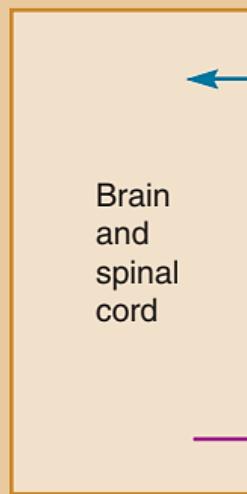


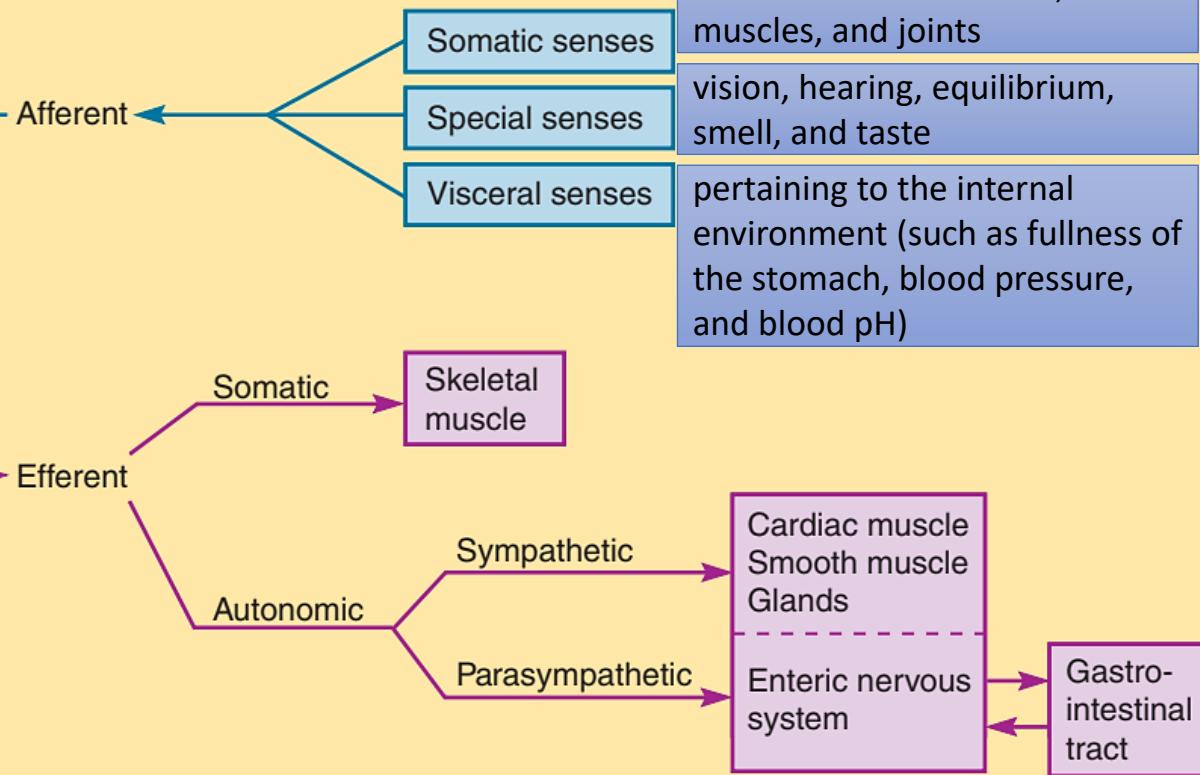
CNS: sensory, motor, higher functions, special senses

Organization of the nervous system

Central Nervous System



Peripheral Nervous System



Neurons

- The human CNS contains about 10^{11} neurons. It also contains 2–10 times this number of glial cells.
- Neurons have four specialized regions:
 1. cell body, or perikaryon or soma
 2. dendrites,
 3. axon, and
 4. presynaptic terminals or telodendria
- Mature neurons have no centrioles and cannot undergo any further mitosis

Dendrites

Typical dendrites are highly branched, with each branch bearing fine 0.5 to 1 μm long processes called **dendritic spines**. CNS neurons receive most of their information primarily at the dendritic spines.

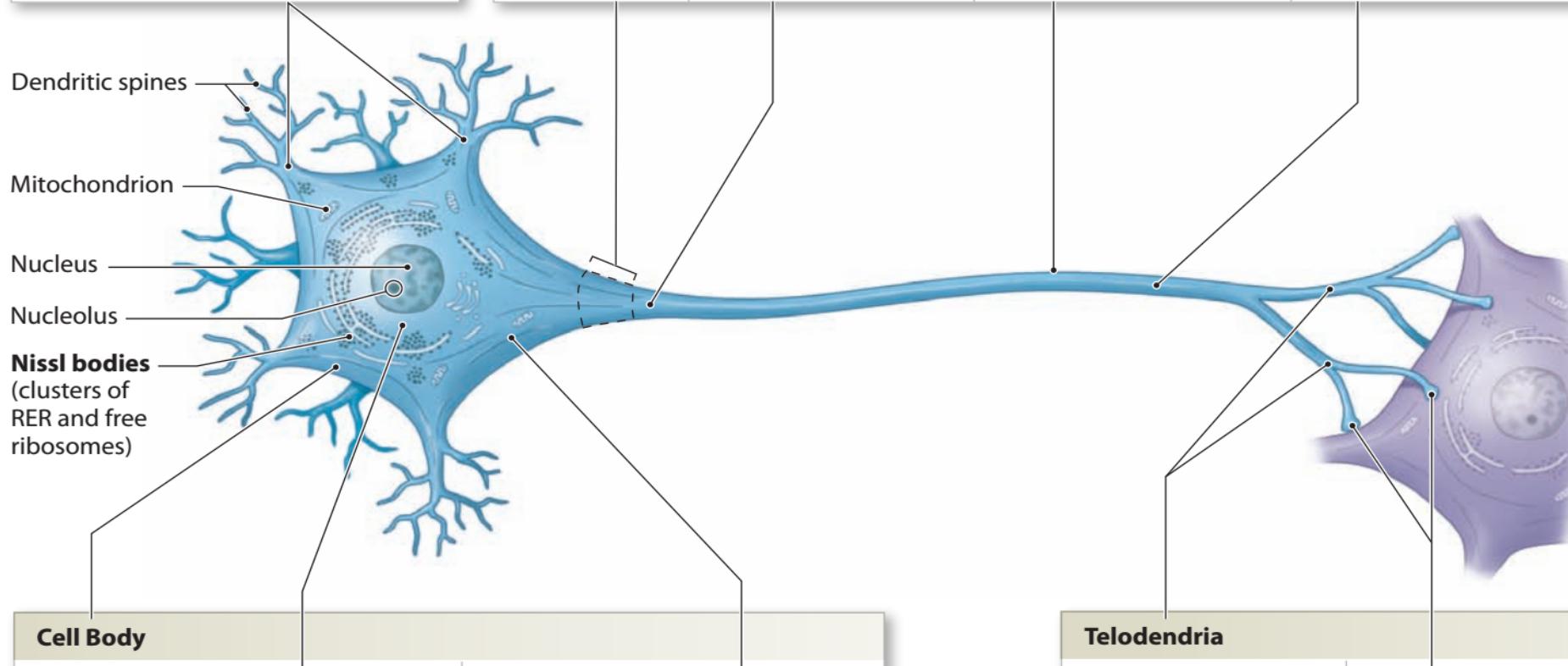
Axon

The **axon hillock** is the origin of the axon from the cell body.

The **initial segment** of the axon lies distally adjacent to the axon hillock. It is where an action potential is initiated.

The **axolemma** (*lemma, husk*) is a specialized portion of the plasma membrane that surrounds the cytoplasm (axoplasm) of the axon.

The **axoplasm** (AK-sō-plazm) contains neurofibrils, neurotubules, small vesicles, lysosomes, mitochondria, and various enzymes.



Cell Body

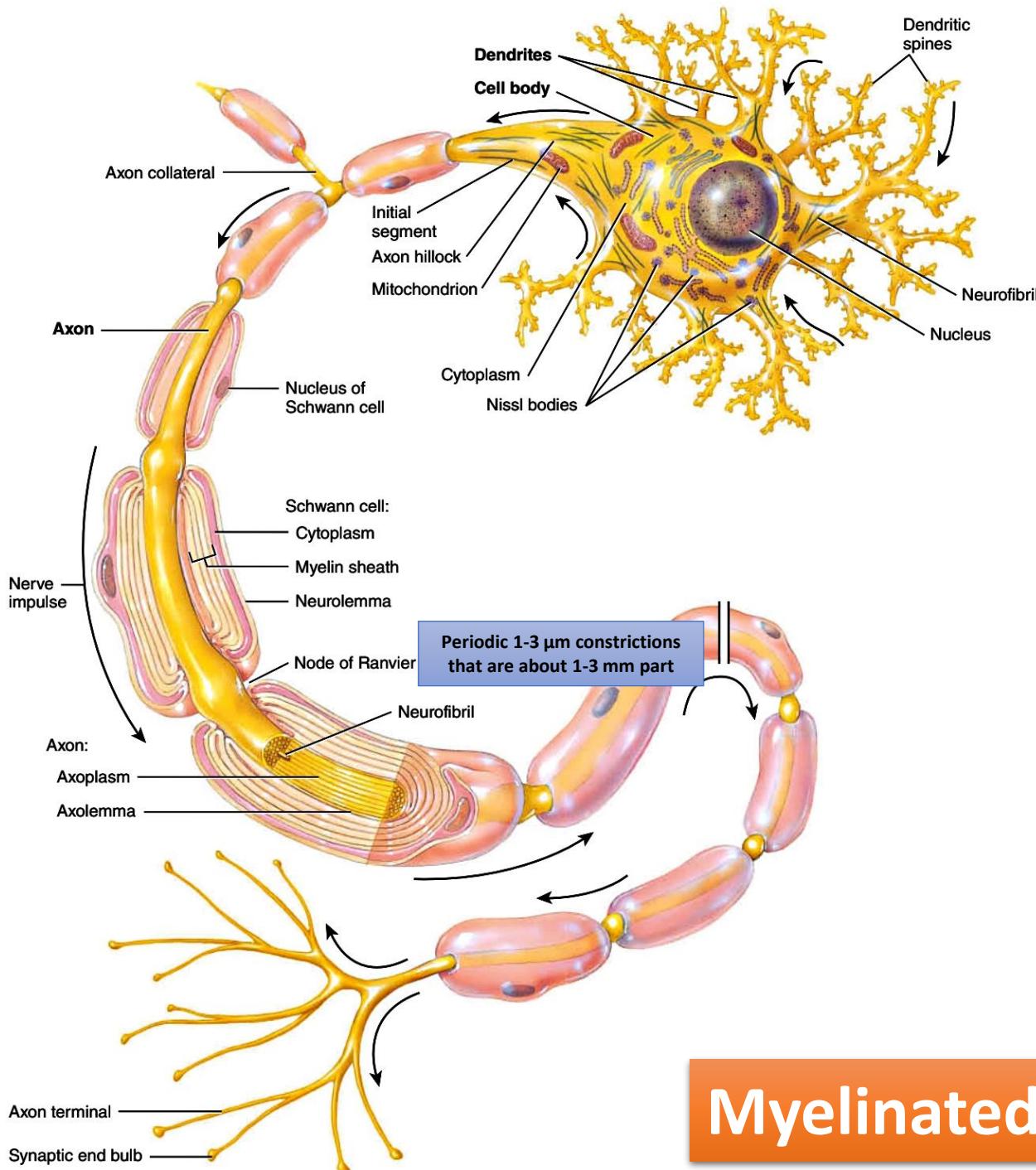
The cytoplasm surrounding the nucleus is called the **perikaryon** (per-i-KAR-ē-on; *peri*, around + *karyon*, nucleus). The perikaryon contains organelles that provide energy and synthesize the chemical neurotransmitters that are important in cell-to-cell communication.

The cytoskeleton of the perikaryon contains **neurofilaments** similar to the intermediate filaments in other cells. **Neurofibrils** are bundles of neurofilaments that extend into the dendrites and axon, providing internal support for these slender processes.

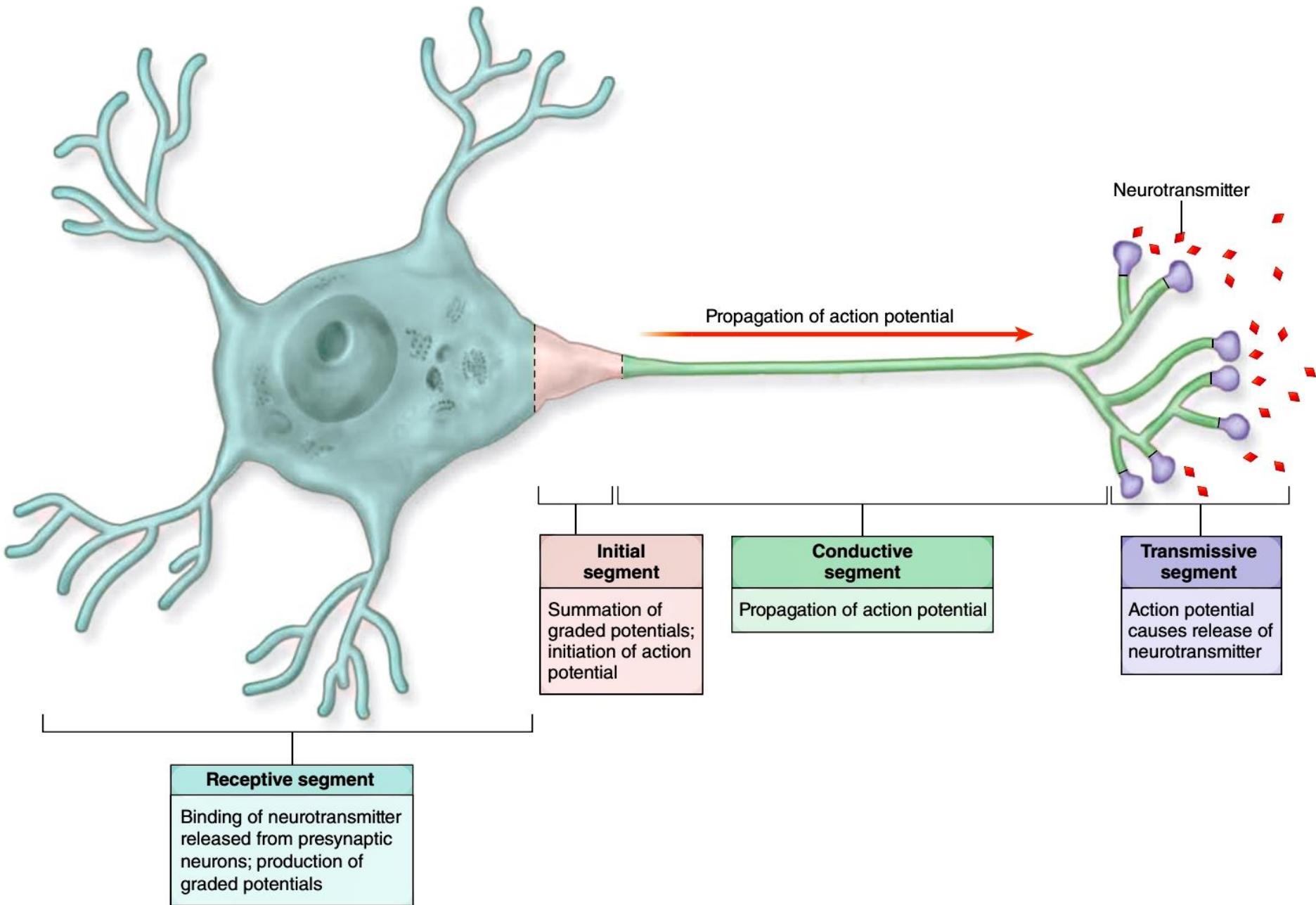
Telodendria

The main axon trunk ends in a series of fine extensions, or **telodendria** (tel-ō-DEN-drē-uh; *telo*, end + *dendron*, tree; singular: **telodendron**).

The telodendria of an axon end at **axon terminals**, or **synaptic terminals**, where the neuron communicates with other cells.



Myelinated neuron

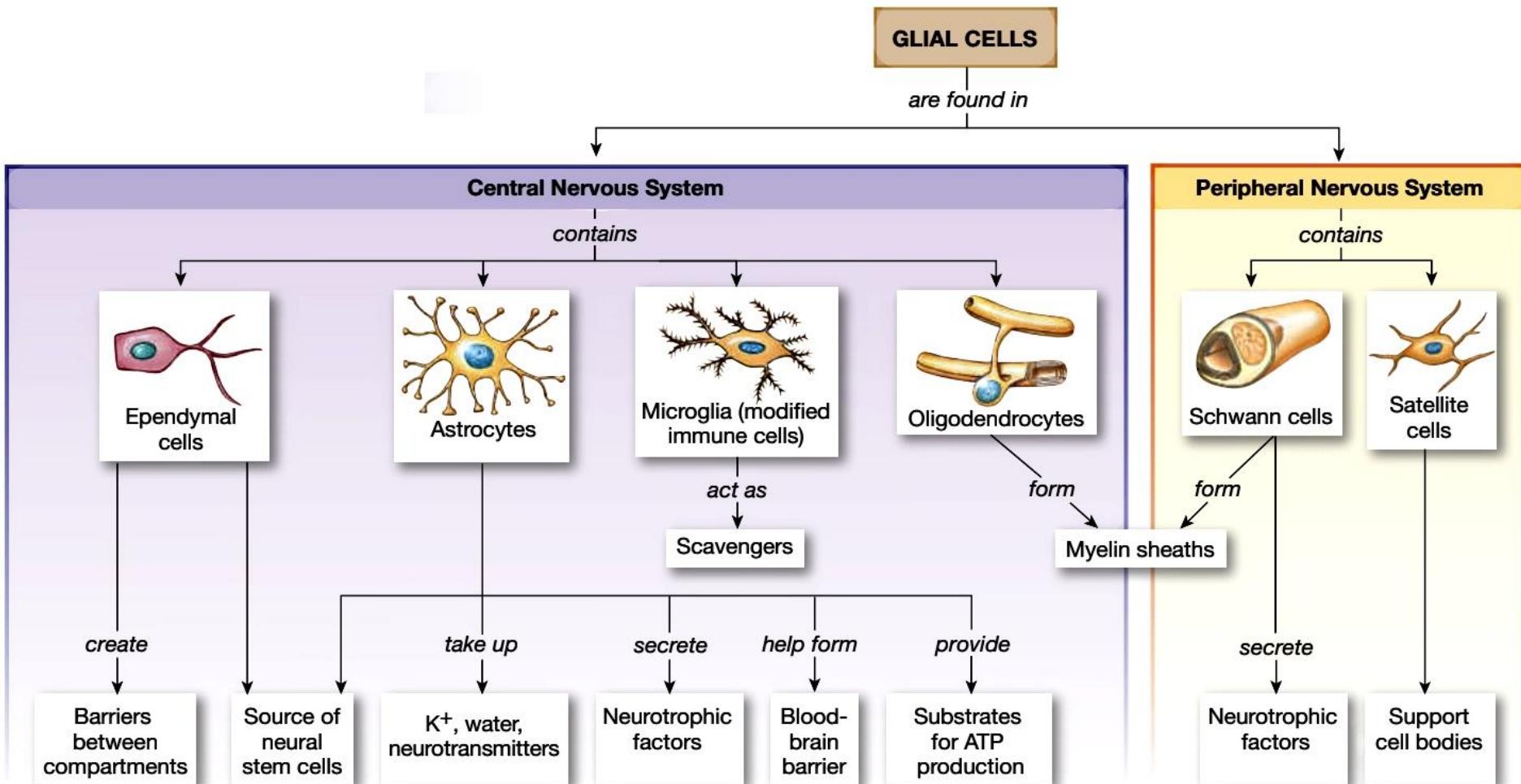


Myelinated nerves

Unmyelinated nerves

- | | |
|--|---|
| 1. Have axons of large diameter. | Have axons of small diameter. |
| 2. Axons surrounded by concentric layers of Schwann cell plasma membrane. | Axons surrounded by cytoplasm of Schwann cells. |
| 3. Nerve impulse jumps from one node to the other node, which is called saltatory conduction. | Nerve impulse travels uniformly along the axolemma. |
| 4. Density of voltage gated Na^+ channels are more (about $350\text{-}500/\mu\text{m}^2$ at initial segment, and 2,000 to $12,000/\mu\text{m}^2$ in node of Ranvier). | Na^+ channels are less in axons (about $110/\mu\text{m}^2$). |
| 5. Saltatory conduction seen in myelinated nerves is fast and consumes less energy. | Conduction seen in unmyelinated nerves is slow and consumes more energy. |
| 6. Examples: All preganglionic fibers in ANS. In PNS, fibers $>1 \mu\text{m}$ in diameter. | All post-ganglionic fibers in ANS. In PNS, fibers $<1 \mu\text{m}$ in diameter. |

