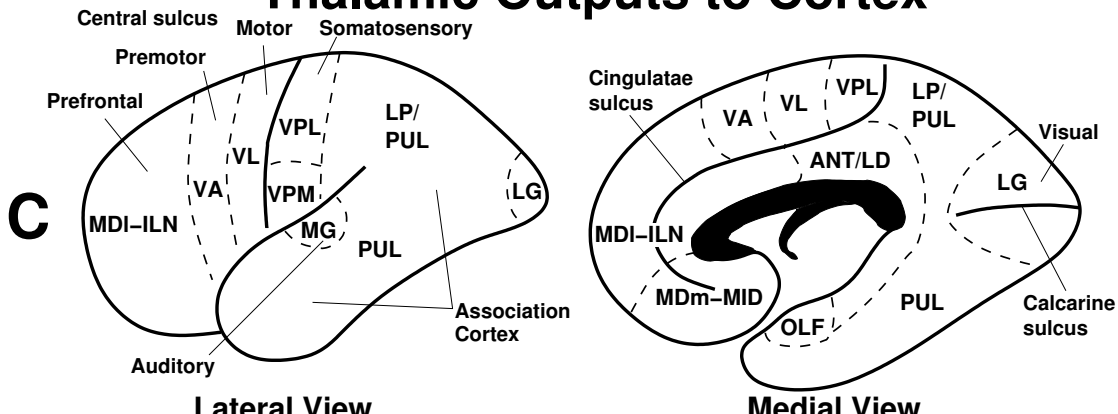
Thalamic Organization on Frontal Section



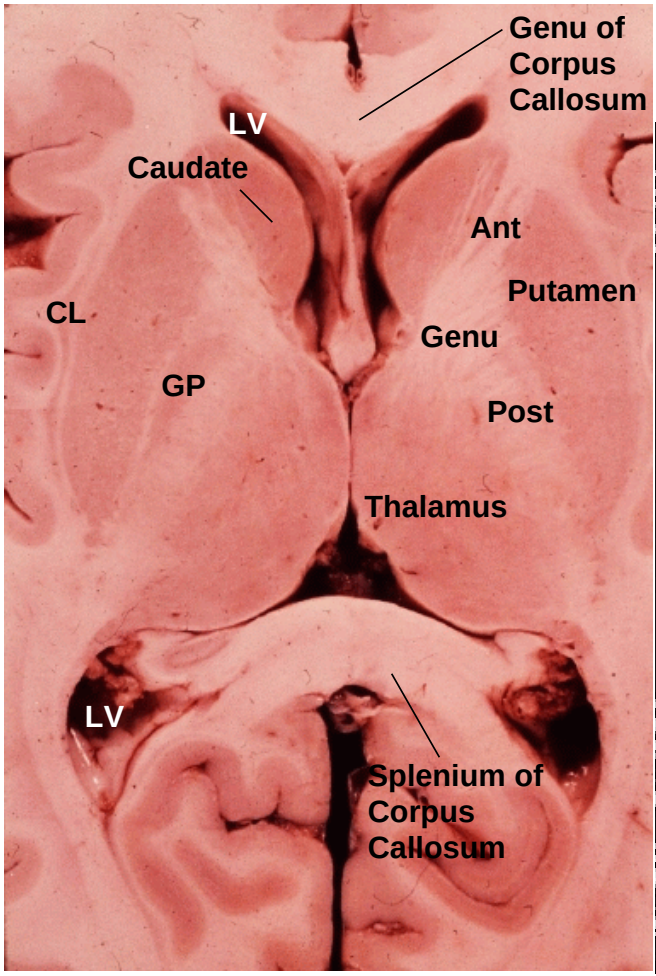
Thalamic Inputs on Horizontal Section

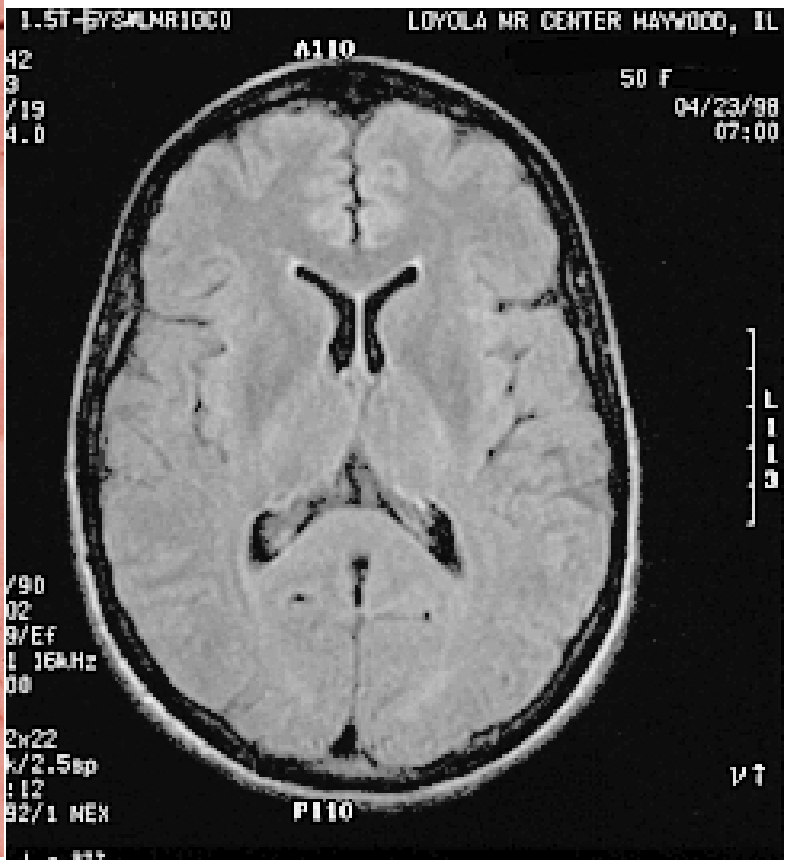


Thalamic Outputs to Cortex

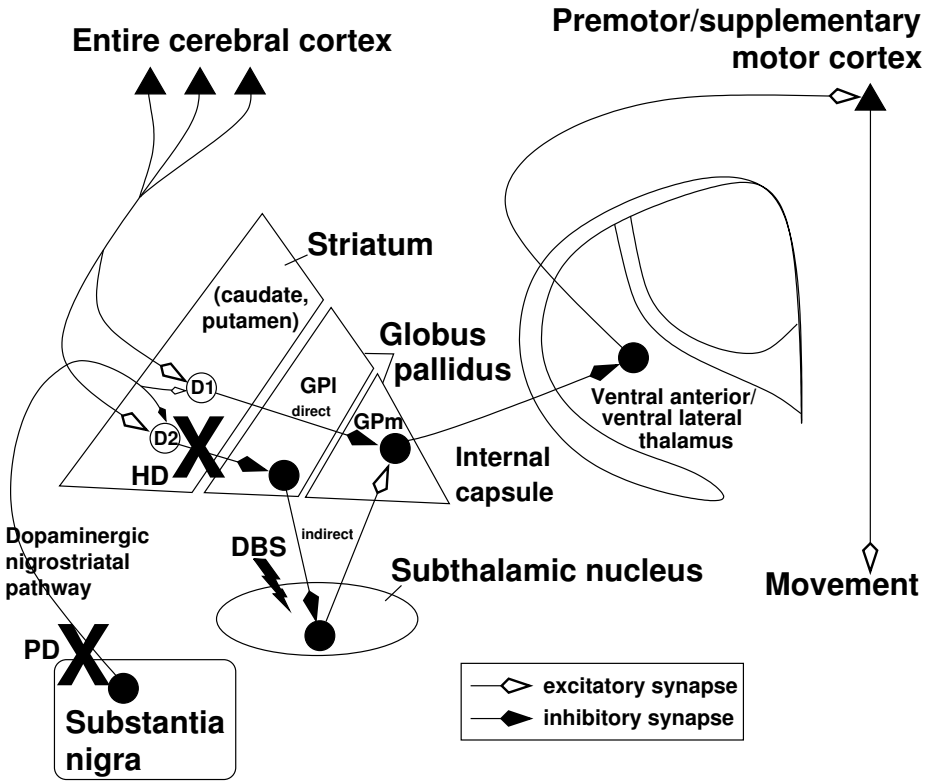


Thal-BG


Genu of
Corpus
Callosum
LV
Caudate
Ant
Putamen
CL
Genu
GP
Post
Thalamus
LV
Splenium of
Corpus
Callosum


