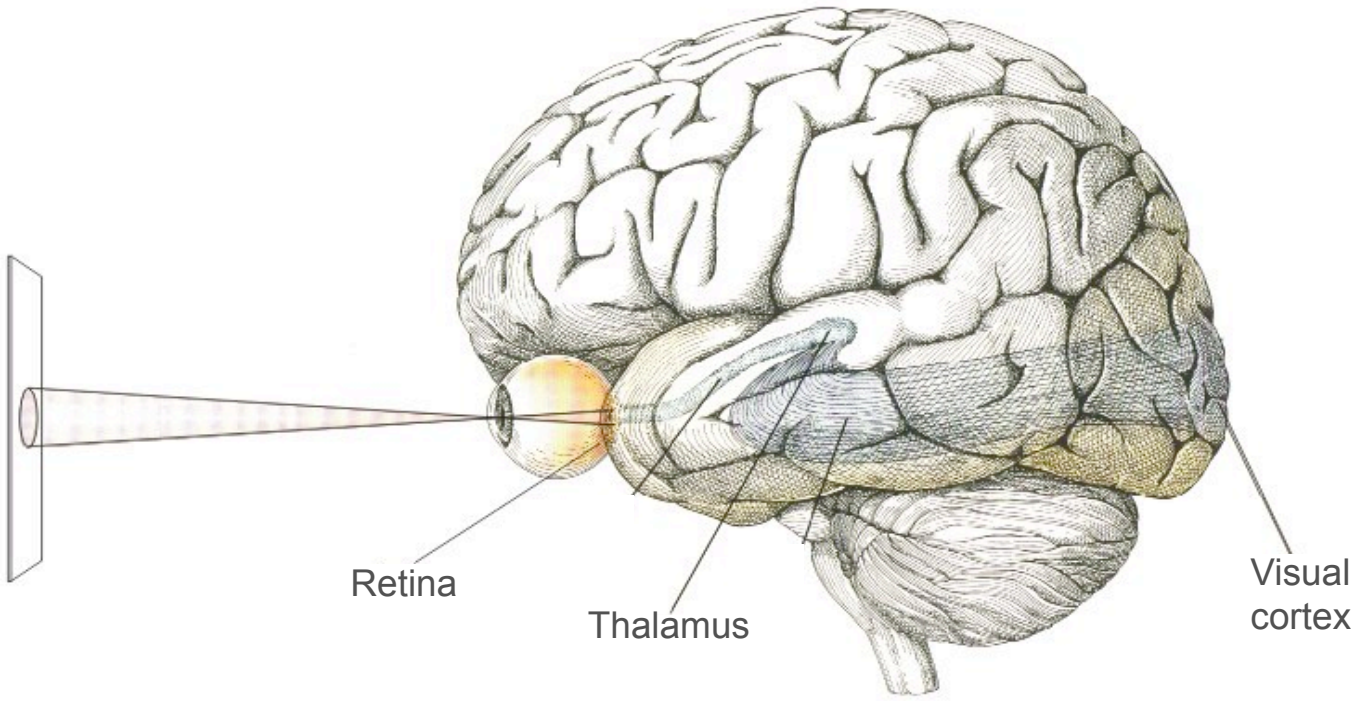
The Lateral Geniculate Nucleus of the Thalamus

(A model for all thalamic ‘relays’)



Why not send projections from retina to visual cortex directly?

2. Vision gone wrong:

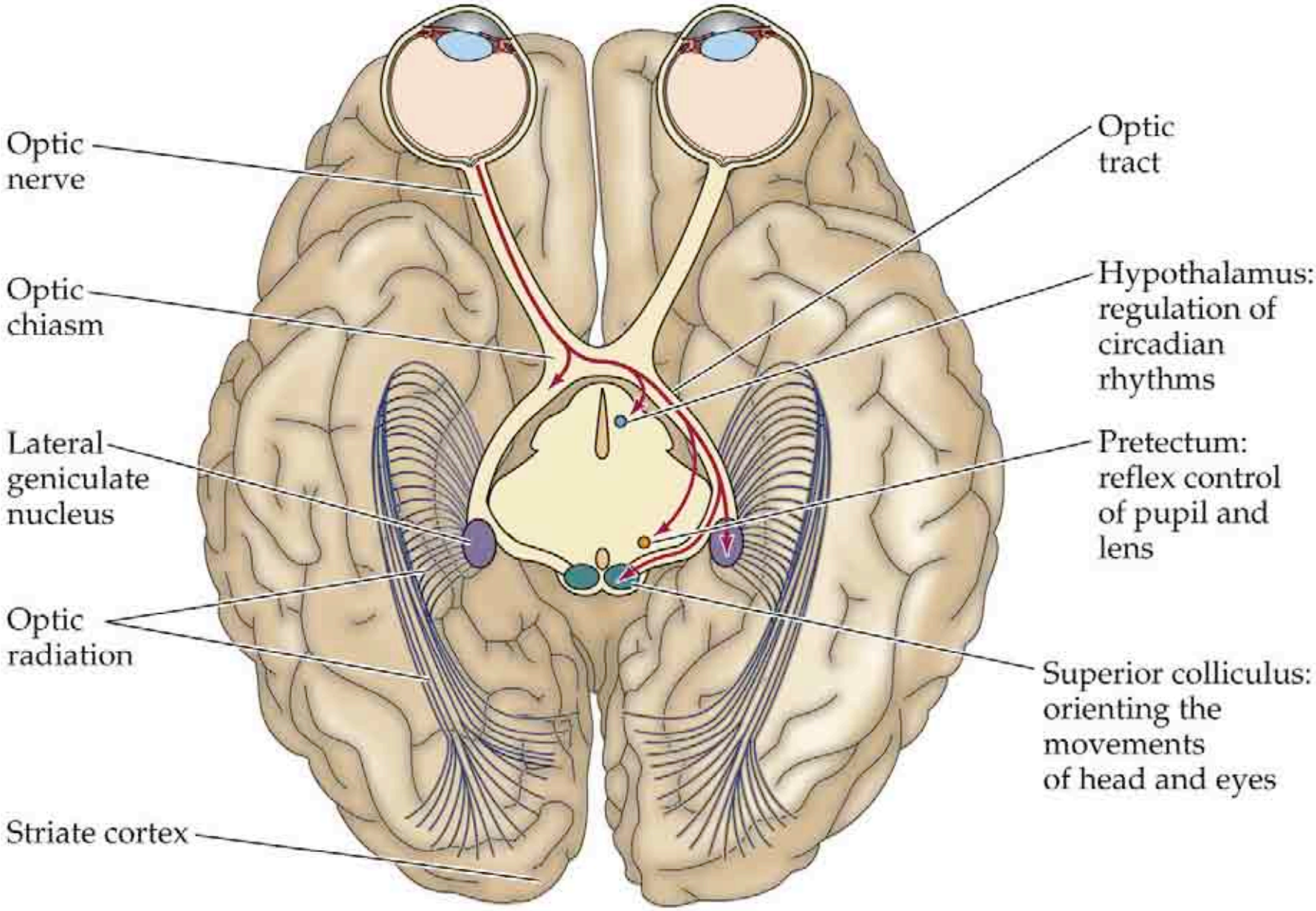
a. Scotoma (lesions along the primary visual pathway from retina through primary visual cortex)

b. Perceptual deficits (motion, depth, places, faces – lesions beyond primary visual cortex)

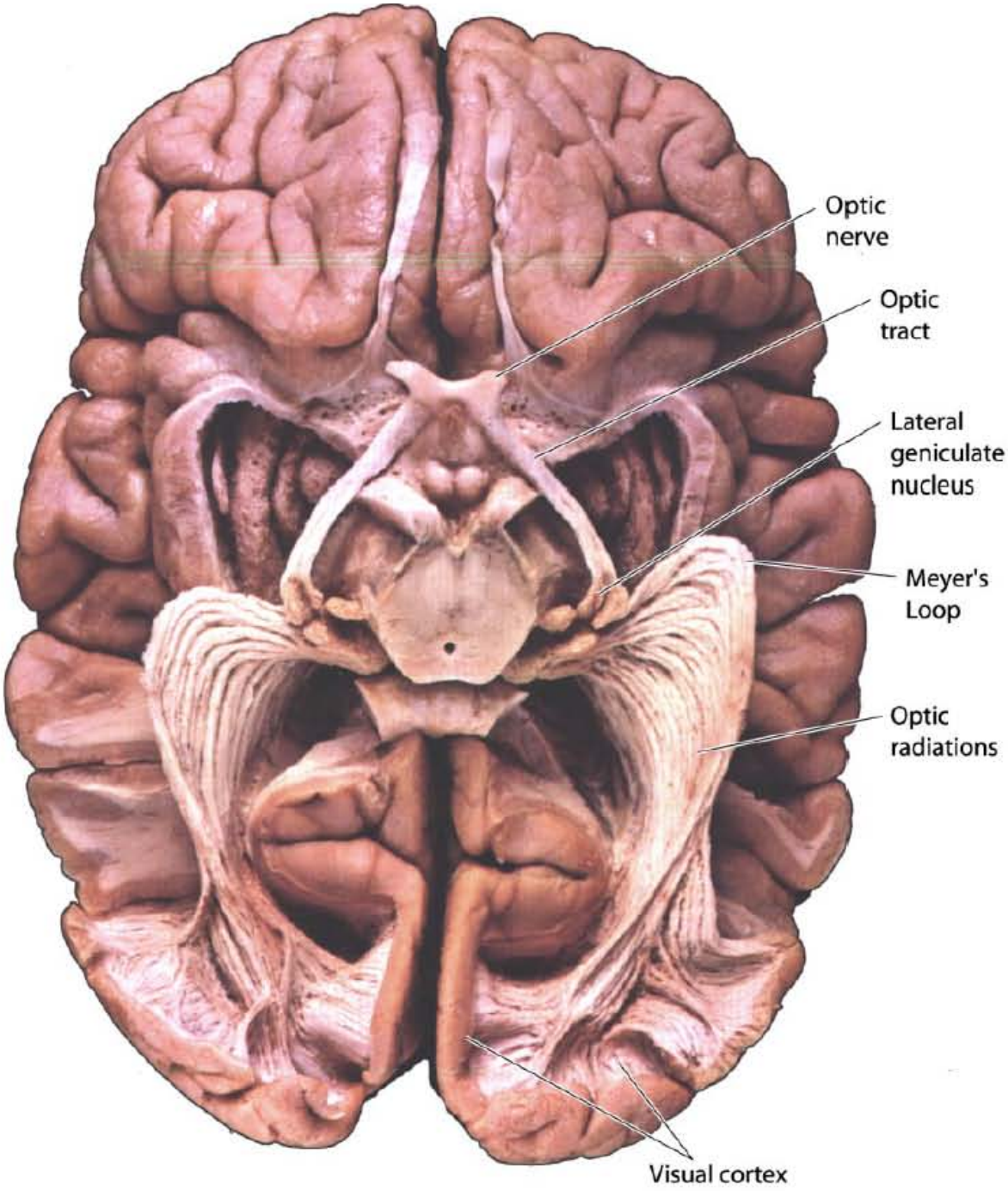
c. Eye movements (saccades, smooth pursuit, vergence, - lesions in SC, FEF, pons, midbrain, SN, cerebellum)

d. Pupil (reflexes – lesions in pretectum, CN III, EW nucl. & "steady state size" – lesions in hypothalamus, RF, T1-T3)

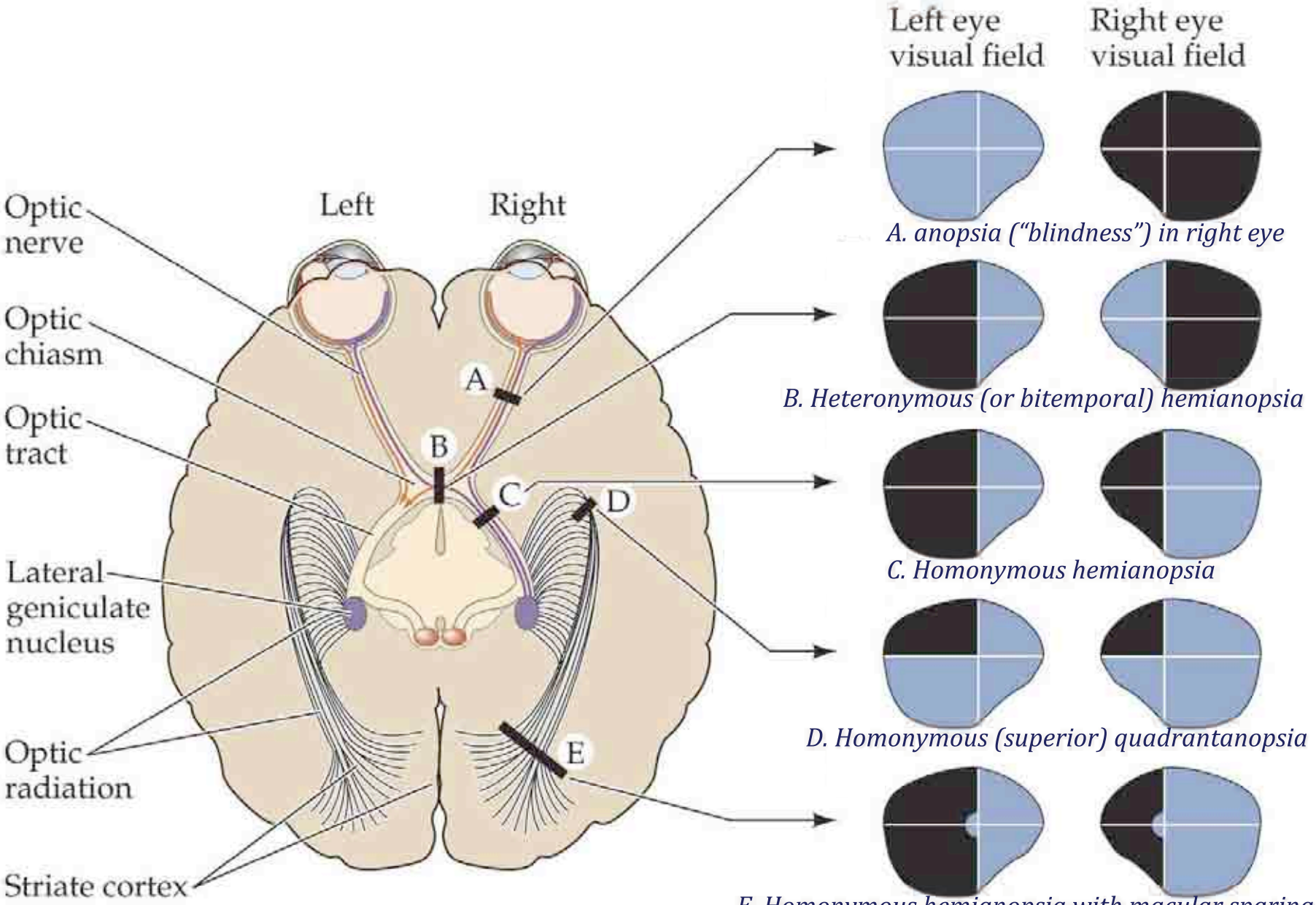
Figure 12.1 Central projections of retinal ganglion cells



***NEUROSCIENCE, Fourth Edition*, Figure 12.1**


Optic nerve
Optic tract
Lateral geniculate nucleus
Meyer's Loop
Optic radiations
Visual cortex

Visual Field Deficits



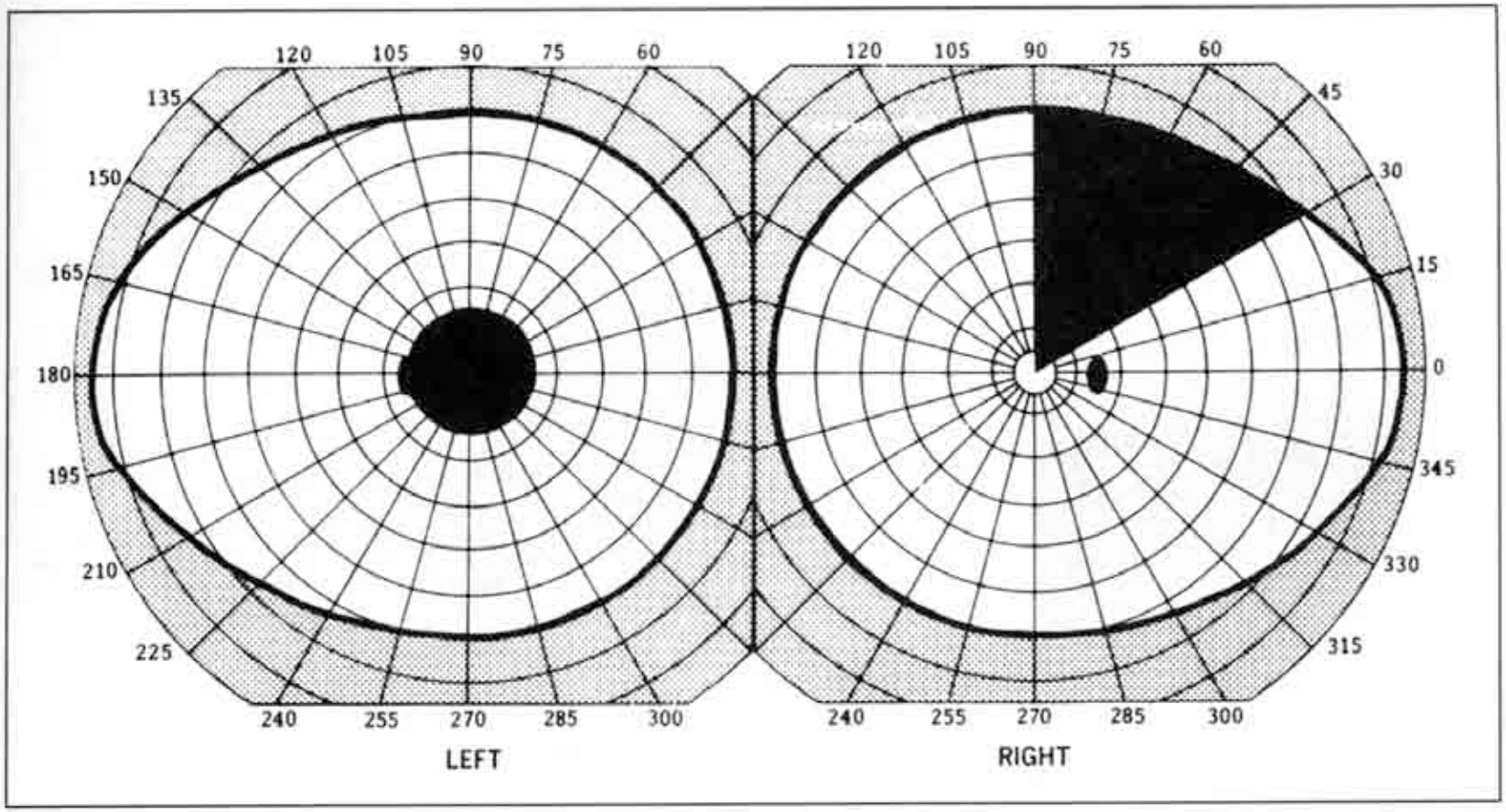
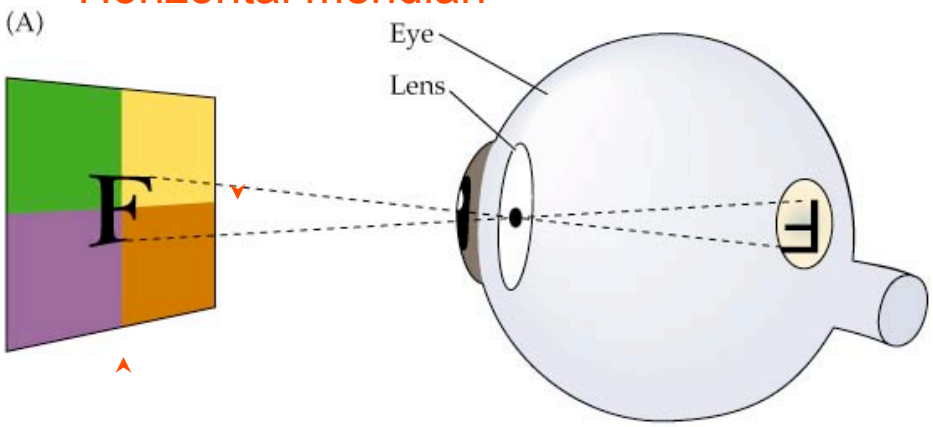