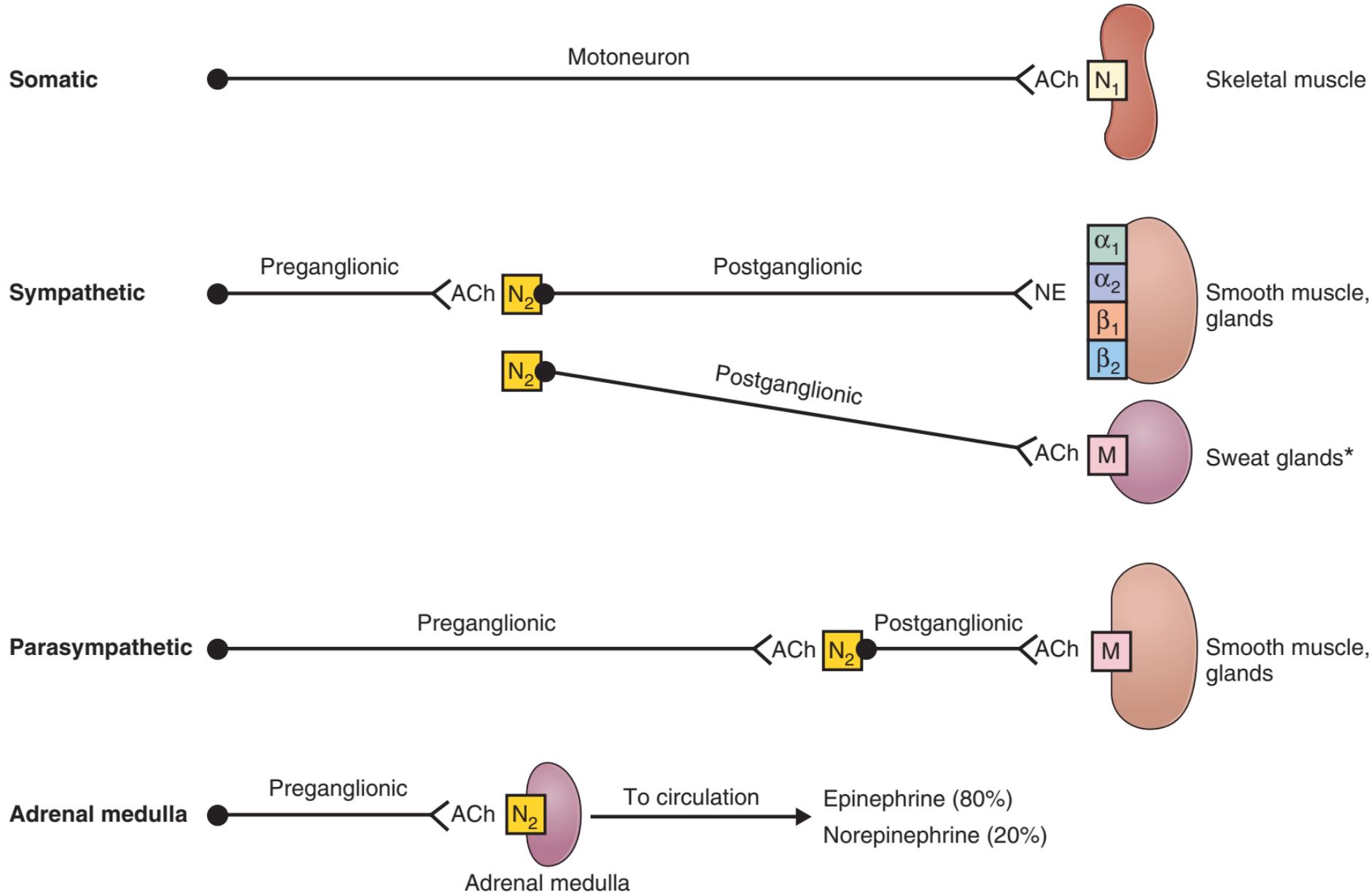
Cells of nervous system: Glial cells



Macroglia: oligodendrocytes, Schwann cells, and astrocytes

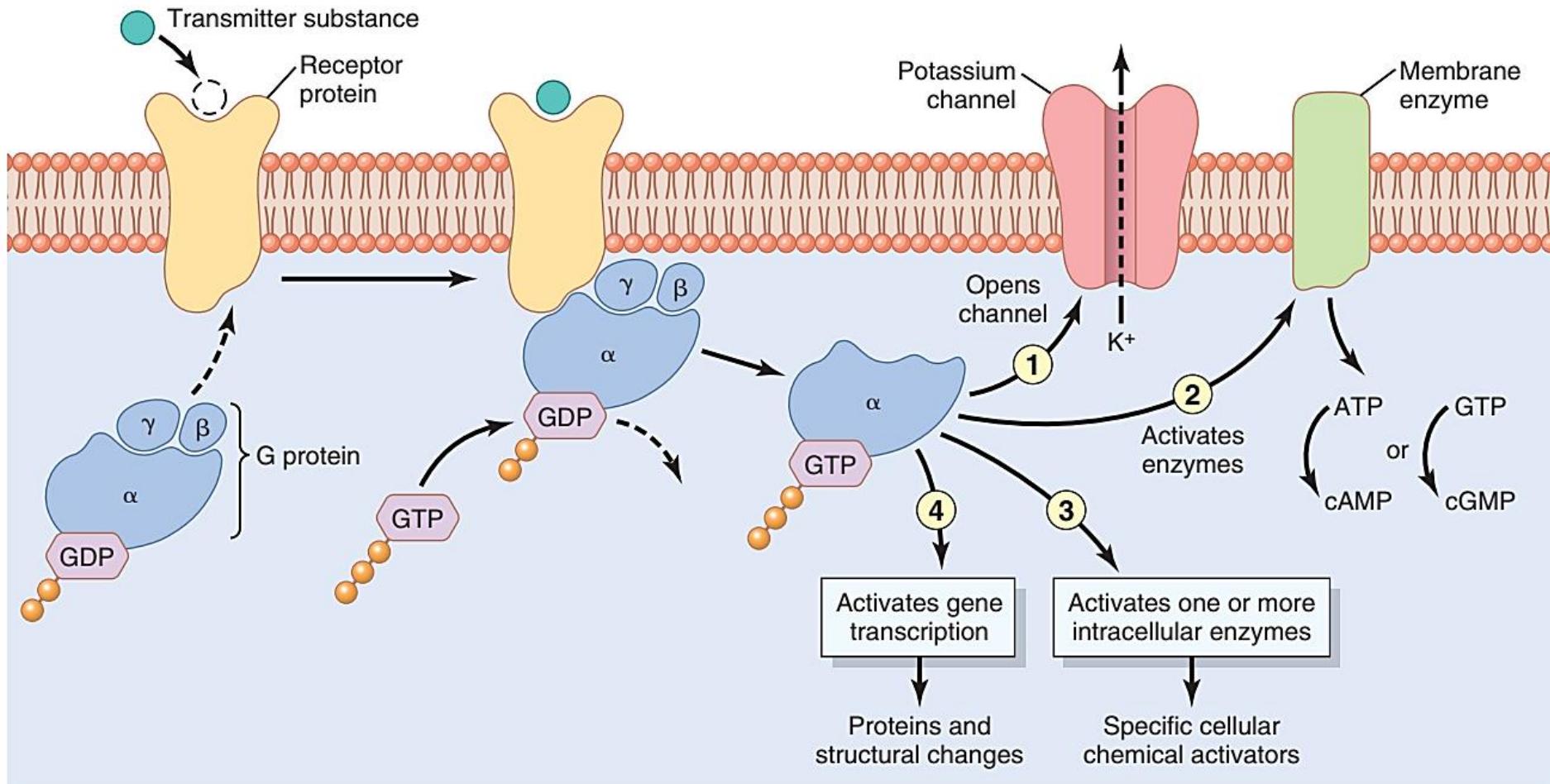
CENTRAL NERVOUS SYSTEM

EFFECTOR ORGANS



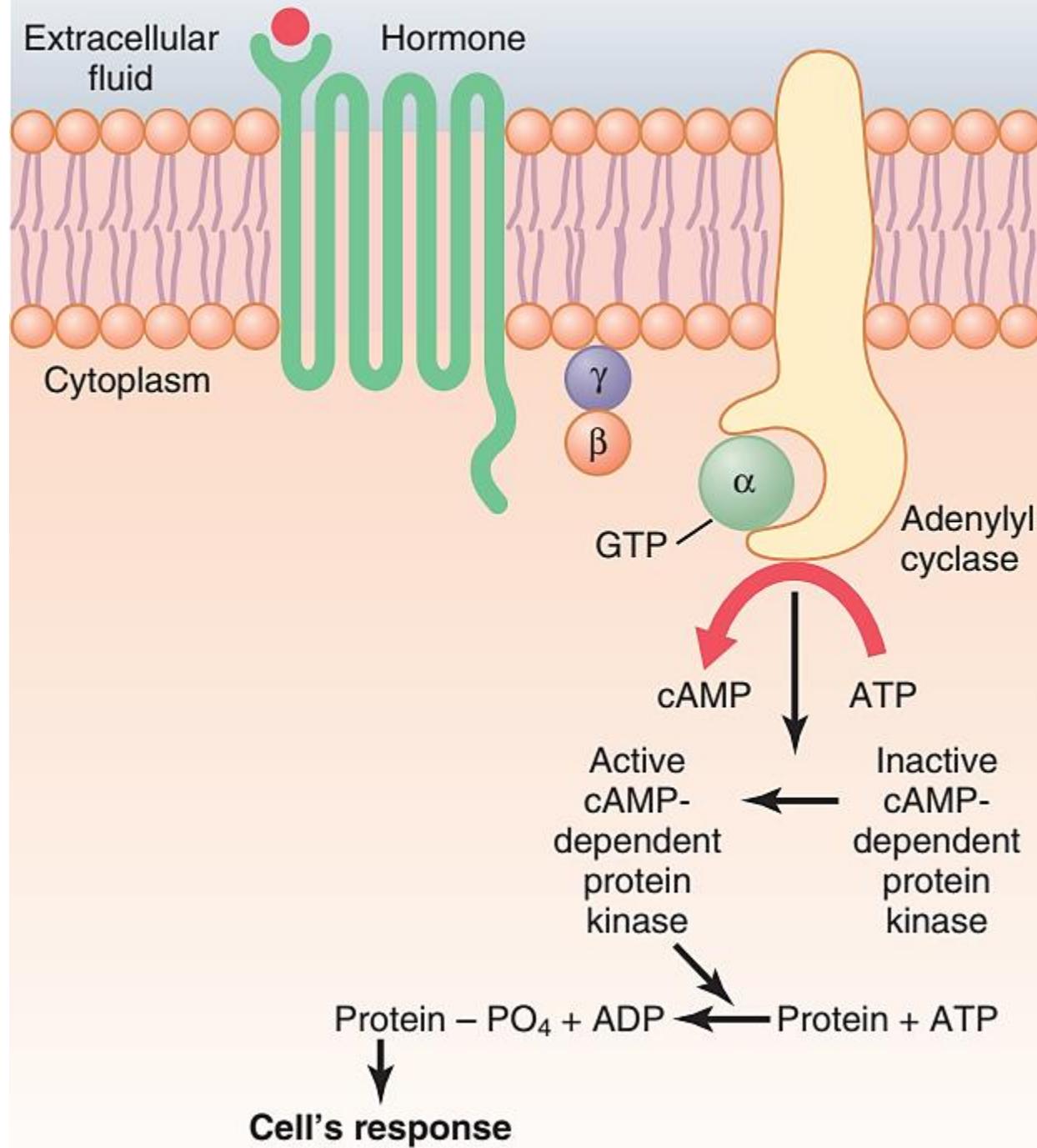
Characteristic	Sympathetic	Parasympathetic	Somatic*
Origin of preganglionic nerve	Nuclei of spinal cord segments T1–T12; L1–L3 (thoracolumbar)	Nuclei of cranial nerves III, VII, IX, and X; spinal cord segments S2–S4 (craniosacral)	
Length of preganglionic nerve axon	Short	Long	
Neurotransmitter in ganglion	ACh	ACh	
Receptor type in ganglion	Nicotinic	Nicotinic	
Length of postganglionic nerve axon	Long	Short	
Effector organs	Smooth and cardiac muscle; glands	Smooth and cardiac muscle; glands	Skeletal muscle
Neurotransmitter in effector organs	Norepinephrine (except sweat glands, which use ACh)	ACh	ACh (synapse is neuromuscular junction)
Receptor types in effector organs	α_1 , α_2 , β_1 , and β_2	Muscarinic	Nicotinic

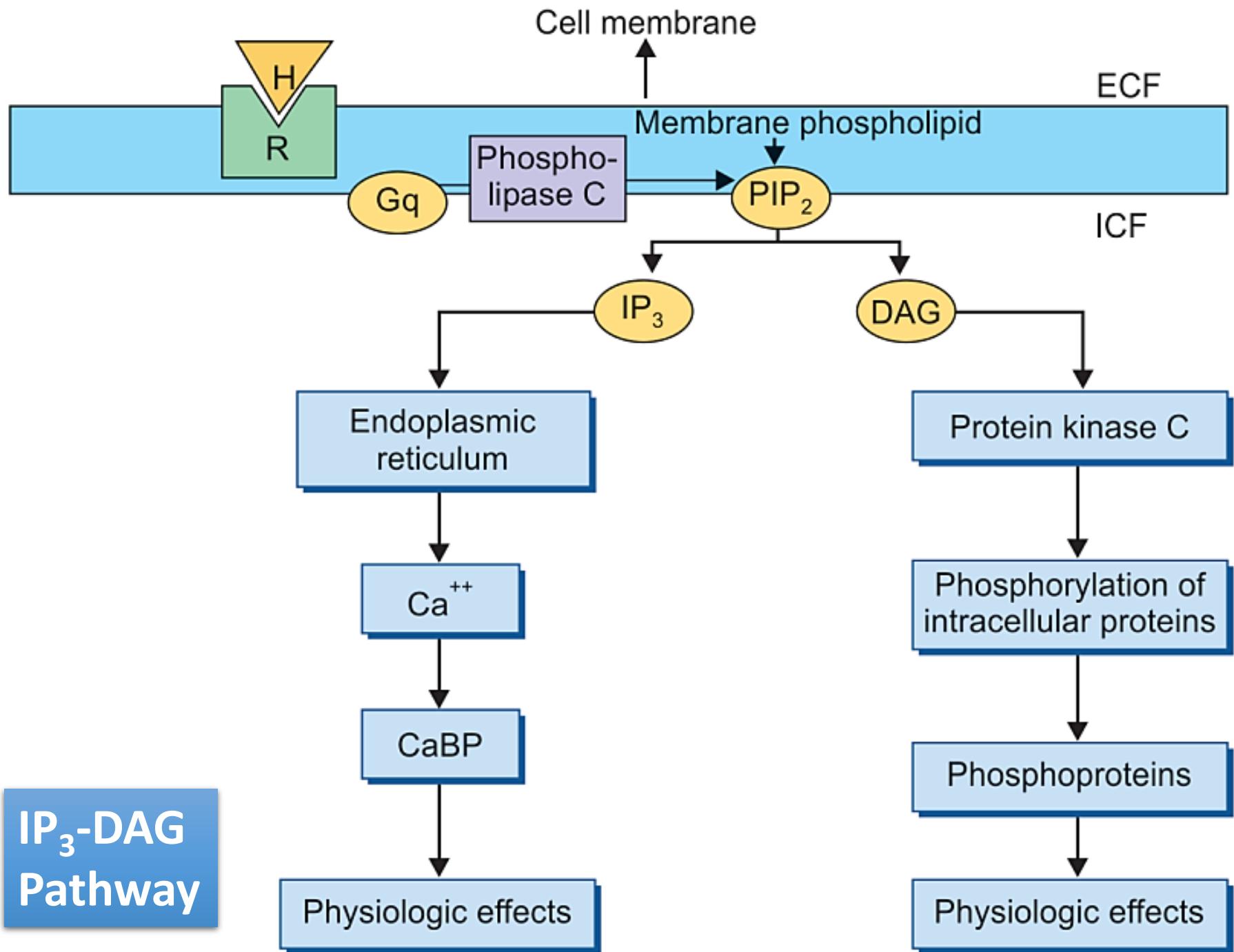
Receptor	Location	G Protein	Mechanism
Adrenergic			
α_1	Smooth muscle	G_q	$\uparrow IP_3/Ca^{2+}$
α_2	Gastrointestinal tract	G_i	$\downarrow cAMP$
β_1	Heart	G_s	$\uparrow cAMP$
β_2	Smooth muscle	G_s	$\uparrow cAMP$
Cholinergic			
$N_M (N_1)$	Skeletal muscle	—	Opening Na^+/K^+ channels
$N_N (N_2)$	Autonomic ganglia	—	Opening Na^+/K^+ channels
M_1	CNS	G_q	$\uparrow IP_3/Ca^{2+}$
M_2	Heart	G_i	$\downarrow cAMP$
M_3	Glands, smooth muscle	G_q	$\uparrow IP_3/Ca^{2+}$



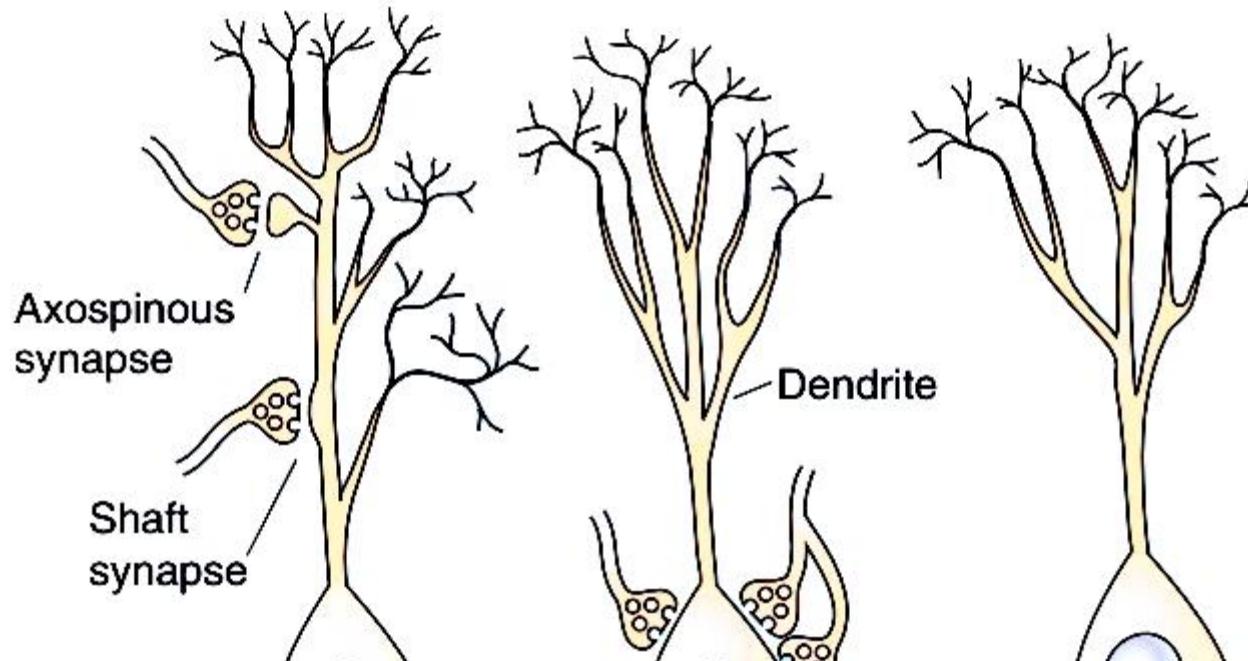
The “second messenger” system by which a transmitter substance from an initial neuron can activate a second neuron by first causing a transformational change in the receptor that releases the activated alpha (α) subunit of the G protein into the second neuron’s cytoplasm. Four subsequent possible effects of the G protein are shown, including 1, opening an ion channel in the membrane of the second neuron; 2, activating an enzyme system in the neuron’s membrane; 3, activating an intracellular enzyme system; and/or 4, causing gene transcription in the second neuron. Return of the G protein to the inactive state occurs when guanosine triphosphate (GTP) bound to the α subunit is hydrolyzed to guanosine diphosphate (GDP) and the β and γ subunits are reattached to the α subunit.

- Types of second messengers:
 1. cAMP
 2. cGMP
 3. Inositol triphosphate (IP₃) and Diacylglycerol (DAG)
 4. Calcium ions

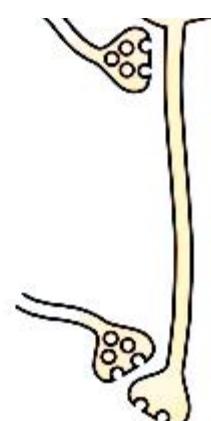
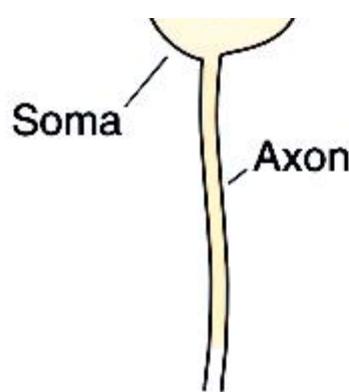
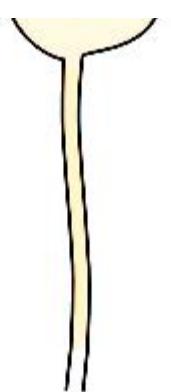




Organ	Sympathetic Action	Sympathetic Receptor	Parasympathetic Action
Heart	↑ heart rate ↑ contractility ↑ AV node conduction	β_1 β_1 β_1	↓ heart rate ↓ contractility (atria) ↓ AV node conduction
Vascular smooth muscle	Constricts blood vessels in skin; splanchnic Dilates blood vessels in skeletal muscle	α_1 β_2	— —
Gastrointestinal tract	↓ Motility Constricts sphincters	α_2, β_2 α_1	↑ Motility Relaxes sphincters
Bronchioles	Dilates bronchiolar smooth muscle	β_2	Constricts bronchiolar smooth muscle
Male sex organs	Ejaculation	α_2	Erection
Bladder	Relaxes bladder wall Constricts sphincter	β_2 α_1	Contracts bladder wall Relaxes sphincter
Sweat glands	↑ sweating	Muscarinic (sympathetic cholinergic)	—
Kidney	↑ renin secretion	β_1	—
Fat cells	↑ lipolysis	β_1	—



commonest type of synapse in brain → Axodendritic SYnapse



Axodendritic
synapses

Axosomatic
synapses

Axoaxonic
synapses

The most common synaptic arrangements in the CNS.

NEUROTRANSMITTERS IN CNS

Two main types of neurotransmitter:

1. Excitatory: Glutamate
2. Inhibitory: GABA, Glycine

All these three neurotransmitters (glutamate, GABA, glycine) are amino acids in nature.

Synapse

- Synapse are the junctions where the axon or some other portion of one cell (the presynaptic cell) terminates on the dendrites, soma, or axon of another neuron or, in some cases, a muscle or gland cell (the postsynaptic cell).
- There are two major types of synapses
 1. Chemical
 2. Electrical
- Conjoint synapse: both electrical and chemical

Genesis of Postsynaptic Potential

- Graded potentials that occur in postsynaptic neurons are specifically called postsynaptic potentials.
- Postsynaptic potentials that result in the neuron becoming more positive (depolarisation) are called excitatory postsynaptic potentials (EPSPs),
- Those result in the neuron becoming more negative (hyperpolarisation) are called inhibitory postsynaptic potentials (IPSPs)

Types of EPSP

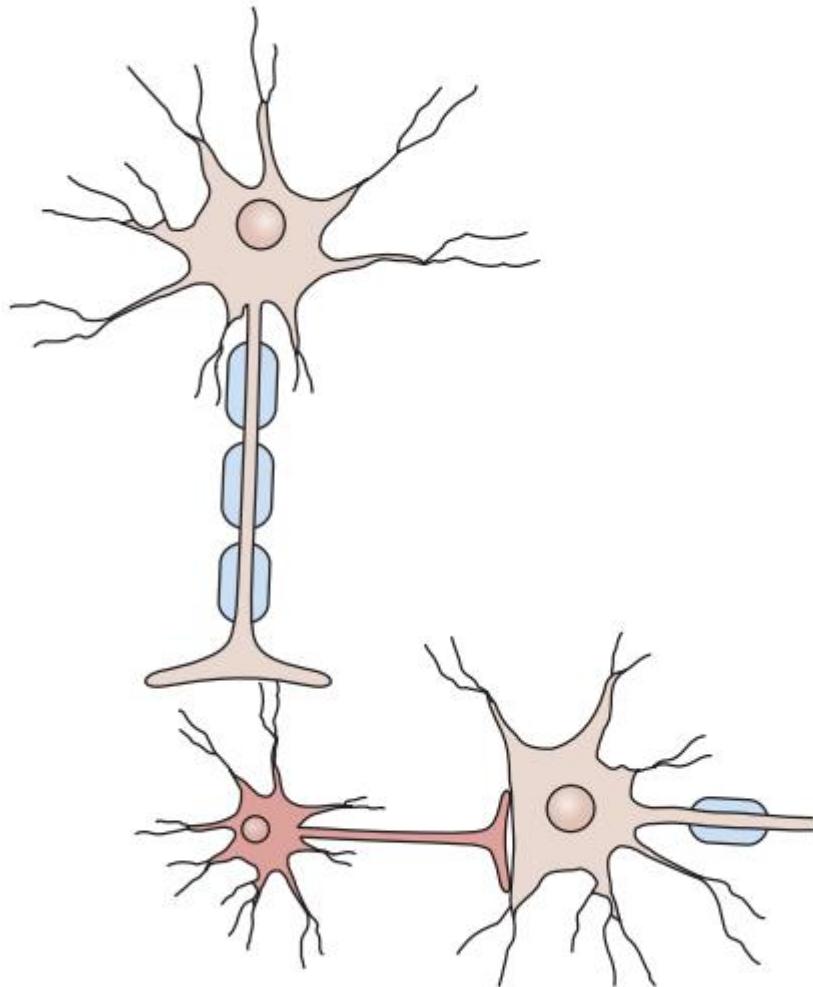
Type	Cause	Site
EPSP	Na and/or, Ca influx	All
Slow EPSP	Decrease K efflux	Autonomic ganglia (muscarinic cholinergic), cardiac muscle, smooth muscle, cortical neurons
Late slow EPSP	Decrease K efflux	Sympathetic ganglia

Types of IPSP

Type	Cause	Site
IPSP	Cl ⁻ influx	All
Slow IPSP	Increased K efflux	Autonomic ganglia (muscarinic cholinergic), cardiac muscle, smooth muscle, cortical neurons

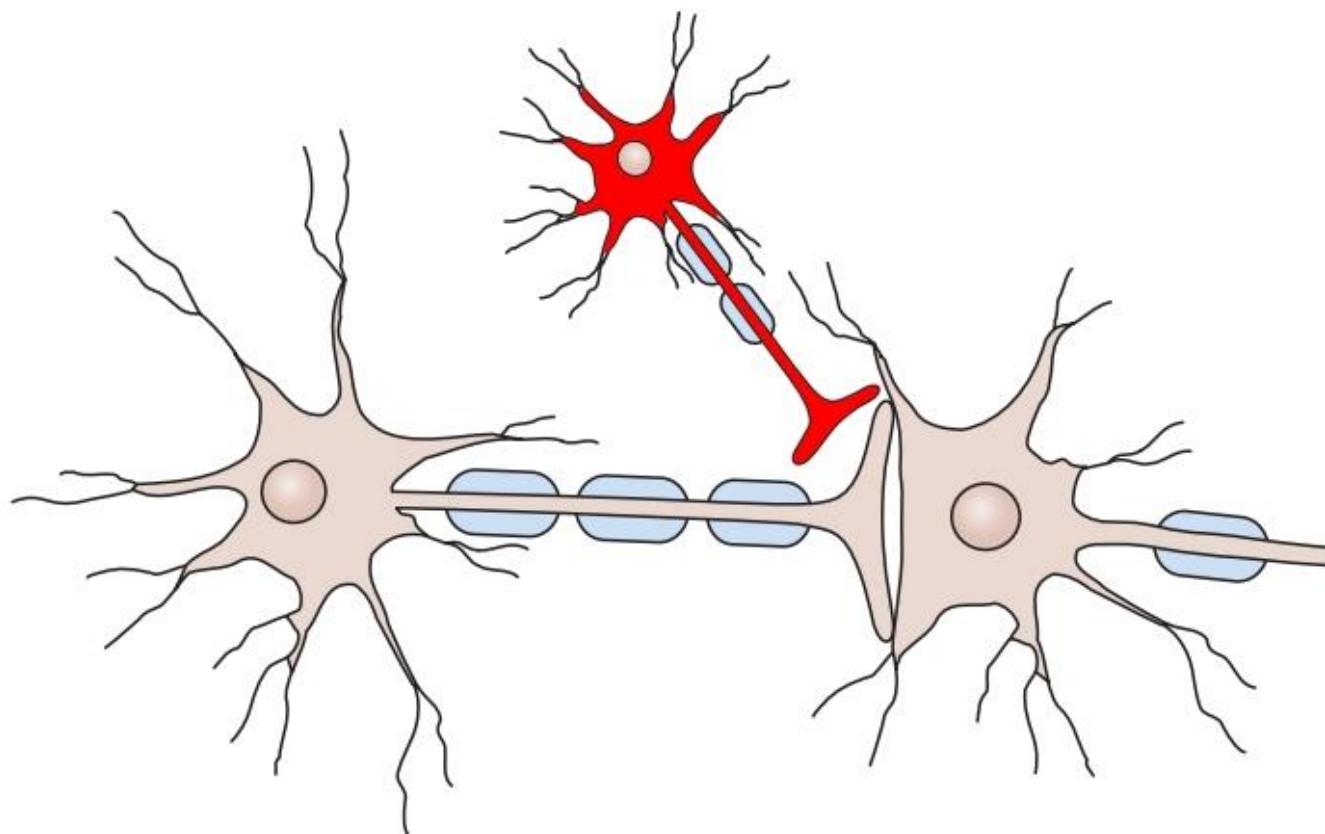
Synaptic Inhibition

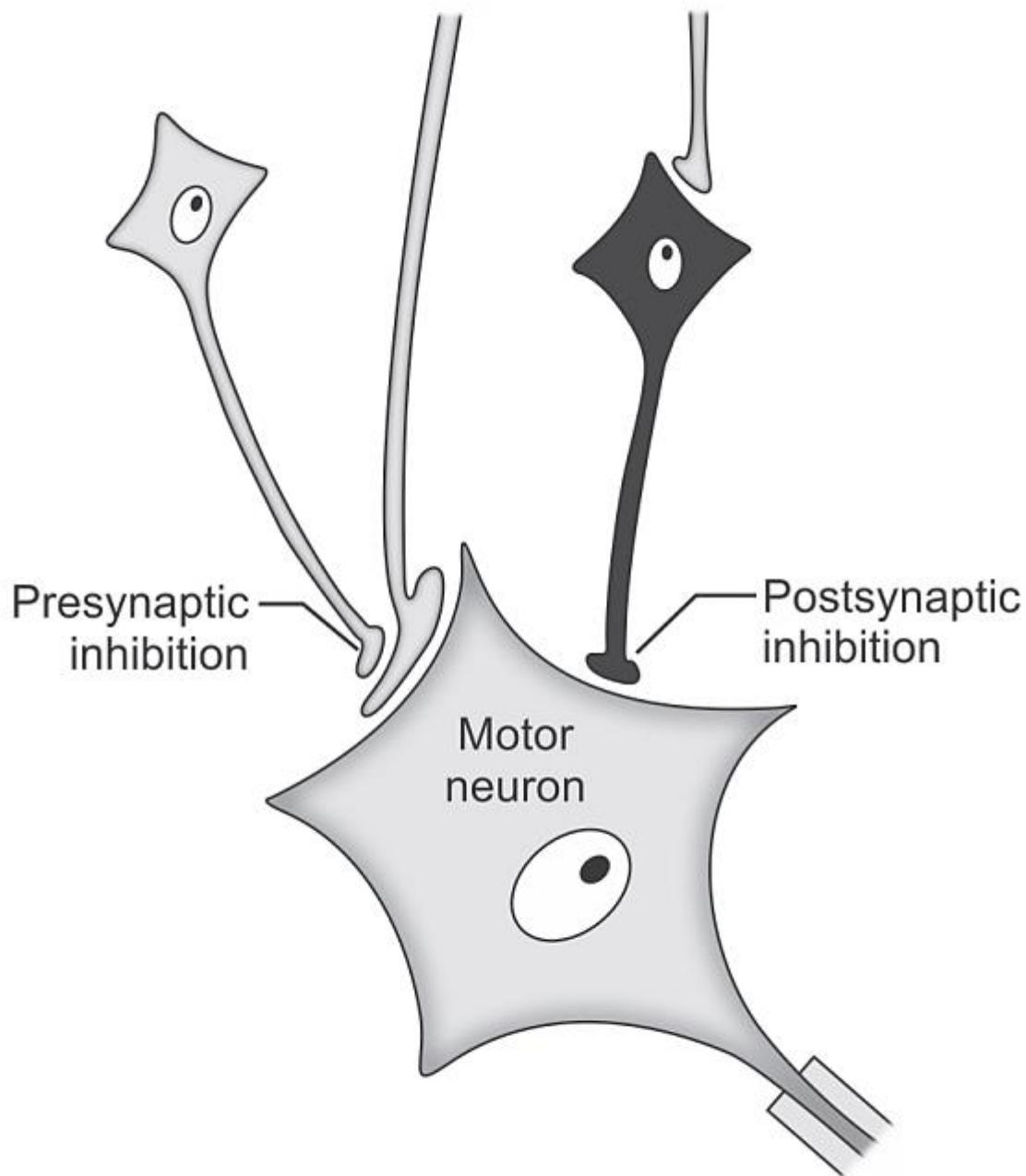
- Types of inhibitions known to occur at synapses in the CNS are:
 1. Post-synaptic inhibition
 2. Pre-synaptic inhibition
 3. Feedback inhibition
 4. Feed forward inhibition



Postsynaptic inhibition occurs when an inhibitory transmitter such as GABA is released from the nerve terminals of an inhibitory interneuron (dark) that synapses on a postsynaptic neuron.