LOYOLA MR CENTER HAYWOOD, IL
A110
50 F
04/23/98
07:00
P110

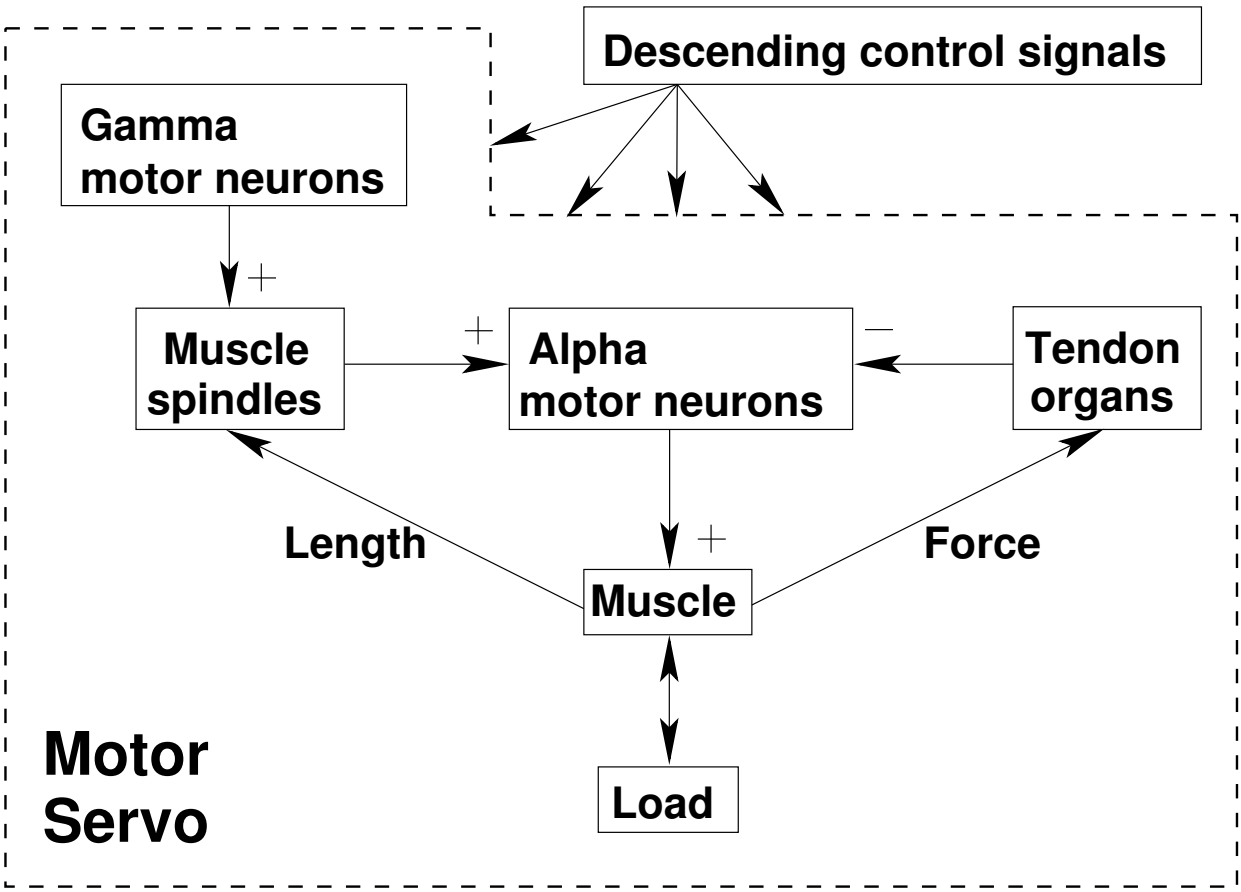
Basal Ganglia Circuitry



Direct Pathway (D1 DA receptor, GABA–SP) facilitates movement.
Indirect Pathway (D2 DA receptor, GABA–ENK) inhibits movement.

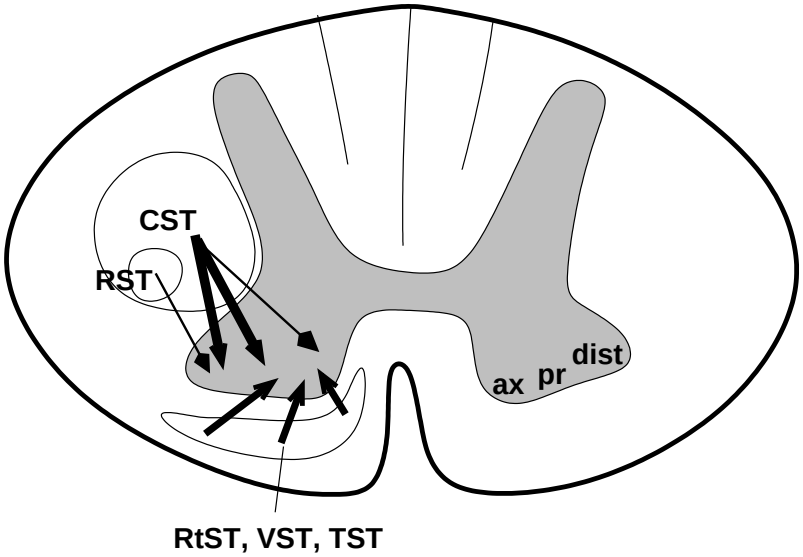
Parkinson's Disease (PD): Loss of nigrostriatal dopamine dysfacilitates Direct Pathway and disinhibits Indirect Pathway, both REDUCING movement (akinesia, bradykinesia). Deep brain stimulation (DBS) of the subthalamic nucleus is a new therapy for PD that reduces excitation of GPm, thereby diminishing its inhibitory output, which relieves akinesia and facilitates movement.

Huntington's Disease (HD): Selective loss of striatal neurons projecting to GPl reduces inhibitory effect of Indirect Pathway, INCREASING movement (chorea).



(Houk, JC and Rymer, WZ 1981. Neural control of muscle length and tension. Handbook of Physiology. Section 1. The Nervous System, Vol. II, Motor Control, Part 1. Am Physiol Soc, Bethesda, pp 257–323)

Motor Pathways

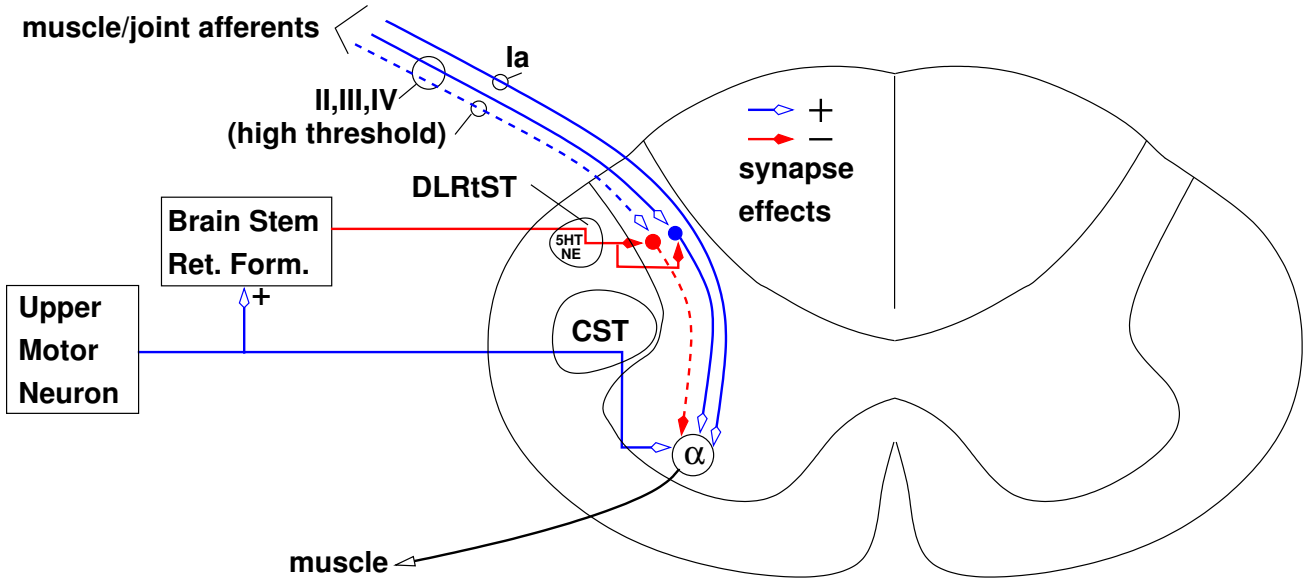


(Lawrence, D.G. and Kuypers, H.G.J.M. The functional organization of the motor system in the monkey, I and II. Brain 91:1−14 and 14−33, 1968.)