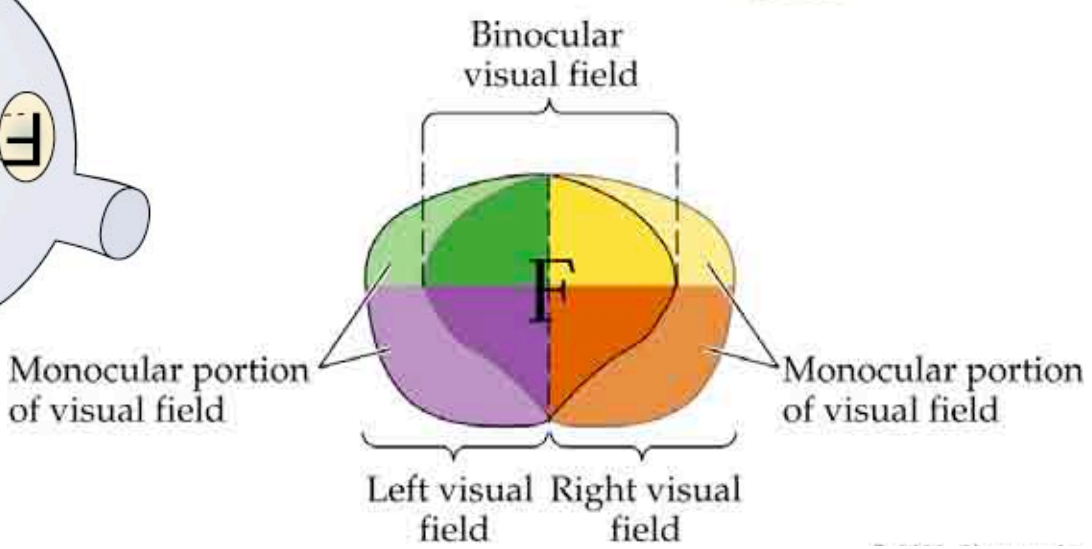
Figure 1-12. Junctional scotoma: a central scotoma in one eye with a superior-temporal defect in the fellow eye; indicates a lesion at the junction of the optic nerve (left eye in this case) and the chiasm.

Horizontal meridian

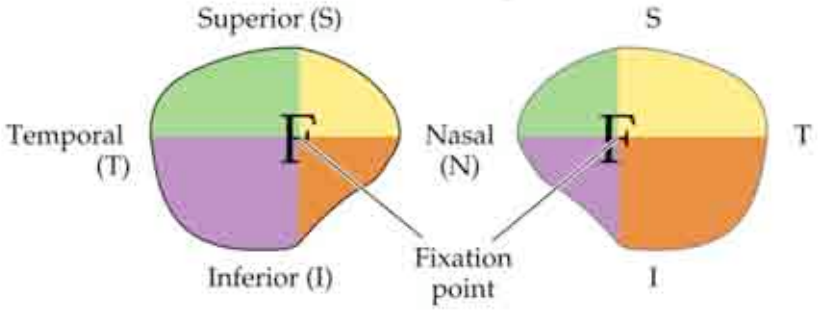


Vertical meridian

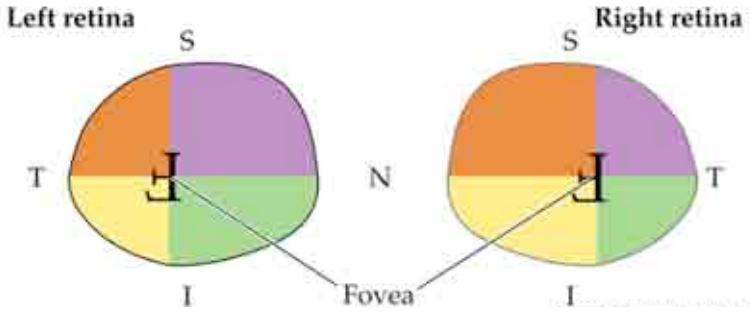
Inversion and reflection



Visual field

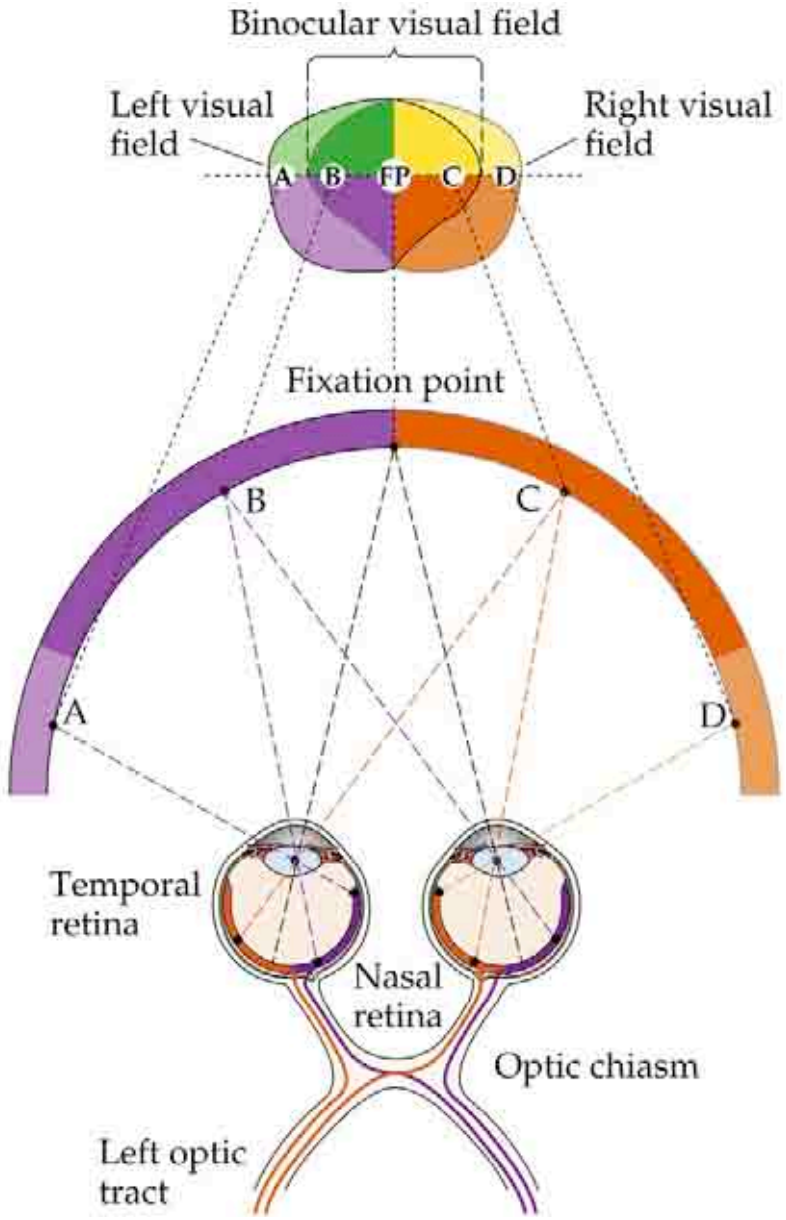


Projections of visual field onto retinas



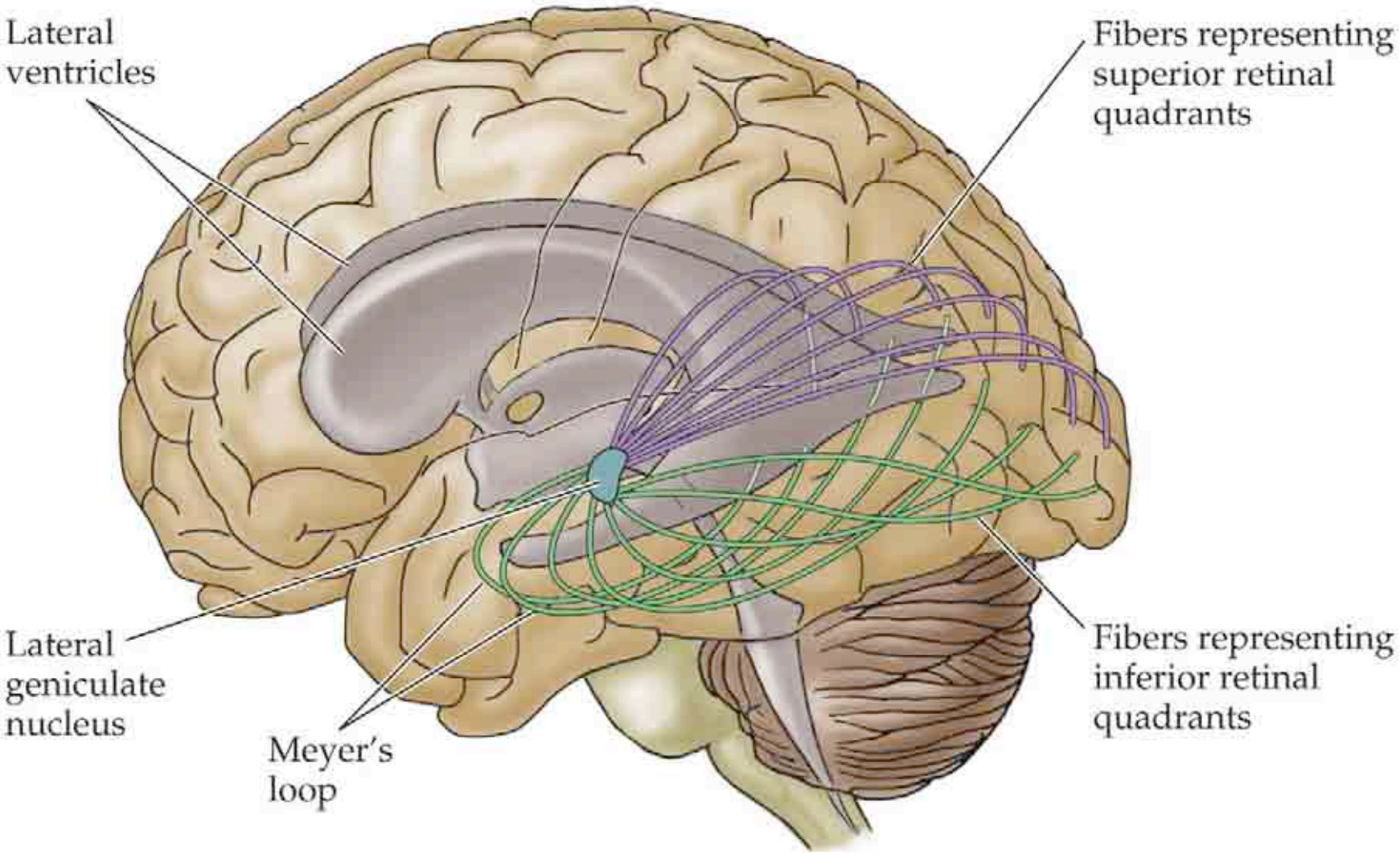
Each eye sees portions of both visual hemifields

Each optic tract represents the contralateral visual hemifield



(D-C-Fp) (Fp-B-A)

Visuotopy in the Optic Radiations



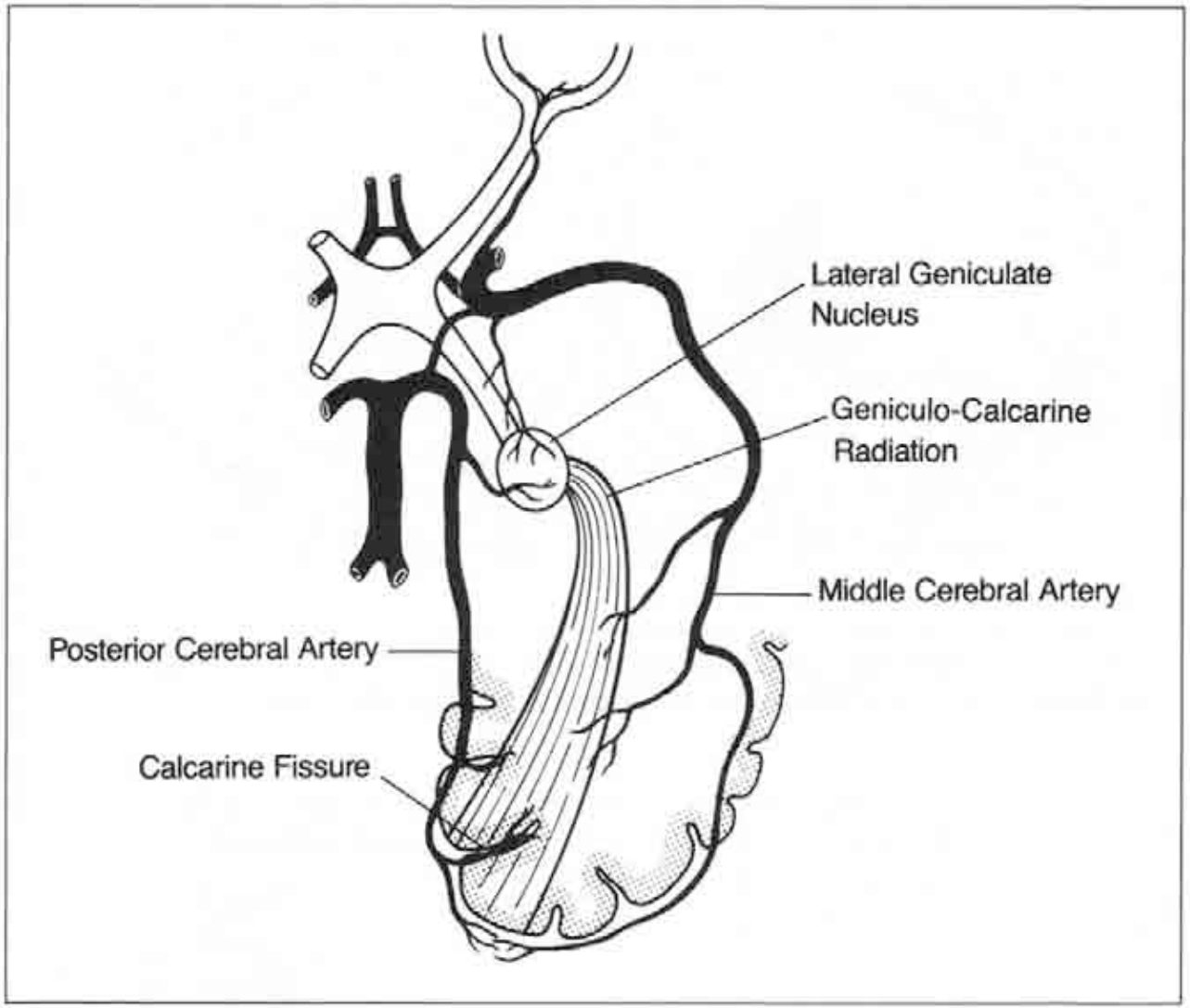


Figure 1-19. The tip of the occipital lobe, where the macular or central homonymous hemifields are represented (see Figure 1-4), is supplied by terminal branches of the middle and posterior cerebral arteries; it is referred to as a watershed area. The mesial surface of the occipital lobe is supplied by more proximal (not terminal) branches of the posterior cerebral artery.