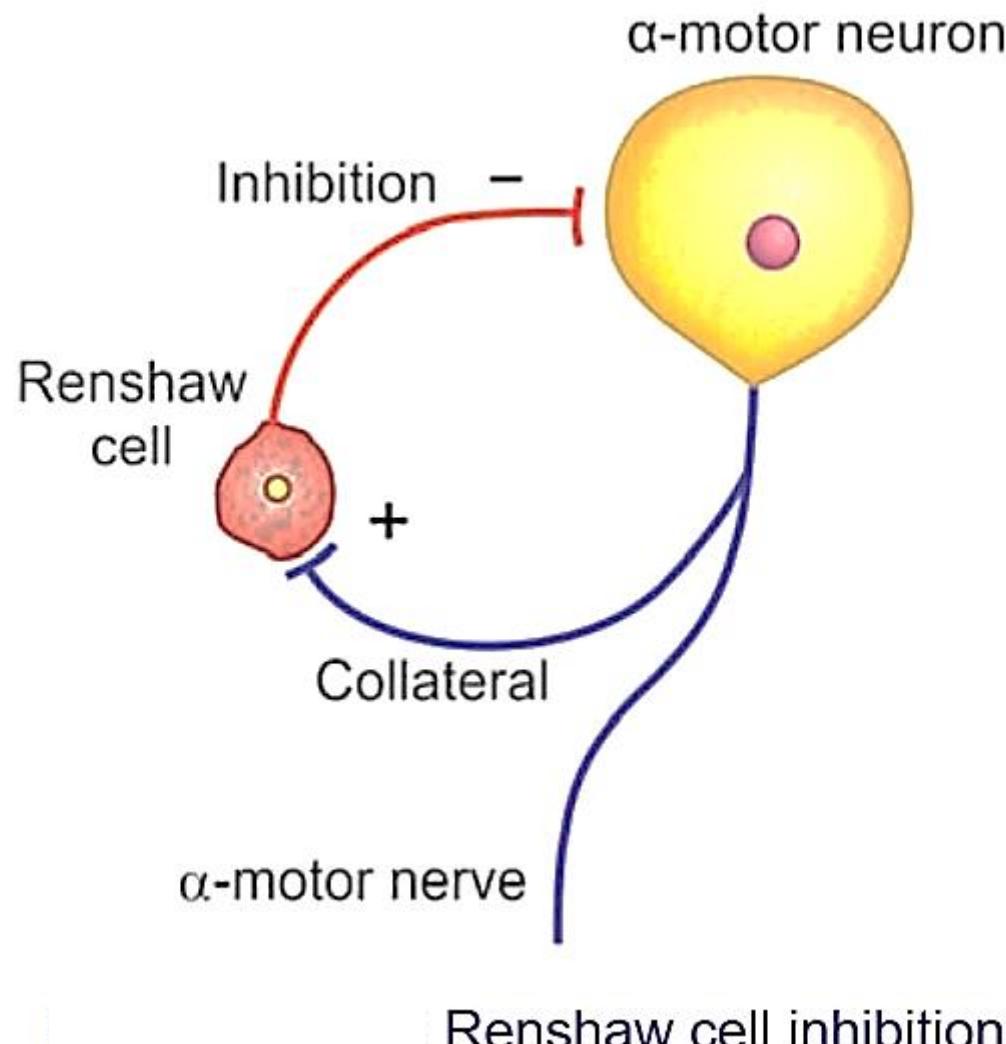
- Presynaptic inhibition is a process mediated by neurons whose terminals are on excitatory endings, forming axo-axonal synapse and reducing transmitter release from the excitatory neuron

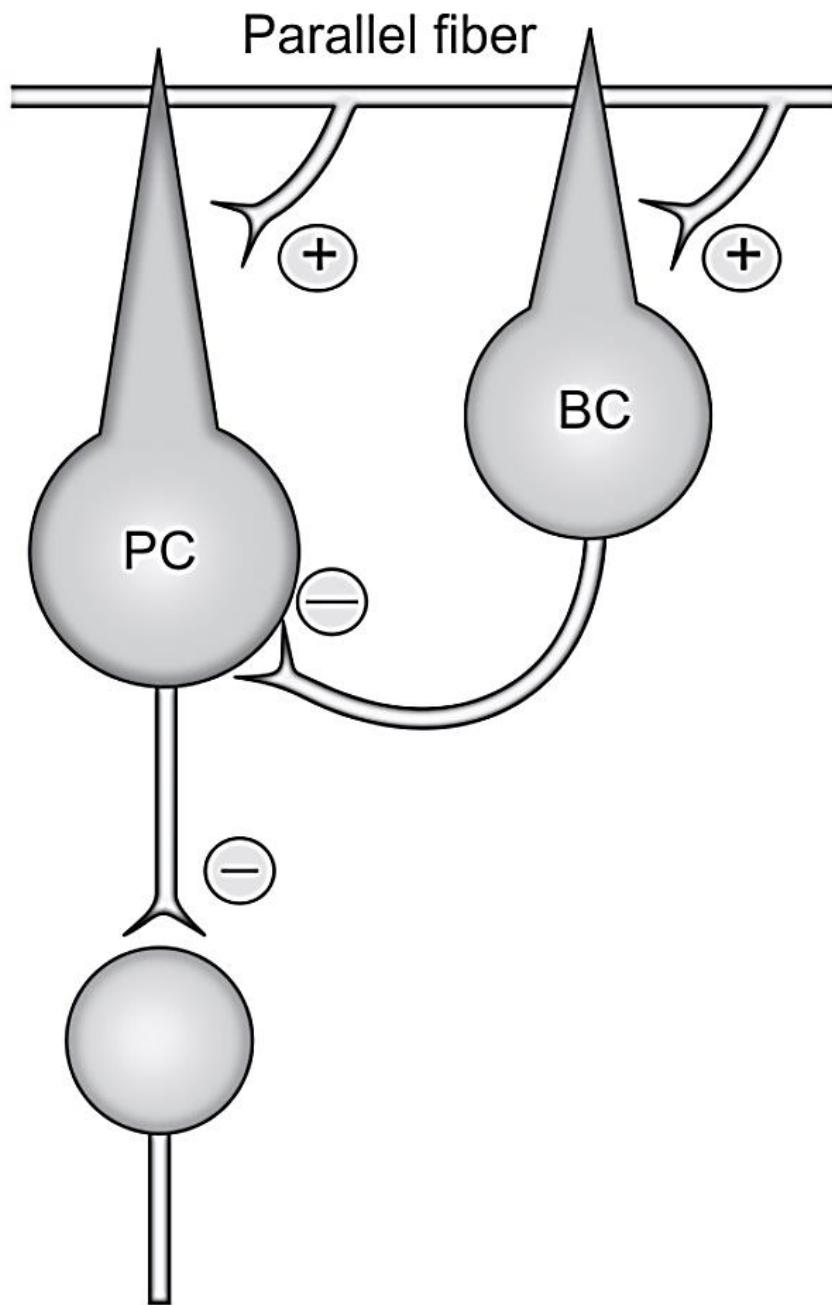




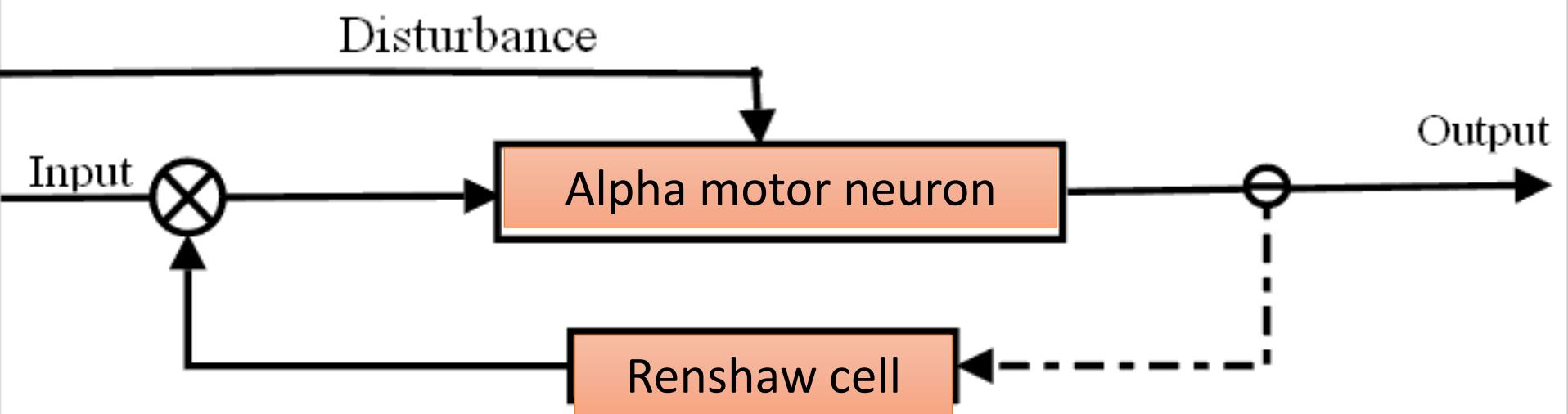
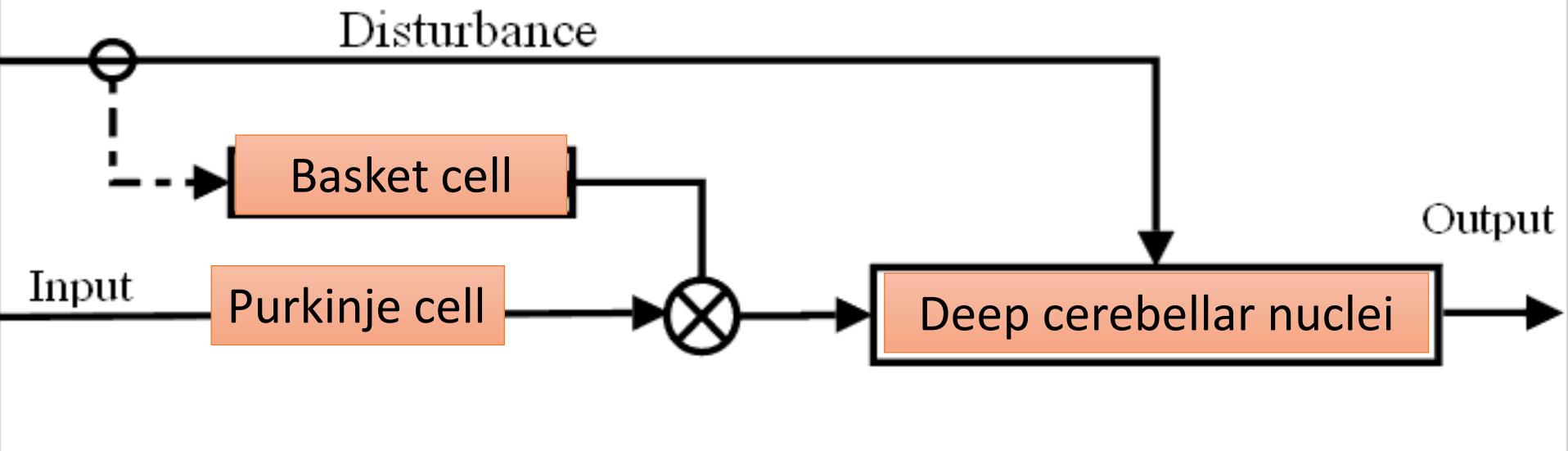
Presynaptic and postsynaptic inhibition

The feedback inhibition, also known as Renshaw cell inhibition, is known to occur in spinal alpha motor neurons through an inhibitory inter-neuron (the Renshaw cell)





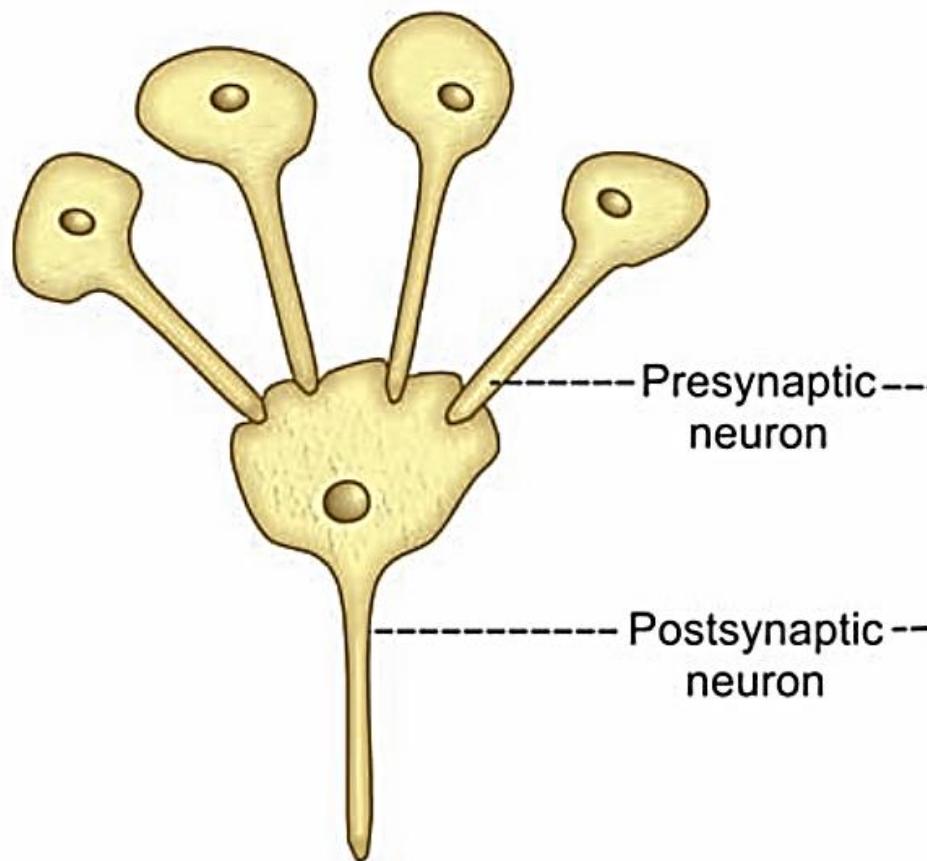
Feed forward inhibition



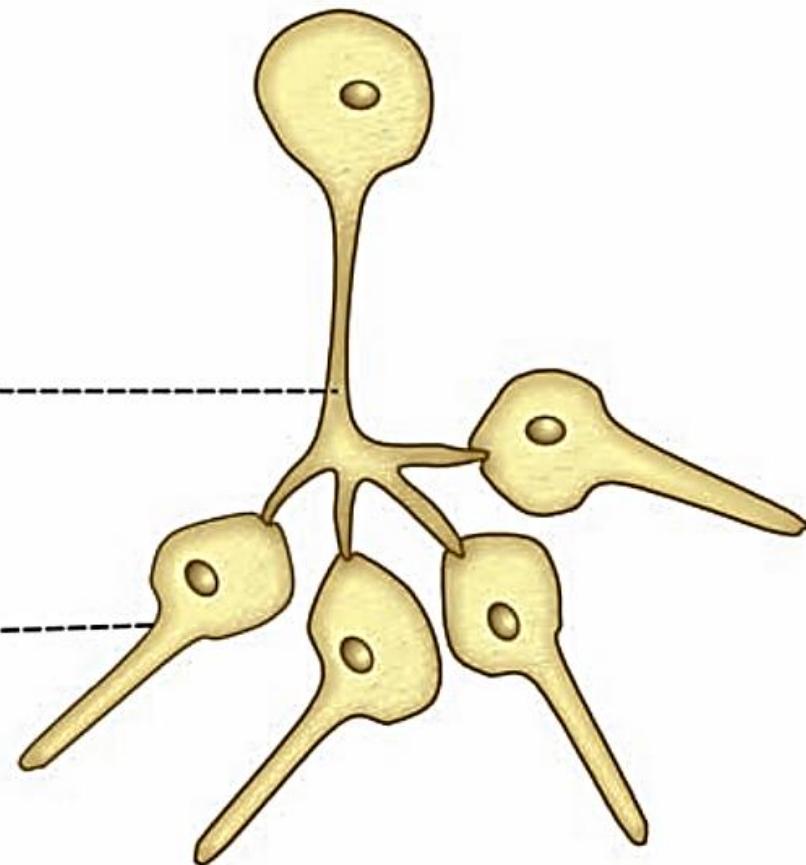
Properties Of Chemical Synaptic Transmission

1. Unidirectional conduction of impulse
2. Synaptic delay (0.5 ms)
3. Synaptic fatigue
4. Convergence and divergence
5. Summation
6. Occlusion
7. Subliminal fringe
8. Synaptic plasticity

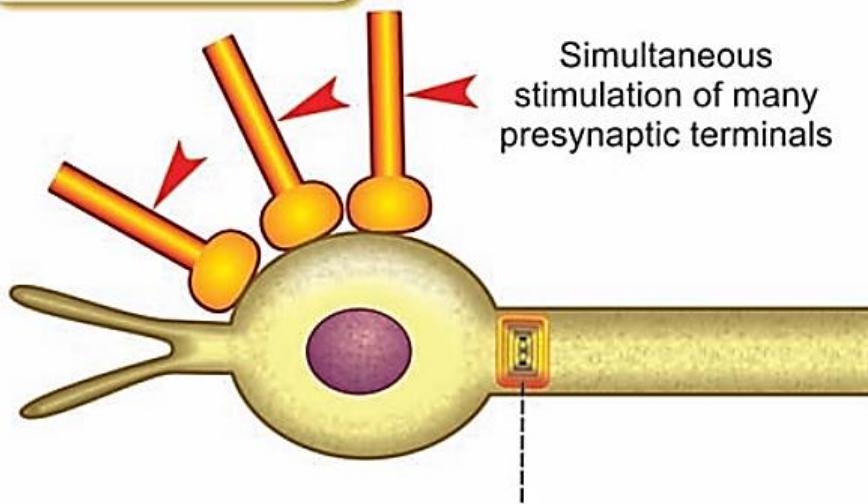
Convergence



Divergence

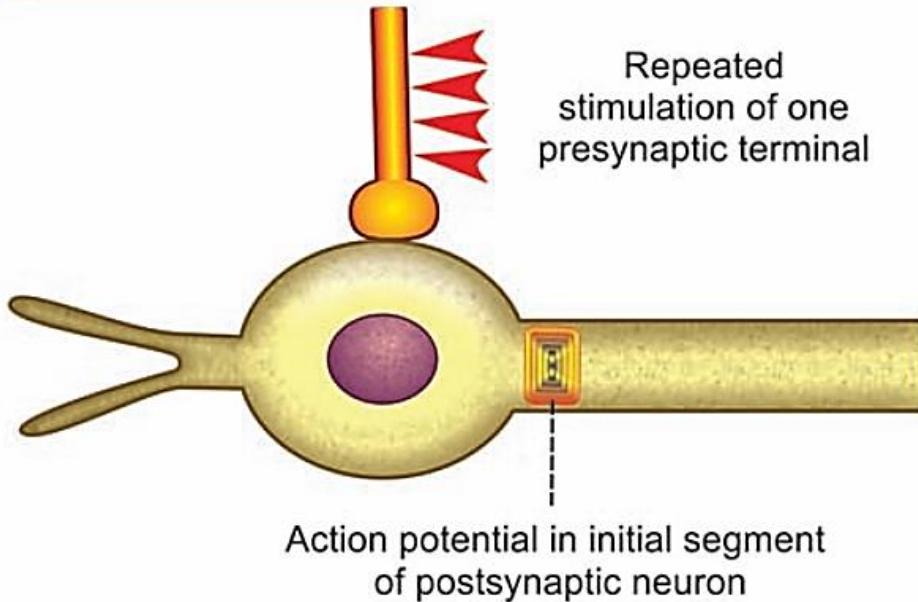


Spatial summation

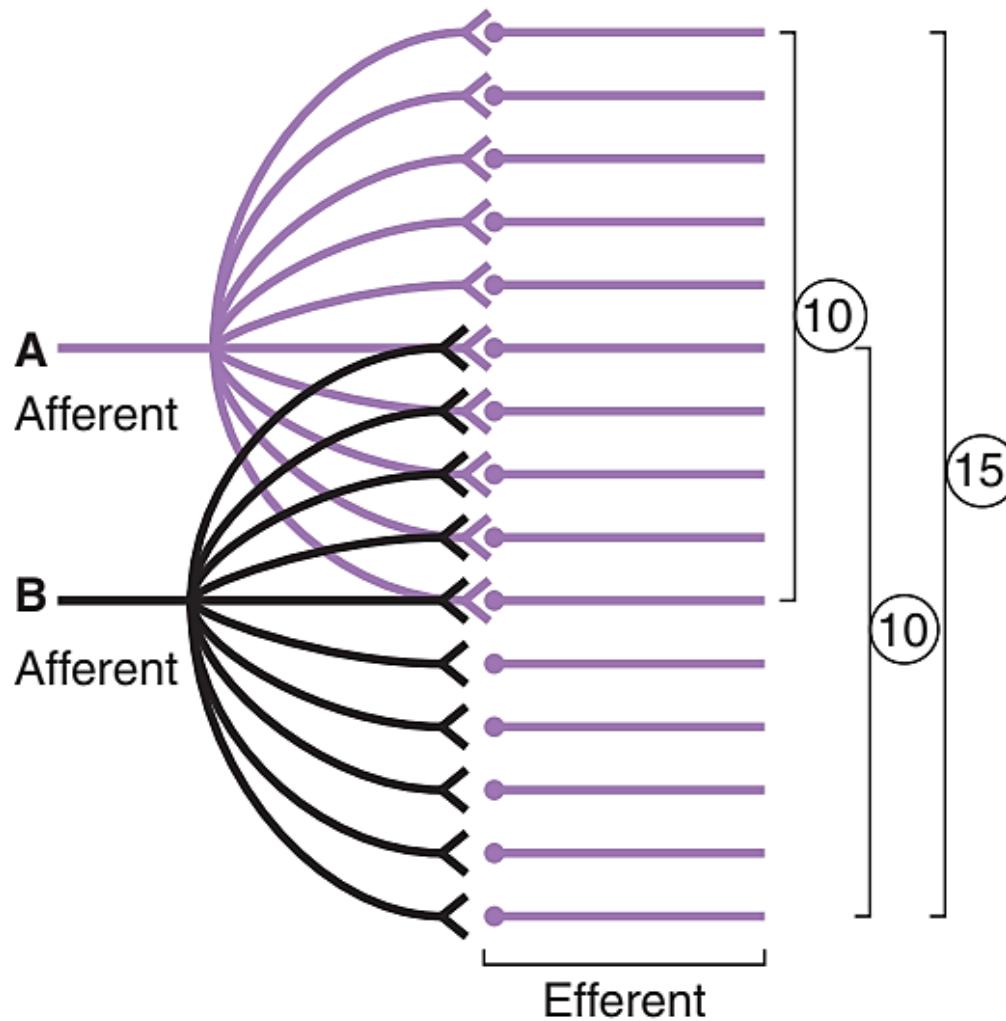


Simultaneous
stimulation of many
presynaptic terminals

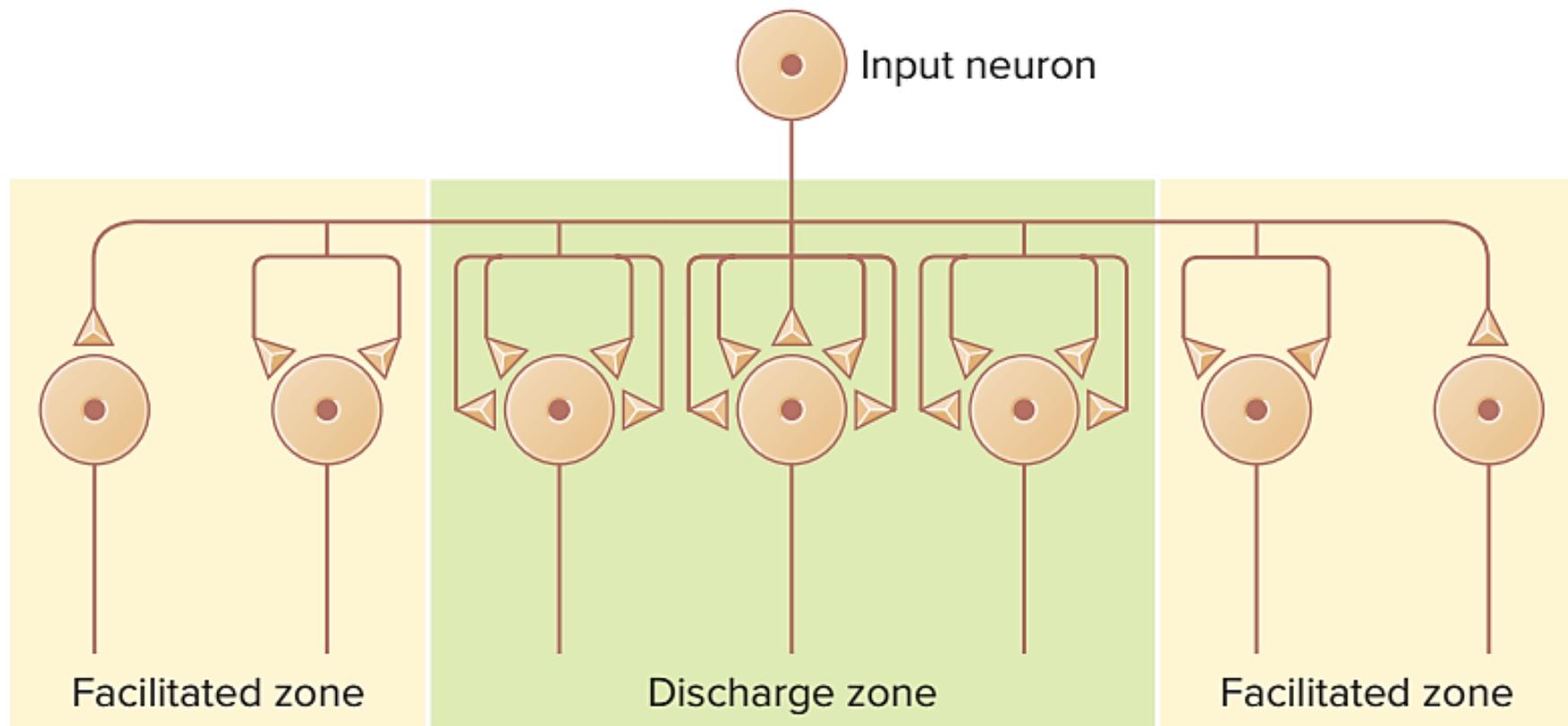
Temporal summation



Repeated
stimulation of one
presynaptic terminal



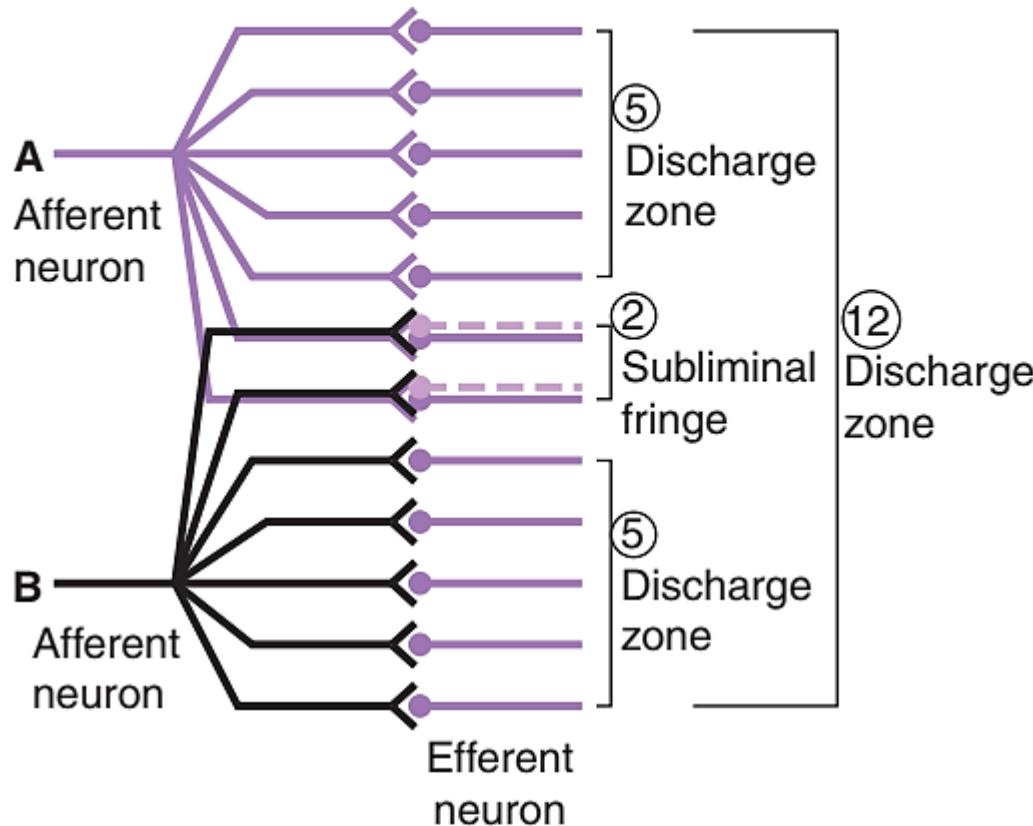
Occlusion phenomenon: stimulation of afferent neuron A and B each excites 10 efferent neurons. Simultaneous stimulation of neuron A and B together excite 15 efferent neurons because five efferent neurons are common to both.