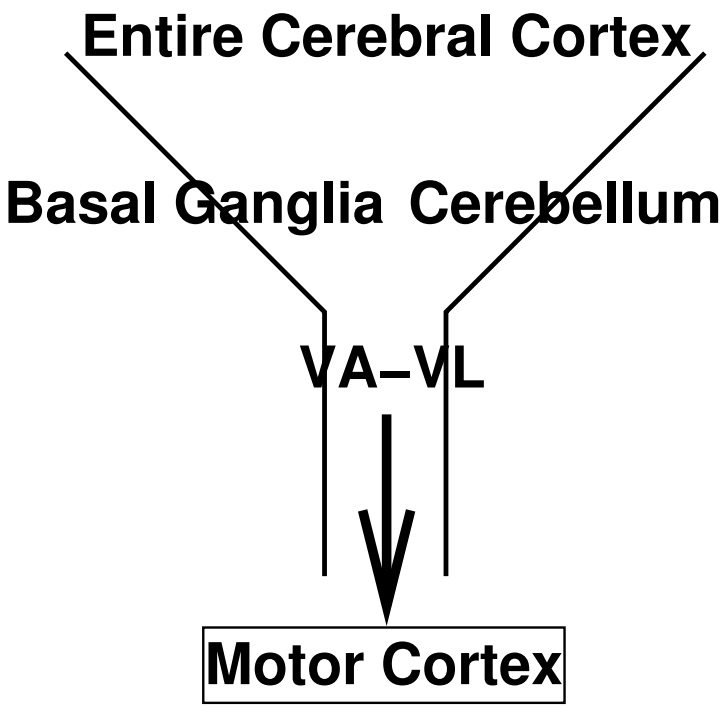
Spasticity = exaggerated stretch reflexes and hypertonia, along with the clasp–knife reflex.

Strictly defined, spasticity is a velocity dependent increase in resistance of a passively stretched muscle that is often associated with a sudden melting of resistance during stretch (clasp knife reflex).

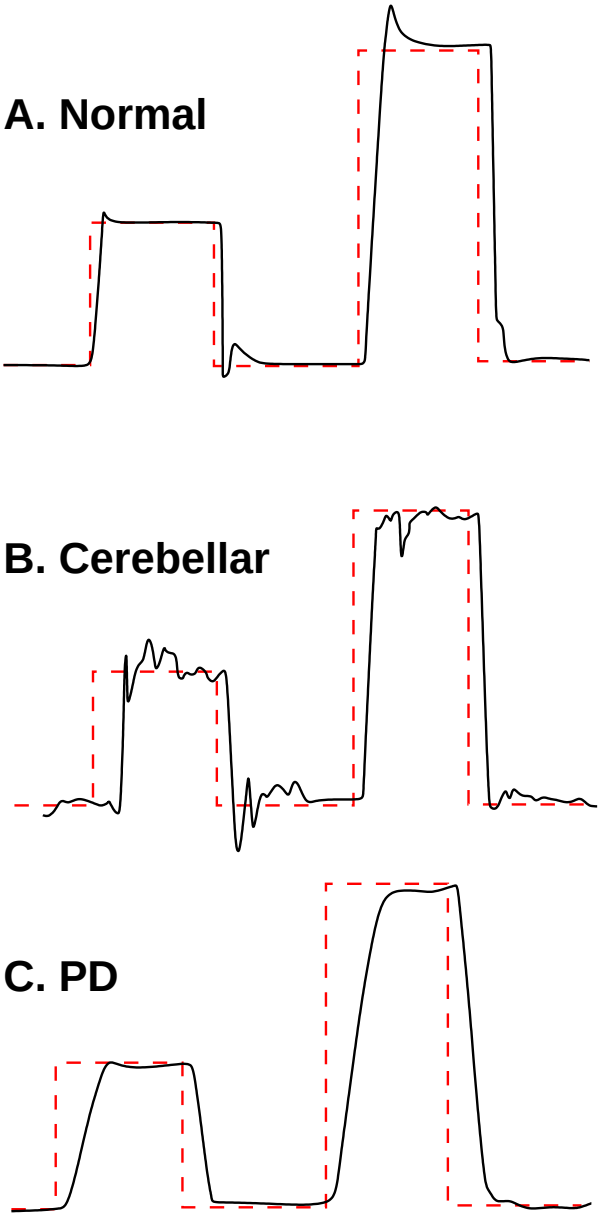
Spasticity is caused by an upper motor neuron lesion that interrupts both the corticospinal tract (CST) and the descending cortical projections to the brain stem reticular formation cells that give rise to the dorsolateral reticulospinal tract (DLRtST). The DLRtST descends in the dorsolateral funiculus and provides tonic inhibition (NE, 5HT) of spinal interneurons activated by Group II, III, and IV afferents. RELEASE from this inhibition causes spasticity's hypertonia and hyperreflexia because of selective facilitation of FR and FF alpha motor neurons. The clasp knife reflex occurs because of loss of inhibition of inhibitory interneurons relaying group II, III, and IV afferent signals activated at relatively high thresholds.



(see Burke et al., Brain 95:31–48, 1972 for more info.)


Entire Cerebral Cortex
Basal Ganglia
Cerebellum
VA–VL
Motor Cortex

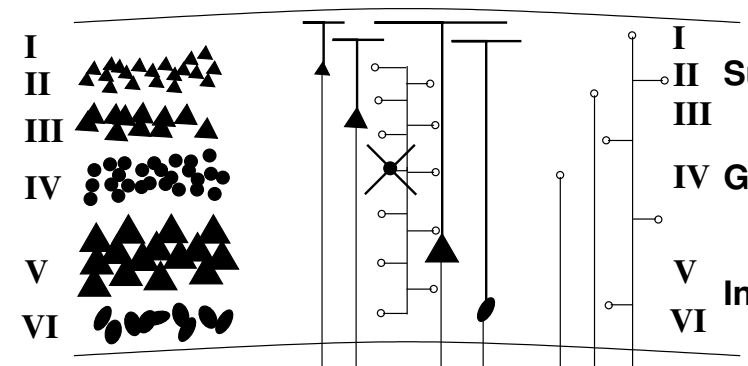
Movement in PD and Cerebellar Damage



Flowers, K. 1975. Neurology 25:413

layers

Cerebral Cortex Layers



I
II Supragranular = Cortico–cortical "Association Cortex"
III
IV Granular = Input or "Sensory Cortex"
V
VI Infragranular = Output or "Motor Cortex"