Optic tract has
Three (+1) main targets:
1. Pretectum
2. SC
3. LGN

(4. hypothalamus – light Sensing ganglion cells, So circadian clock resetting is intact in photoreceptor blindness)

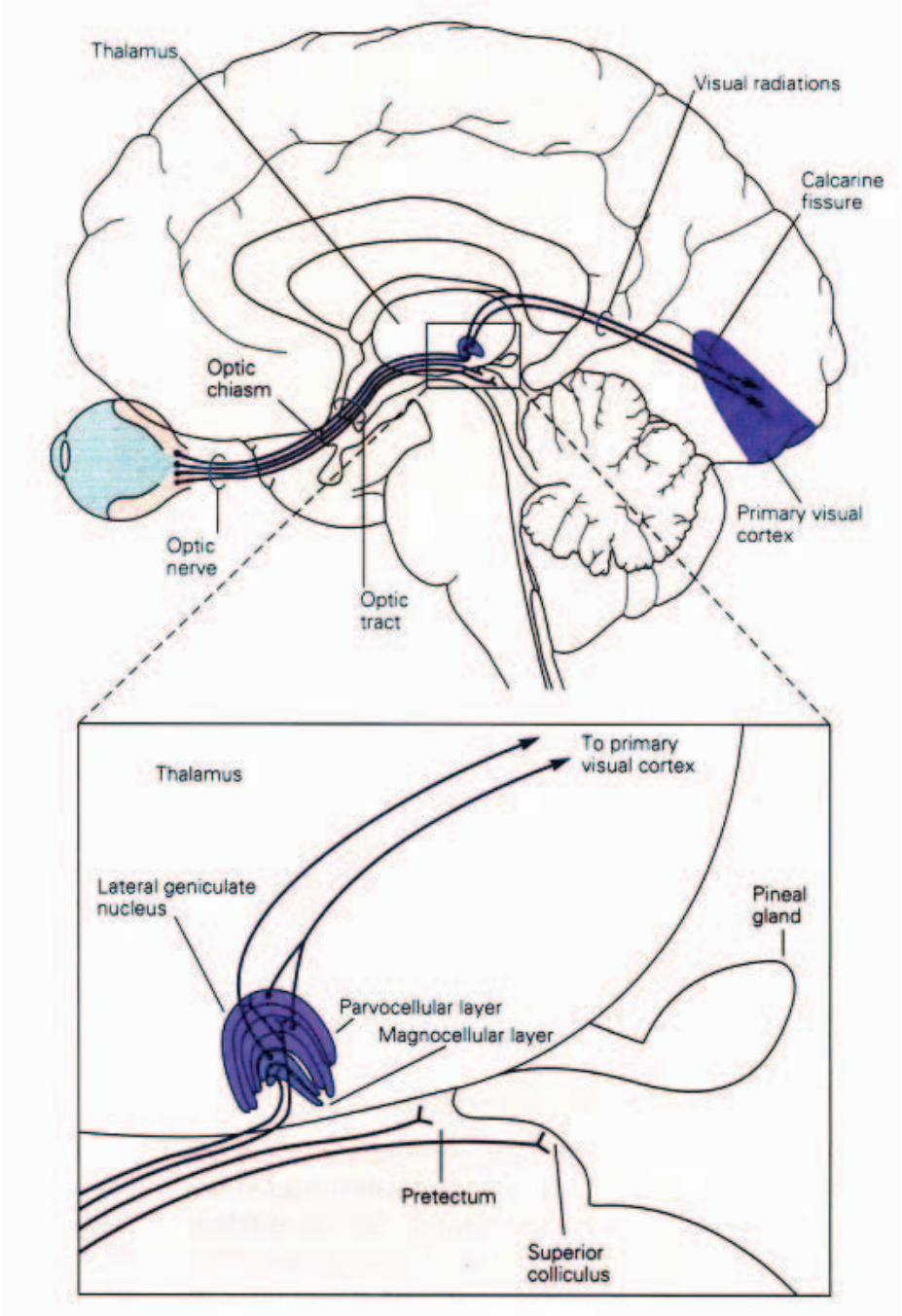
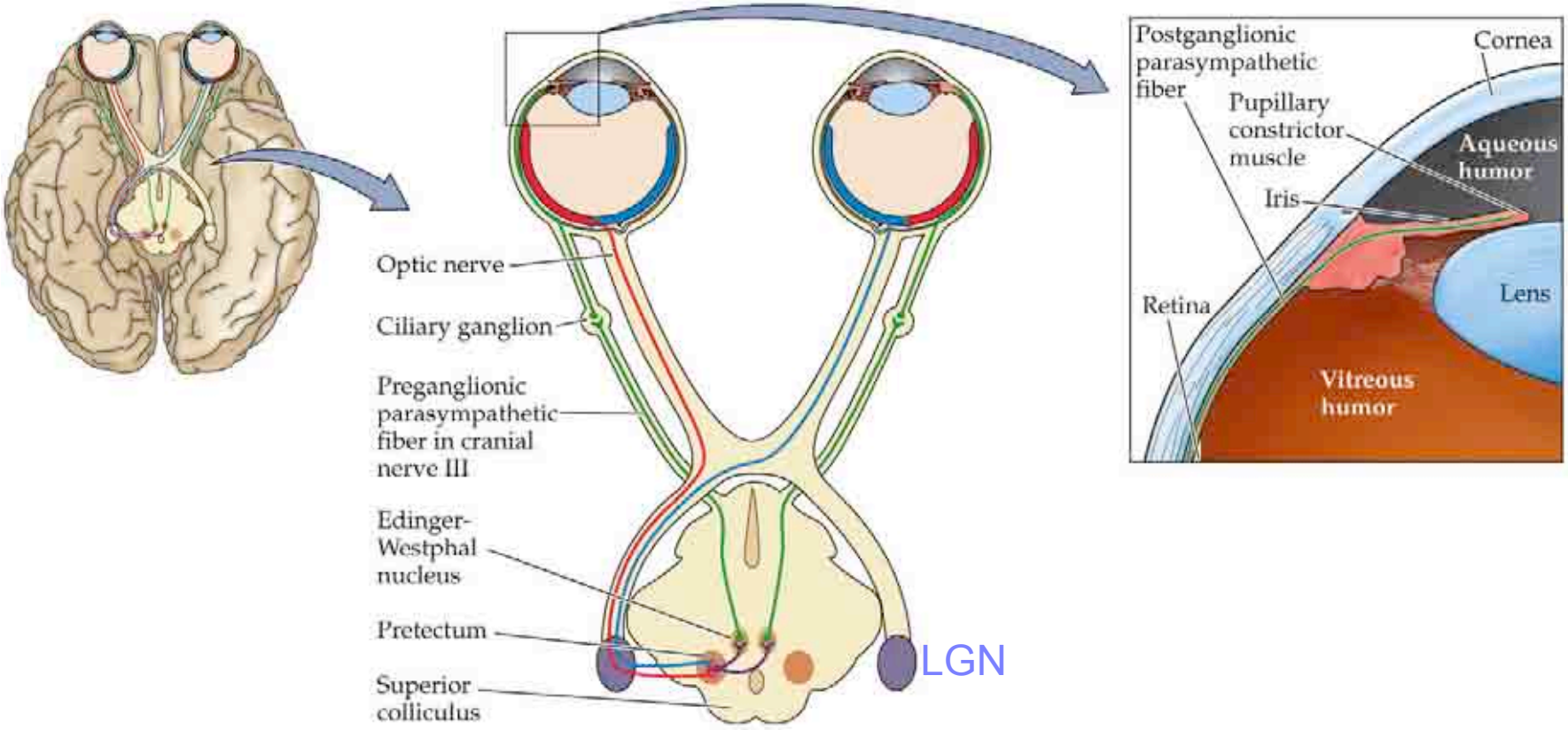
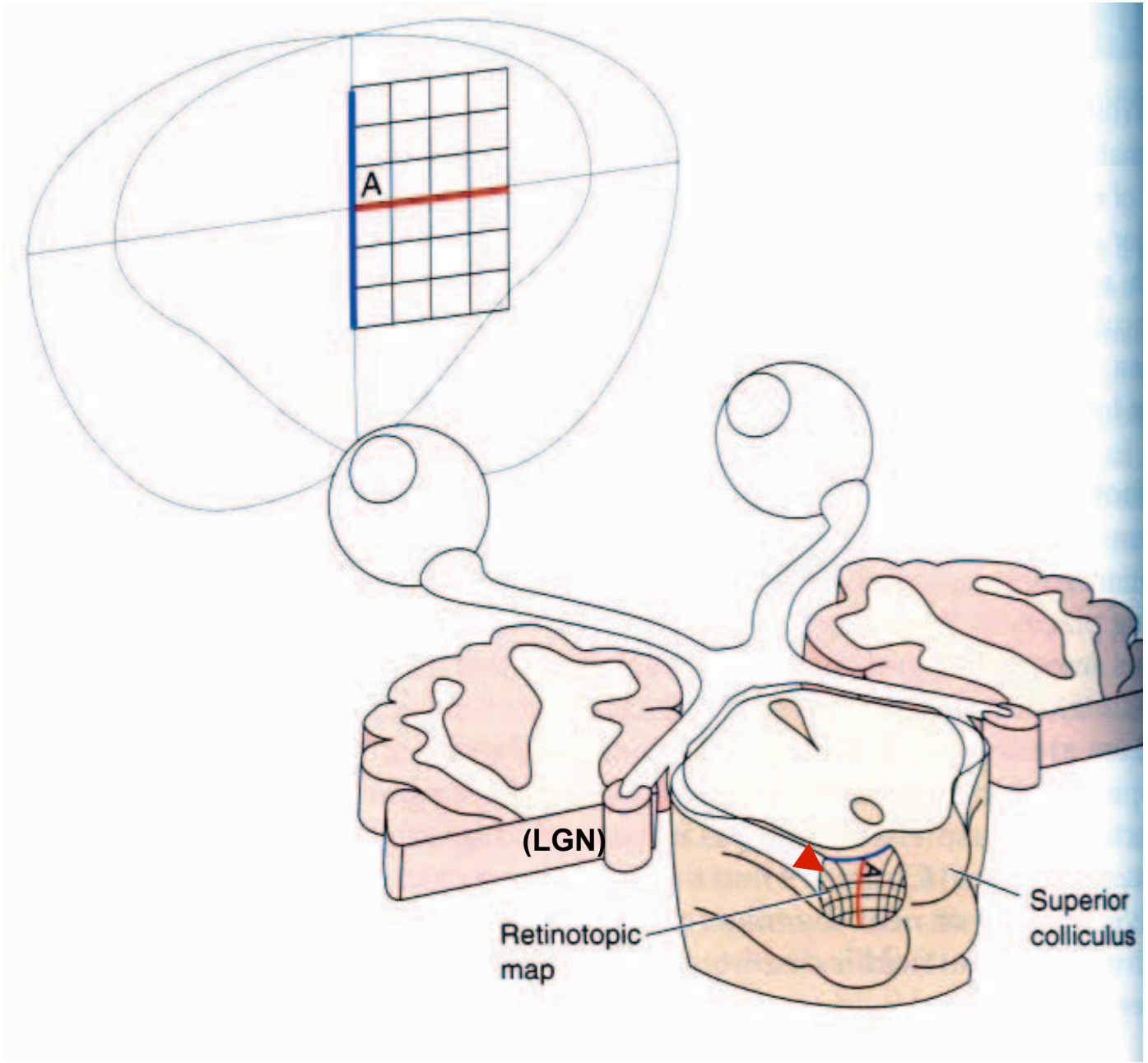