Facilitated and Discharge Zones in a Neural Pool.

In the discharge zone, the presynaptic input neuron has so many synaptic contacts with each postsynaptic neuron that it alone can induce the postsynaptic cell to fire.

In a facilitated zone, the presynaptic neuron lacks enough synaptic contacts with a postsynaptic neuron to induce firing by itself. However, it can collaborate with other presynaptic neurons, facilitating each other in making the postsynaptic cell fire.



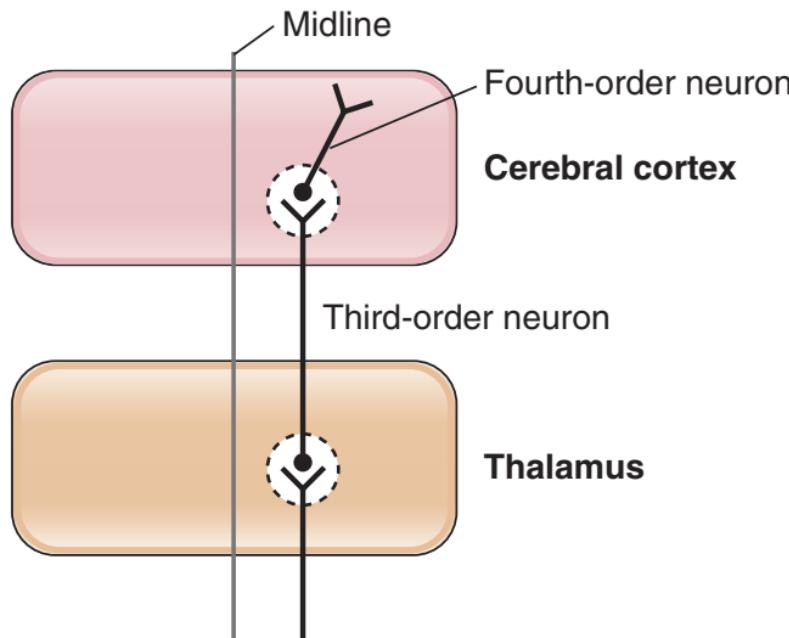
Subliminal fringe effect: stimulation of afferent neuron A and B each excites five efferent neurons and subliminal fringe effect on two efferent neurons (which are common to both A and B neurons). Simultaneous stimulation of neuron A and B together excites 12 efferent neurons because the subliminal fringe effect on two neurons gets summated to produce threshold stimulation.

Chemical Synapse

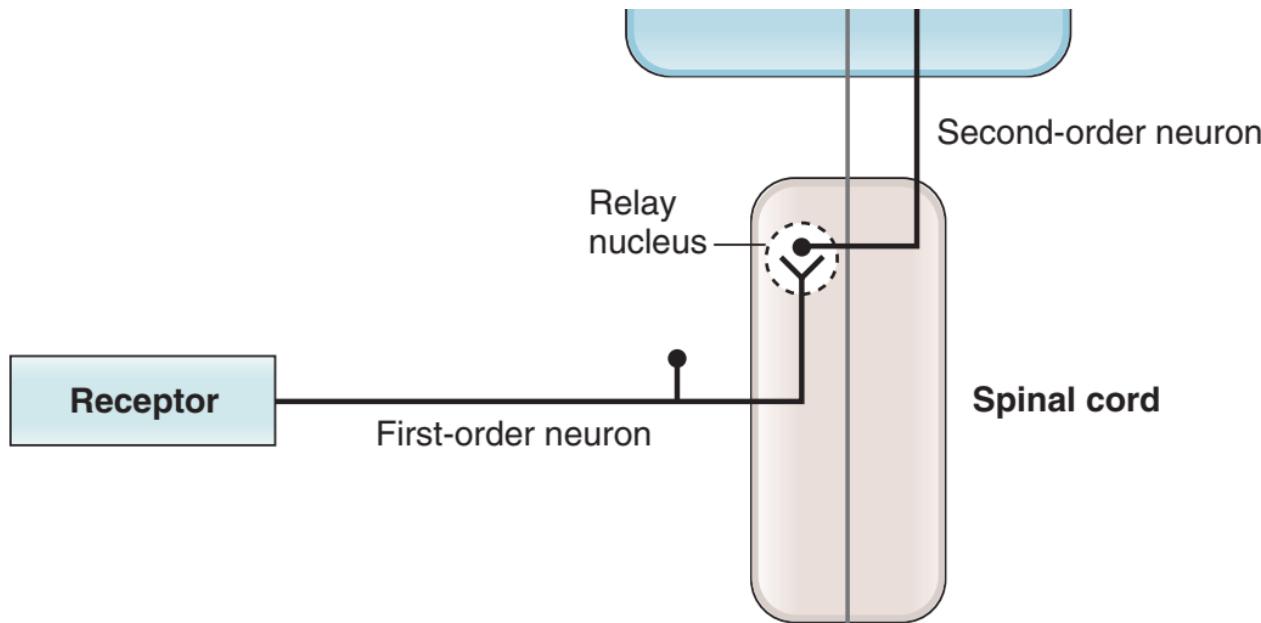
Electrical Synapse

Exhibits synaptic delay eg at NMJ reveal a delay of 0.5 to 4.0 mili sec	Almost no delay in transmission.
20- to 40-nanometer distance that separates cells	Cells approach within about 3.8 nm of each other
Two separate cells that do not touch	Gap junctions are intercellular connection that directly connect the cytoplasm of cells
Slower than Electrical	Faster: many neurons fire synchronously
Mostly unidirectional	Mostly bidirectional
More complex behaviors	Are fast, but can produce only simple behaviors
Act on receptors which are specific	Without the need for receptors to recognize chemical messengers
The response may not be the same as the source.	The response is always the same sign as the source.
The response in the postsynaptic neuron is variable.	Lack Gain the signal in the postsynaptic neuron is the same or smaller than that of the originating neuron

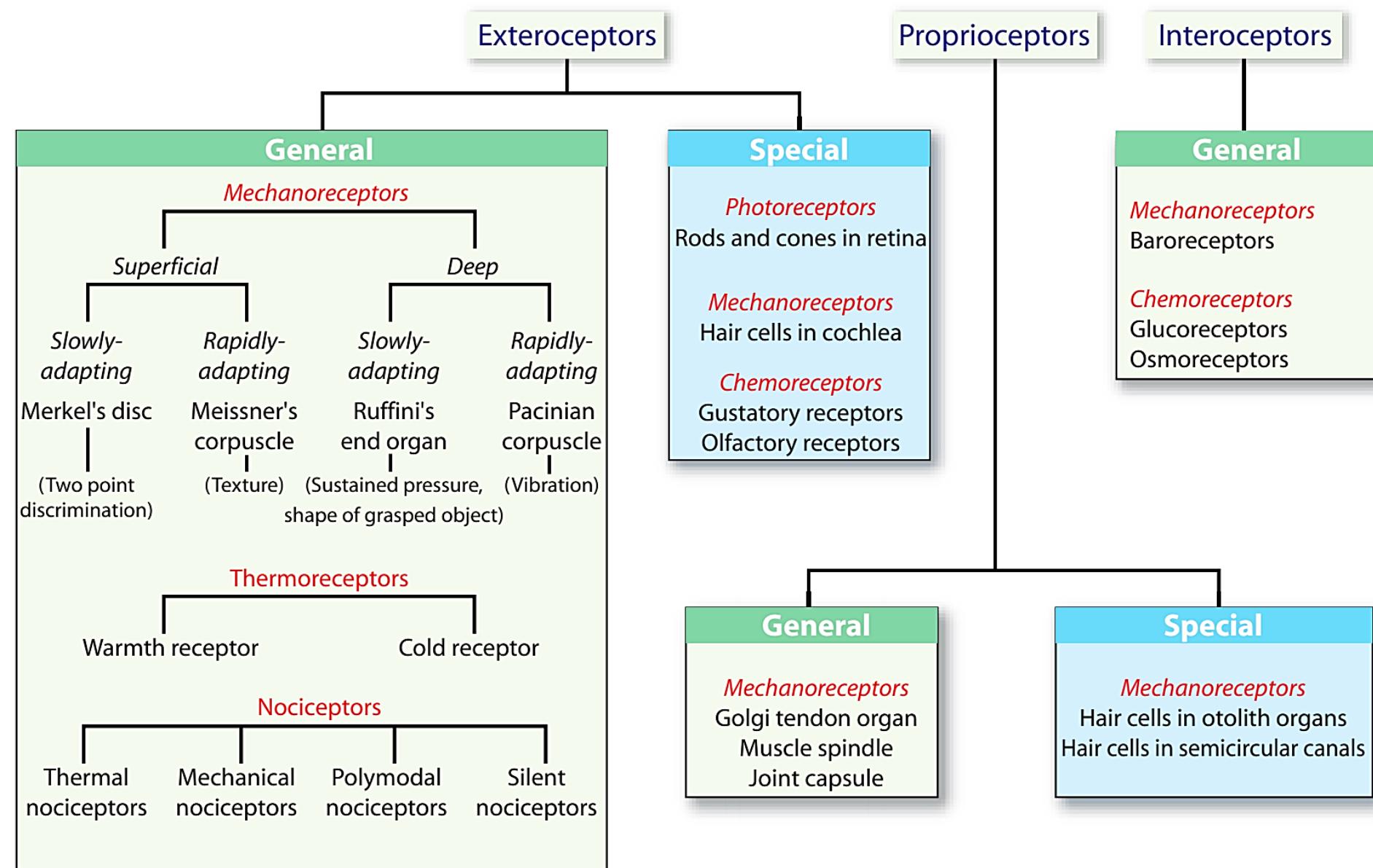
SENSORY SYSTEM



THALAMUS is the OBLIGATE RELAY STATION for all general & special senses
Except OLFACTION



Classification of sensory receptors based both on stimulus source and stimulus energy.



TELE RECEPTORS

- ↳ source of stimulus is at a certain distance from the body
- ↳ Ex: Vision Hearing Smell

LAWS → encodes intensity, duration, location & modality

BELL MEGENDIE LAW → DORSAL ROOTS ARE SENSORY & VENTRAL ROOTS ARE MOTOR

LABELLED LINE PRINCIPLE

- encodes modality, location (to some extent)
- EACH SENSORY MODALITY IS CARRIED BY A SPECIFIC TRACT IN CNS LOCATED IN A SPECIFIC PART OF CNS
- EX: Fine touch carried by dorsal columns

MULLER'S DOCTRINE OF SPECIFIC NERVE ENERGIES

- NO MATTER WHAT FORM OF ENERGY IS APPLIED, EACH SENSORY PATHWAY CONVEYS THE SAME FORM OF ENERGY THAT IT SUPPOSED TO CONVEY

LAW OF PROJECTION

- PHANTOM LIMB
- NO MATTER WHERE YOU APPLIED A STIMULUS, CORTEX ALWAYS PROJECTS THE SENSATION ONTO THE RECEPTOR FROM WHICH THE PATHWAY STARTS

Phantom pain sensation may disappear after 6 months

↳ CORTICAL PLASTICITY

- gradually impulses from amputated area diminishes
- the dendritic geometry in cortex area changes & neurons shrink in size
- Neurons of neighbouring area starts encroaching the area effectively & training of that area obliterated

INTENSITY DISCRIMINATION

- Intensity discrimination in CNS occurs by changing AP FREQUENCY

WEBER FECHNER LAW

$$S = K \times \log(I)$$

PERCEPTION FELT BY THE CORTEX ABOUT THE INTENSITY OF STIMULUS CHANGES IS LOGARITHMIC SCALE OF THE ACTUAL INTENSITY APPLIED IN PERIPHERY

$$I = 100 \rightarrow S = 1 \times 2 = 2$$

IF the perceptⁿ felt is doubled,
actual intensity is ↑ by
10 times

RECEPTOR	LOCATION	RECEPTIVE FIELD SIZE	SPEED OF ADAPTATION	SENSATION ENCODED
Merkel's disc	Epidermis	smallest	slowly	locat'n of touch
meissner corpuscle	Dermis	small	Rapidly	speed of applicat'n of touch Lips and finger tips.
Pacinian corpuscle	Dermis & Deeper tissues	large	very rapidly	vibrat'n
Ruffini's corpuscle	Ligaments muscles tendons	large	slowly	Deep pressure (massage)

Rapidly adapting (Phasic) receptor

- Sense rate of change of stimulus
- Detect motion and vibration
- They are encapsulated

Slow adapting (tonic) receptor

- Sense steady stimulus
- Detect object pressure and form (shape, size, texture)
- They are expanded

Examples:

Examples:

BRAILLE

→ meissner corpuscle > ruffini's corpuscle

THERMORECEPTORS

- Detect temperature



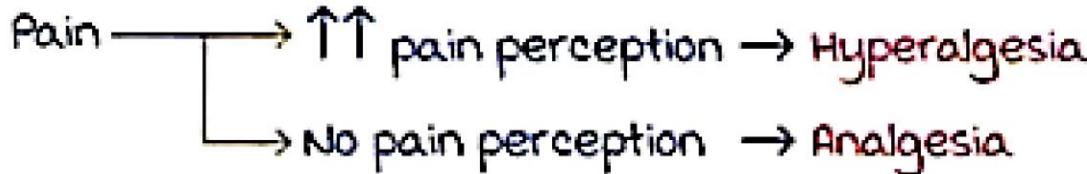
- A δ and C fibres
 - 10 times more in number
 - Detect temperature between 10-24°C
 - TRPM channels (Transient receptor potential menthol)
- C fibres
 - Detect temperature between 30-45°C
 - TRPV₁ channels
v → vanilloid (Capsaicin activates these channels)
- * Temperature < 5°C → Cold pain
 - * Temperature > 45°C → Warm pain

PAIN RECEPTORS

- Free nerve endings of Aδ and C fibres

A-delta fibres	C fibres
<ul style="list-style-type: none">• myelinated• Fast pain / 1st pain• Sharp / Pricking / Acute pain• New in evolution → Called neospinothalamic pain• Neurotransmitter: Glutamate	<ul style="list-style-type: none">• Unmyelinated• Slow pain / 2nd pain• Dull / Diffuse / Burning pain• Oldest → Called paleospinothalamic pain• Neurotransmitter: Substance P

Pain perception:



Algogens

Bradykinin —————→ Most potent
K⁺ (direct depolarization of nerve terminal)
5-HT (platelets) Serotonin
Prostaglandins
Histamine

ANALGESIA SYSTEMS

GATE CONTROL THEORY OF PAIN

→ Accupressure Therapy is based on this

DESCENDING ANALGESIA SYSTEM

→ 3 COMPONENTS

1. Midbrain: Periaqueductal gray (PAG) → enkephalin

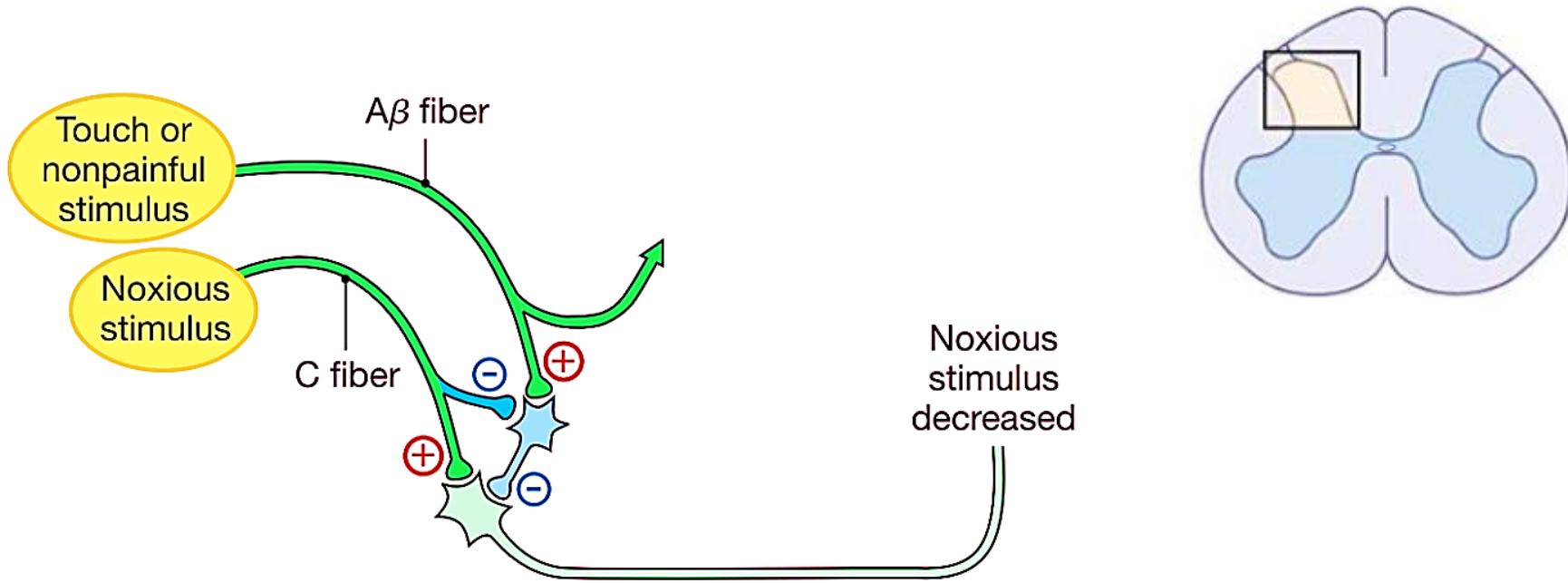
2. Medulla: Nucleus raphe magnus (NRM) → serotonin

Rostro ventrolateral medulla (RVLM) → Catecholamine

3. Spinal Cord: Enkephalin → pre and post synaptic inhibition

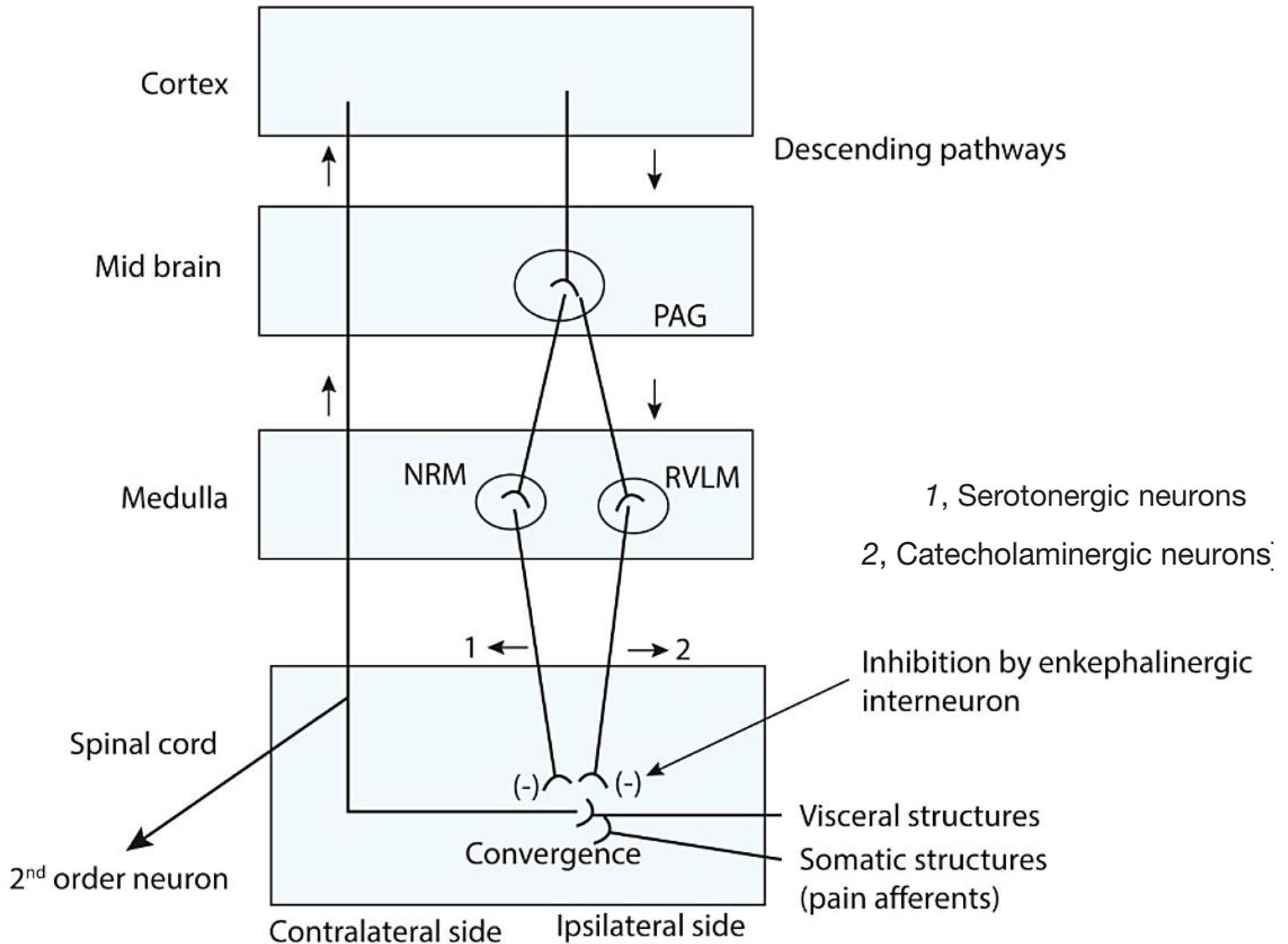
*Endogenous Opioids: Endorphins, enkephalin, dynorphin