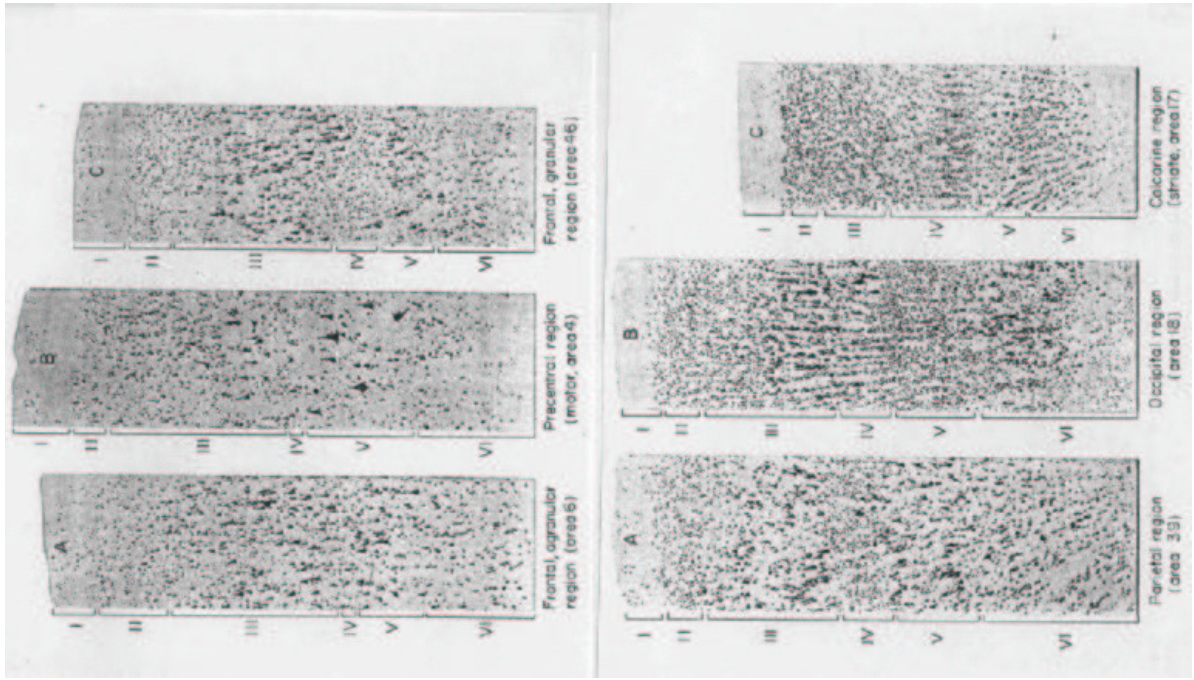
Ipsilateral Cortex
Contralateral Cortex (callosal)
Striatum, Brainstem, Spinal Cord
Thalamus and Claustrum