Figure 12.2 The circuitry responsible for the pupillary light reflex

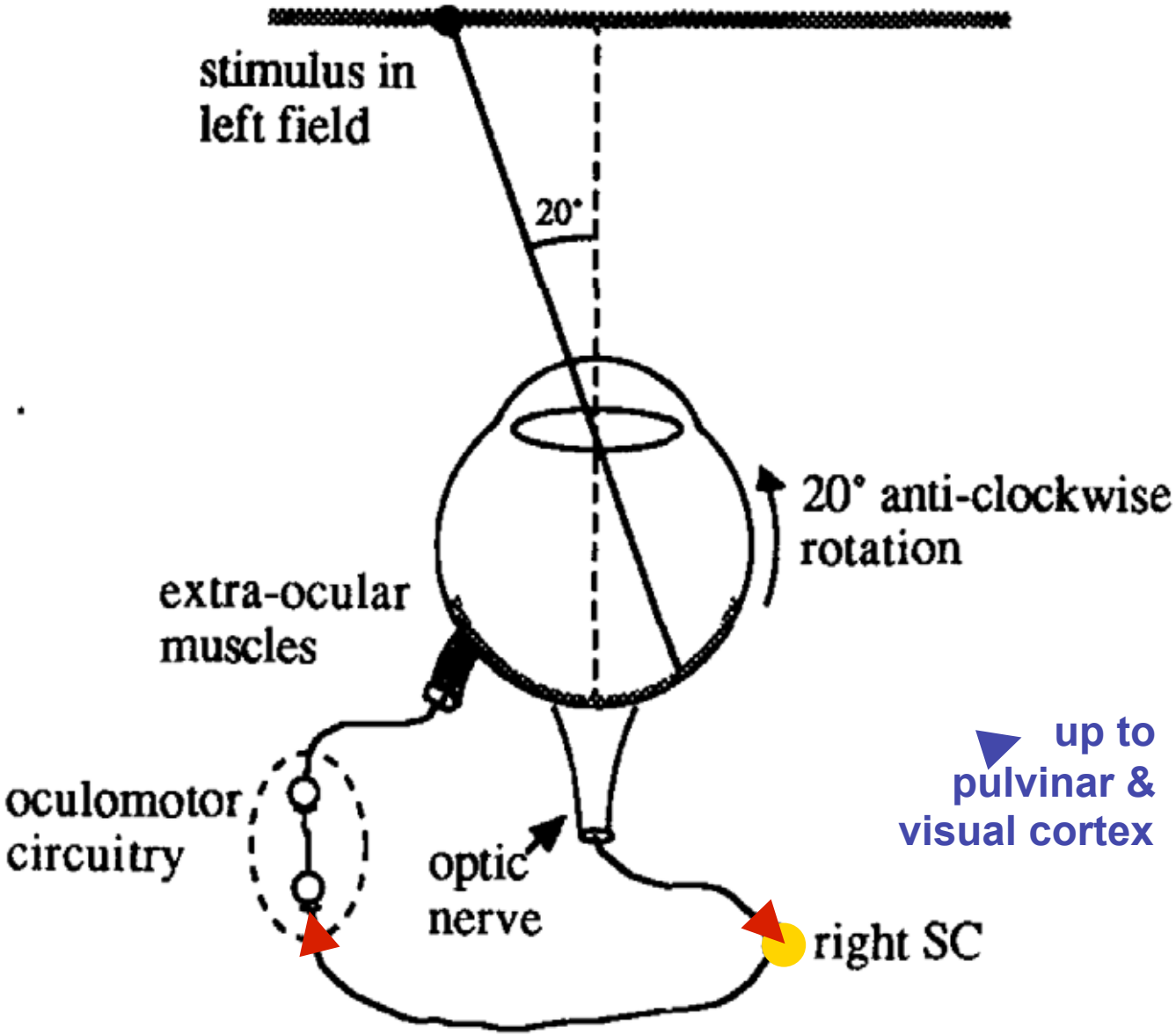


***NEUROSCIENCE, Fourth Edition*, Figure 12.2**

2. Superior Colliculus: eye movements & 2nd visual pathway



2. Superior Colliculus: eye movements & <u>2nd visual pathway (blindsight)</u>

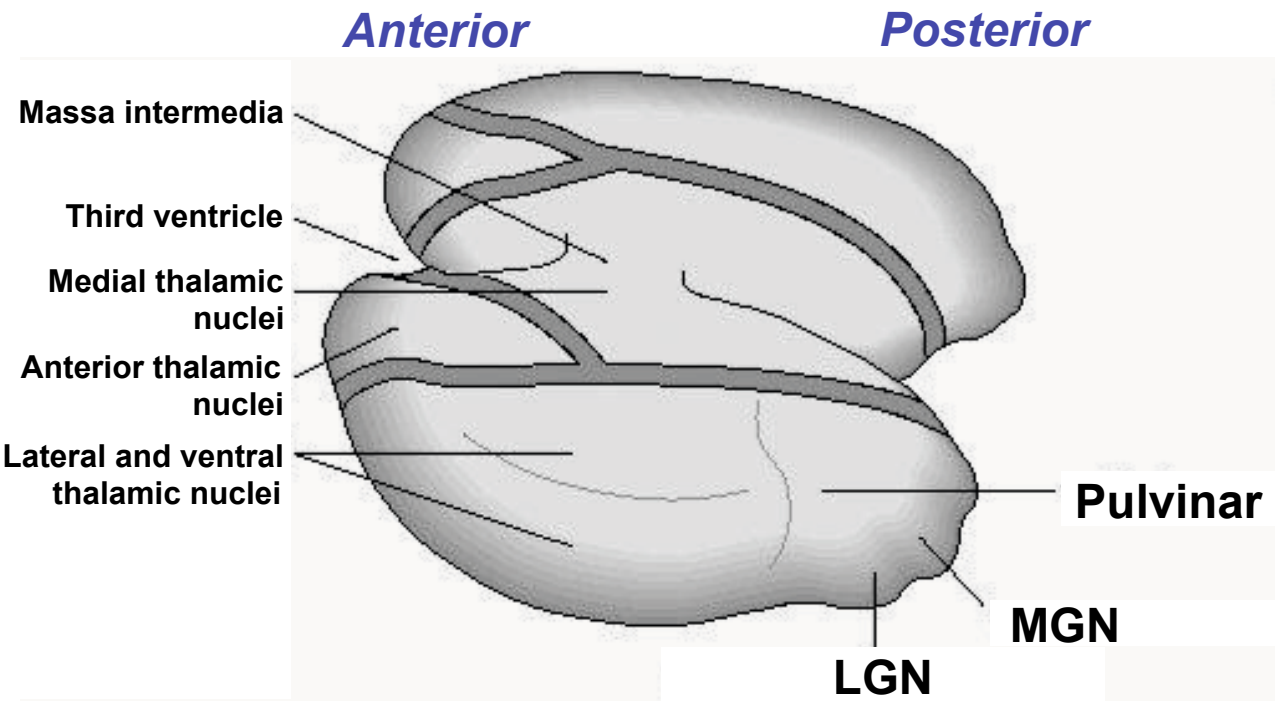


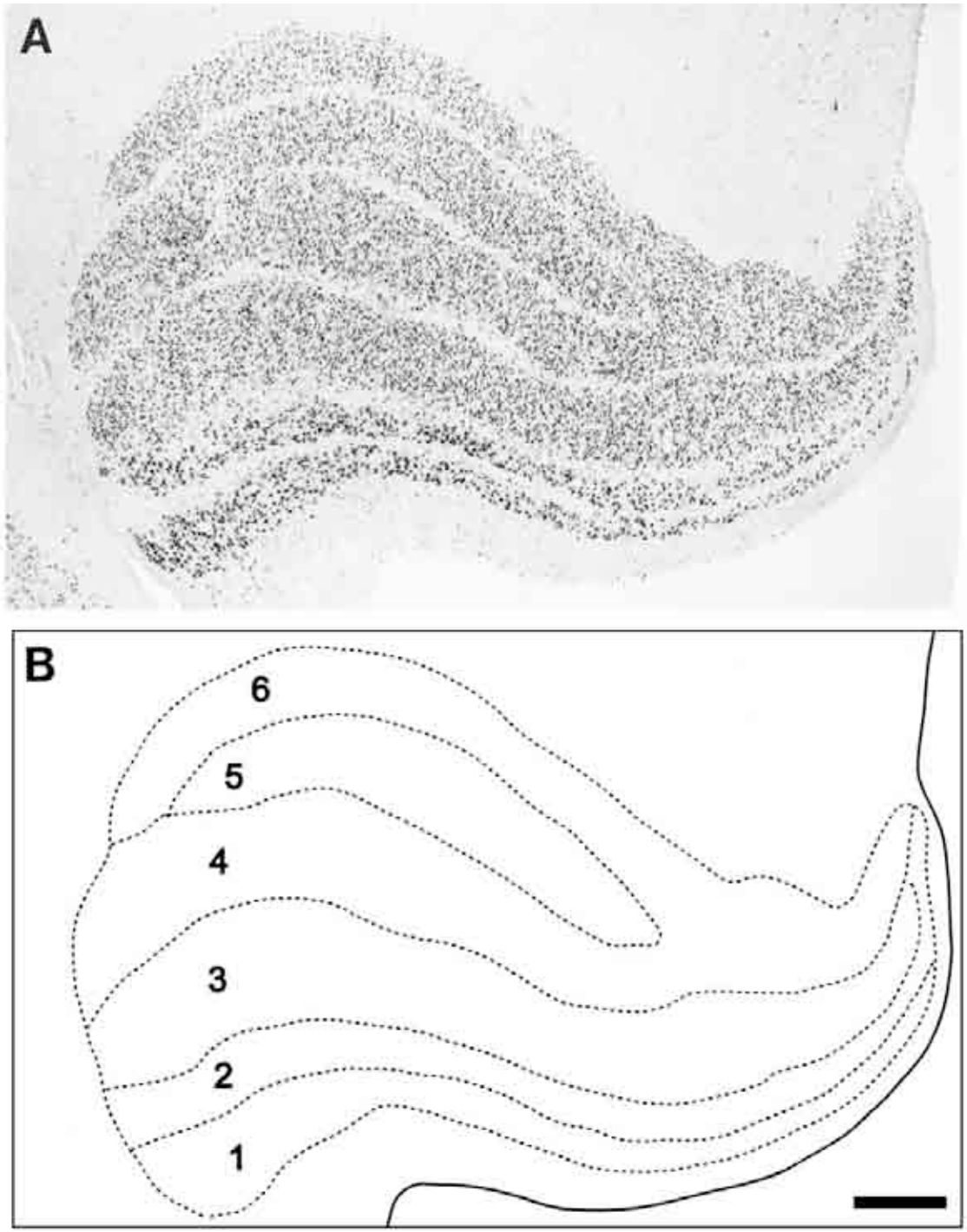
LGN: sometimes also called the “dLGN” because it is in the dorsal thalamus. But because there is no similarly large ventral counterpart (at least in primates, probably), usually omit the “d”.

LGN lesions: extremely rare. (significant damage around the thalamus leads to coma etc. and then visual field defects might be the last of your worries).

Can result in incongruous homonymous hemianopias, and sometimes other strange horizontal or hourglass-shaped field defects.

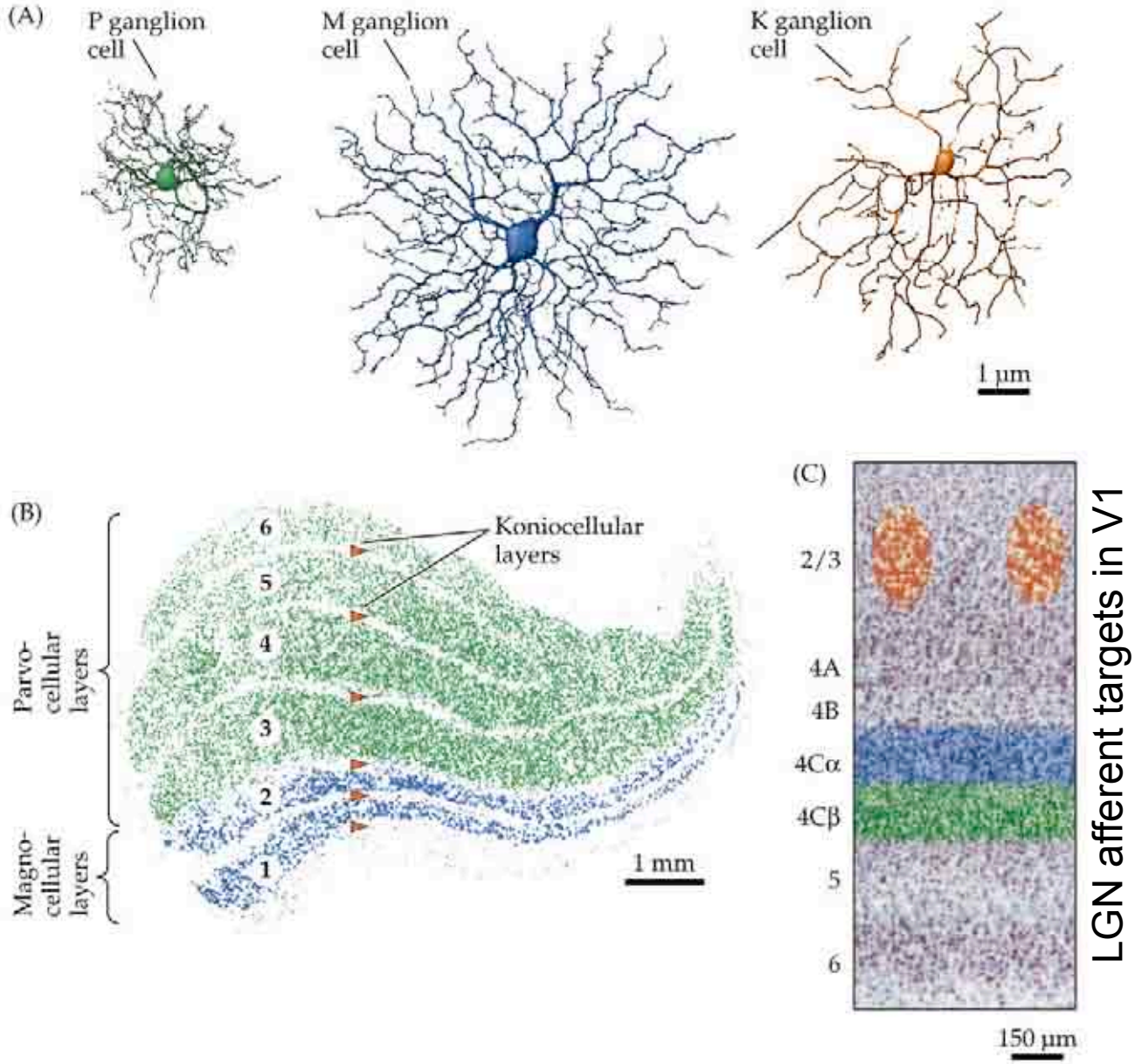
3. Lateral Geniculate Nucleus: Part of the thalamus





Human LGN
(Nissl stain)

Figure 12.15 Magno-, parvo-, and koniocellular pathways

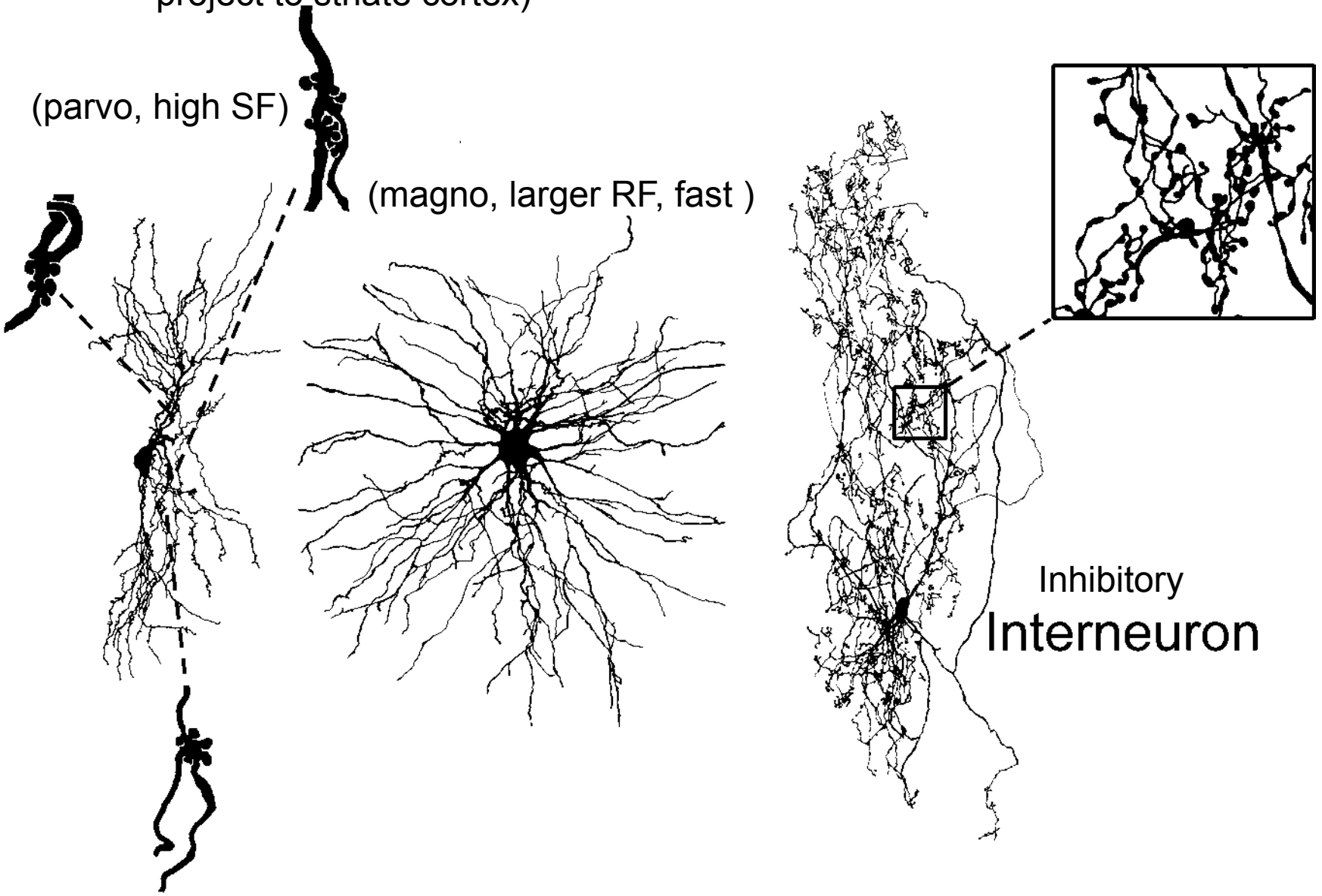


NEUROSCIENCE, Fourth Edition, Figure 12.15

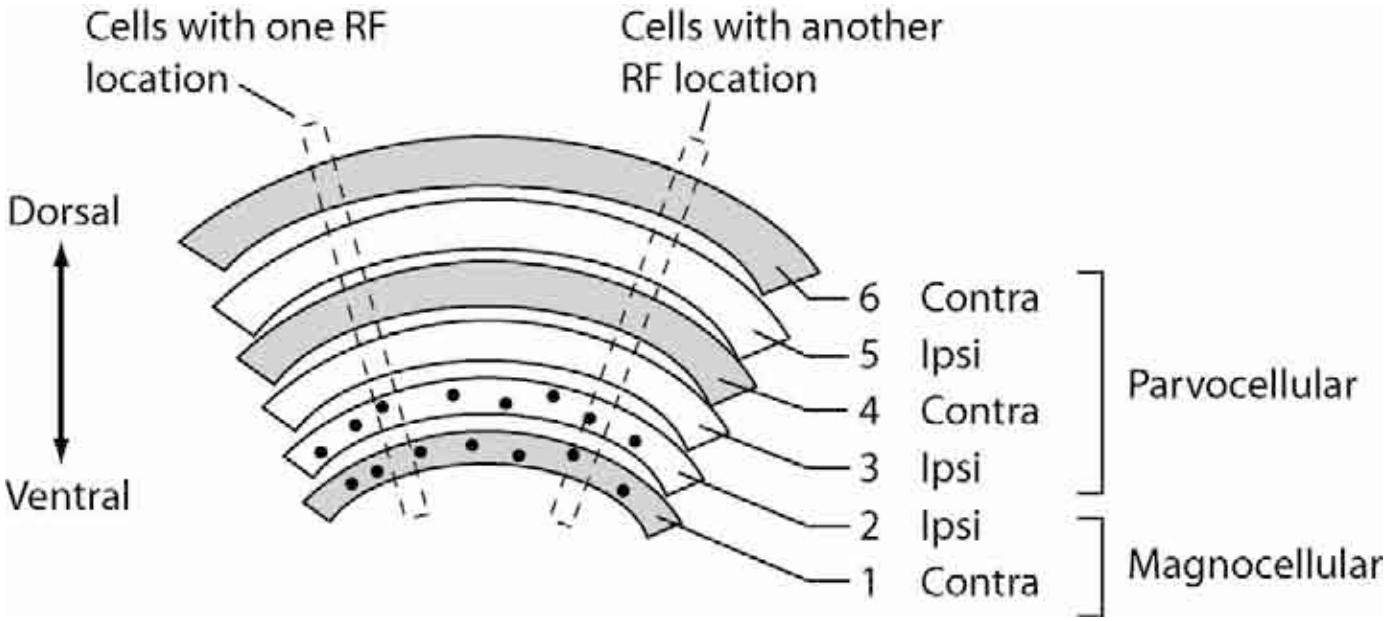
Excitatory Neurons (relay cells,
project to striate cortex)

(parvo, high SF)

(magno, larger RF, fast)



Inhibitory
Interneuron

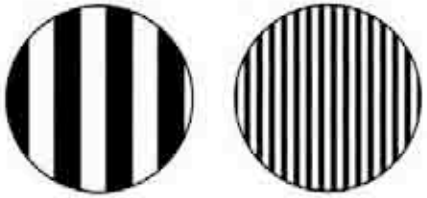


LGN: 1,4,6 (Contralateral eye!)

2,3,5, stay on the same side (Ipsilateral eye).

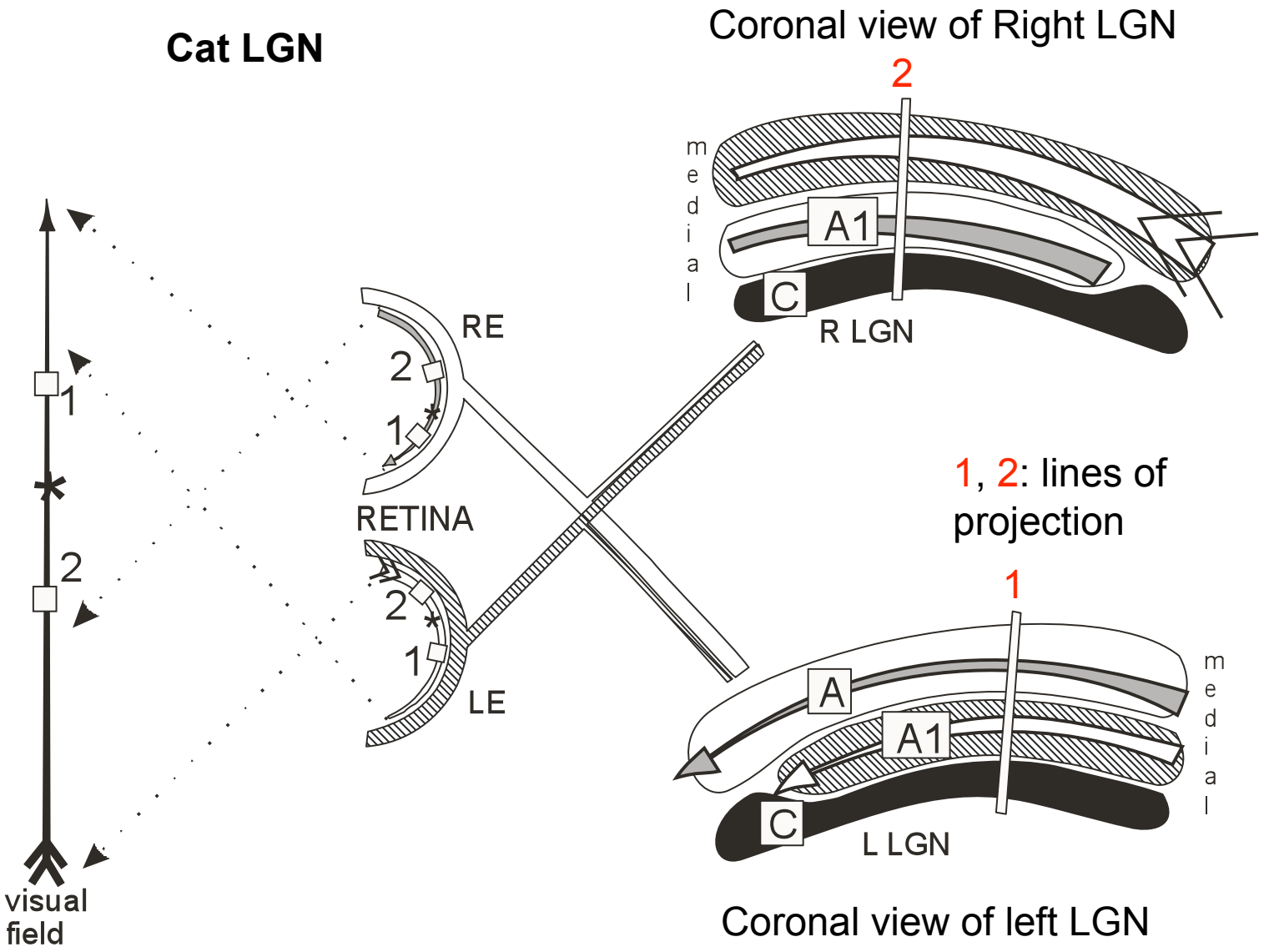
Magno cells are ventral, layers 1 + 2. (Fast, no color, no high spatial frequencies, respond strongly to low contrasts, large cell bodes, big, fast myelinated axons).