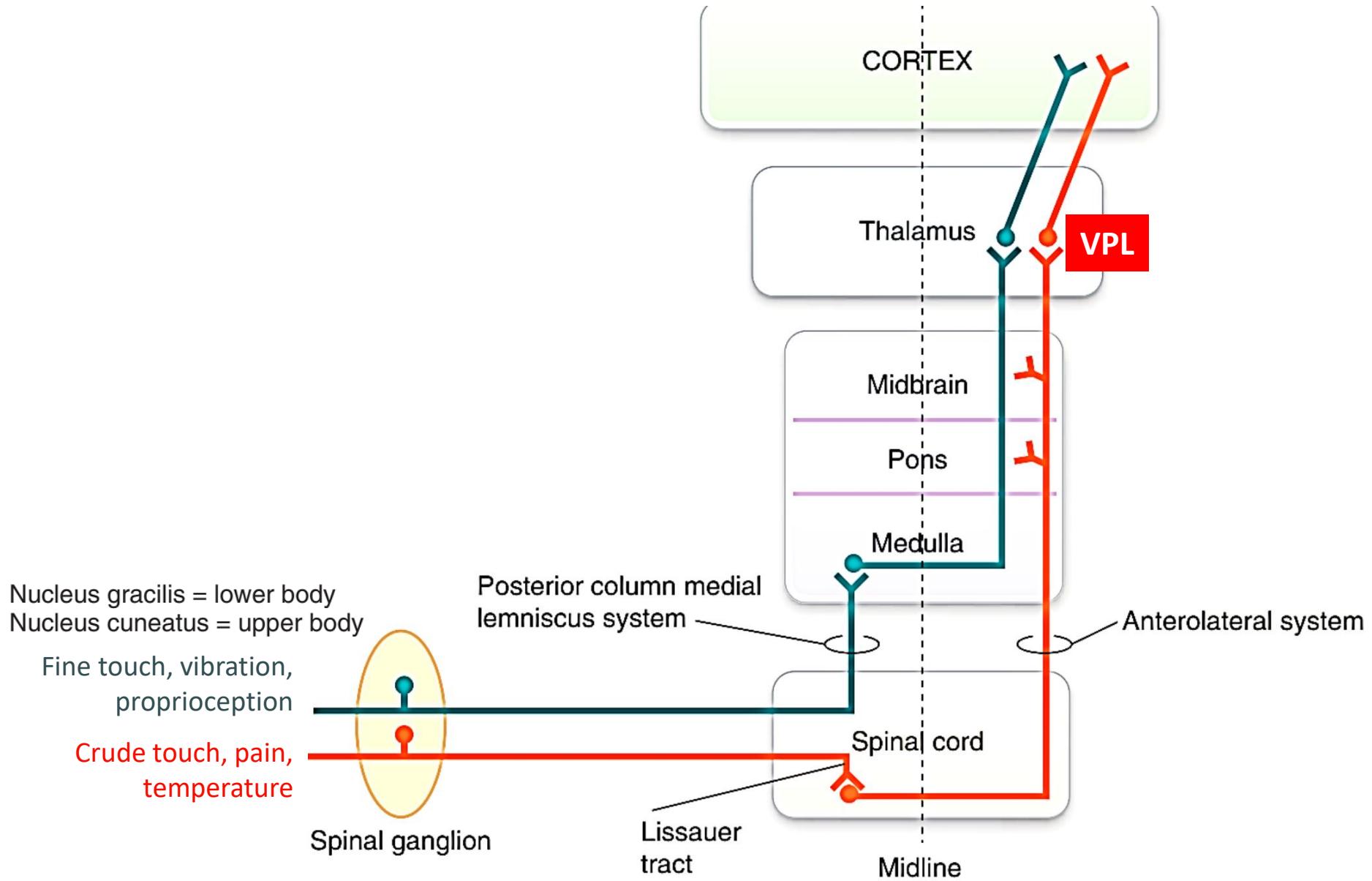
*Endogenous Cannabinoid: Anandamide



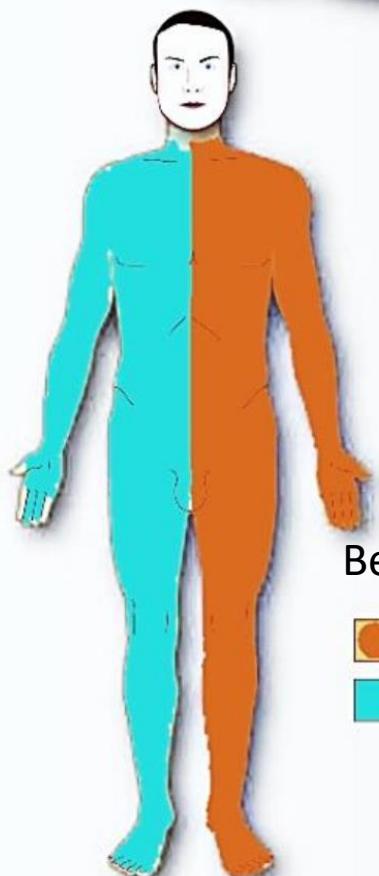
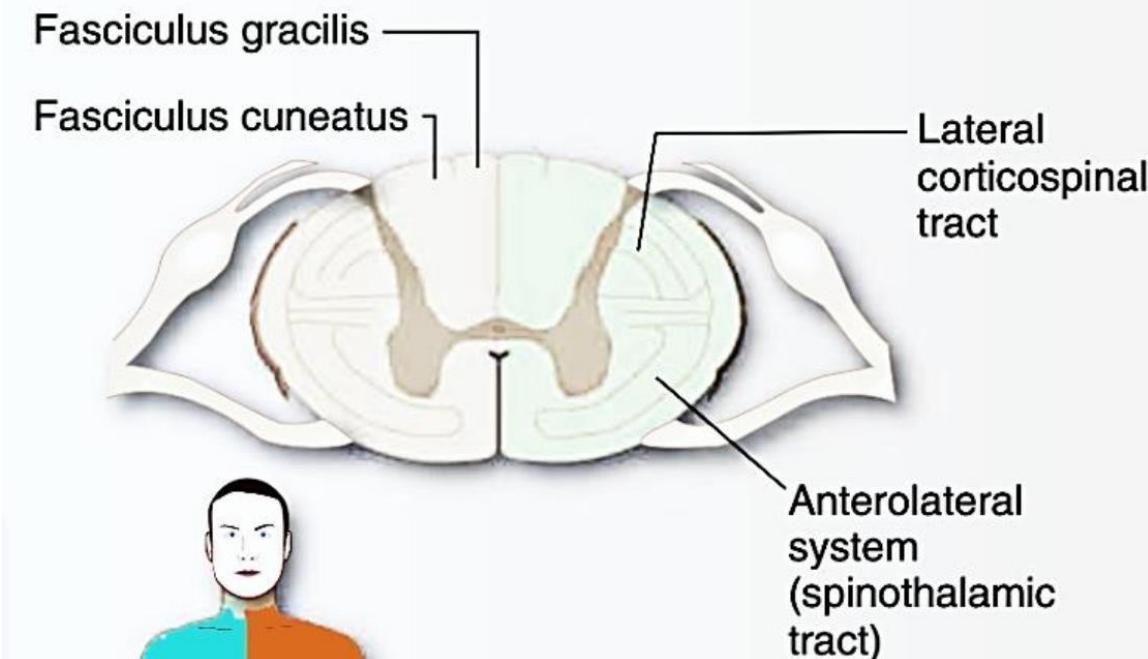
Lamina II or substantia gelatinosa acts as the gate where pain modulation occurs.

In TENS (transcutaneous electrical nerve stimulation), large-diameter (A α and A β) fibers are stimulated to relieve pain. Thus, it is based on gate control theory.





Hemisection (Brown-Séquard syndrome)

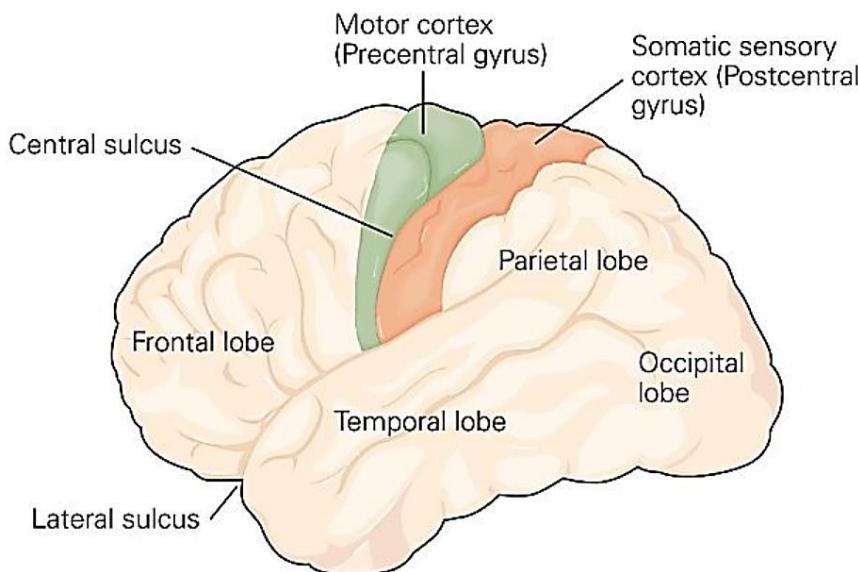


Below the level of hemisection:

- Loss of pain, temperature Contralateral
- Loss of motor, position, vibration, proprioception Ipsilateral

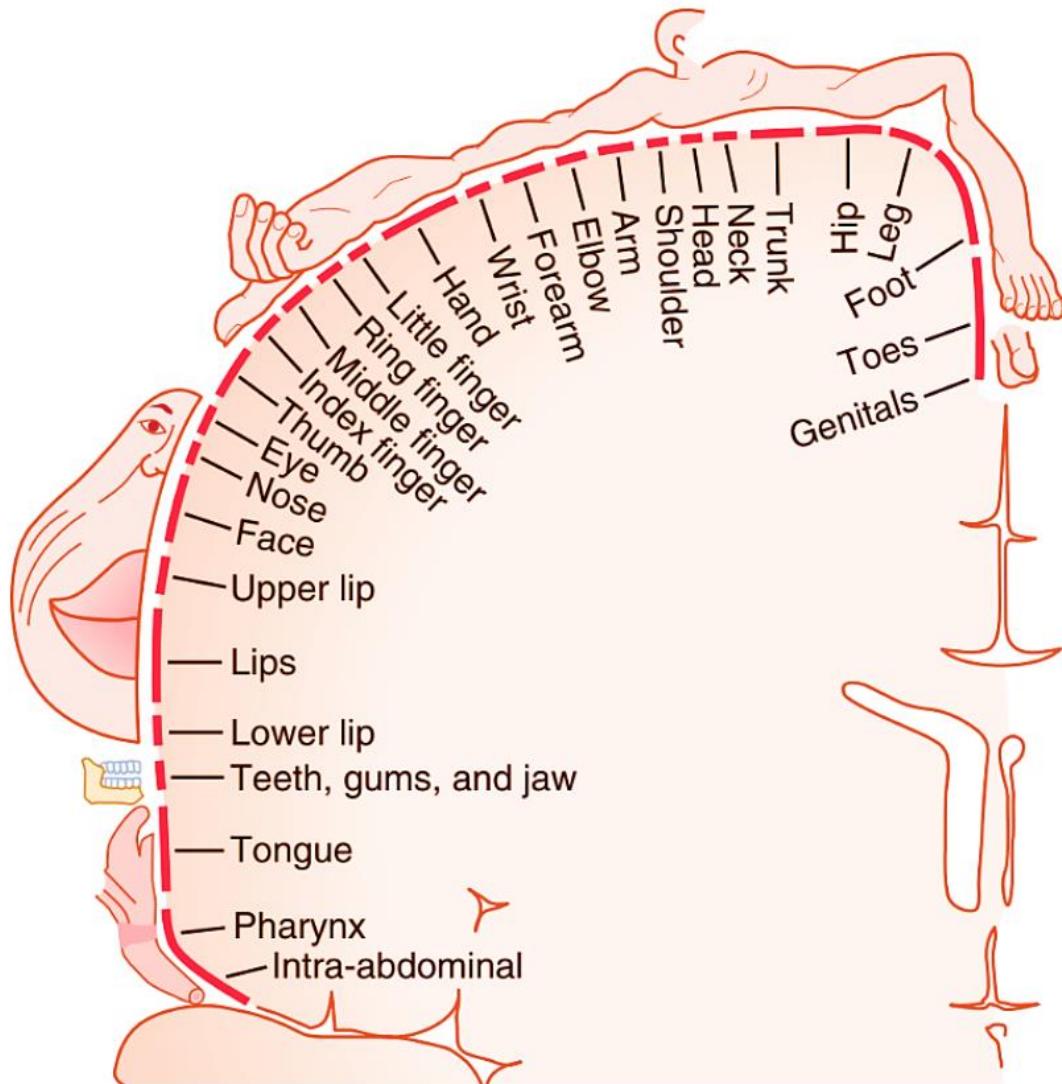
SOMATOSENSORY CORTEX

The primary somatosensory cortex (SI): the postcentral gyrus and the posterior paracentral lobule of the parietal lobe (Brodmann area 3,1,2).

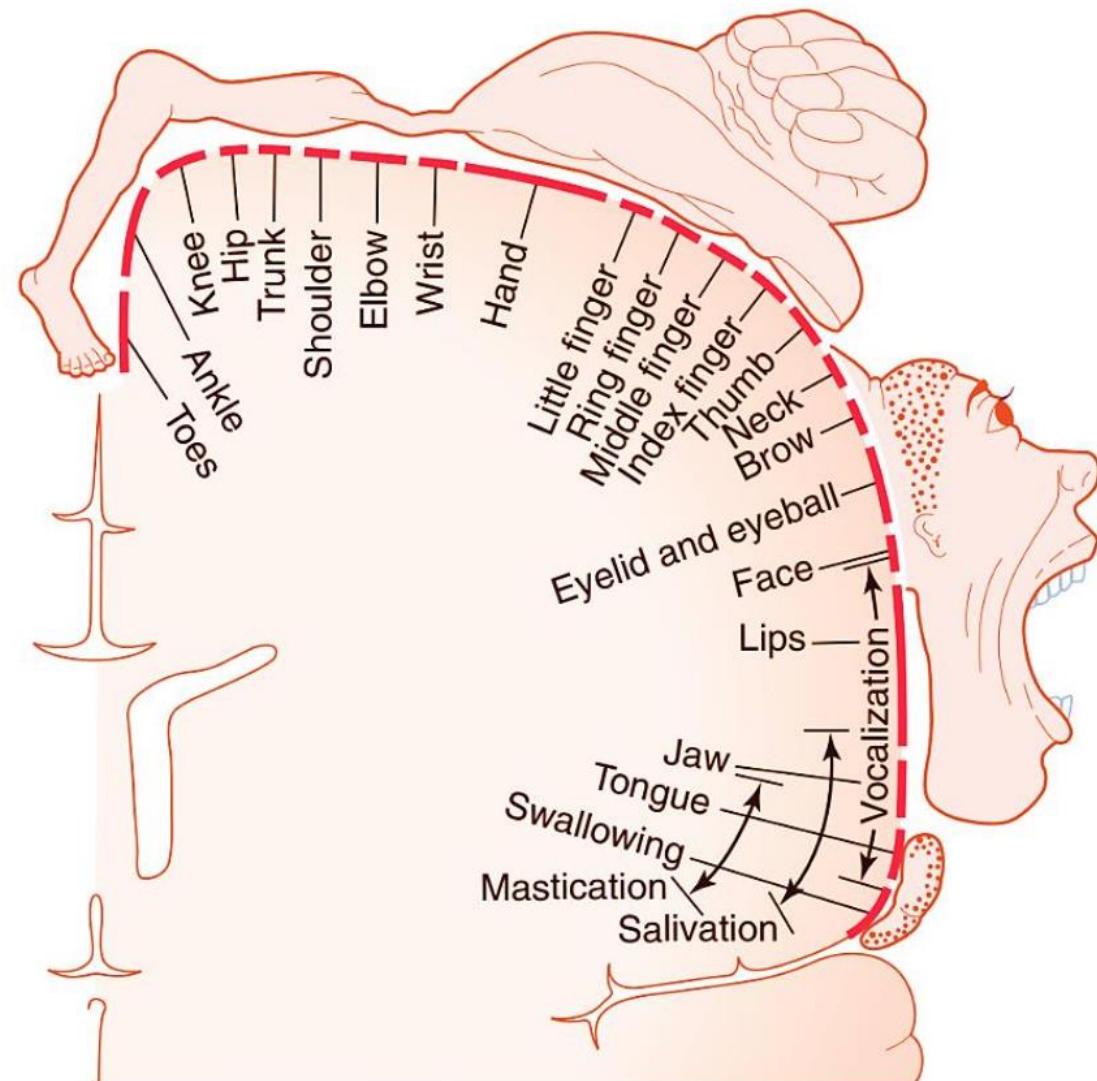


Sensory Homunculus: Some areas of the body are represented by large areas in the somatic cortex—the lips the greatest of all, followed by the face and thumb—whereas the trunk and lower part of the body are represented by relatively small areas.

The sizes of these areas are directly proportional to the number of specialized sensory receptors in each respective peripheral area of the body.



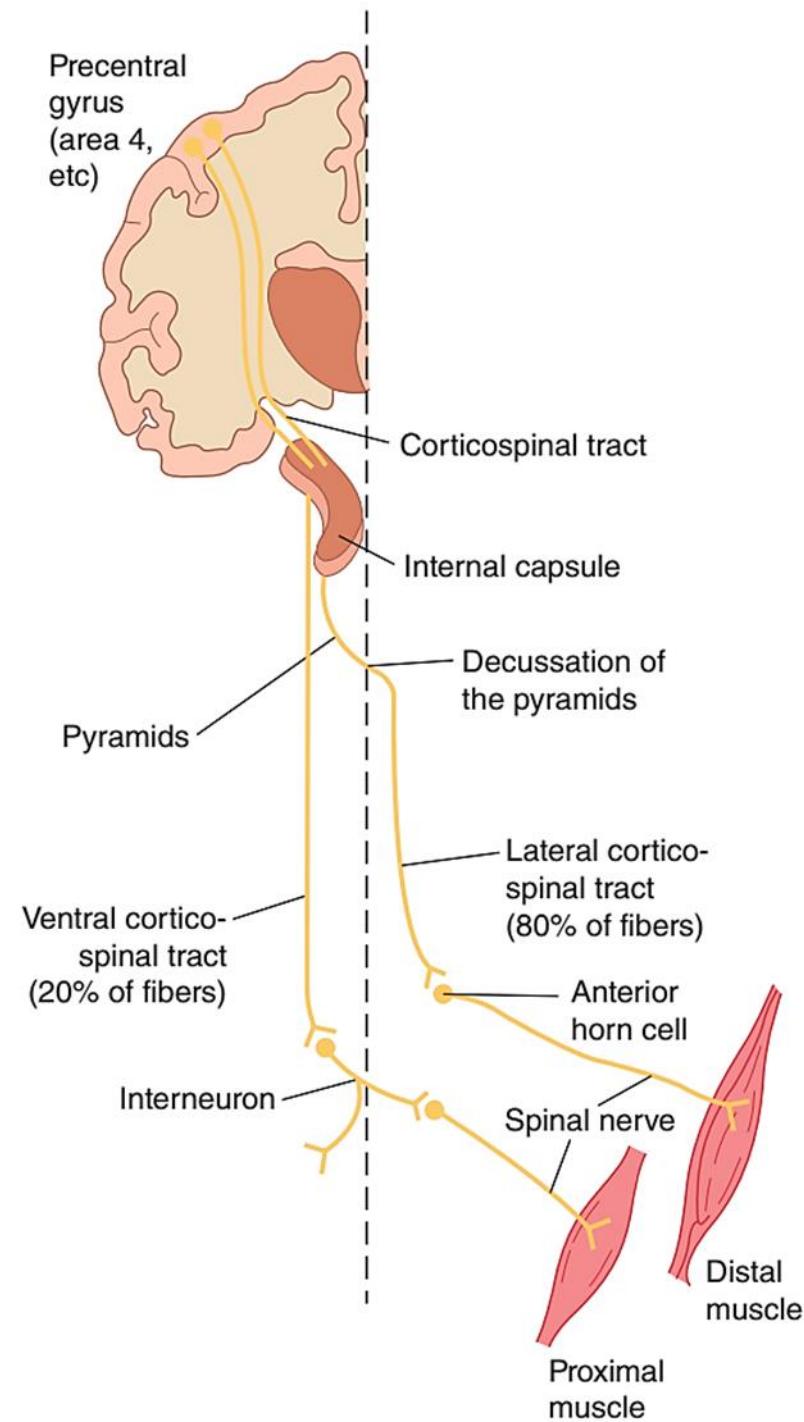
Motor Homunculus: The distortion of the various body parts in the homunculus indicates approximately how much of the cortex is devoted to their motor control. Note that more than one half of the entire primary motor cortex is concerned with controlling the muscles of the hands and the muscles of speech. Precise movements of the hand require the separate innervation of many small muscles, whereas the innervation of the trunk requires less precise regulation. Therefore, the cortical representation of the trunk is significantly smaller than that of the hand.