Non–specific (locus ceruleus, raphe, nucleus basalis)
Cortico–cortical
Thalamus

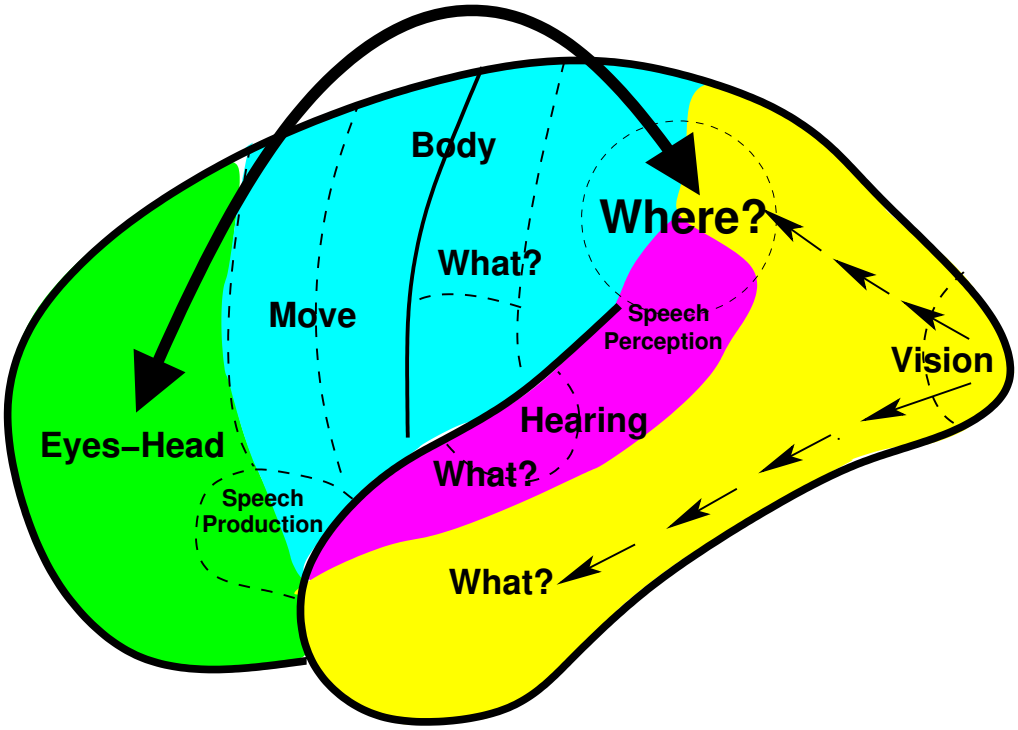
Outputs from cortical layers

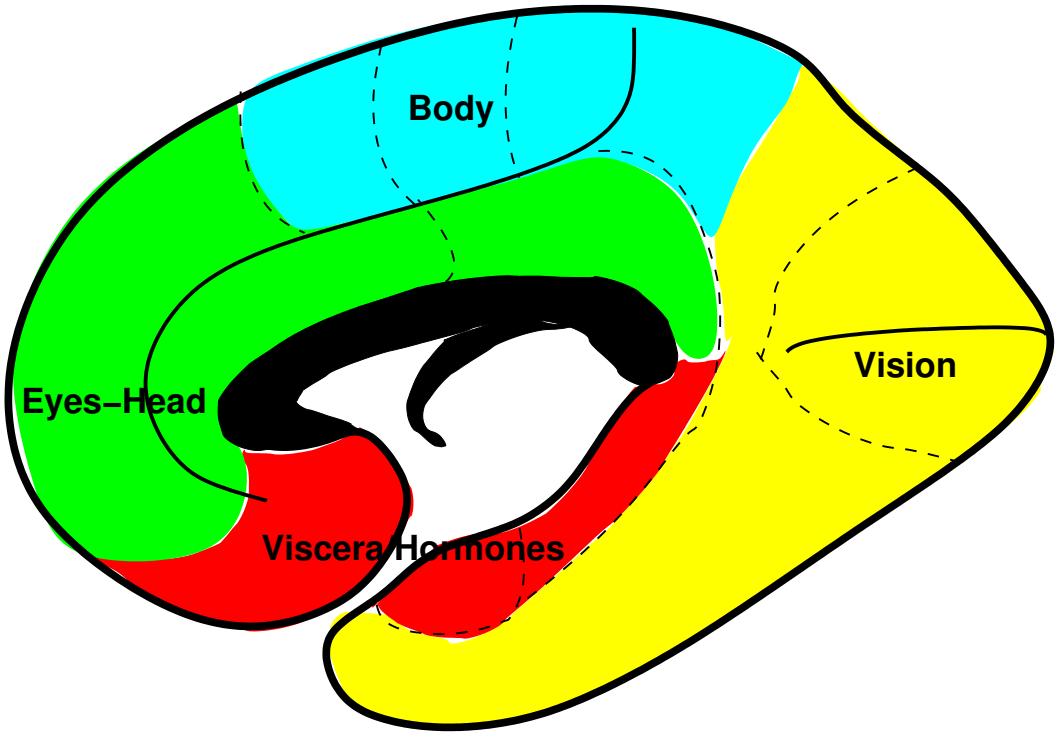
Inputs to cortical layers

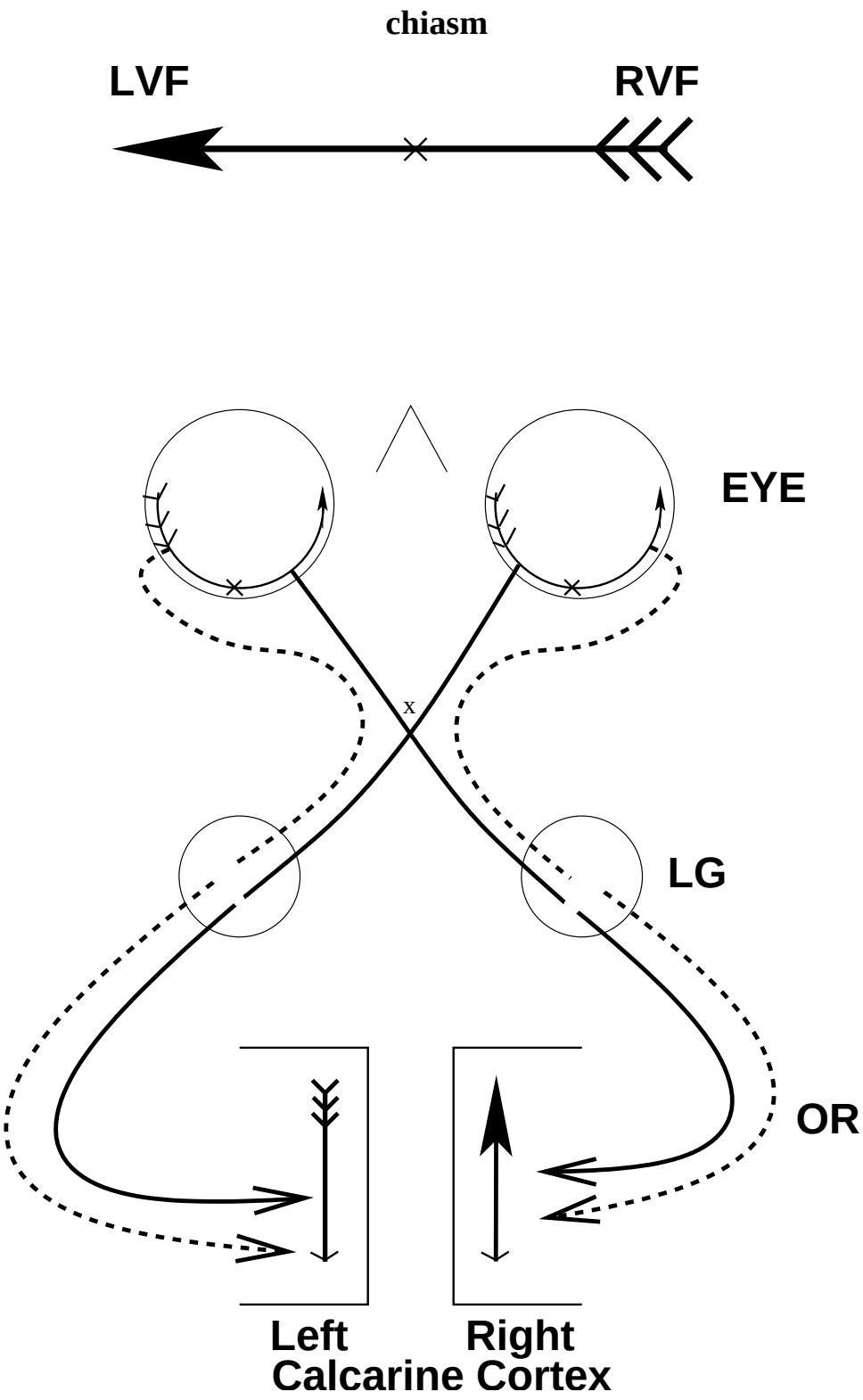
Cerebral Cortex Cytoarchitecture



Cortex Big Picture

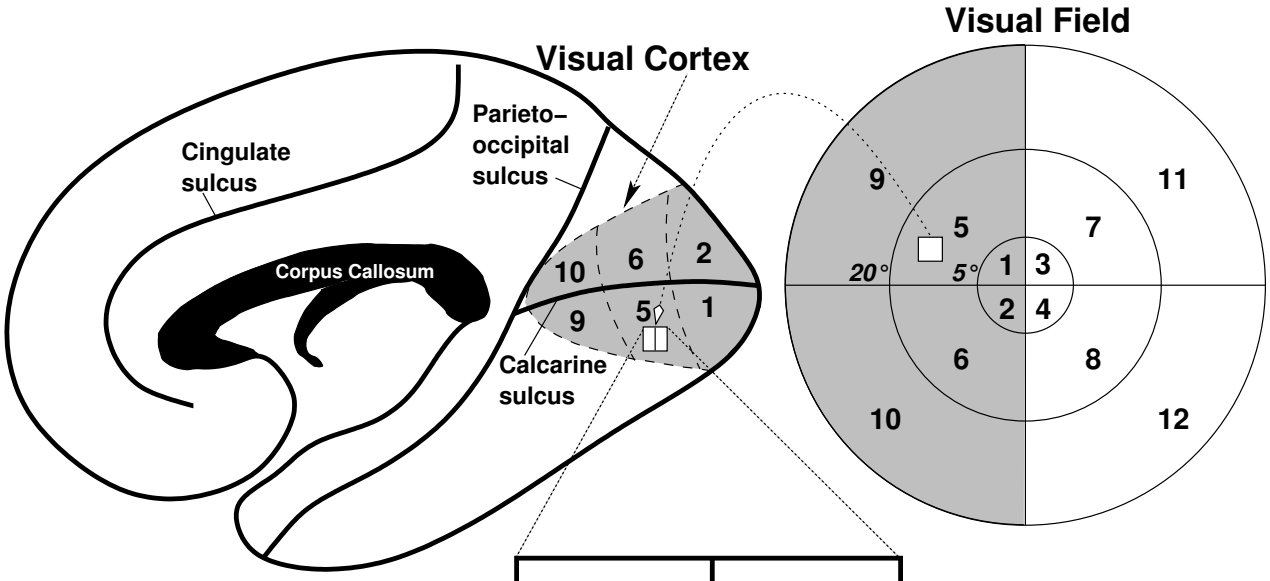




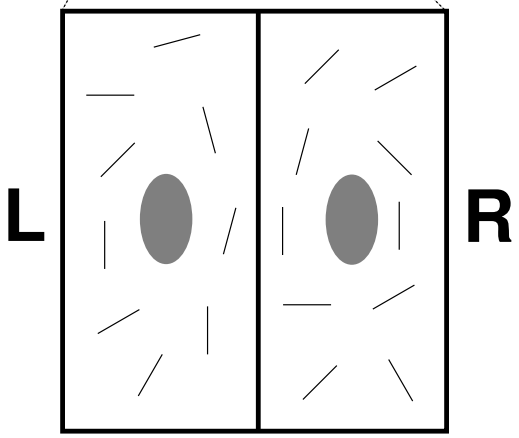

chiasm
LVF
RVF
EYE
x
LG
OR
Left
Right
Calcarine Cortex

Visual Cortex

The upper and lower banks of the calcarine sulcus are the location of the prima visual cortex (area 17). It is organized into a map or "representation" of the contralateral visual field in which the fovea is represented most posteriorly. Note the UPPER hemifield maps to the LOWER bank of the calcarine sulcus.

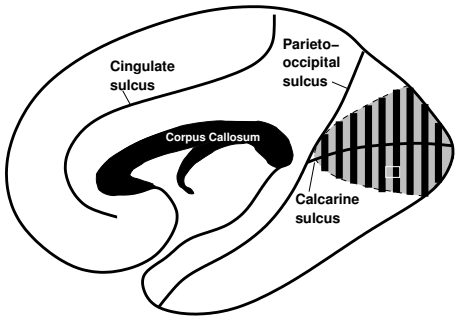


Enlarged view of pial surface of "hypercolumn" that processes visual input from one small area of contralateral visual field. Hypercolumn extends from pia to to white matter.