Parvo cells are dorsal, layers 3,4,5,6. (Slow, color, high spatial frequencies, respond poorly to low contrasts, smaller cell bodies hence slower conduction through axons).

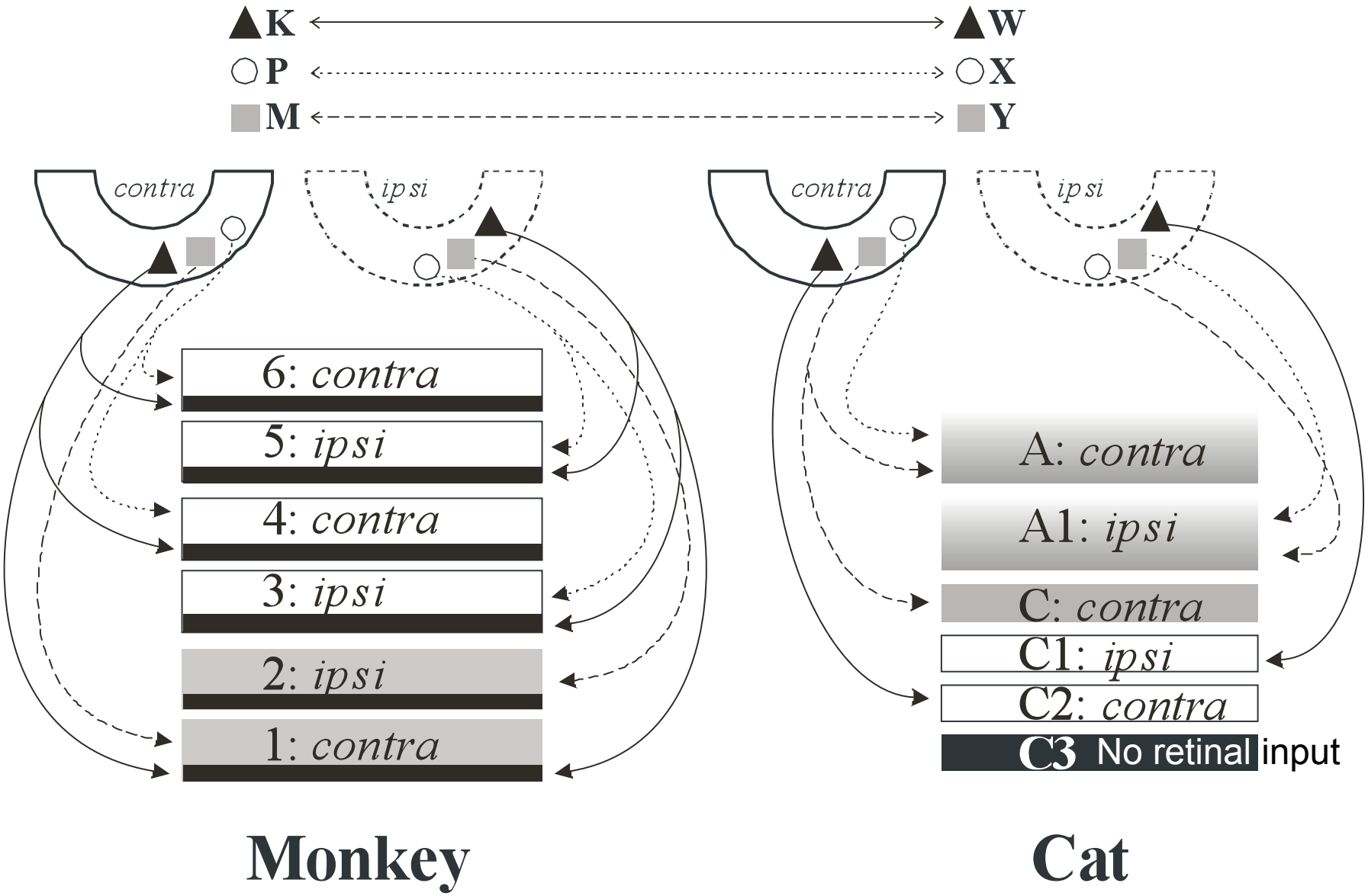
Low SF  High SF (parvo)

Parvocellular: Specialized for fine spatial details & color.
Magnocellular: Specialized for flickering/moving objects

Even though each of the two LGNs receive input from both eyes, each layer and each LGN relay cell only receives feedforward **monocular** input from the retina.

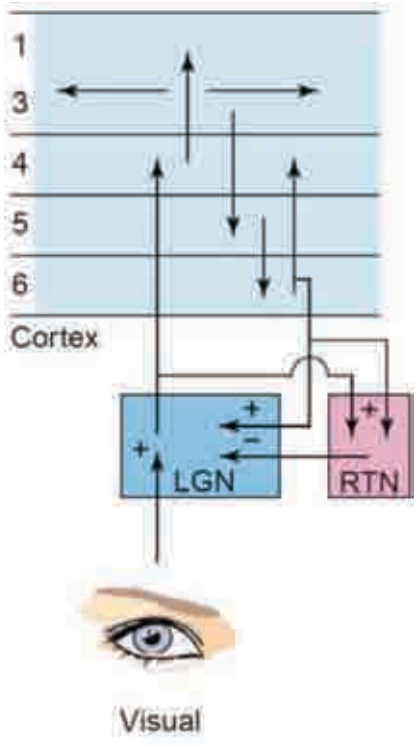


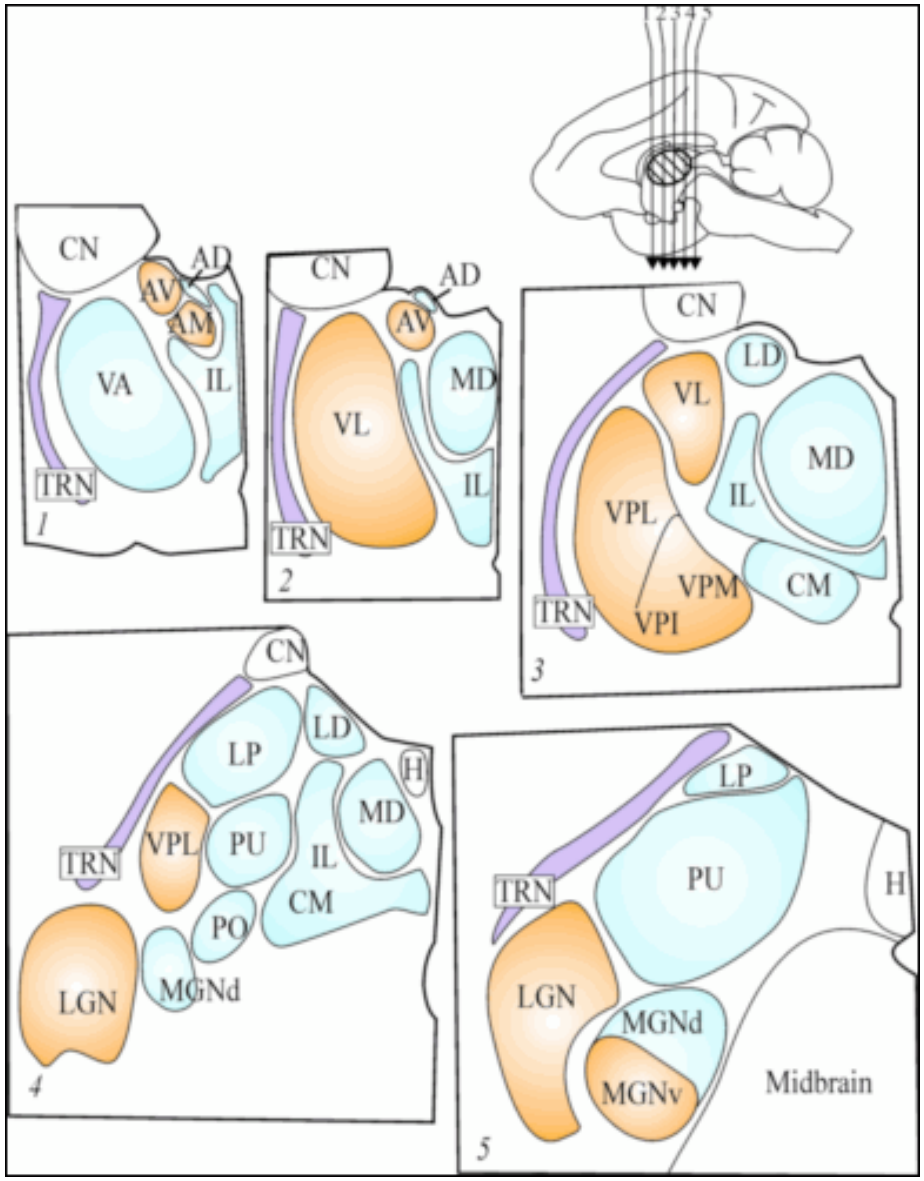
Guess the number of layers in the rodent LGN?



Each LGN layer receives input from one eye only, parallel pathways to cortex

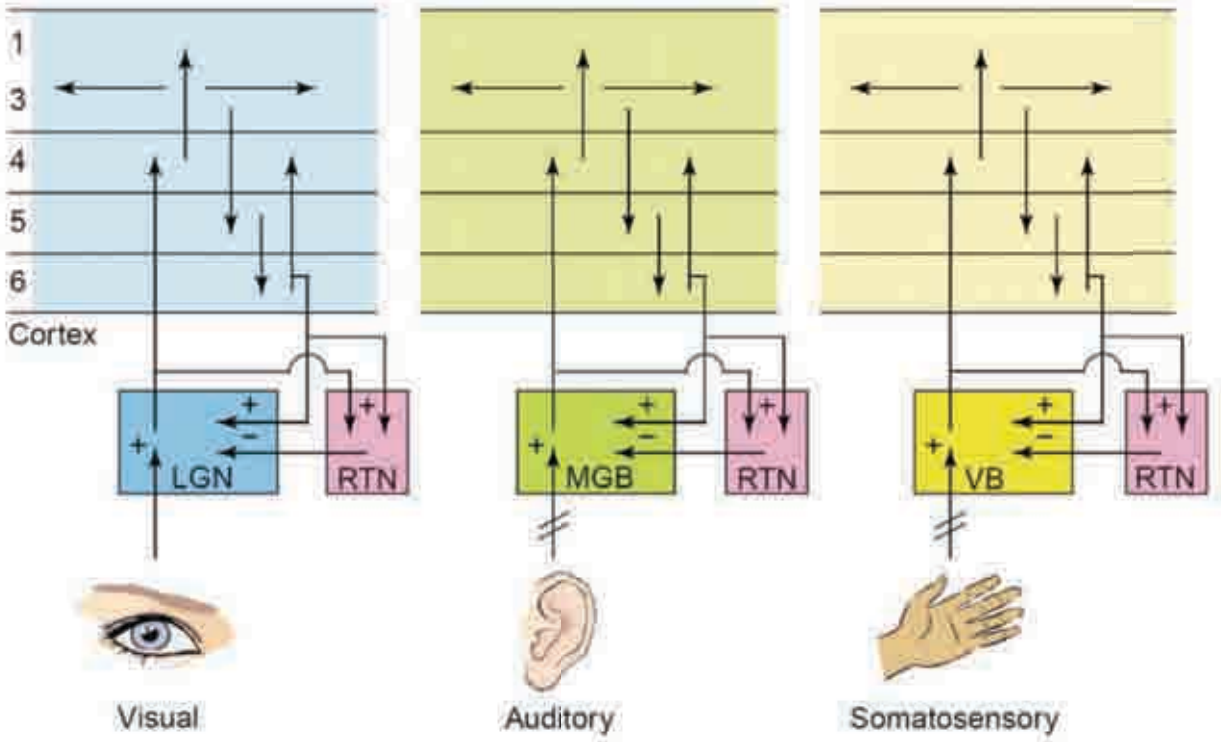
***Biggest* mystery of LGN:**
over 90% of its inputs come
from cerebral cortex, brainstem, TRN,
not retina!



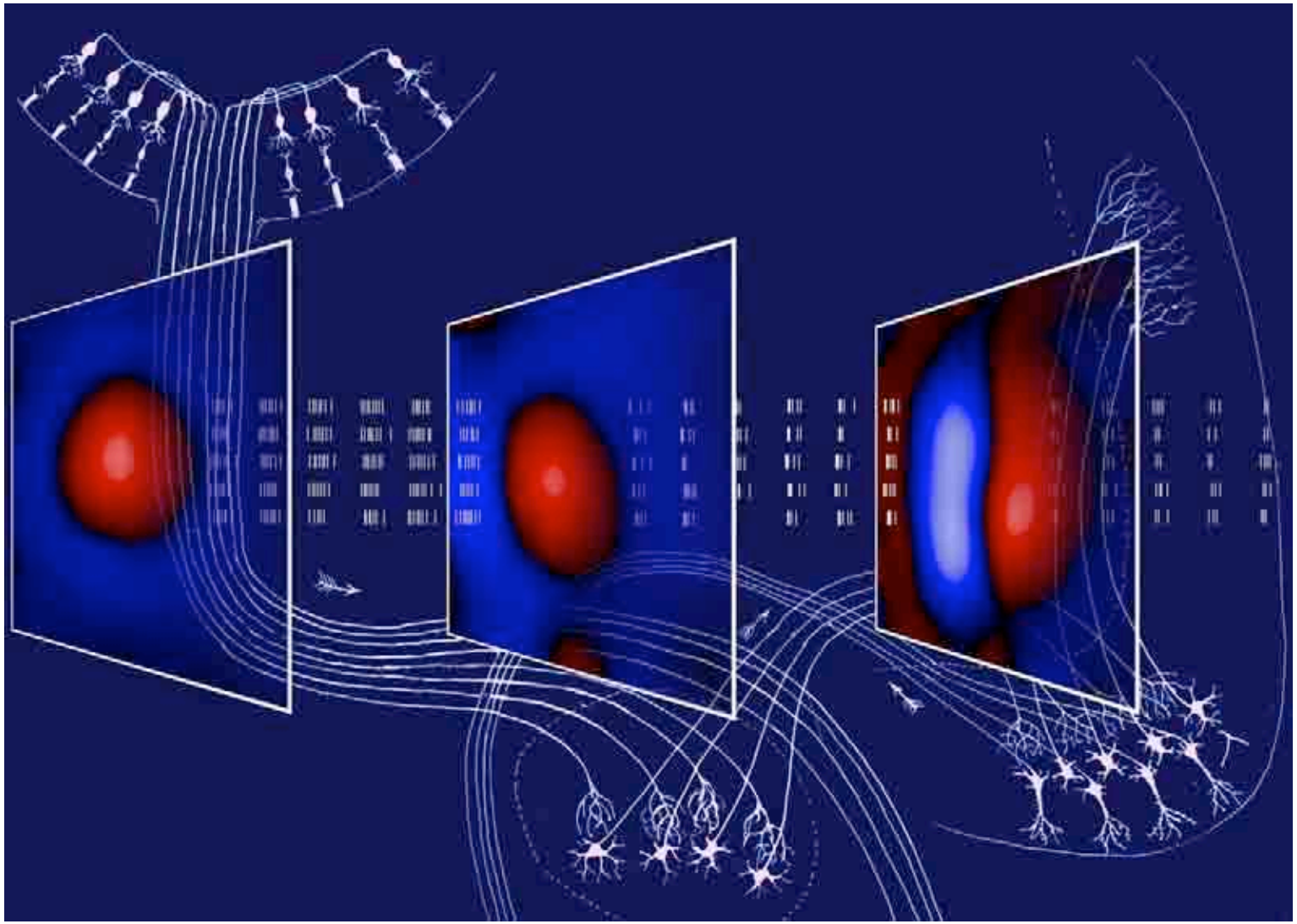


(RTN = TRN,
both abbreviations
are commonly used.
Reticular Nucleus of Thalamus)

This is a circuit that is common in sensory systems.