CORTICO SPINAL TRACT

ORIGIN

- 30% Fibers → Area 4
- 30% fibers → Area 6
- 40% Fibers → Sensory Cortex



EXTRA PYRAMIDAL TRACT

→ all fibers outside the pyramids in medulla

1. vestibulo spinal tract

↳ controls posture & Equilibrium }

2. Reticulospinal tract

↳ controls trunk muscles }

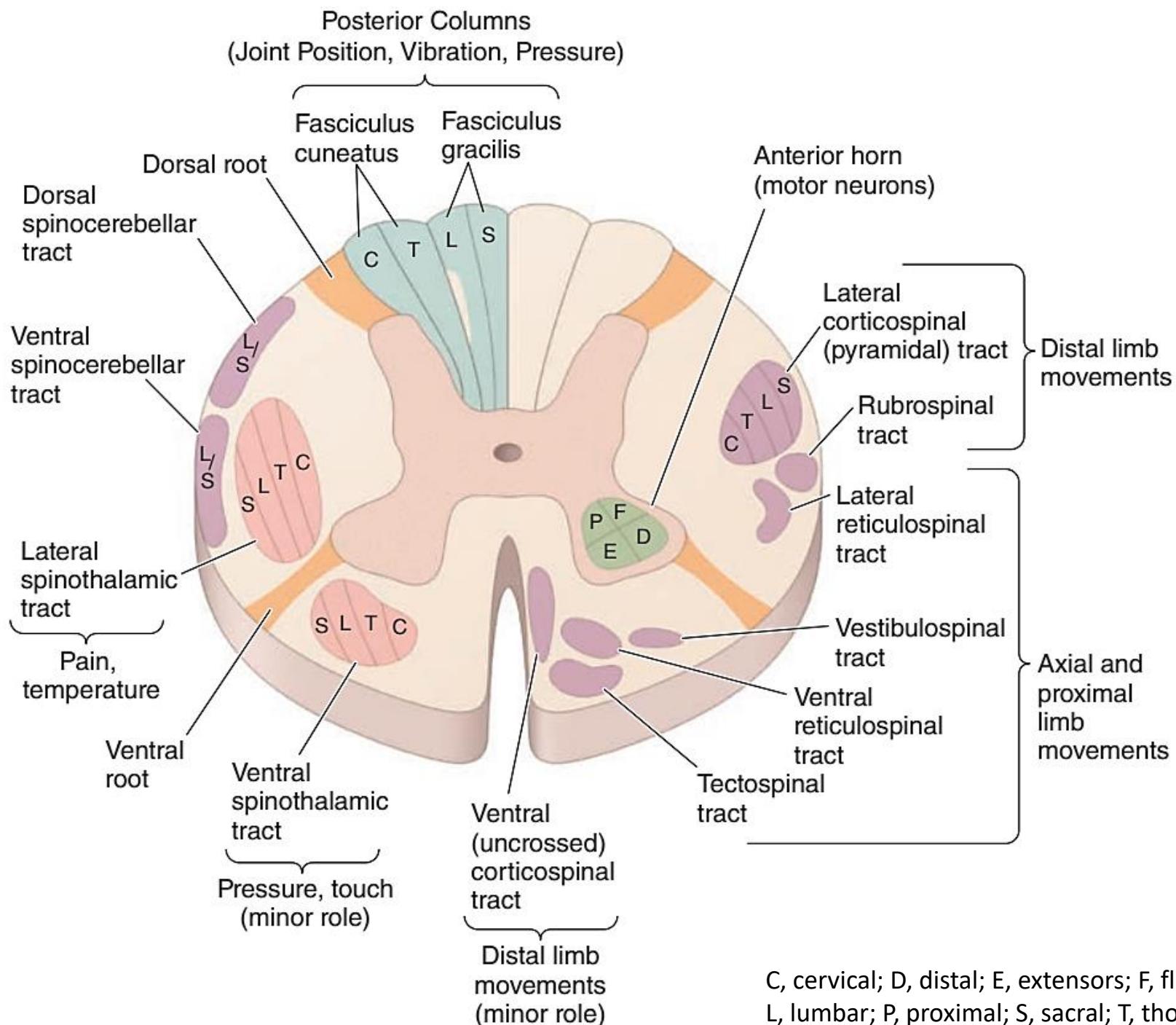
3. Rubrospinal tract

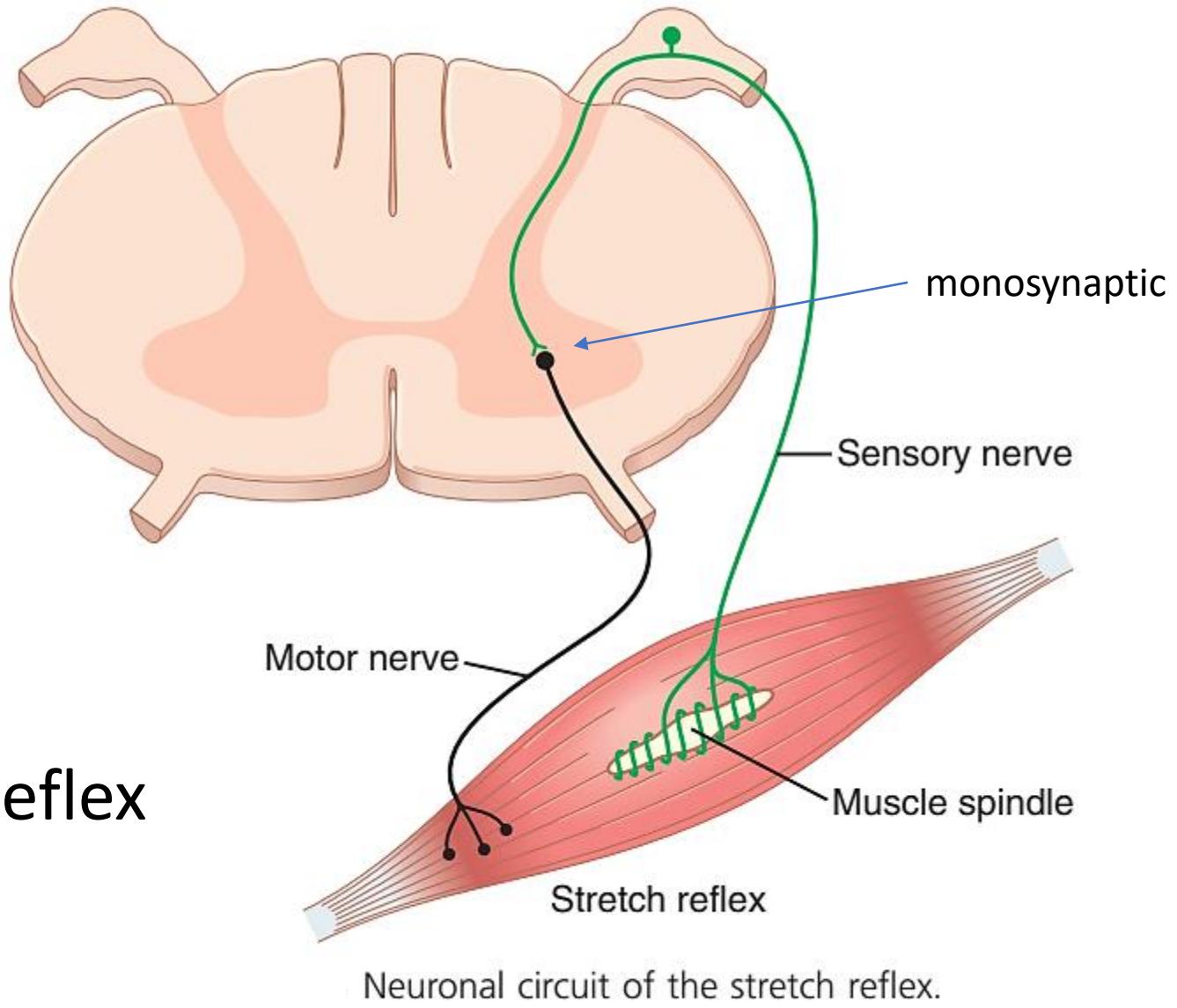
↳ controls proximal muscle tract

4. Tectospinal tract

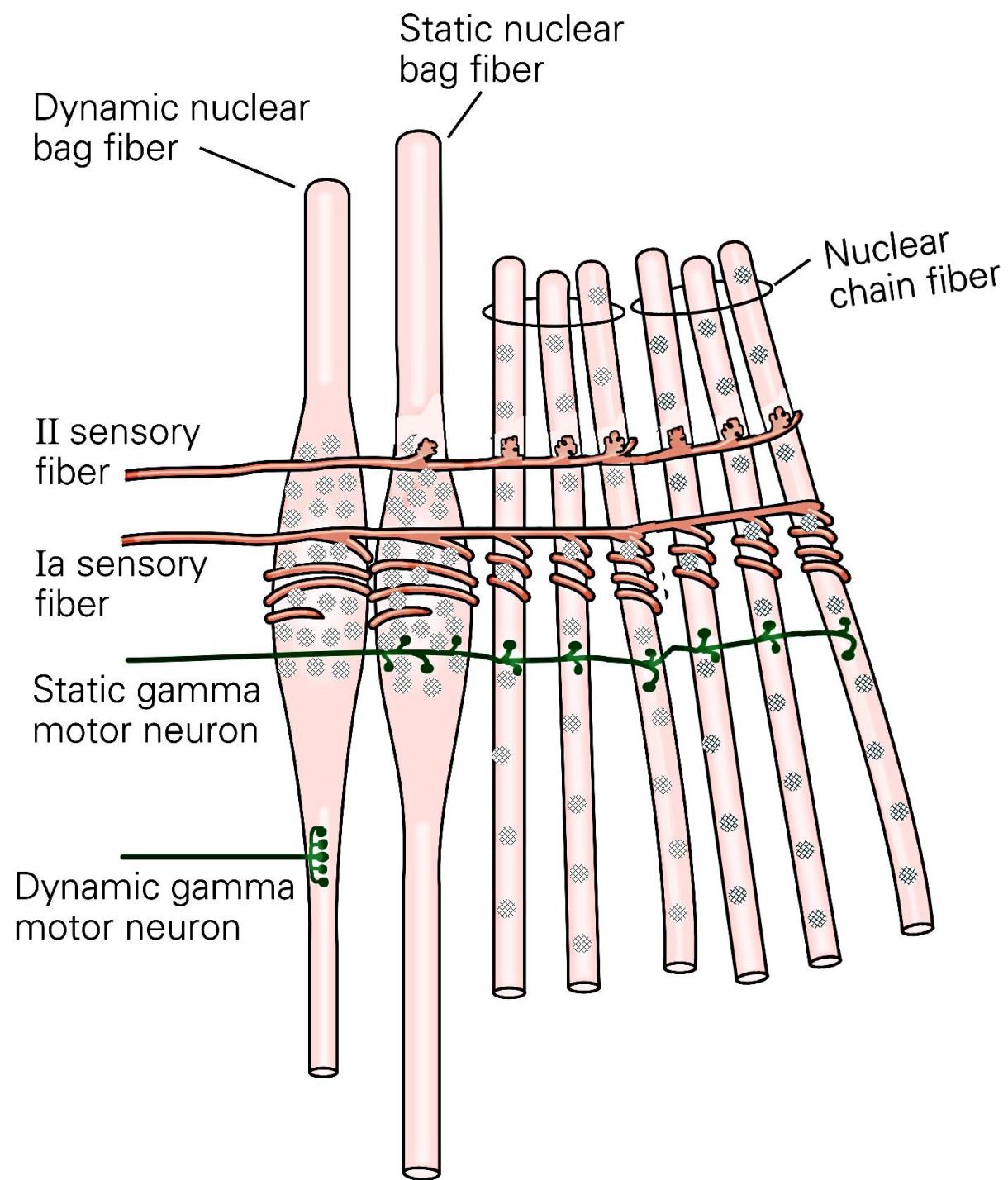
↳ tectum → ROOF OF MID BRAIN

controls
axial
muscles



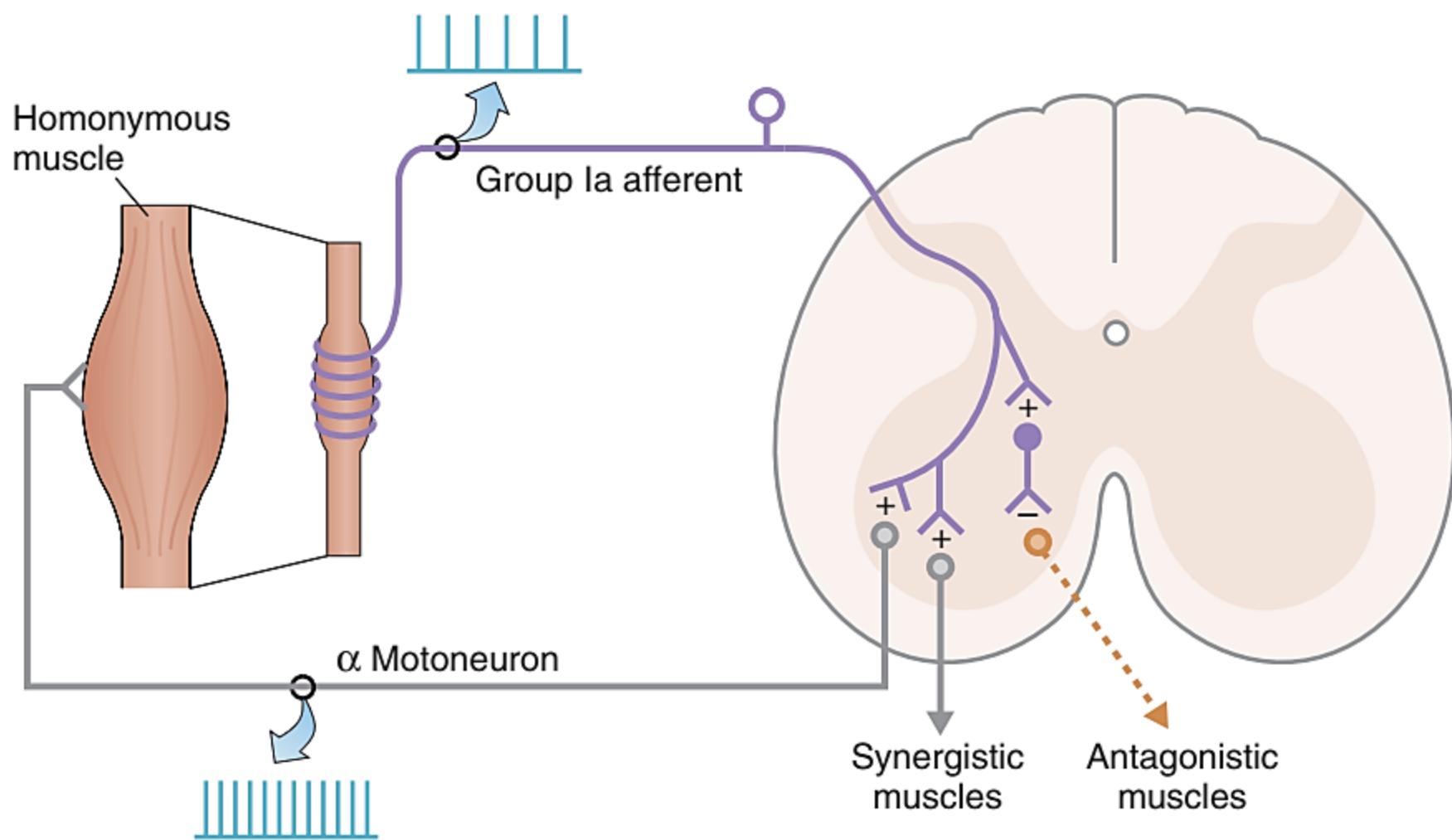


Stretch Reflex



- 3 to 12 intrafusal muscle fibers per muscle spindle
- 1-3: Nuclear bag
- 3-9: Nuclear chain
- Central region: non-contractile
- Afferent Ia ($A\alpha$): Annulospiral ending, dynamic response
- Afferent II ($A\beta$): Flower spray ending, static response
- Efferent γ static: Trail ending
- Efferent γ dynamic: Plate ending
- Stimulus: stretch to the muscle

STRETCH REFLEX



Muscle tone

- Even at rest, muscles normally exhibit some level of contractile activity driven by reflex arcs (gamma motor neuron discharge) from the muscle spindles. This reflex sustained partial contraction of skeletal muscle is known as tone.
- The muscles are generally hypotonic when the rate of γ -motor neuron discharge is low and hypertonic when it is high
- Isolated (i.e., denervated) unstimulated muscles are flacid i.e., without tone.

Upper Motor Neuron



UP

MORE

HYPER

- More Muscle Contraction → Spasticity
- More Muscle Tone → Hypertonicity
- More Muscle Reflexes → Hyperreflexia
- + Babinski – Toes Point Up

SPASTICITY

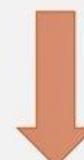
Seen in pyramidal tract lesion

unidirectional

involves one group of muscles
(agonists)

velocity dependent

Lower Motor Neuron



DOWN

LESS

HYPO

- Less Muscle Contraction → Flaccid
- Less Muscle Tone → Hypotonicity
- Less Muscle Reflexes → Hyporeflexia
- Loss of innervation → Denervation Atrophy
- Toes Point Down

RIGIDITY

Seen in Extra pyramidal tract lesion

Bidirectional

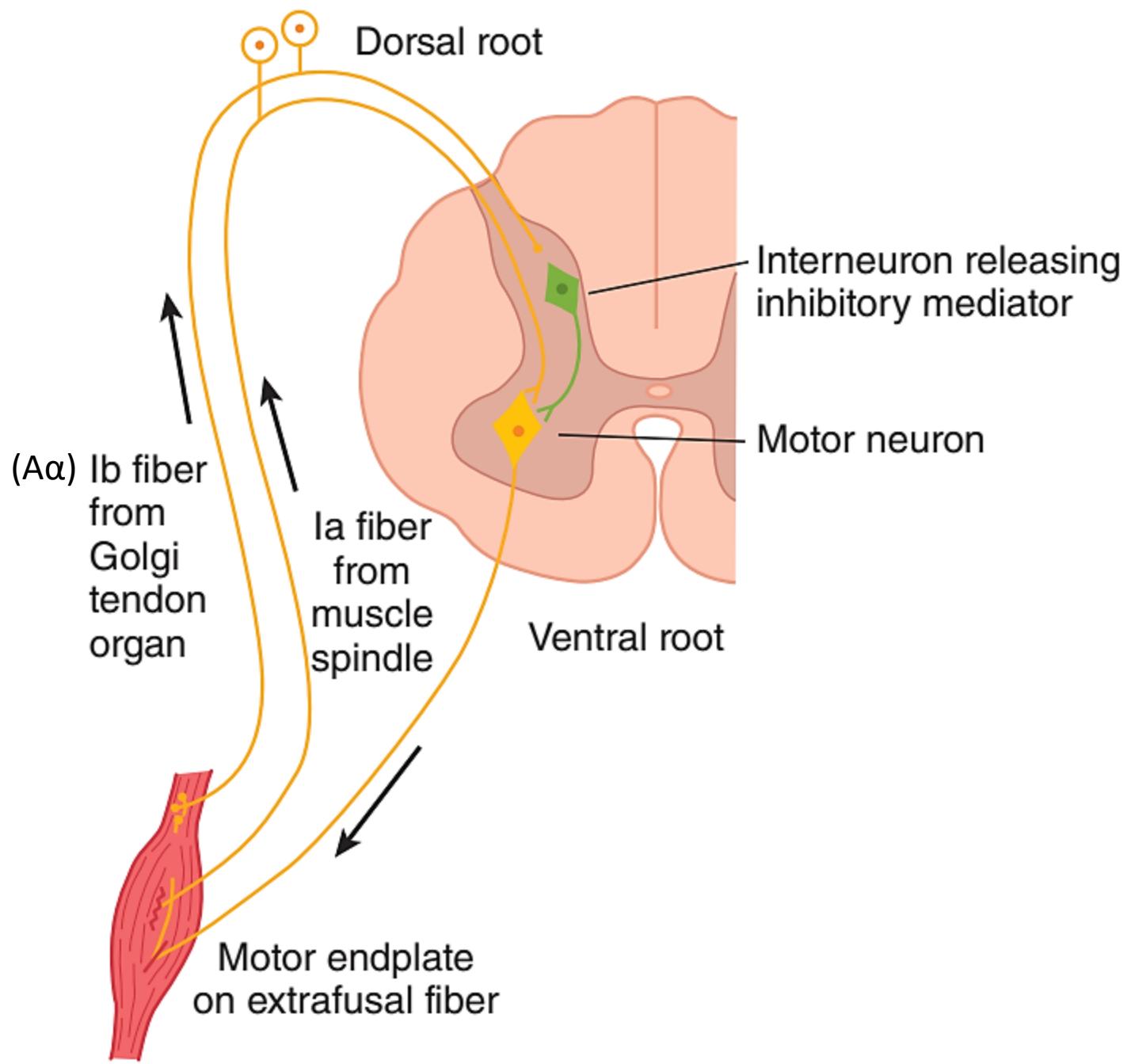
Involves both agonists & antagonists

↳ Lead pipe rigidity → cogwheel rigidity
not velocity dependent

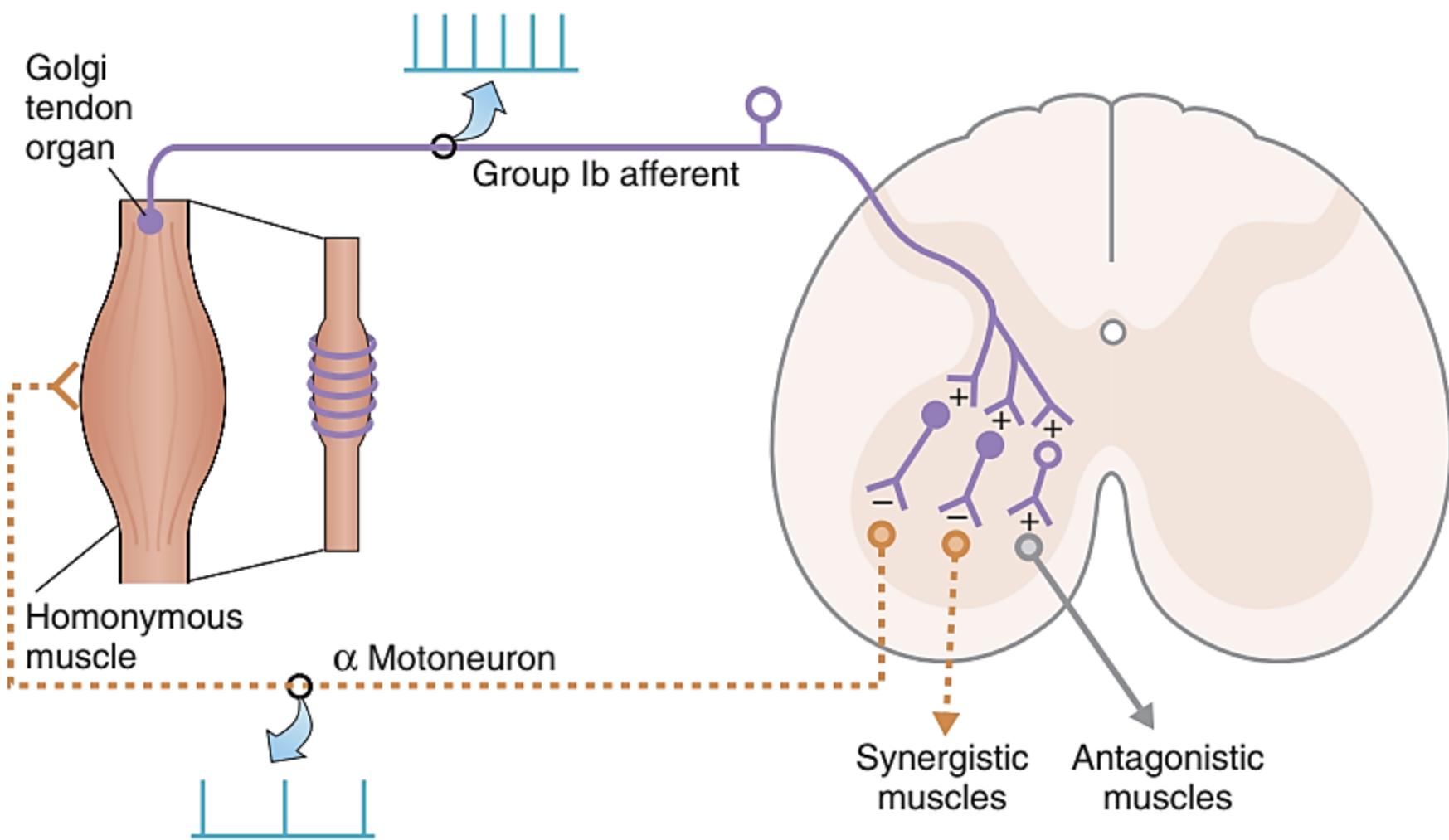
	UMN Paralysis	LMN Paralysis
1. Muscles affected	In groups (not individual muscles)	Individual muscles are affected
2. Size of the muscles	No atrophy	Atrophy is a pronounced feature
3. Type of paralysis	Spastic paralysis (hypertonia)	Flaccid paralysis (hypotonia)
4. Tendon reflexes	Exaggerated	Diminished or absent
5. Superficial reflexes	Absent	Absent
6. Babinski's sign	Extensor planter response	Flexor planter response
7. Involuntary movements	Absent	Present (in the form of fascicular twitches)
8. Clonus	Present	Absent
9. Nerve conduction study	No abnormalities	Decreased nerve conduction

Golgi Tendon Reflex

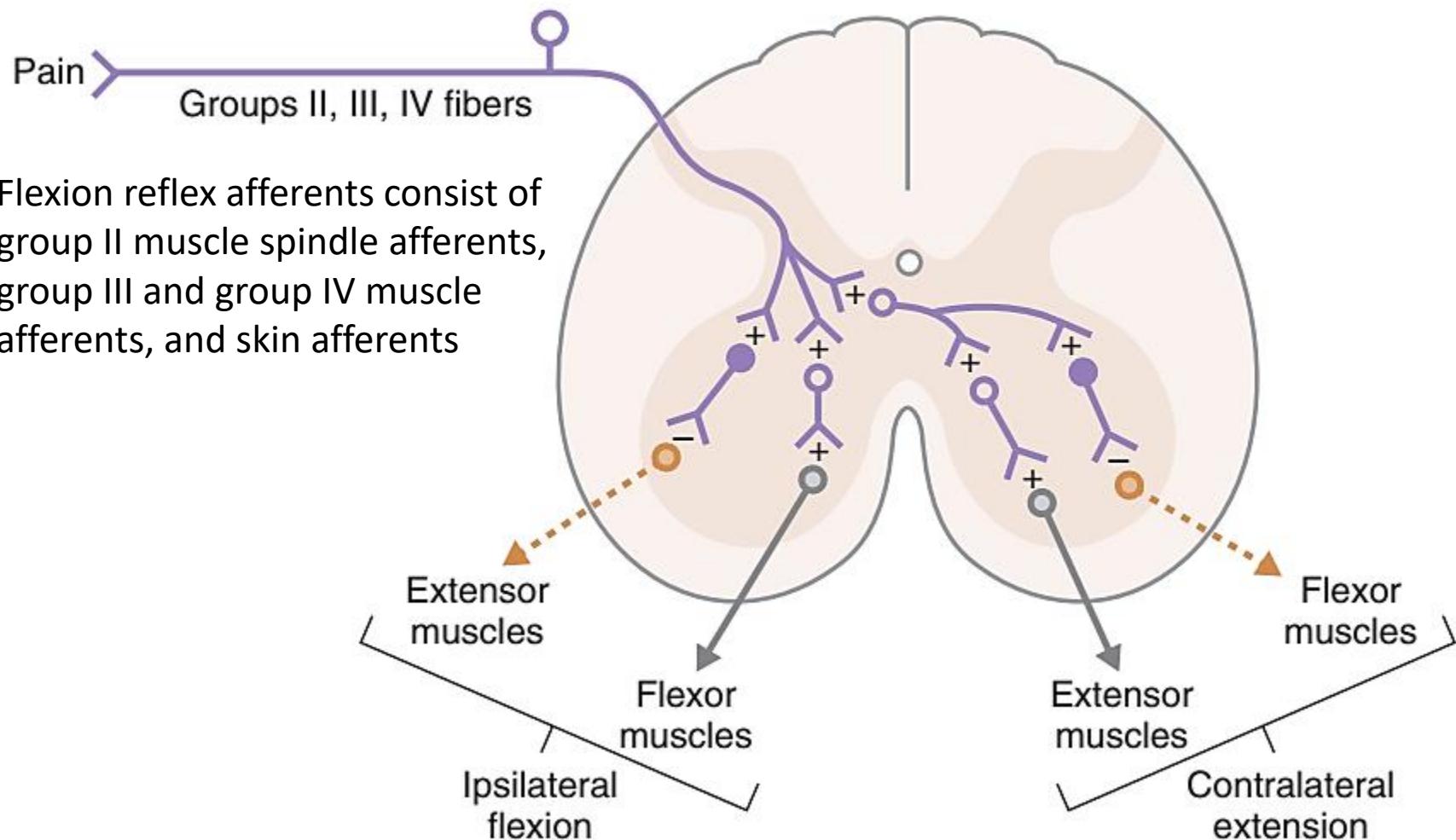
- Also called as inverse stretch reflex
- Golgi tendon organs are located in the tendons
- There are 3–25 muscle fibers per tendon organ.
- Golgi tendon organs, unlike the spindles, are in series with the muscle fibers.
- The tendon organ, like the primary receptor of the muscle spindle, has both a dynamic response and a static response
- The spindle detects muscle length and changes in muscle length, whereas the tendon organ detects muscle tension

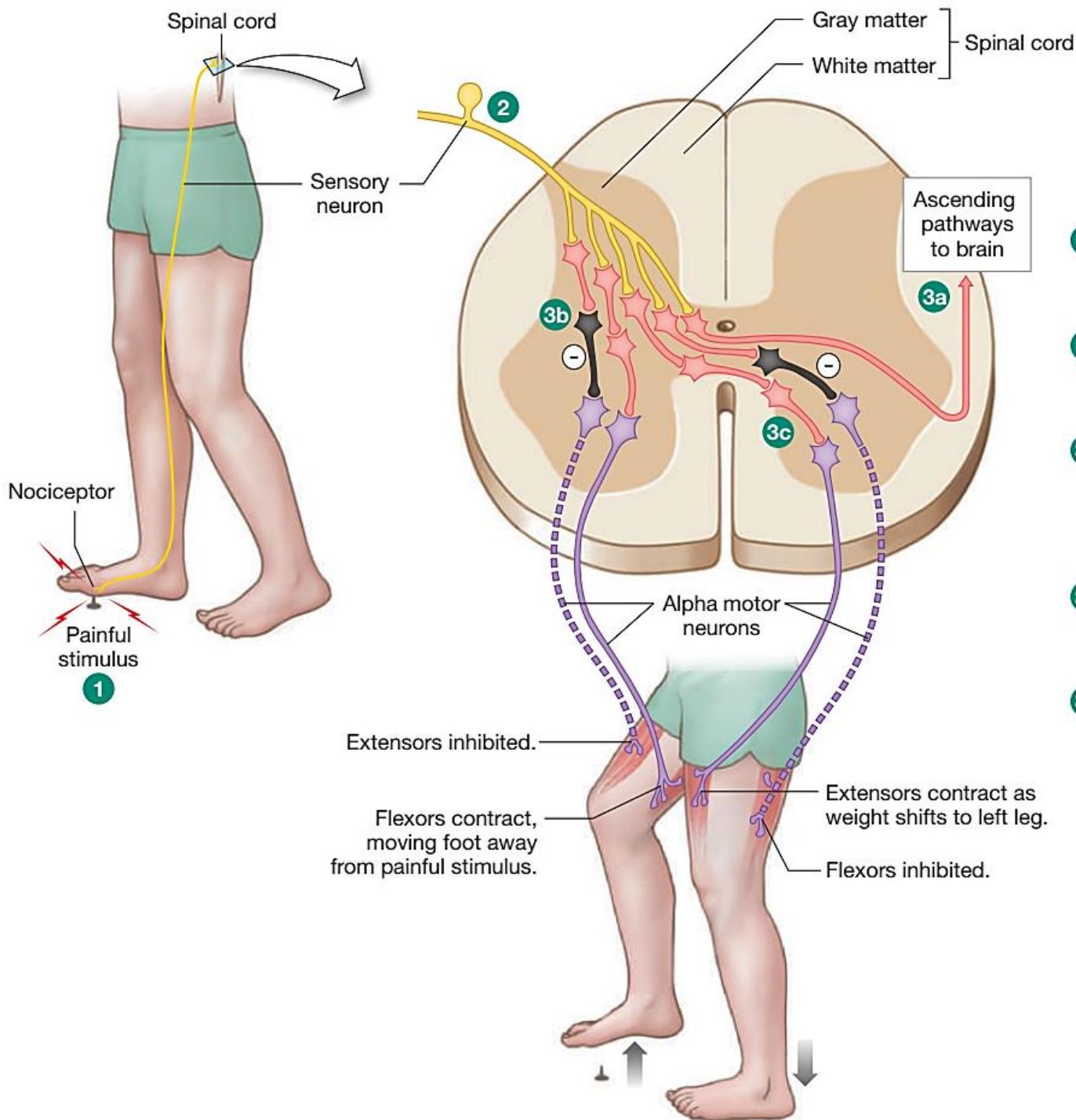


GOLGI TENDON REFLEX



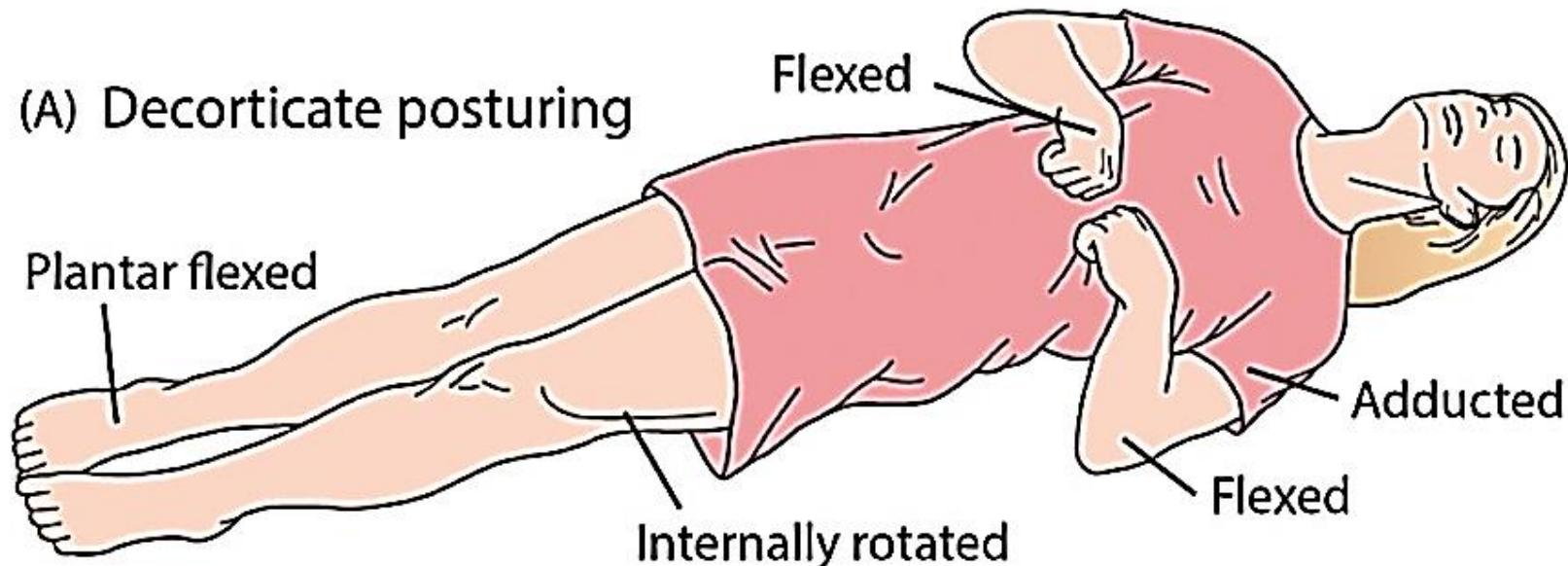
FLEXOR-WITHDRAWAL REFLEX





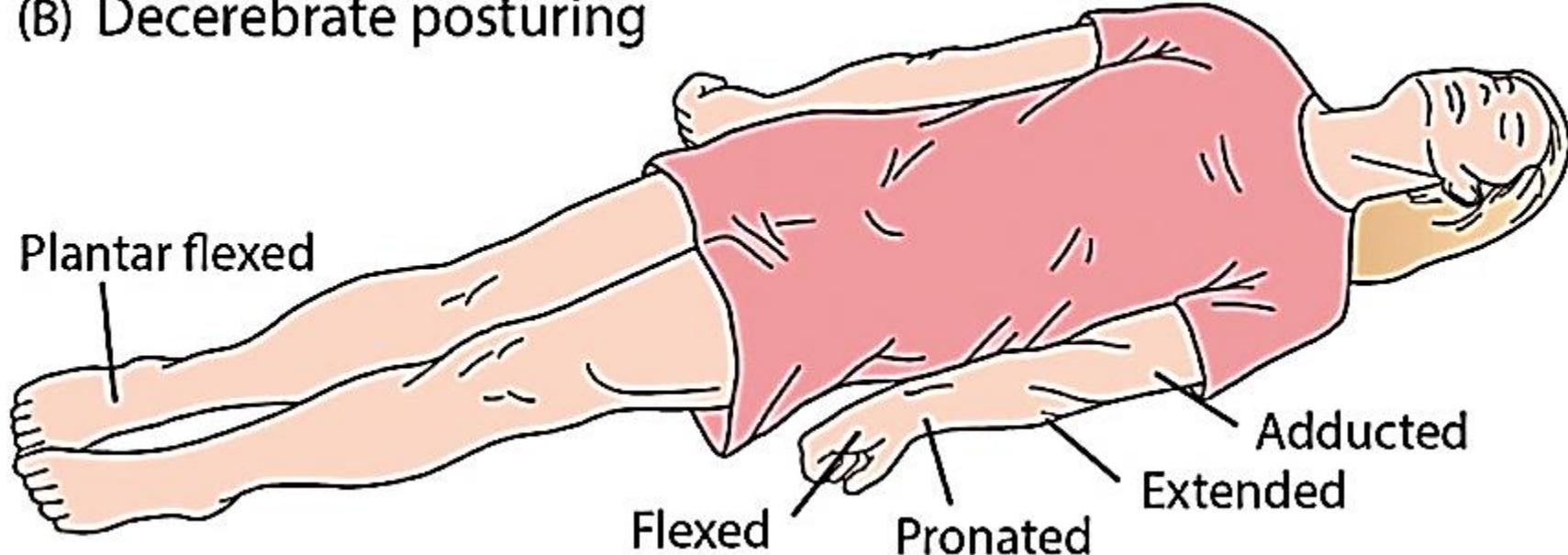
- 1 Painful stimulus activates nociceptor.
- 2 Primary sensory neuron enters spinal cord and diverges.
- 3a One collateral activates ascending pathways for sensation (pain) and postural adjustment (shift in center of gravity).
- 3b Withdrawal reflex pulls foot away from painful stimulus.
- 3c Crossed extensor reflex supports body as weight shifts away from painful stimulus.