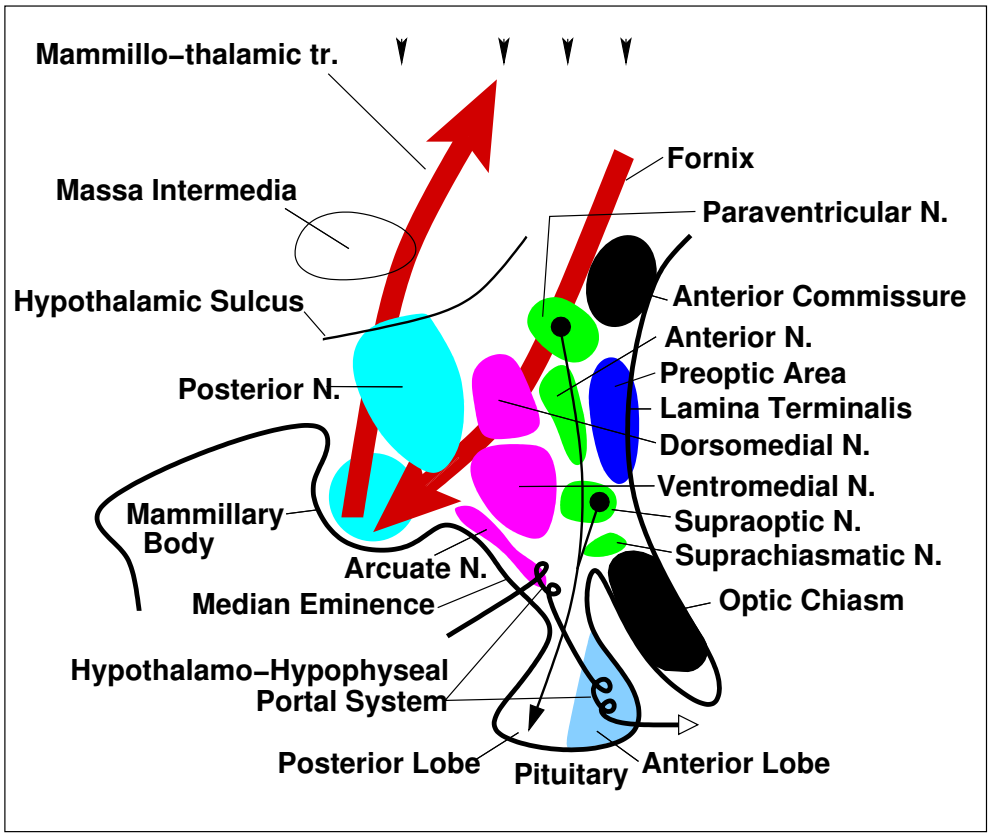
All cells in a hypercolumn respond to same part of visual field, but each hypercolumn can be subdivided into two "ocular dominance columns" in which cells respond more strongly to inputs from left or right eye (L, R). In addition, each hypercolumn can also be subdivided into many "orientation columns" whe cells respond more strongly to lines or edges with a particular orientation (vertic horizontal, or oblique, depicted as short line segments inside hypercolumn). Finally, each hypercolumn also contains color–sensitive "blobs" (shaded ovals) where cells respond to color but not orientation. The visual cortex begins proce making "lines and shapes" out of retinal "points," allowing us to see Seurat's famous painting.

odpattern

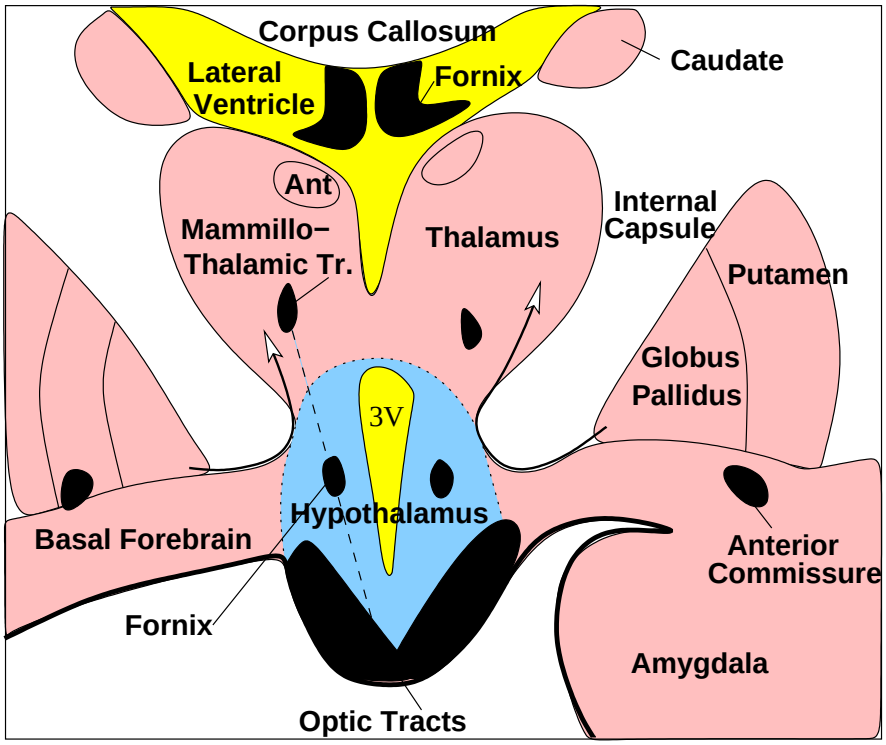


Simplified view of overall pattern of ocular dominance "stripes" in primary visual cortex. Light band represents LEFT eye dominance, and black band represents RIGHT eye dominance. Small white rectangle corresponds to hypercolumn on previous figure. Note that stripes "flow" into calcarine sulcus and then reemerge onto cortical surface.