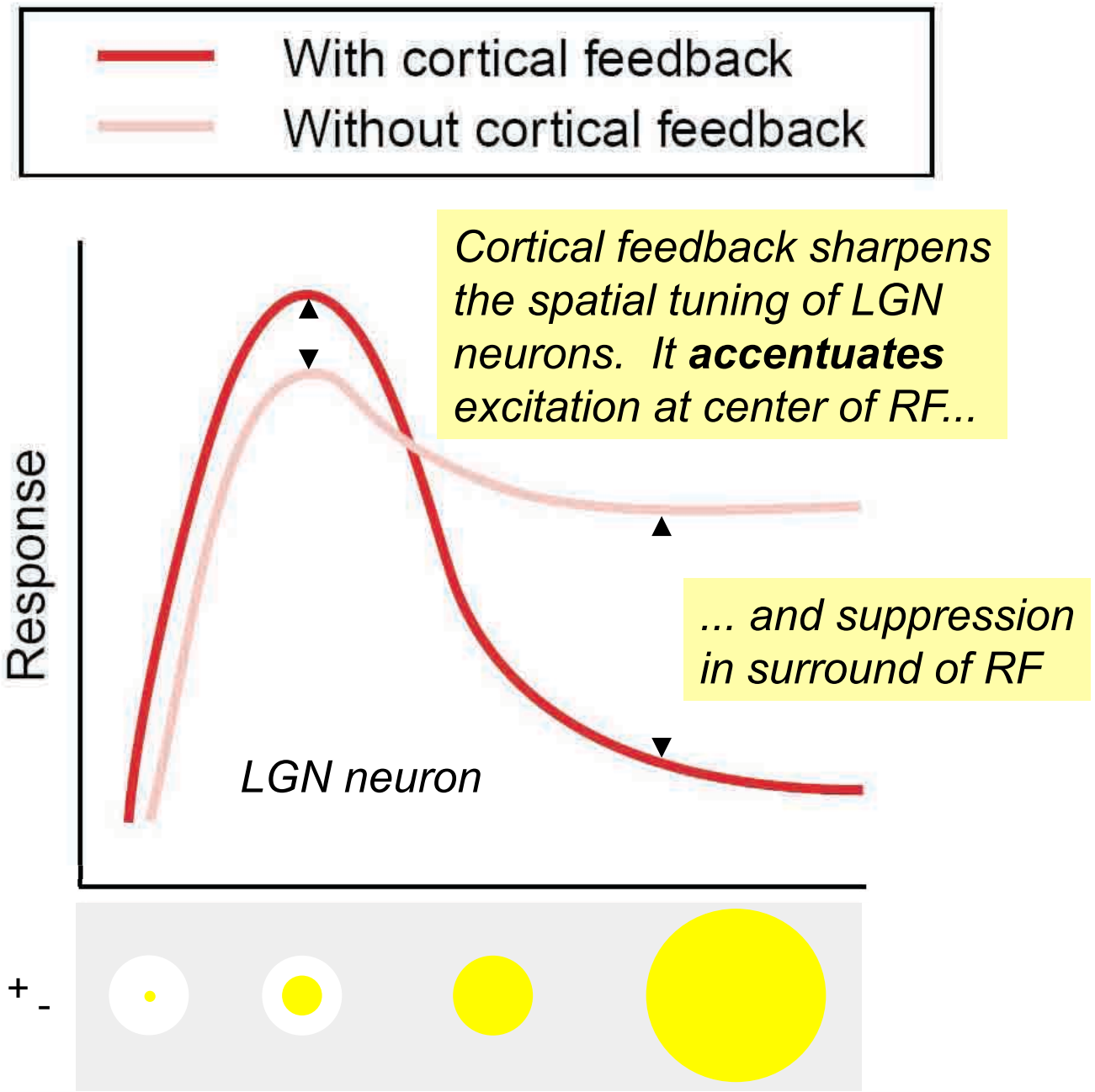


Spatial receptive field structure of LGN relay cells is very similar to RGCs, but…



Kara et al. 2000 *Neuron*

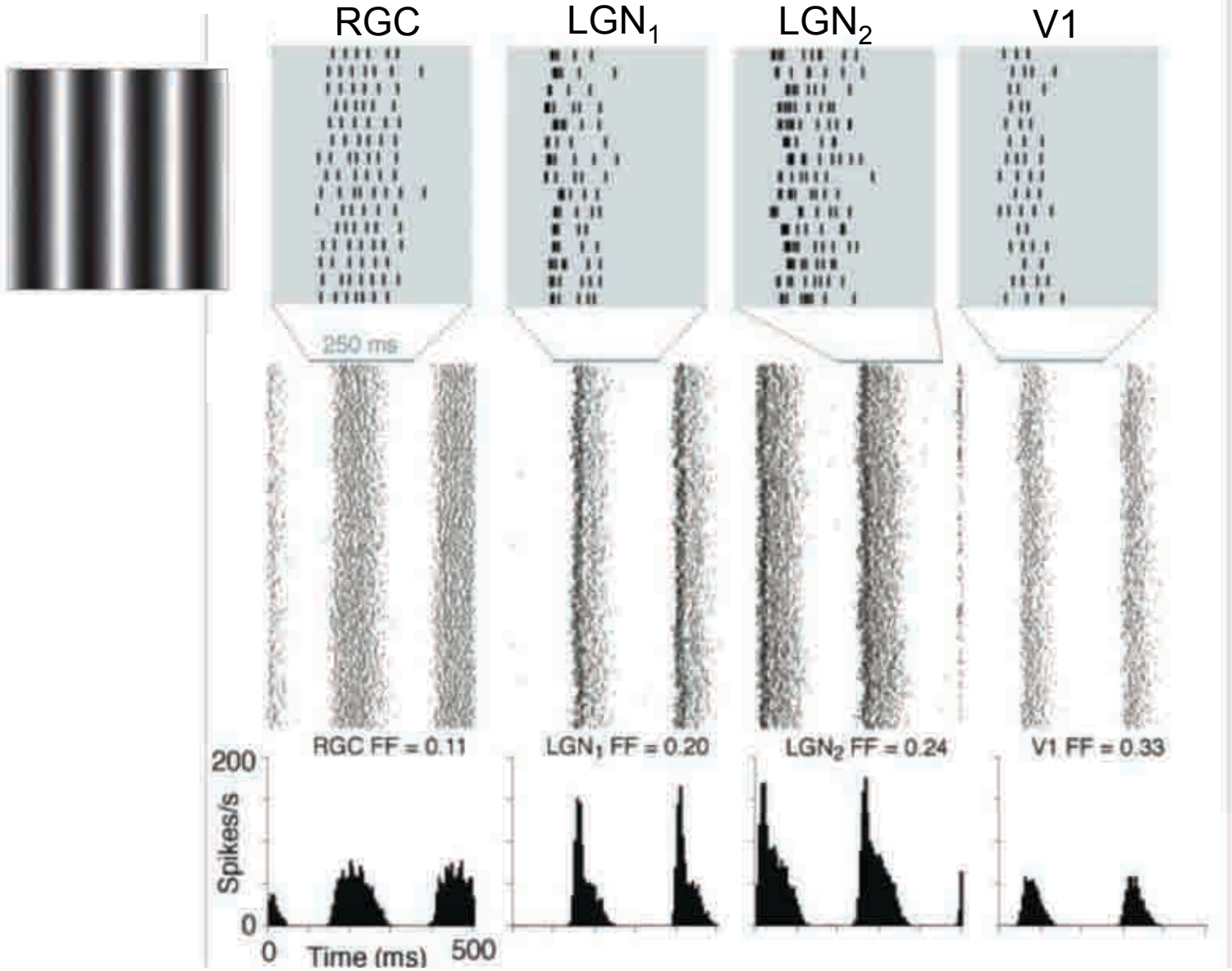
We know only a little bit about the functions of corticothalamic feedback.
For example:



*Cortical feedback sharpens the spatial tuning of LGN neurons. It **accentuates** excitation at center of RF...*

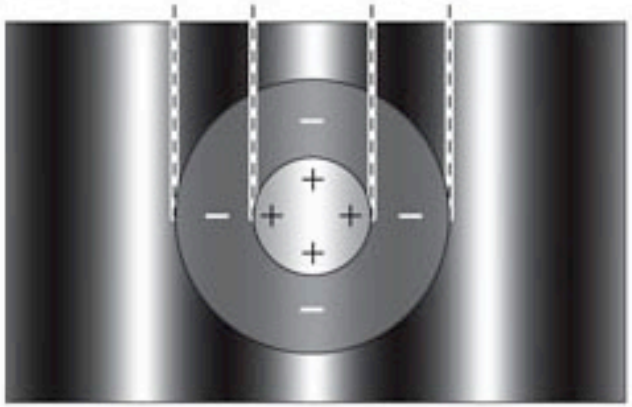
... and suppression in surround of RF

Timing of spikes in the LGN

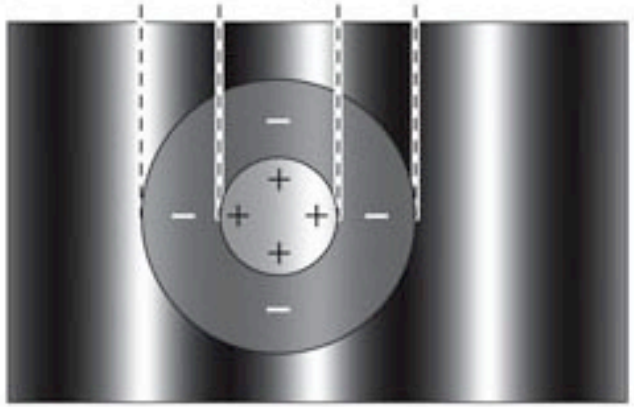


Kara et al. 2000 *Neuron*

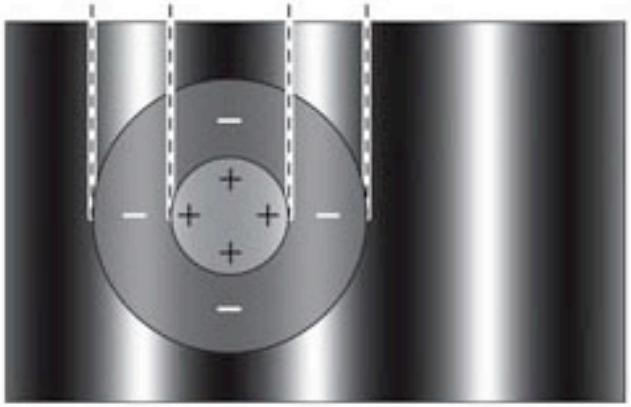
(*a*) 0° – Positive response



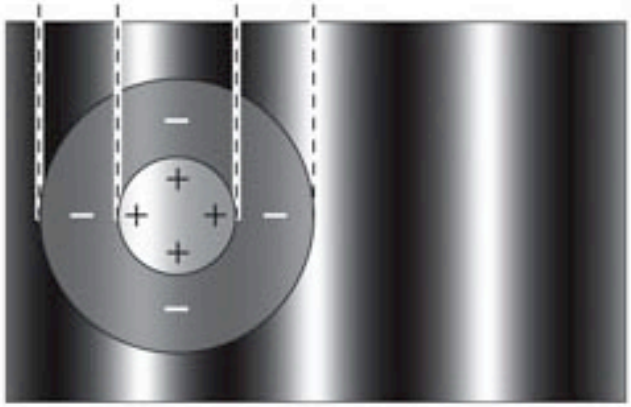
(*b*) 90° – No response



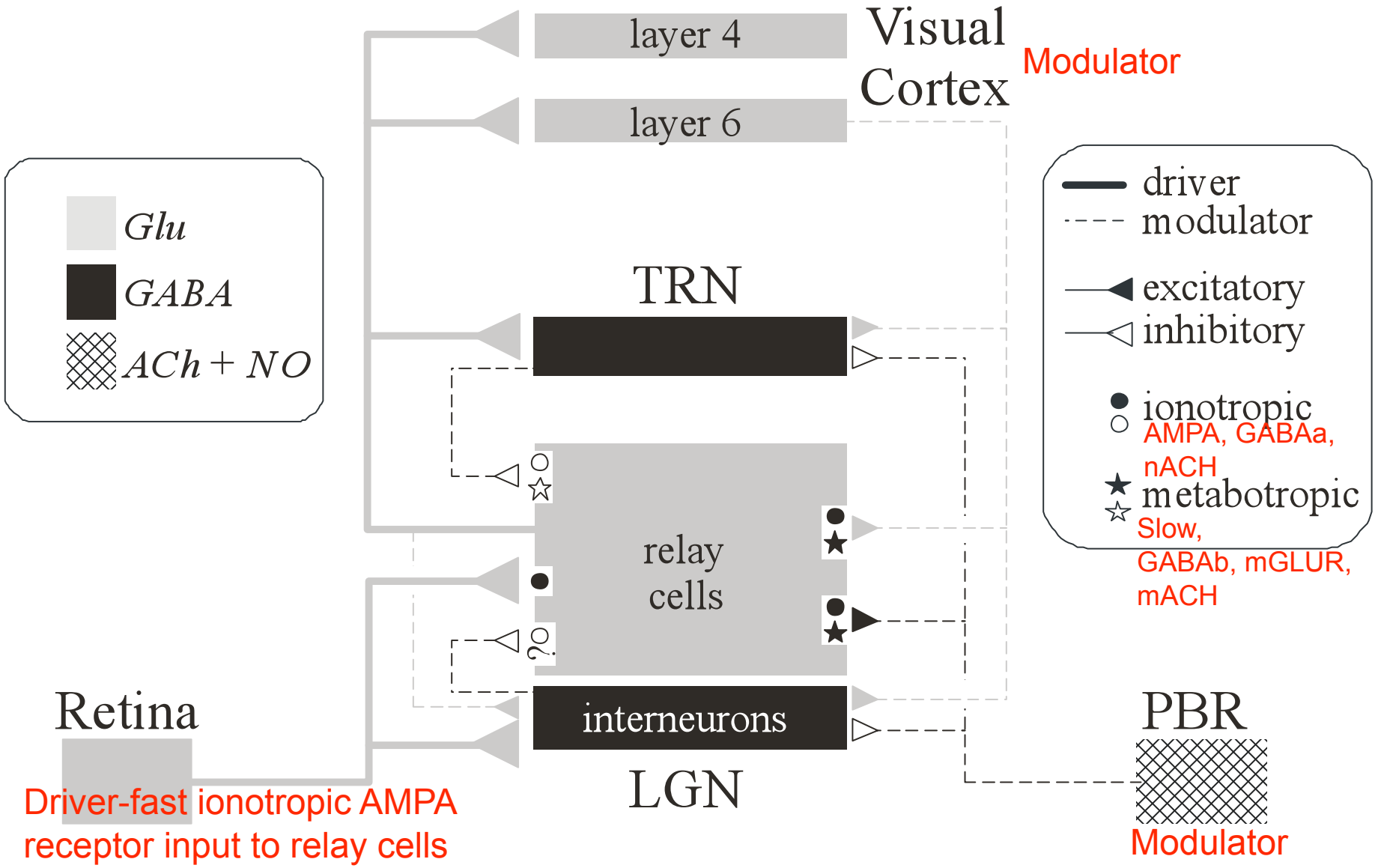
(*c*) 180° – Negative response



(*d*) 270° – No response

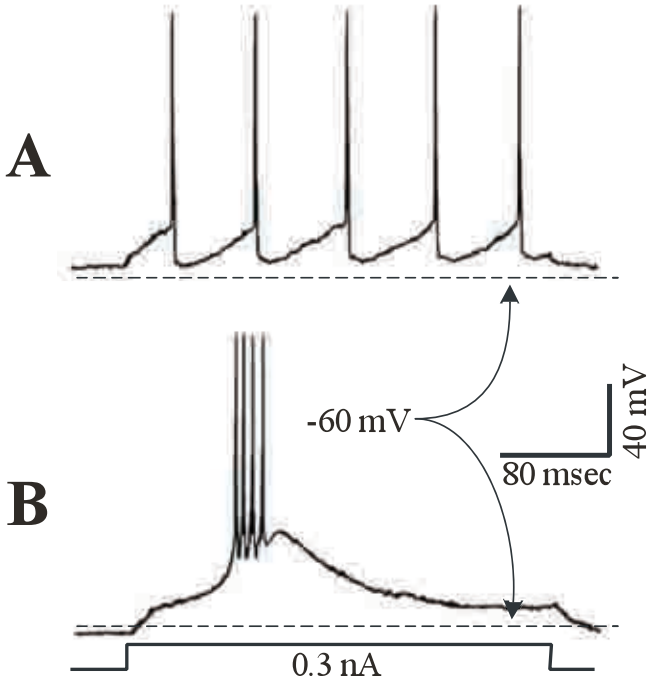


LGN “A” layers circuitry

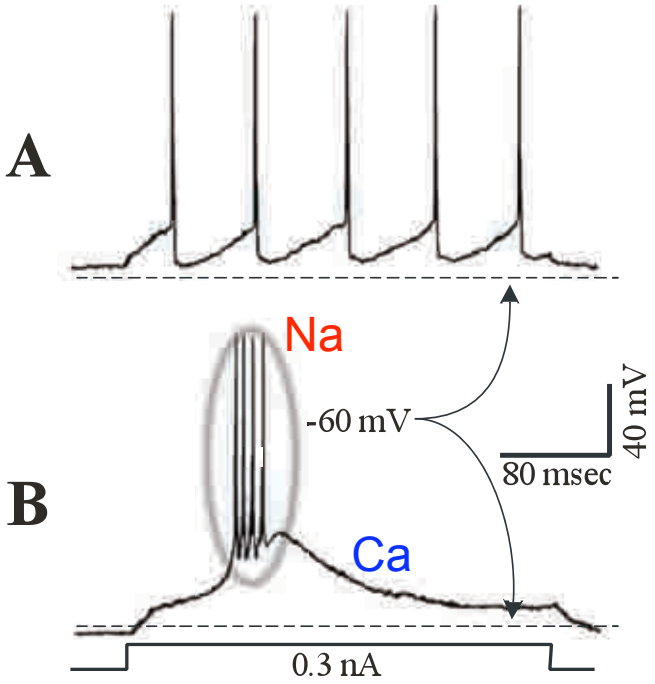


Most synapses onto relay cells are from non-retinal sources
Retinal afferents = 5-10% of synapses on LGN relay cells

Same depolarizing pulse but opposite effects ('condition' membrane for >= 100 ms)

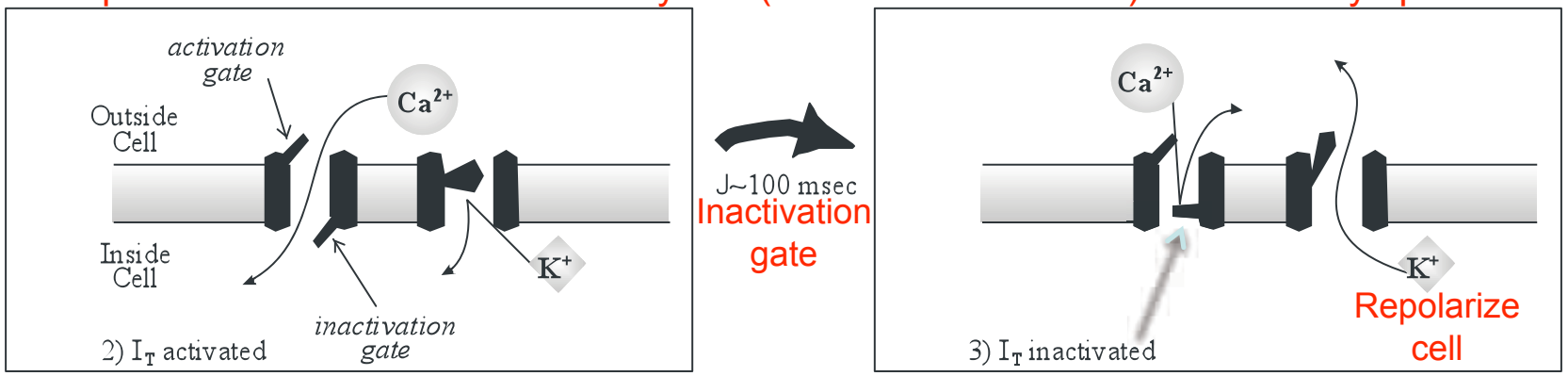


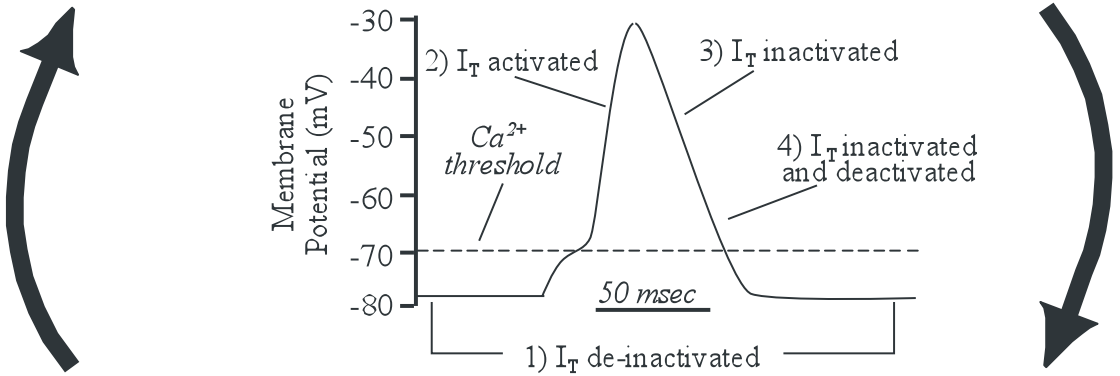
Same depolarizing pulse but opposite effects ('condition' membrane for >= 100 ms)