(A) Decorticate posturing



Decorticate rigidity—Flexion in upper limb and extension of lower limb (flexion because of stimulation of rubrospinal tract due to positive input at red nucleus from cerebellum)

(B) Decerebrate posturing



Transection between superior and
inferior colliculi (mid brain)

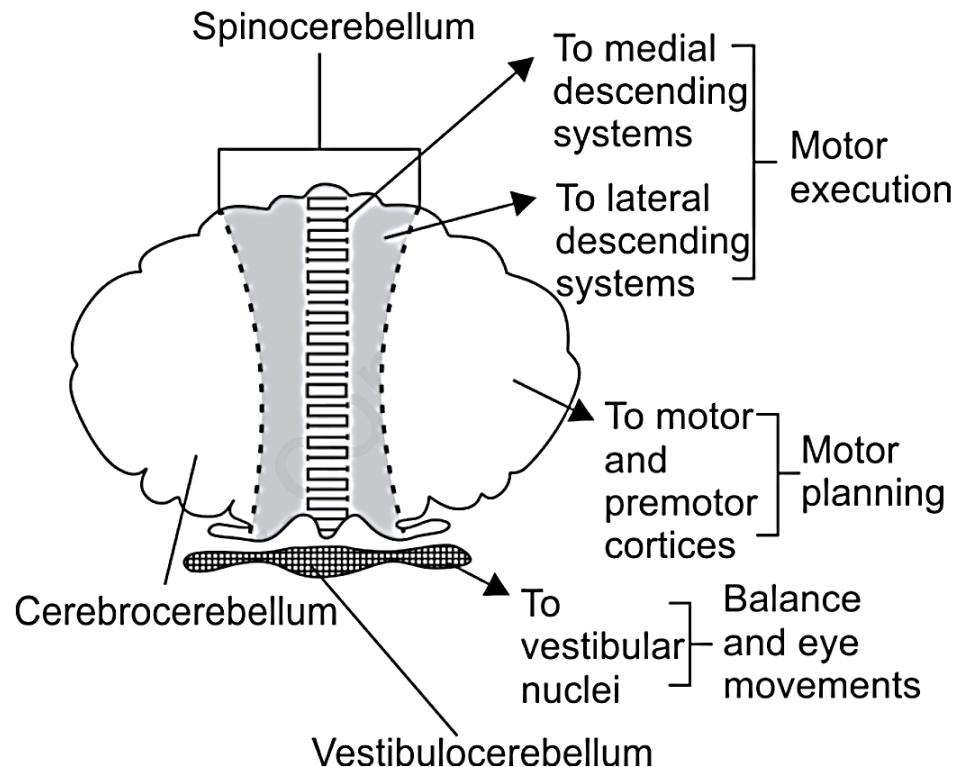
Postural Reflexes and Centres

Reflexes	Integrated in
Stretch reflex	Spinal cord
Positive supporting reaction (Magnet)	
Negative supporting reaction	
Tonic labyrinthine and tonic neck reflexes (Antigravity reflexes attitudinal reflexes)	Medulla
Righting reflexes (except optical righting reflex)	Midbrain
Labyrinthine righting reflexes	
Neck righting reflexes	
Body on head righting reflexes	
Body on body righting reflexes	
Conditioned reflex	Cortex
Optical righting reflexes	
Hopping and Placing reaction	

CEREBELLUM

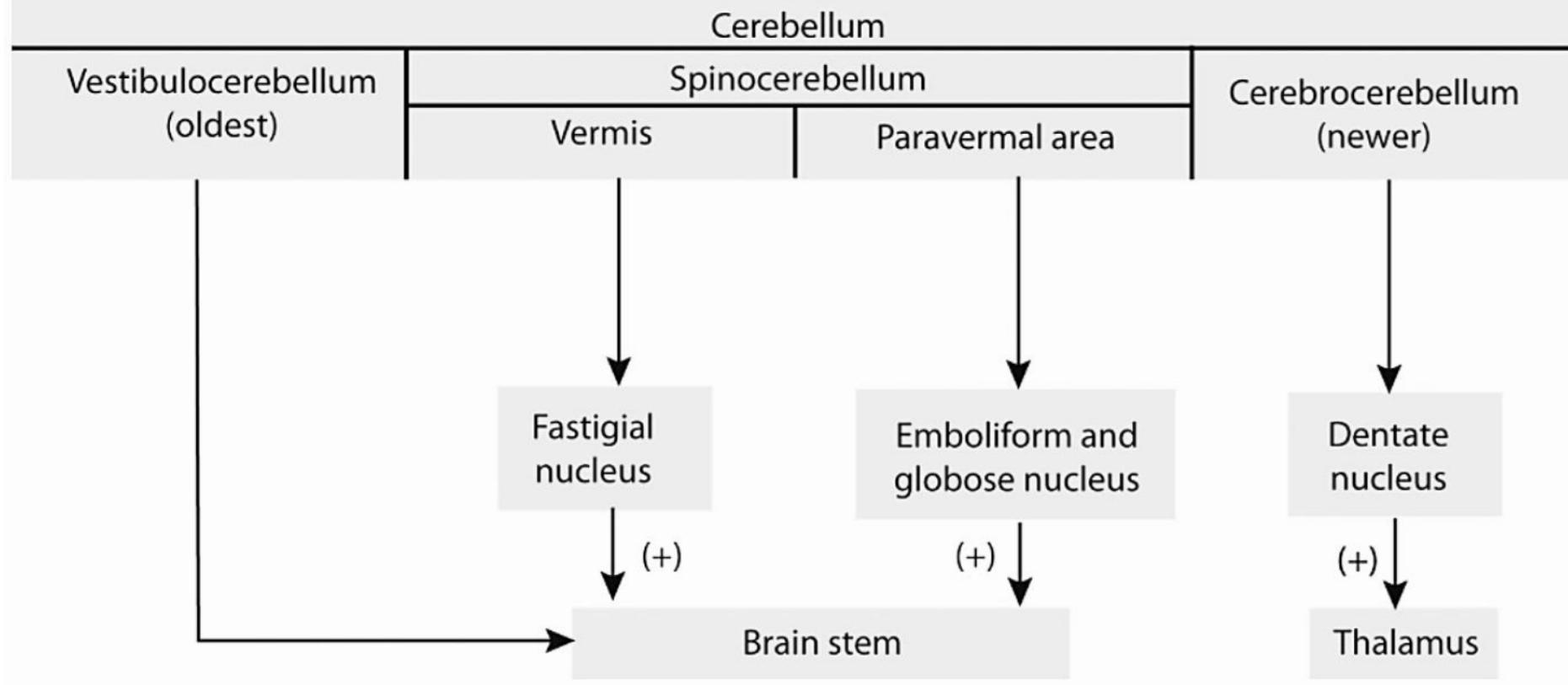
The cerebellar has 3 layers, 4 nuclei and 5 types of cells:

- ❖ **The 3 layers of cerebellar cortex are:** outer molecular layer, middle Purkinje layer, inner granular.
- ❖ **The 4 deep nuclei are** (4 on each side): Dentate, Emboliform, Fastigial, Globose (*emboliform and globose are together referred to as the Interposed*)
- ❖ **The 5 cells are:** Purkinje, Granular, Golgi, Stellate, Basket



Layers of cerebellar cortex (outside to inside):

1. Molecular layer—Basket cells (BC), stellate cells (SC)
2. Purkinje layer—Purkinje cells (PC)—biggest
3. Granular layer—Granule cells (GrC, only excitatory cell that release glutamate), golgi cells (GC)



vestibulocerebellum = flocculonodular lobe concerned with equilibrium (postural balance) and eye movements

spinocerebellum: smooths and coordinates movements

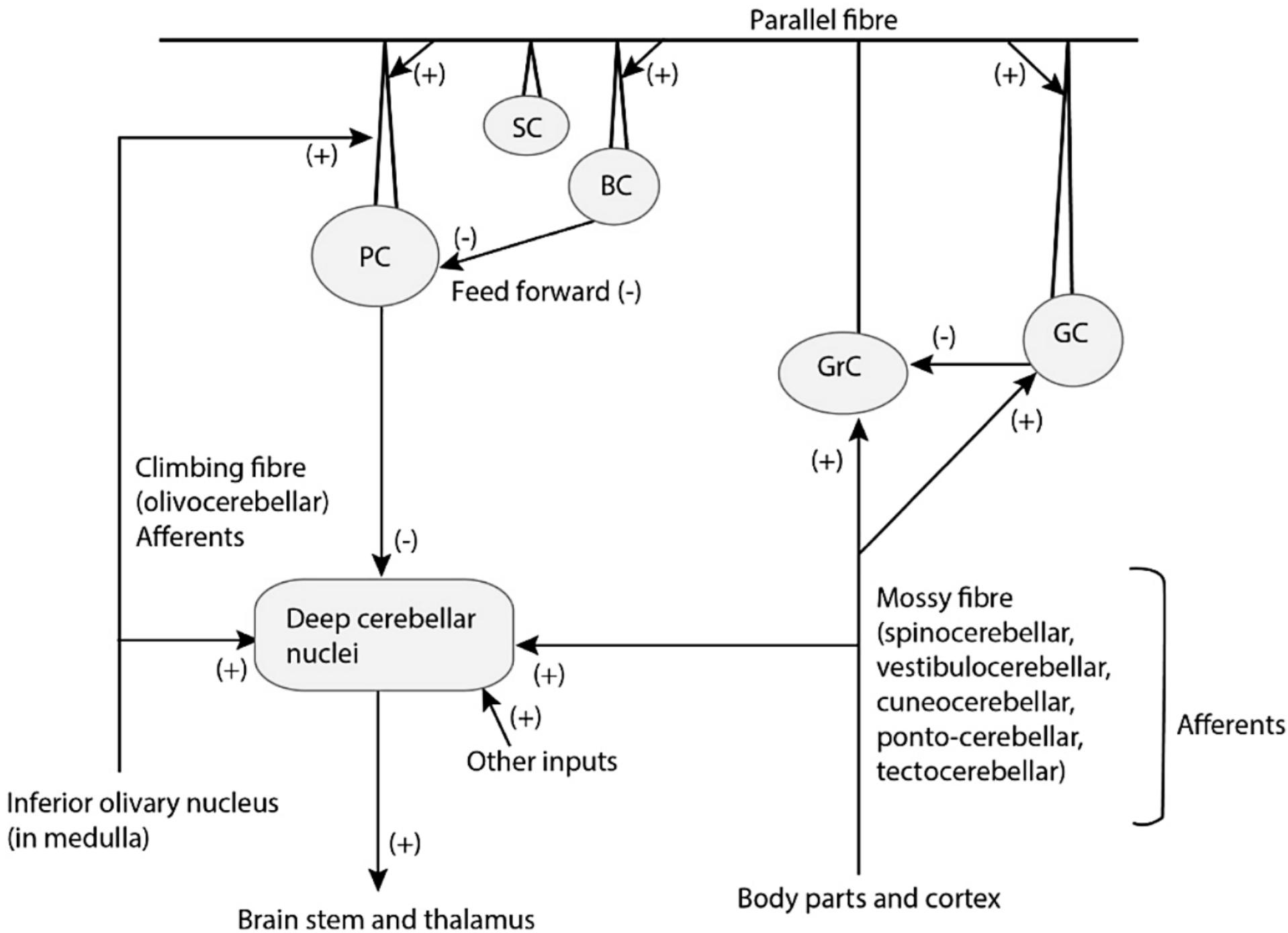
cerebrocerebellum: planning and programming movements

Initiation of skilled voluntary movements: cortical association area

Afferents or input to cerebellum

1. Climbing fibres carry proprioceptive input from whole body (olivocerebellar tract).
 2. Mossy fibres carry input to cerebellum through rest of the tracts
-
- Climbing fiber inputs exert a strong excitatory effect on single Purkinje cells, whereas mossy fiber inputs exert a weak excitatory effect on many Purkinje cells via the granule cells
 - The basket and stellate cells are also excited by granule cells via their parallel fibers; and the basket and stellate cells, in turn, inhibit the Purkinje cells (feedforward inhibition)

The output of the Purkinje cells is in turn inhibitory to the deep cerebellar nuclei: dentate, globose, fastigial, emboliform



Lesion of cerebellum produces:

1. Hypotonia
2. Ataxia
3. Intention tremor
4. Dysmetria
5. Dysdiadochokinesia
6. Rebound phenomenon
7. Disequilibrium
8. Nystagmus
9. Decomposition of movements

BASAL GANGLIA

→ subcortical masses of grey matter

1. caudate nucleus }
2. Putamen } corpus striatum

Lentiform
nucleus

3. Globus Pallidus

↳ GP Externa

↳ GP Interna

4. substantia nigra

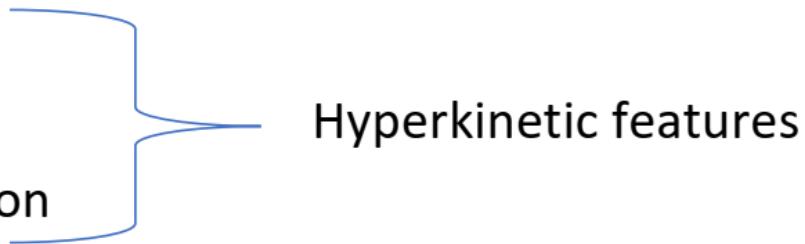
↳ Pars compacta

↳ Pars reticulata

5. Subthalamic nucleus / Body of Luys

Basal ganglia disorder:

1. resting tremor (Hallmark of Parkinsonism)
2. rigidity: cogwheel and lead pipe type
3. akinesia or bradykinesia
4. athetosis: putamen lesion
5. chorea: caudate nucleus lesion
6. hemiballismus: subthalamic nucleus lesion



- Parkinsonism: loss of dopamine containing neurons in substantia nigra (nigrostriatal tract)
- Huntington disease: degeneration of the striatum (mainly caudate nucleus) with selective loss of GABAergic and cholinergic neurons
- Wilson's disease or hepatolenticular degeneration: lenticular nuclei (pallidus and putamen) affected due to excess copper deposition

HYPOTHALAMUS

Paraventricular and supraoptic nuclei

- regulate water balance
- produce ADH and oxytocin
- destruction causes diabetes insipidus
- paraventricular nucleus projects to autonomic nuclei of brainstem and spinal cord

Anterior nucleus

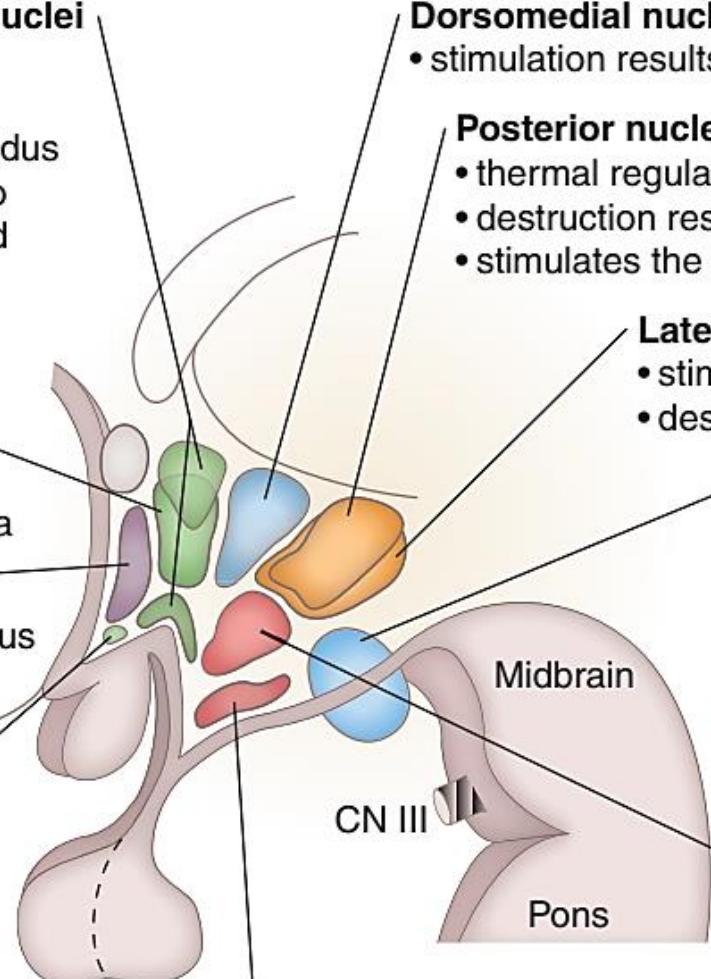
- thermal regulation (dissipation of heat)
- stimulates parasympathetic NS
- destruction results in hyperthermia

Preoptic area

- contains sexually dimorphic nucleus
- regulates release of gonadotropin-releasing hormone

Suprachiasmatic nucleus

- receives input from retina
- controls circadian rhythms



Dorsomedial nucleus

- stimulation results in obesity and savage behavior

Posterior nucleus

- thermal regulation (conservation of heat)
- destruction results in inability to thermoregulate
- stimulates the sympathetic NS

Lateral nucleus

- stimulation induces eating
- destruction results in starvation

Mammillary body

- receives input from hippocampal formation via fornix
- projects to anterior nucleus of thalamus
- contains hemorrhagic lesions in Wernicke's encephalopathy

Ventromedial nucleus

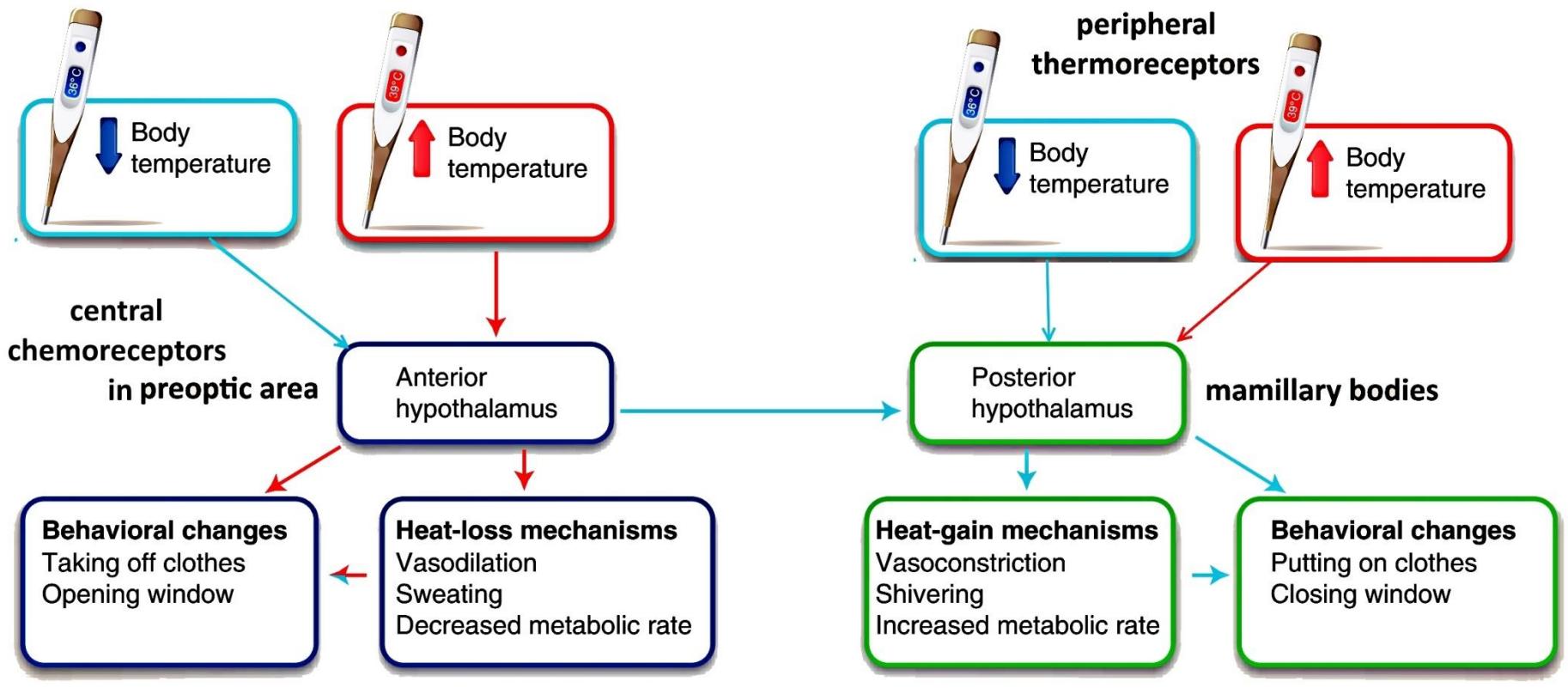
- satiety center
- destruction results in obesity and savage behavior

Arcuate nucleus

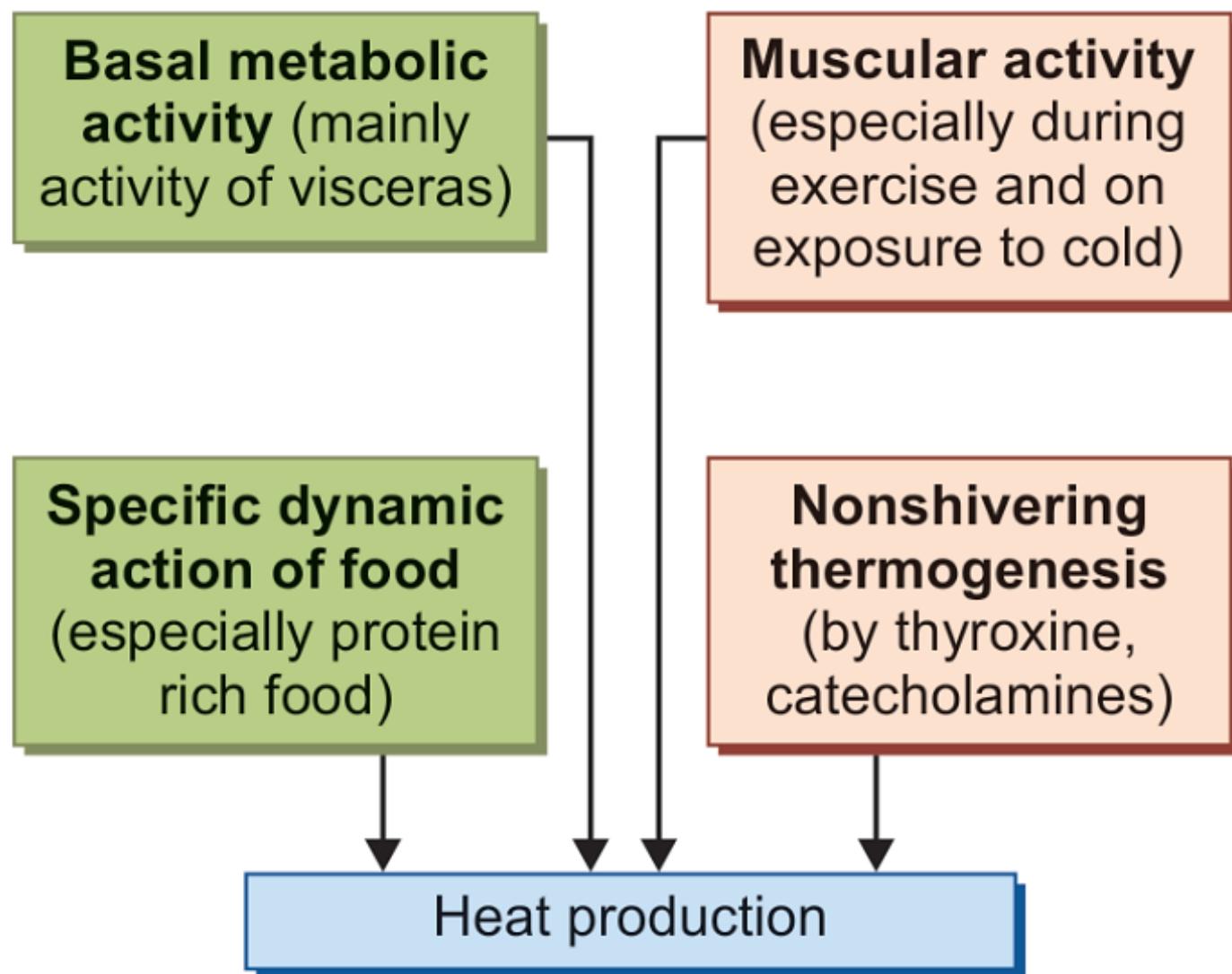
- produces hypothalamic releasing factors
- contains DOPA-ergic neurons that inhibit prolactin release

Body temperature regulation

- Peripheral thermoreceptors: concerned with preventing hypothermia. The skin has far more (upto 10 times) cold receptors than warmth receptors.
- Central thermoreceptors: anterior hypothalamic-preoptic area contains large numbers of heat-sensitive neurons, as well as about one third as many cold-sensitive neurons
- Anterior hypothalamus: heat loss centre (response to heat)
- Posterior hypothalamus: heat gain centre (response to cold)



Mechanisms of heat production in the body.