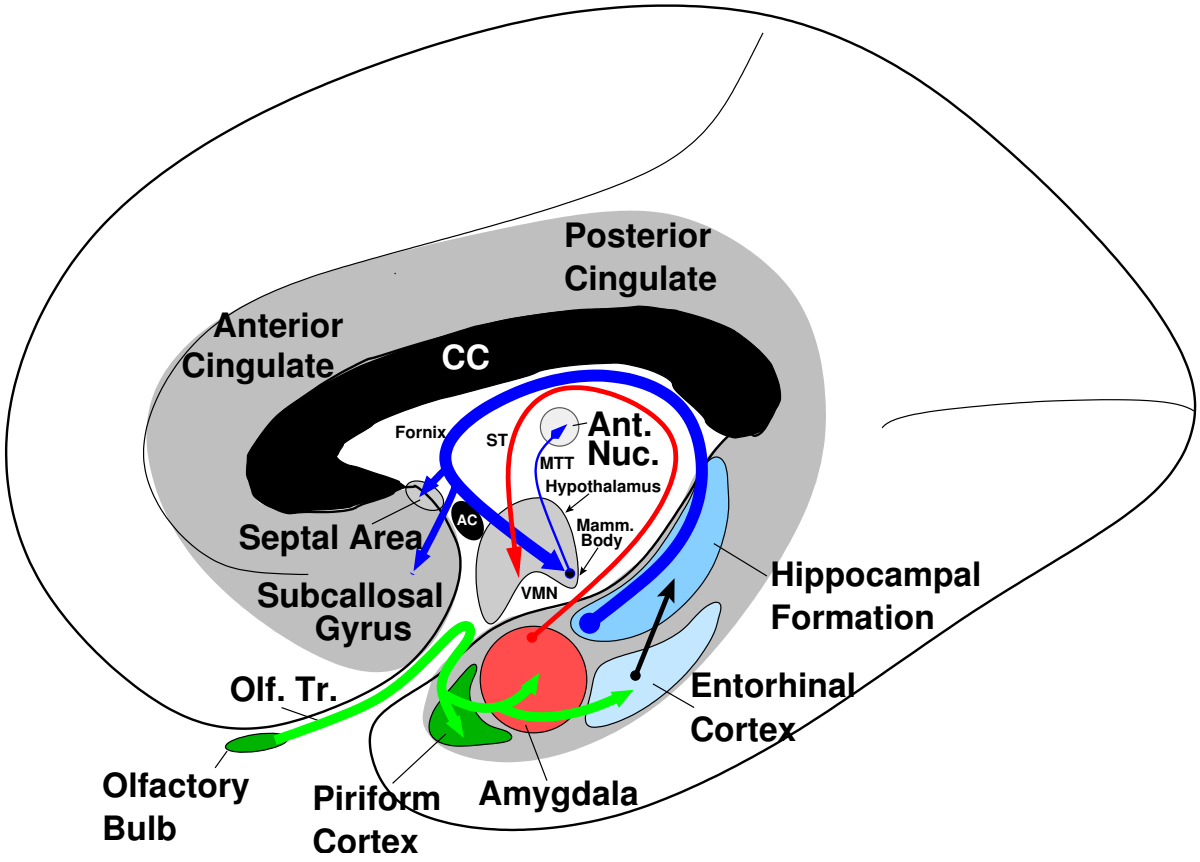
Hypothalamus-Midsag



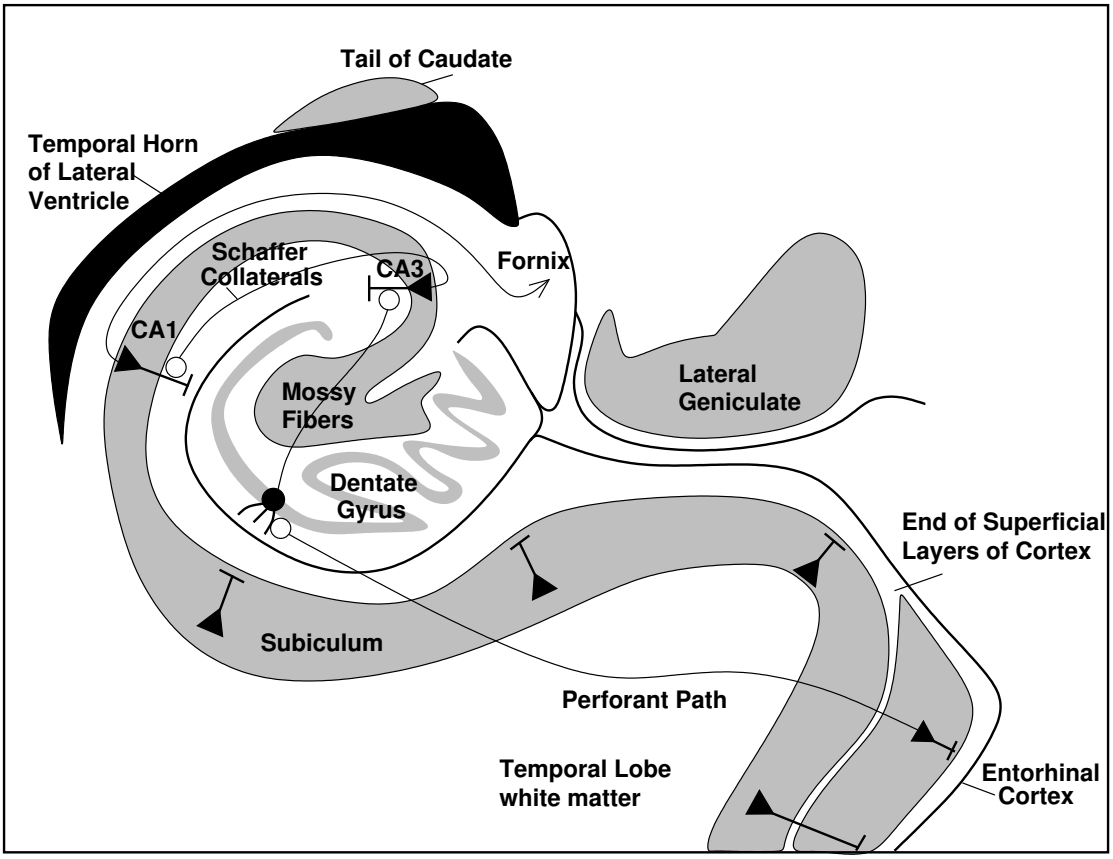
Hypothalamus-Xsec



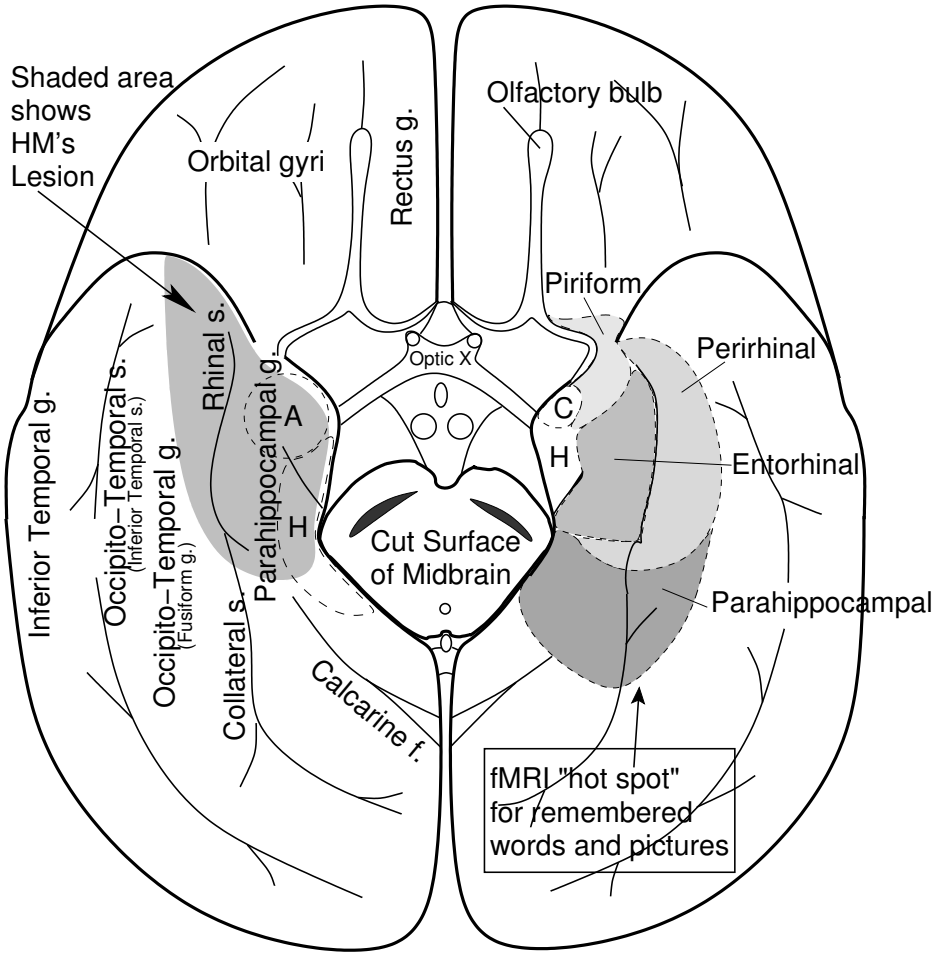
Limbic System



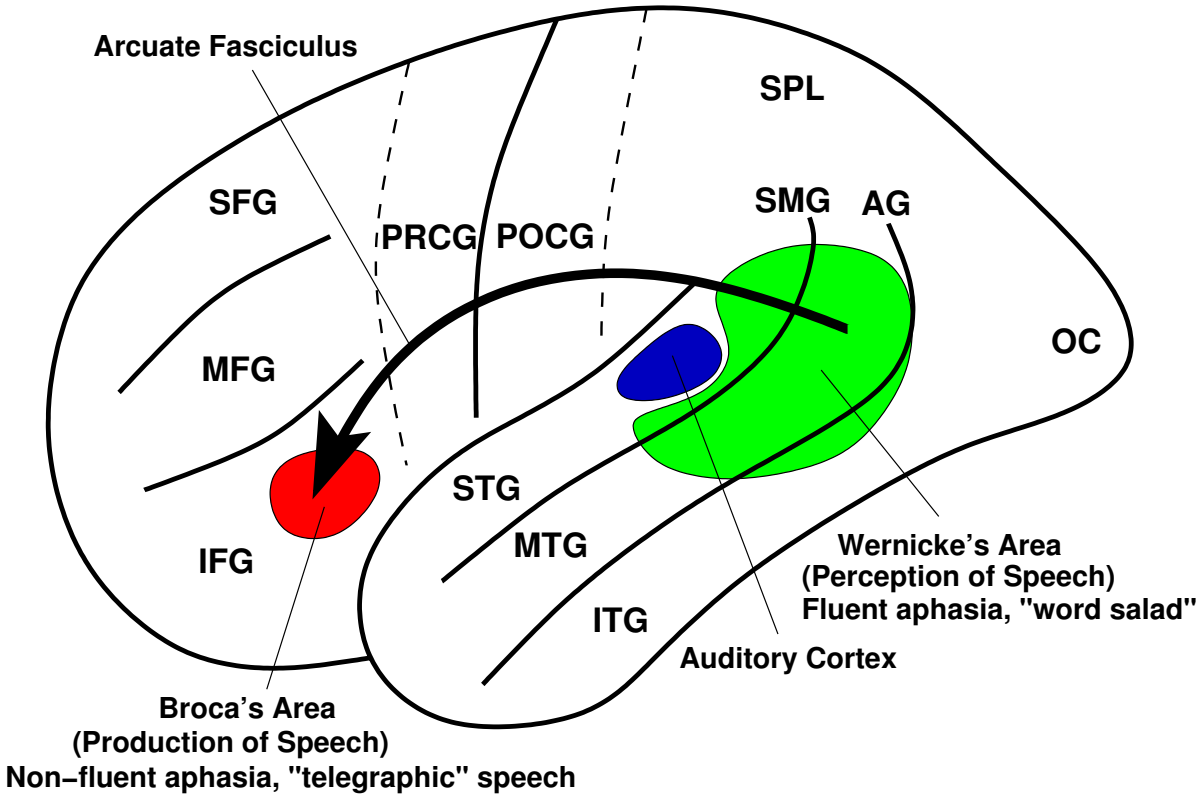
Hippocampal Trisynaptic Pathway



HM


Shaded area shows HM's Lesion
Orbital gyri
Rectus g.
Olfactory bulb
Piriform
Perirhinal
Optic X
C
H
Entorhinal
Rhinal s.
Parahippocampal g.
A
H
Inferior Temporal g.
Occipito–Temporal s. (Inferior Temporal s.)
Occipito–Temporal g. (Fusiform g.)
Collateral s.
Cut Surface of Midbrain
Parahippocampal
Calcarine f.
fMRI "hot spot" for remembered words and pictures

aphasia


Arcuate Fasciculus
SPL
SFG
PRCG
POCG
SMG
AG
OC
MFG
STG
MTG
IFG
ITG
Wernicke's Area
(Perception of Speech)
Fluent aphasia, "word salad"
Auditory Cortex
Broca's Area
(Production of Speech)
Non−fluent aphasia, "telegraphic" speech