The Low Threshold Ca^{2+} Spike

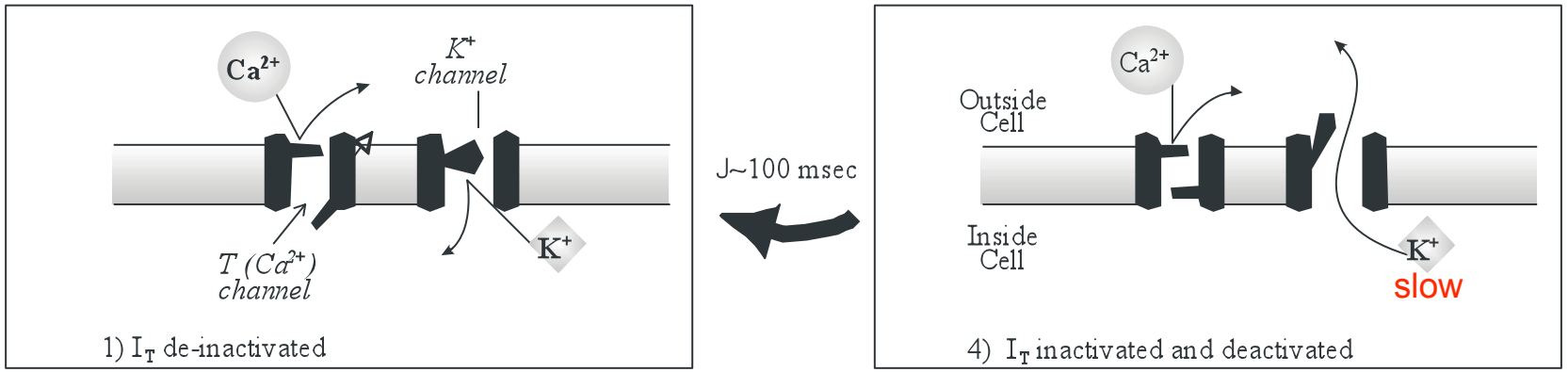
T channel present in EVERY thalamic relay cell (in all thalamic nuclei) and in every species studied





Activ. & inactiv. gates have opposite voltage dependency

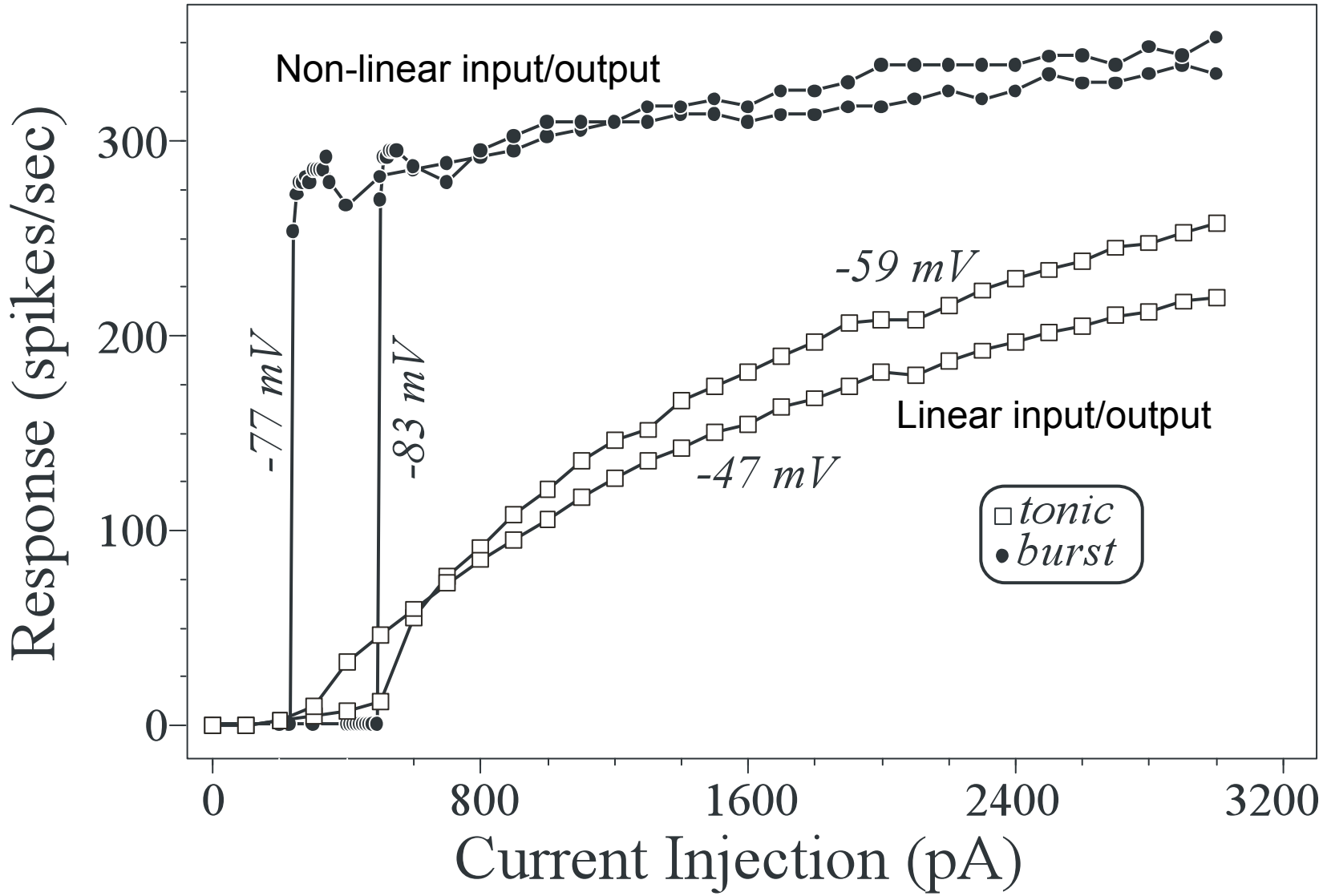
1



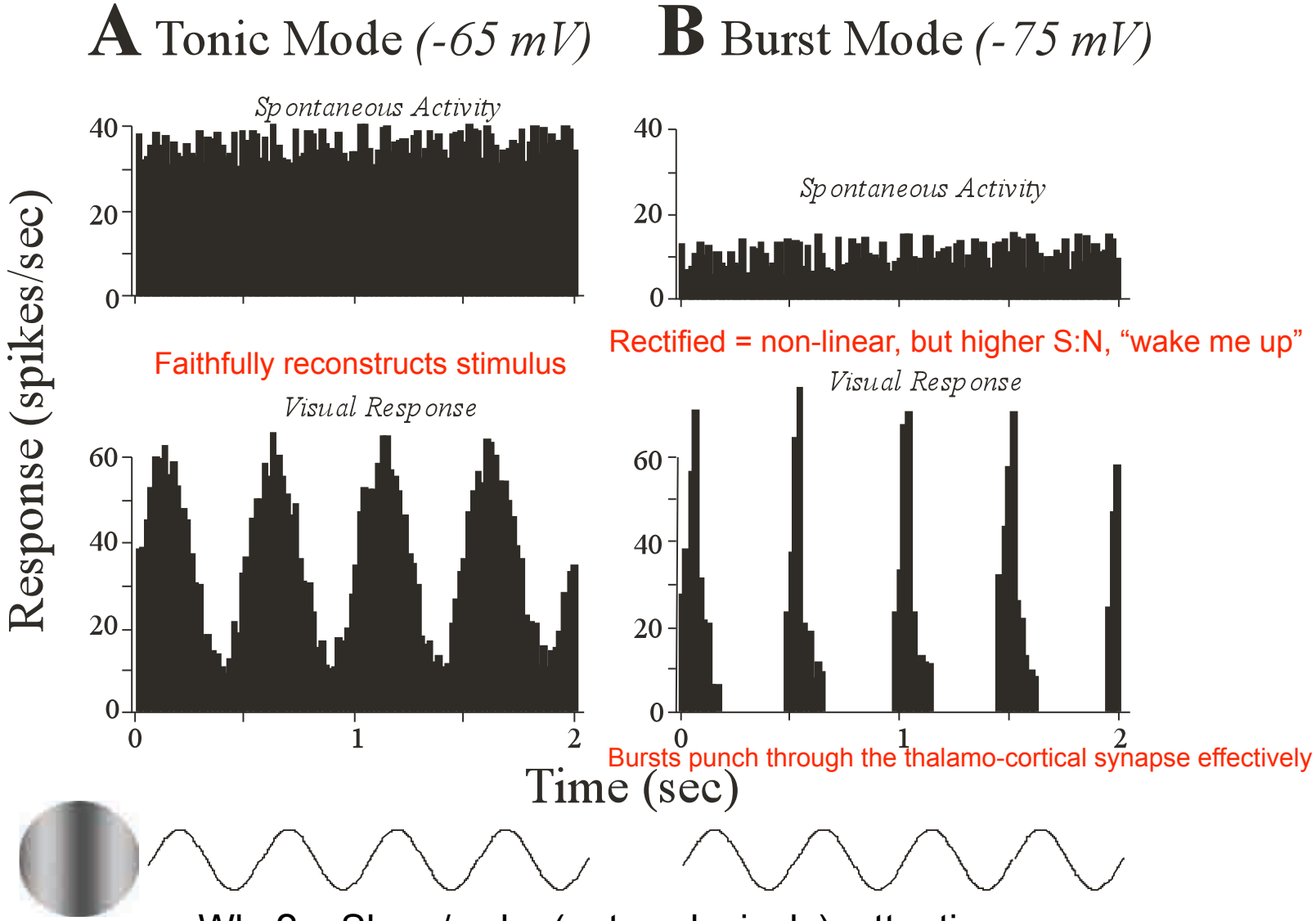
Activation gate closed, inactivation gate OPEN

The LT calcium spike in thalamic relay cells is *qualitatively* similar to the Na-K spike found in most/all neurons but *quantitatively* different:

- Calcium spike is slower (activation and inactivation)
- Calcium spike is not present in the axon initial segment, but only in the cell body
- It does not propagate down into the axon and affect the postsynaptic target
- It operates in a more hyperpolarized regime (i.e., low threshold)
- It needs to be in a low or high state for > 100 ms for activation/inactivation

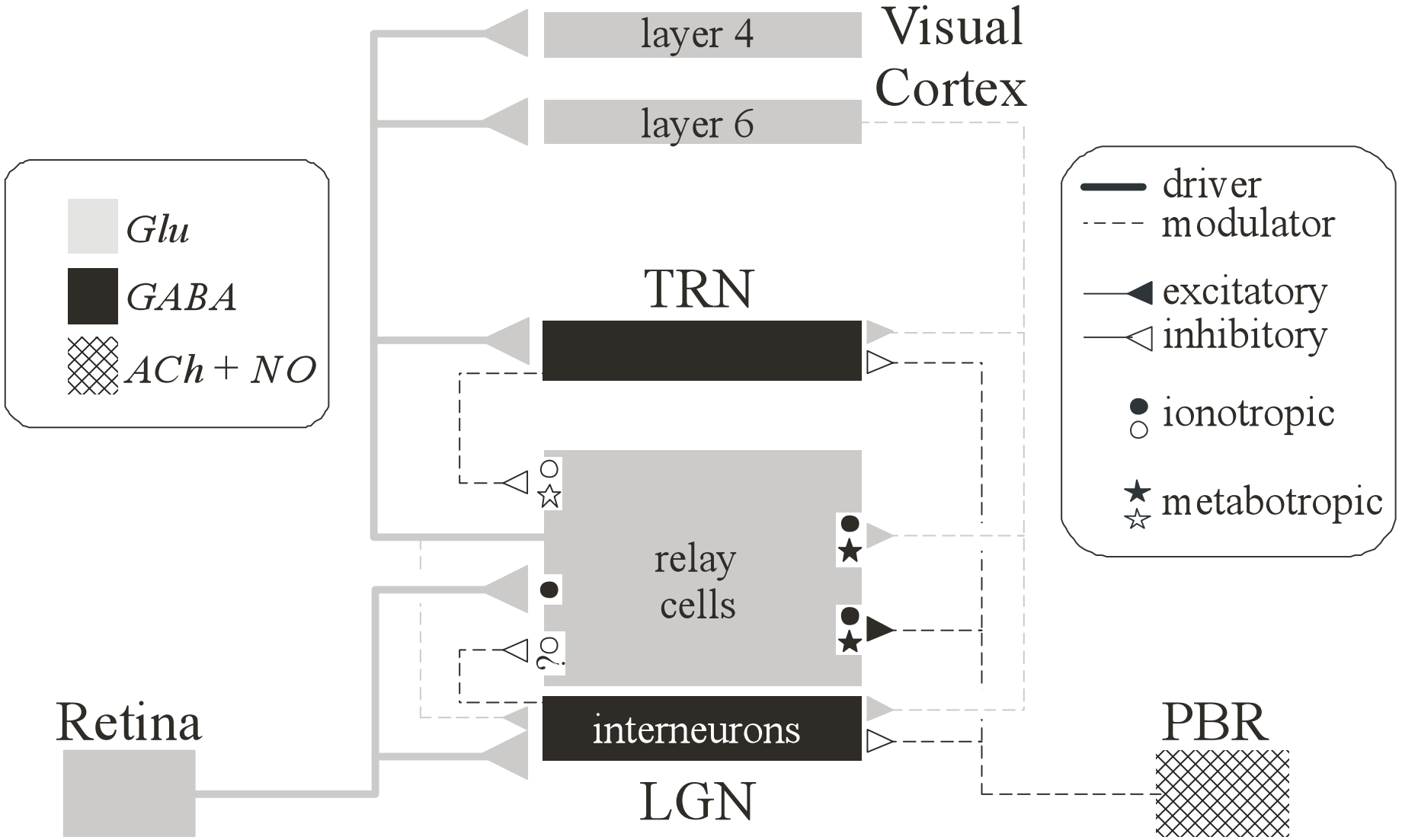

Non-linear input/output
Linear input/output
-77 mV
-83 mV
-59 mV
-47 mV
tonic
burst
Response (spikes/sec)
Current Injection (pA)
0
100
200
300
0
800
1600
2400
3200

Relay cell responses to sensory stimuli (sine gratings)



Why? Sleep/wake (not exclusively), attention (yes), detecting new stimuli in the environment

Iontropic receptors (inputs) CANNOT cause mode switches:
mGLUR from cortex or mACH from parabrachial region cause switch from burst to tonic
& GABAb from brainstem reticular formation and local interneurons – opposite.

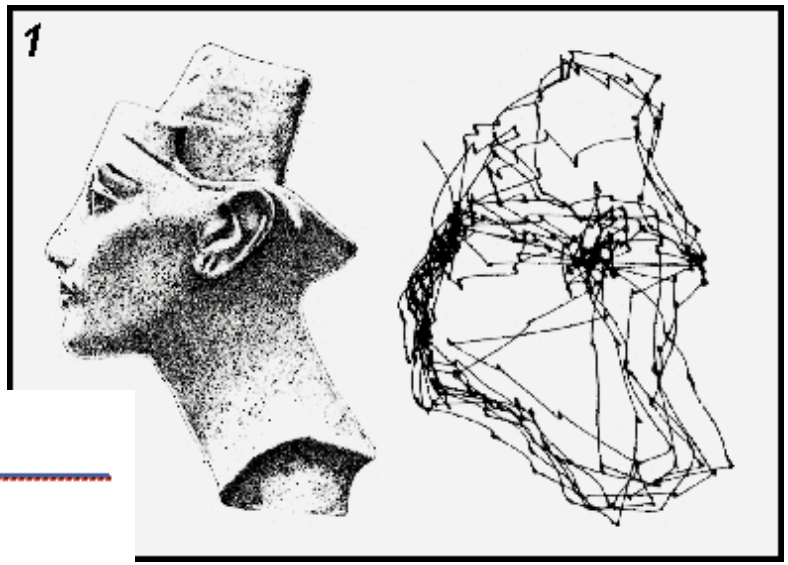


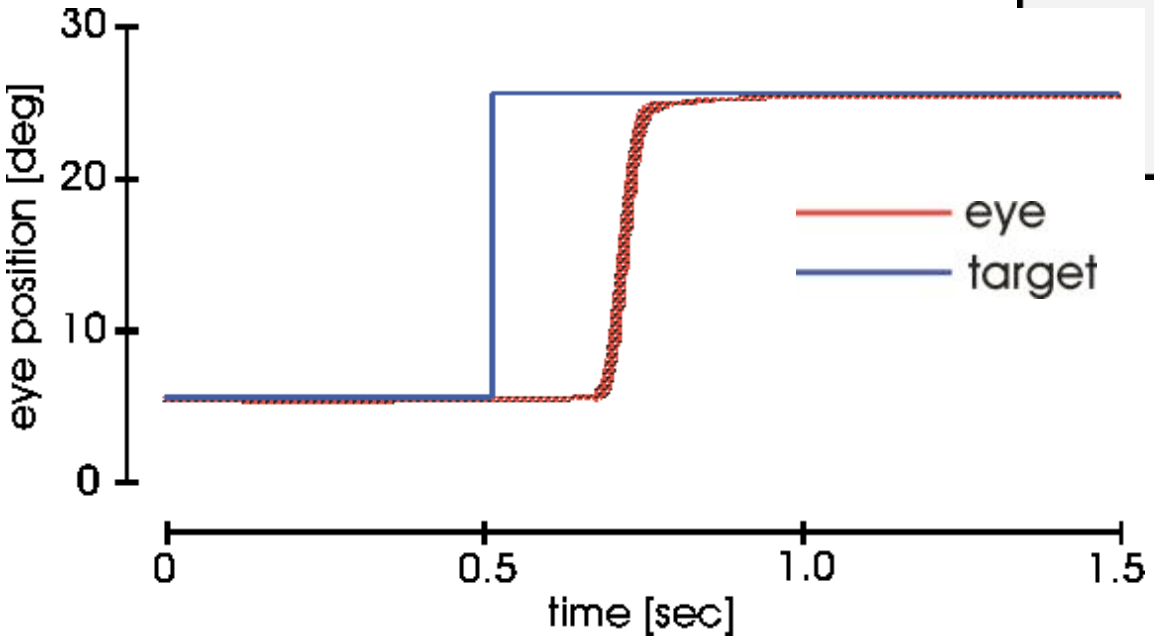
Thalamus = last bottleneck for behavioral states to affect information processing:
Much fewer cells (than cortex) to gate