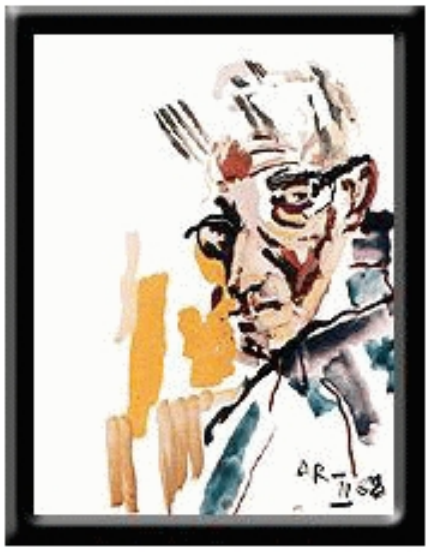
Radersheidt: Contralateral Neglect

Anton Raderscheidt's self–portraits following right parietal cortex damage.

2mo

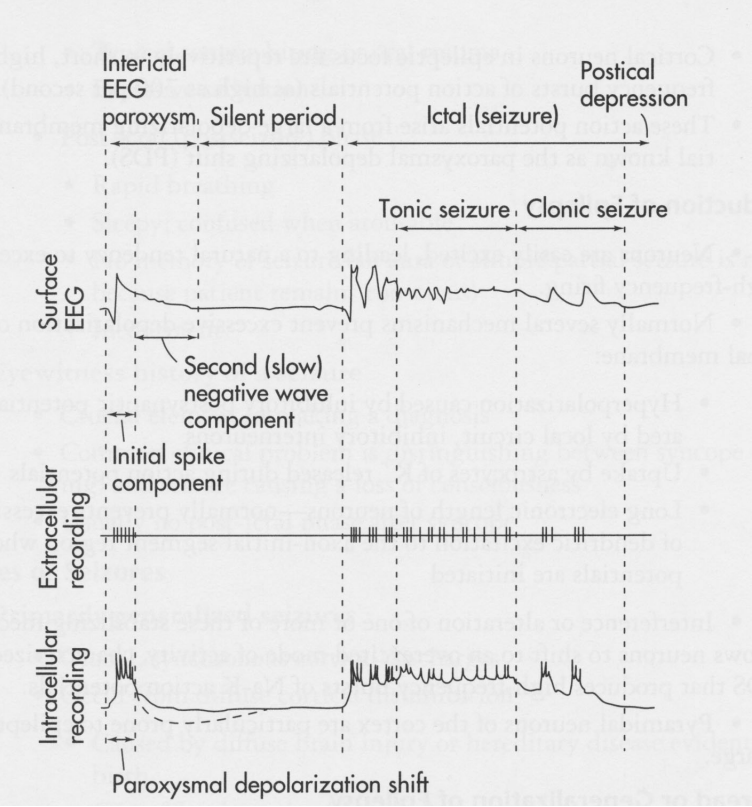
3.5mo

6mo

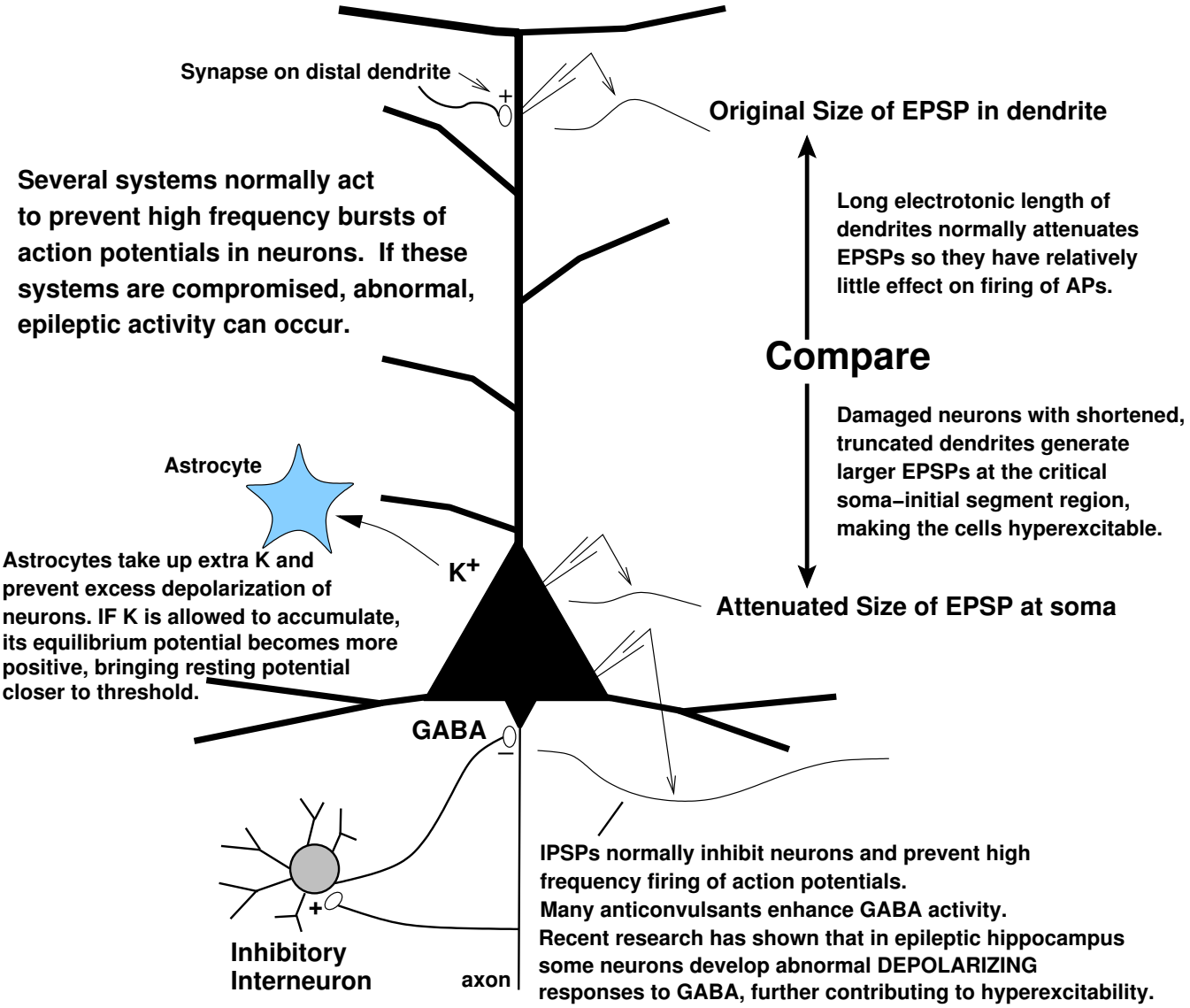


9mo

Epilepsy EEG



Mechanisms



Pyramidal neurons also have a "natural tendency" to fire in high frequency bursts of action potentials. An intrinsic capability to generate long duration, calcium action potentials contributes to this tendency.

Inherited "channelopathies" due to mutations in genes for K, Ca, and Na channels and other membrane proteins also contribute to various types of inherited epilepsies.