Two main kinds of eye movements

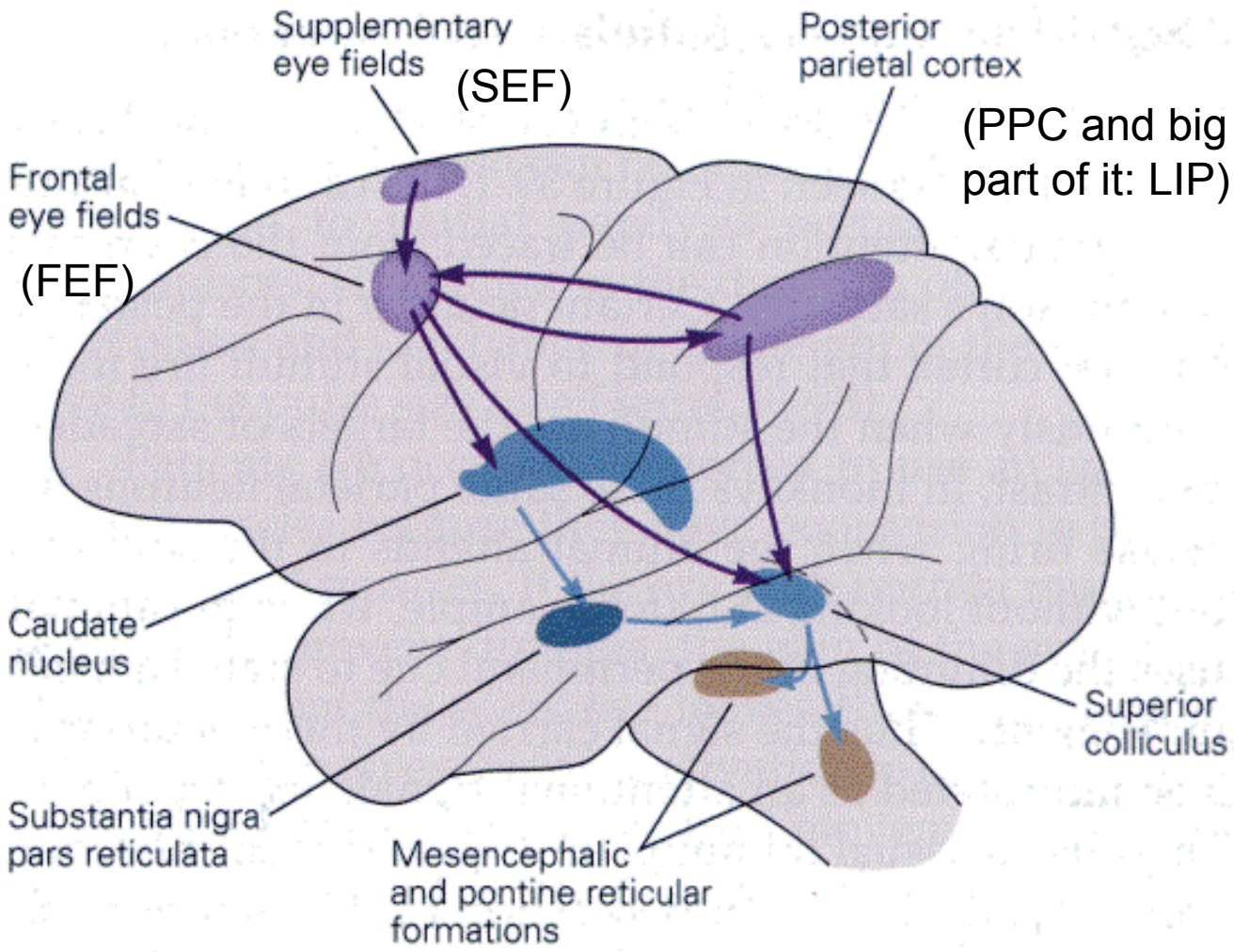
- ***Saccades***

  (microsaccades)



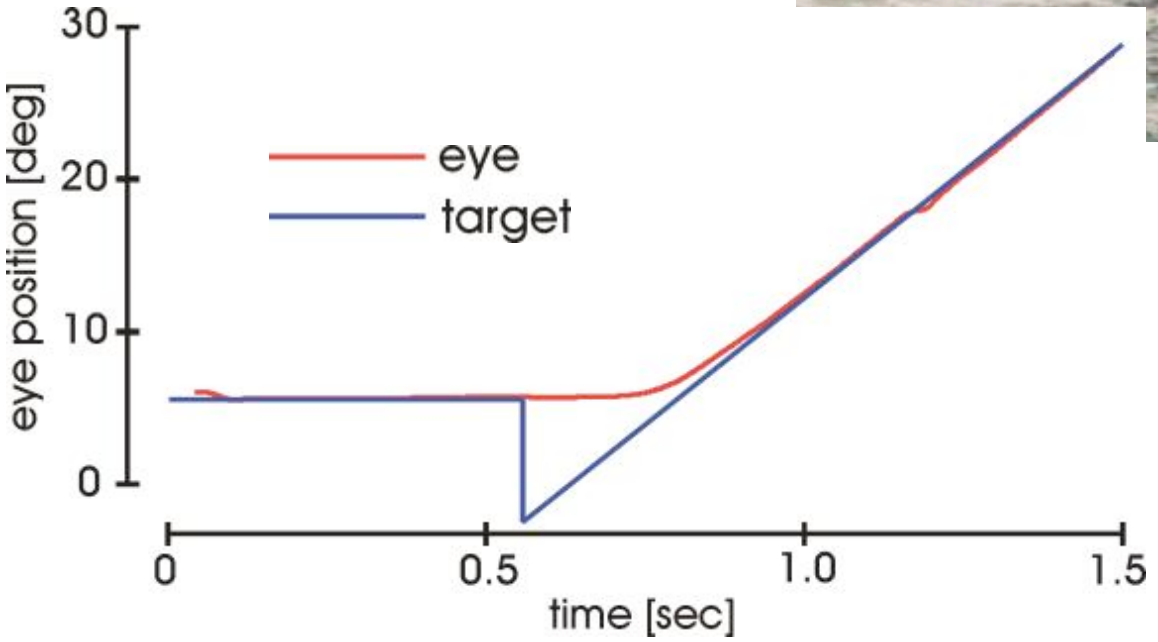


Simplified outline of saccadic system

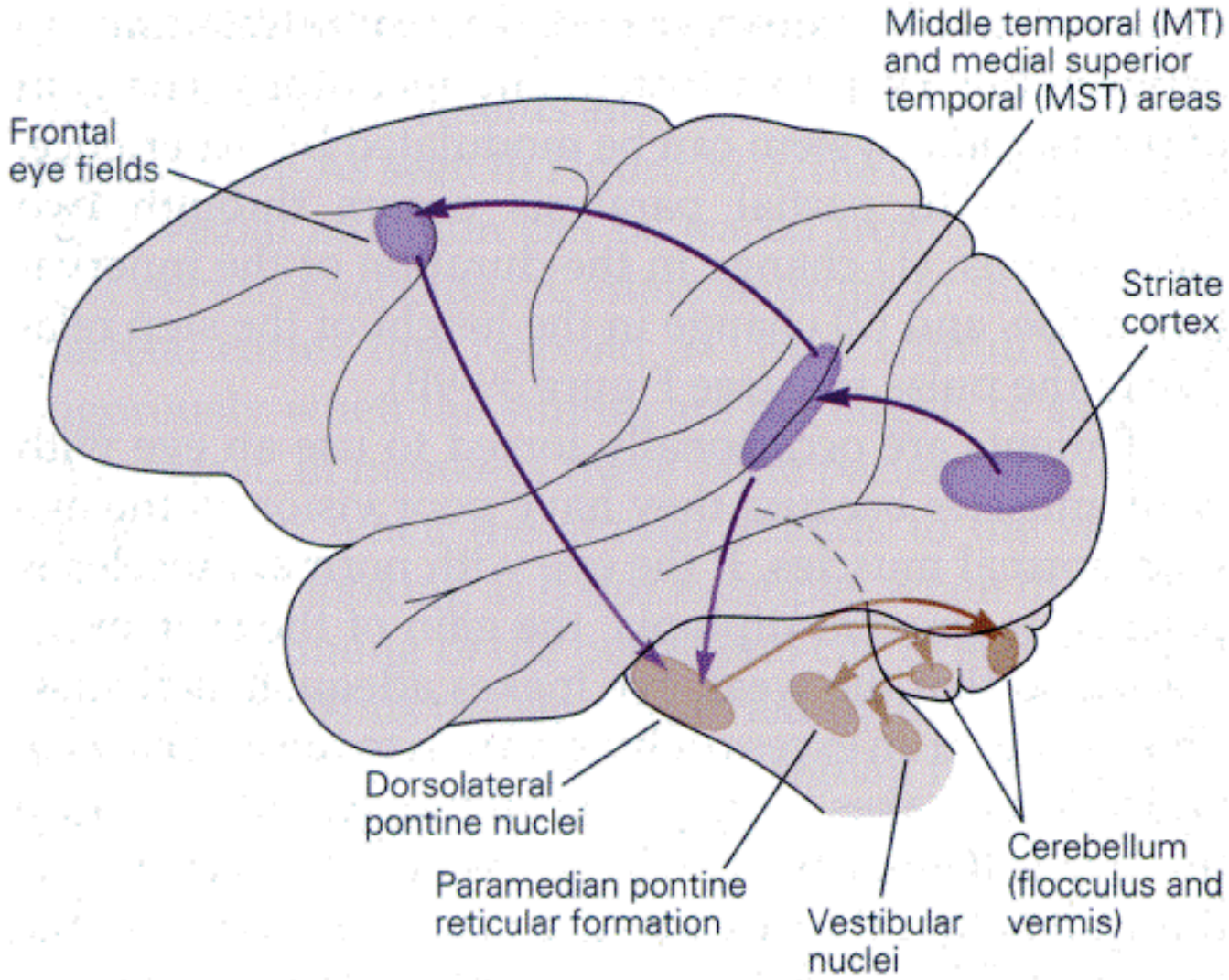


Two main kinds of eye movements

- ***Smooth pursuit***



Outline of smooth pursuit system

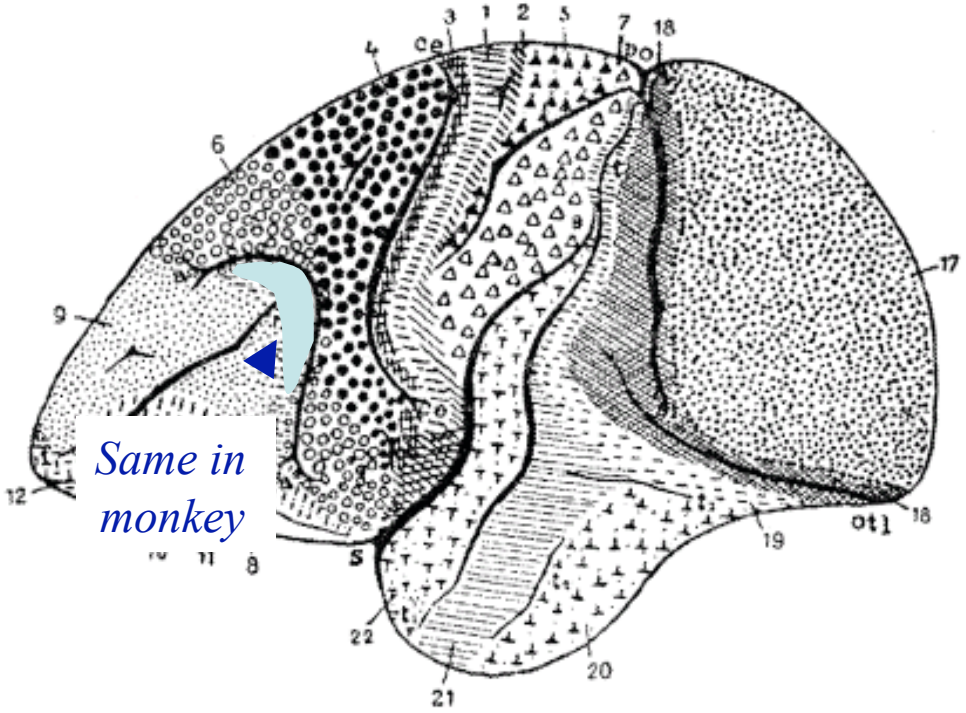


Frontal Eye Field

- At the intersection of prefrontal cortex and premotor/motor cortex.

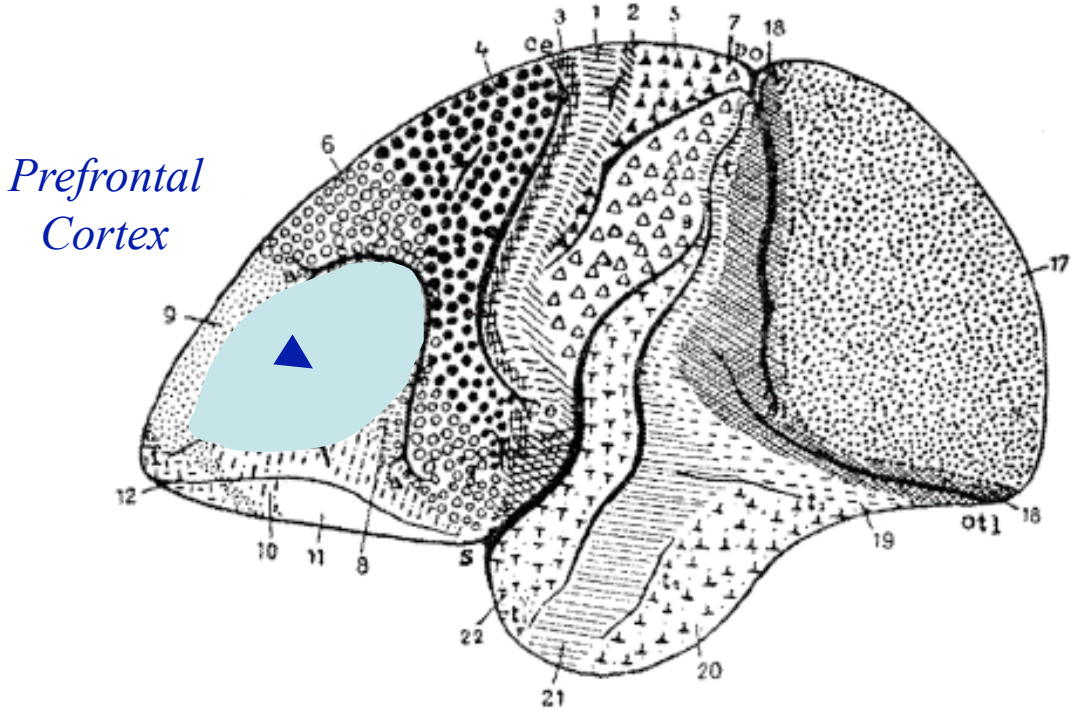
MONKEY

Brodmann's areas (from 1905)



MONKEY

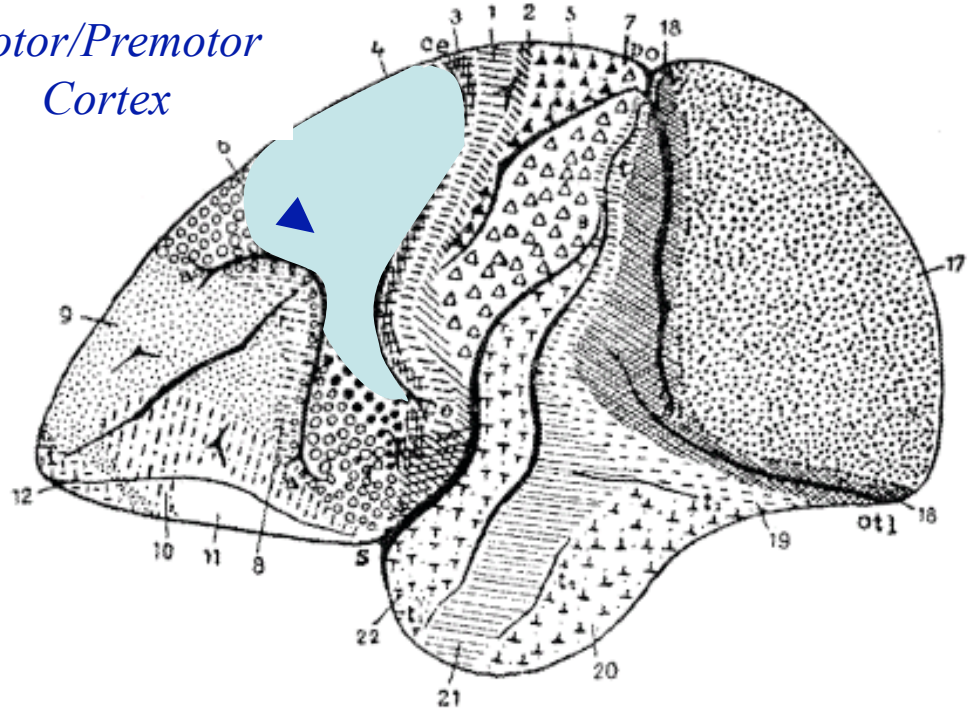
Brodmann's areas (from 1905)



MONKEY

Brodmann's areas (from 1905)

Motor/Premotor Cortex



Frontal Eye Field

- This position at juncture of “cognition” area and “movement” area thought to be critical, because saccades are so important for our visual analyses and decisions.

Exploring the Visual Scene

(Yarbus 1967)

Exploring the Visual Scene

Baseline: No Instructions

"Estimate Ages of People"