Walls

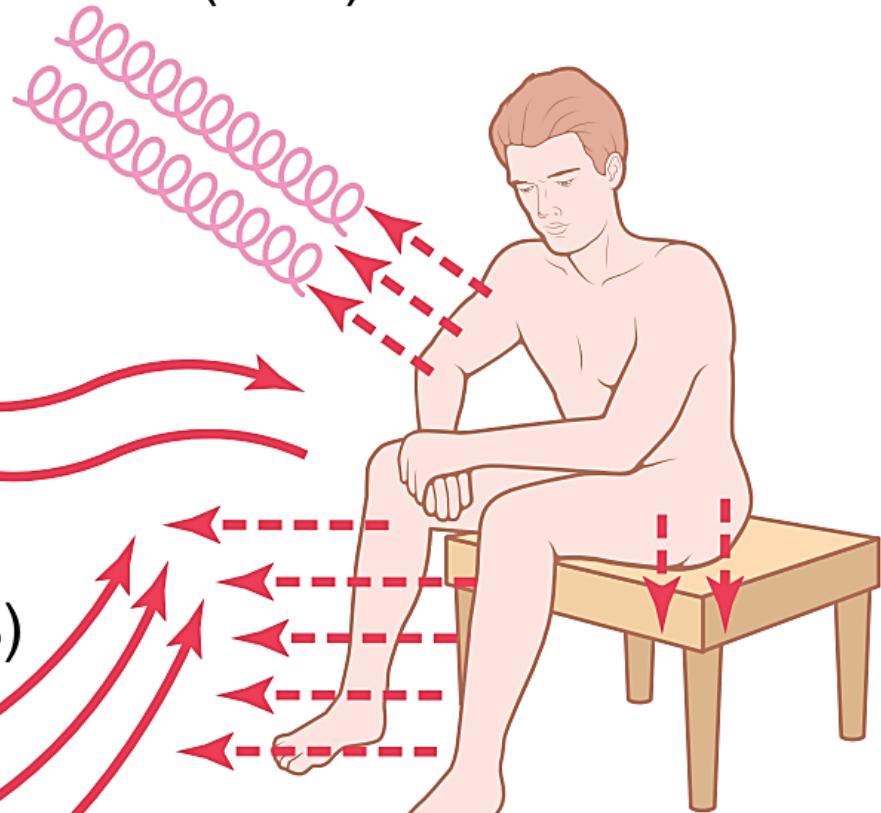
Evaporation (22%)

Radiation (60%)
heat waves

Conduction to air (15%)

Air currents
(convection)

Conduction to
objects (3%)



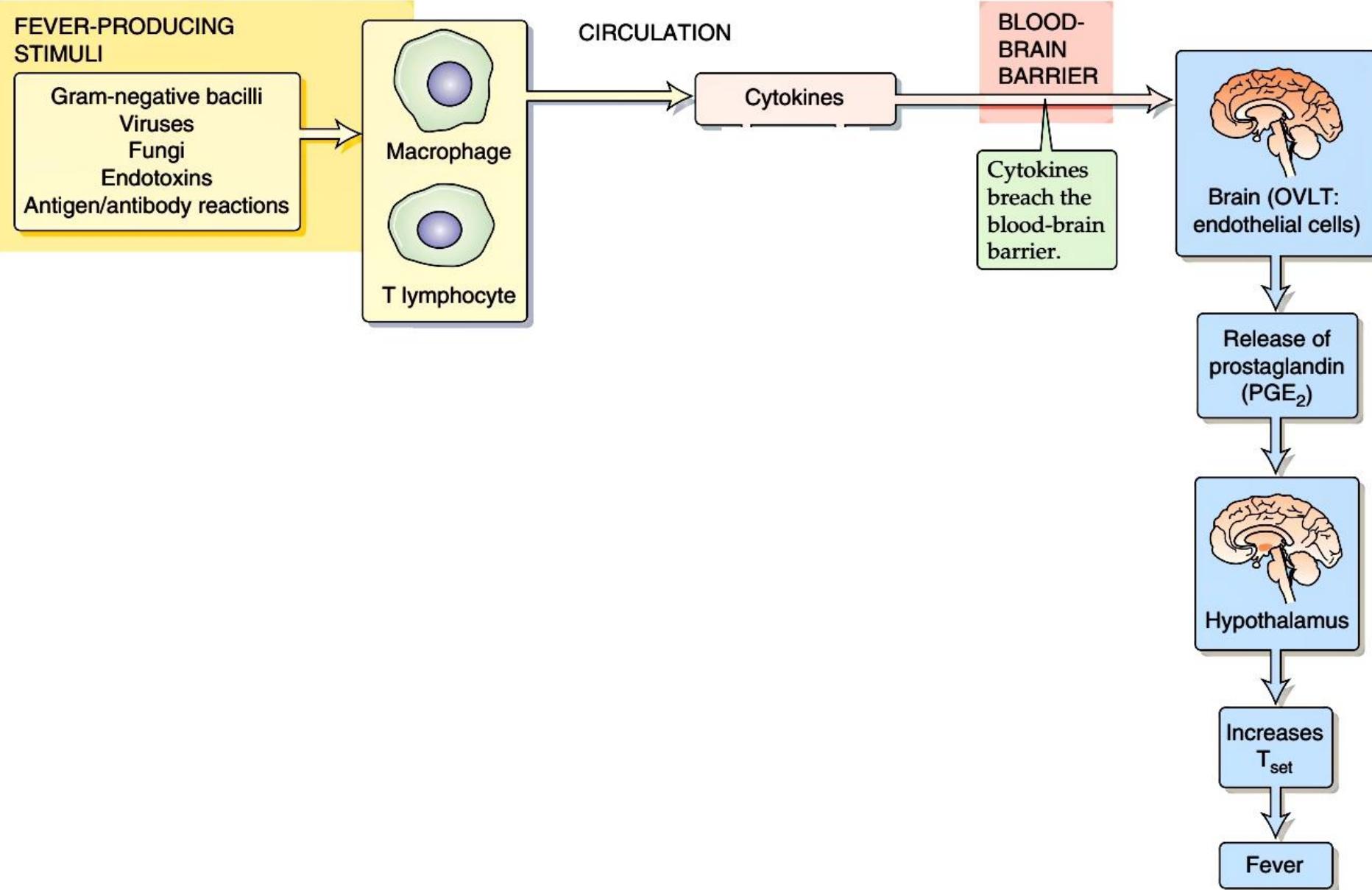
Mechanisms of heat loss from the body.

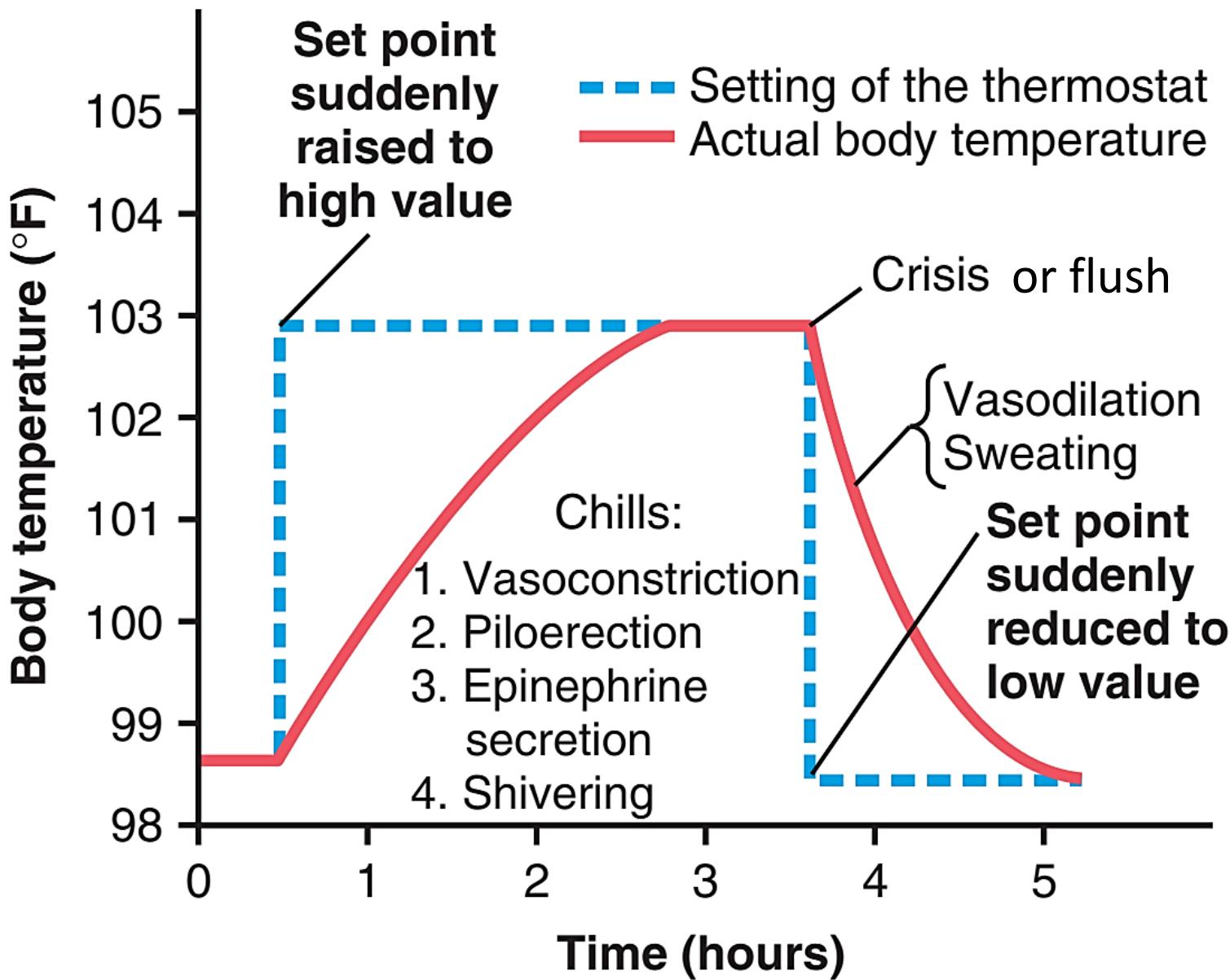
Factors affecting heat balance

Heat gain	Heat loss
<p><i>Increased heat production</i></p> <ol style="list-style-type: none">1. Shivering*2. Neurohumoral stimulation of metabolic rate*3. Exercise ↑ voluntary activity4. Thermic effect of feeding Hunger	<p><i>Increased heat dissipation</i></p> <ol style="list-style-type: none">1. Cutaneous vasodilation*2. Sweating*3. Panting**4. Air movement5. Cooler environment6. Increased surface area <p>7. Increased respiration</p>
<p><i>Decreased heat loss</i></p> <ol style="list-style-type: none">1. Cutaneous vasoconstriction*2. Increased insulation (clothing/fur) Horripilation3. Reduced air movement4. Warmer environment5. Reduced surface area Curling up	<p><i>Decreased heat conservation</i></p> <p>Reduced insulation (clothing/fur)</p> <p>Anorexia</p> <p>Apathy and inertia</p>

*Basic physiological mechanisms of temperature regulation in man.

**A basic physiological mechanism of temperature regulation in dog and related animals.

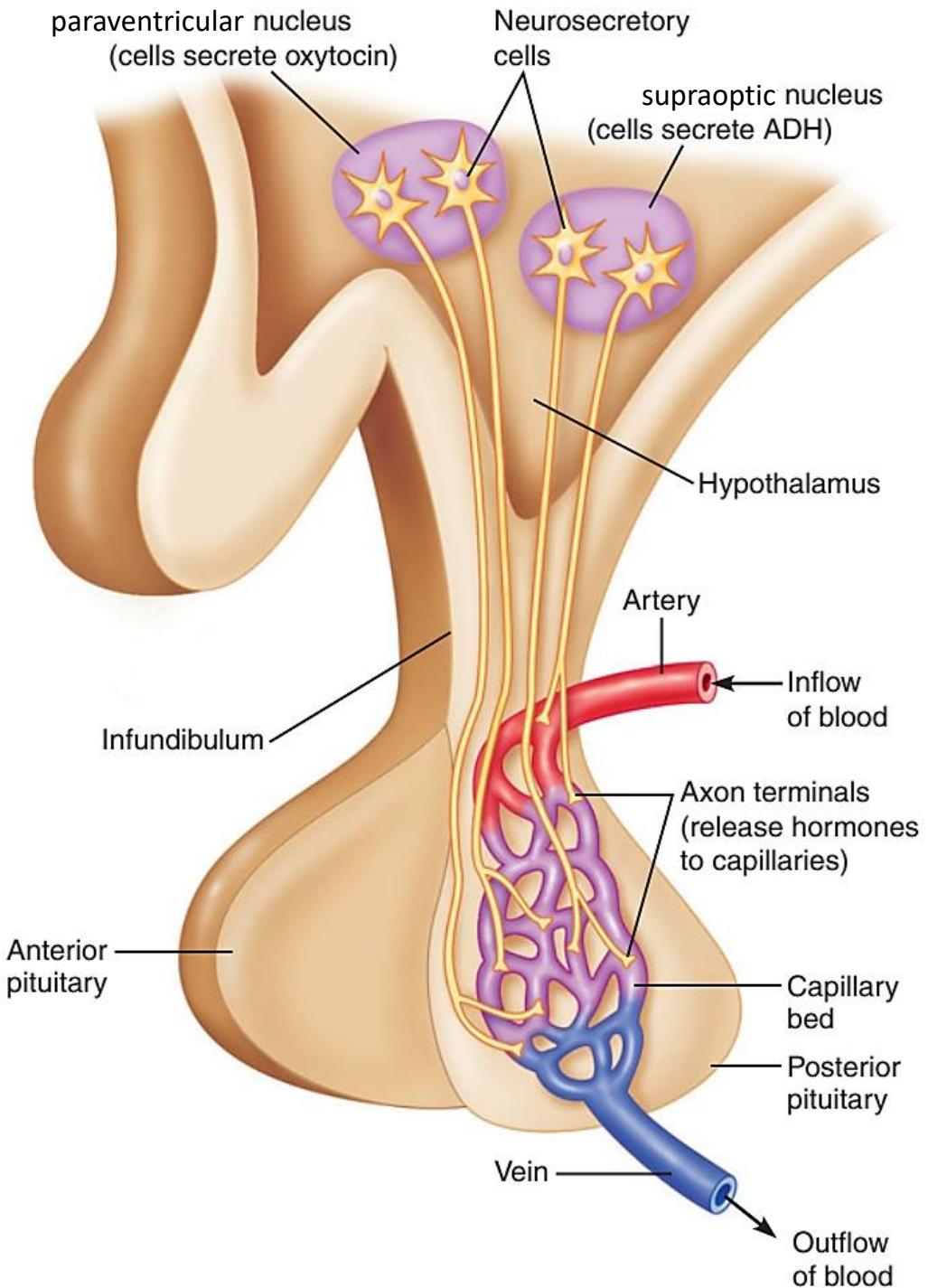




Effects of changing the set point of the hypothalamic temperature controller.

Posterior pituitary hormone synthesis

- ADH: from supraoptic nucleus
- Oxytocin: from paraventricular nucleus



Control of Behavior

- Stimulation in the lateral hypothalamus → overt rage and fighting
- Stimulation in the ventromedial nucleus → tranquillity (state of being quite and peaceful)

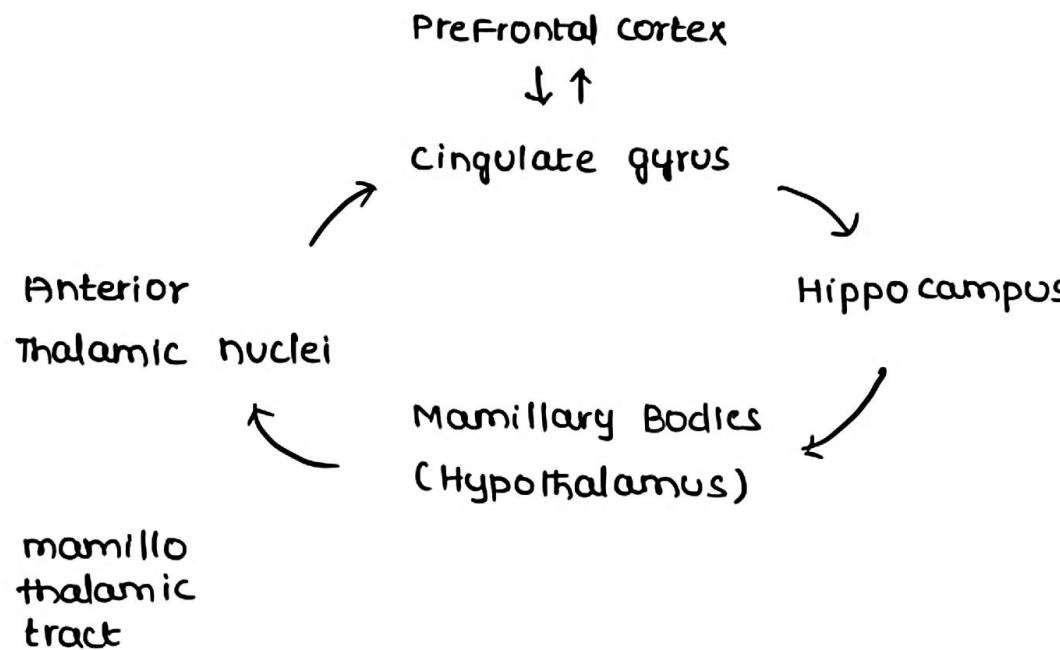
- Punishment centre: located in periventricular zones of the hypothalamus
- Reward centre: located along the course of medial forebrain bundle, especially in lateral and ventromedial nucleus of hypothalamus
- Sexual drive can be stimulated from several areas of the hypothalamus, especially the most anterior and most posterior portions

LIMBIC SYSTEM

- present in Archi cortex
- controls emotions
- LIMBIC SYSTEM → HYPOTHALAMUS + surrounding structures
- Amygdala → Emotional window through which we see the external world

PAPEZ CIRCUIT

- responsible for emotional basis
- connects Neo cortex & Archi cortex

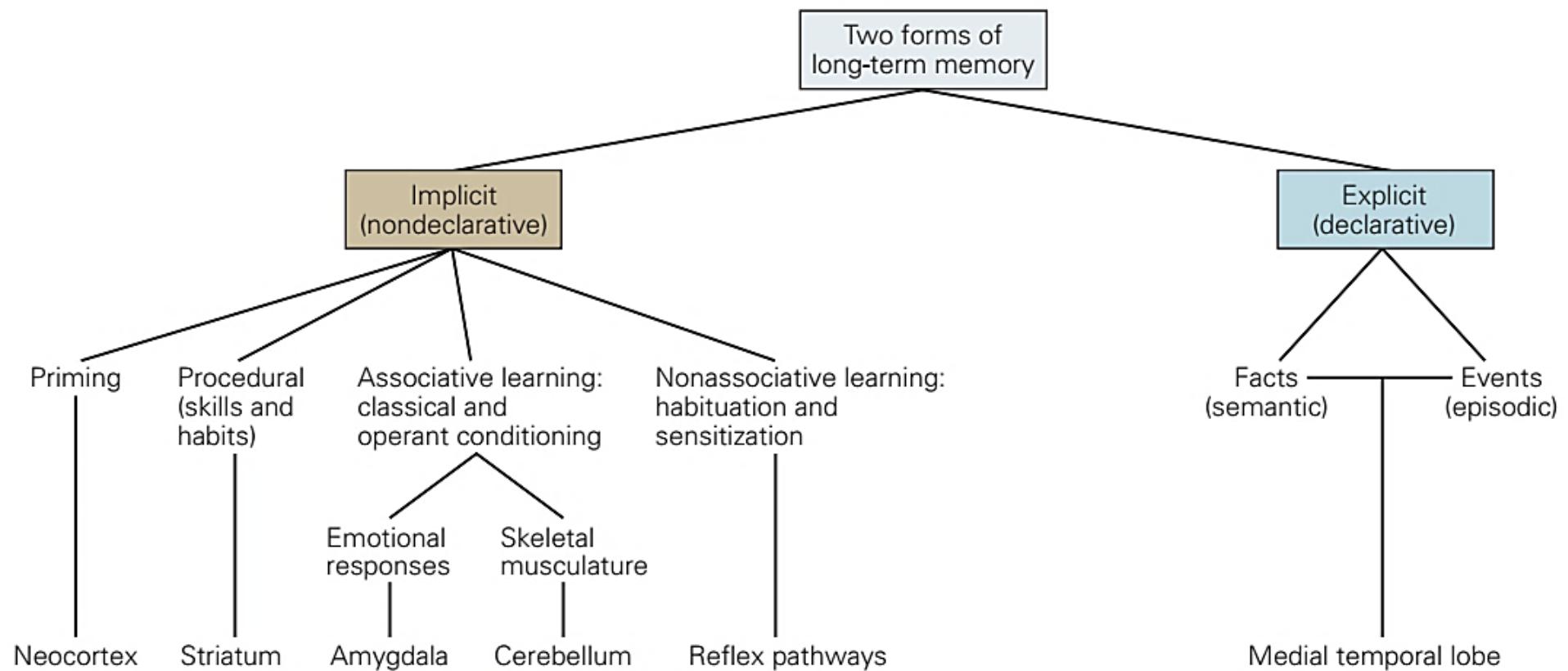


Lesion of amygdala will result in Kluver-Bucy syndrome which is characterised by:

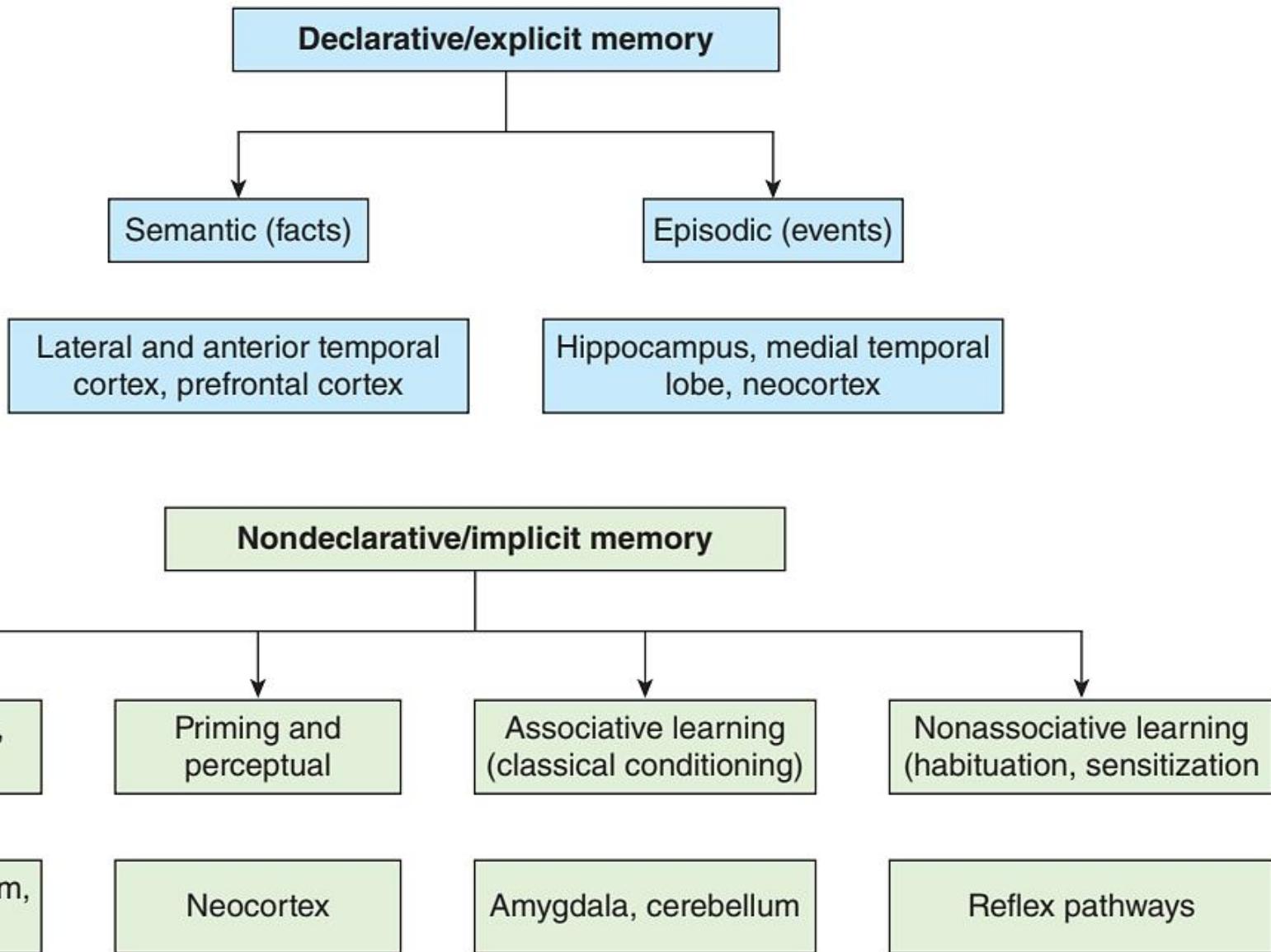
1. Visual agnosia: not able to recognise objects by sight
2. Hyperorality: excessive tendency to examine objects by mouth
3. Placidity: not afraid of anything
4. Hyperphagia
5. Hypersexuality
6. Hypermetamorphosis: has extreme curiosity about everything

LEARNING & MEMORY

→ acquisition of new information & skill → learning
Storage of the skill → memory



Brain regions involved in different kinds of learning and memory



- Depending upon permanency of storage memory is:
 1. **Short-term memory**, also termed as primary memory, lasts up to minutes
 2. **Intermediate long-term memory** (or secondary memory) lasts up to weeks but is eventually lost
 3. **Long-term memory** (or tertiary memory), which once stored, can be recalled years later or for a lifetime
 4. **Working memory** is a complex type of short-term memory. A person plans actions based on working memory. Central executive: *Prefrontal cortex*

CLASSICAL CONDITIONING (PAVLOV)

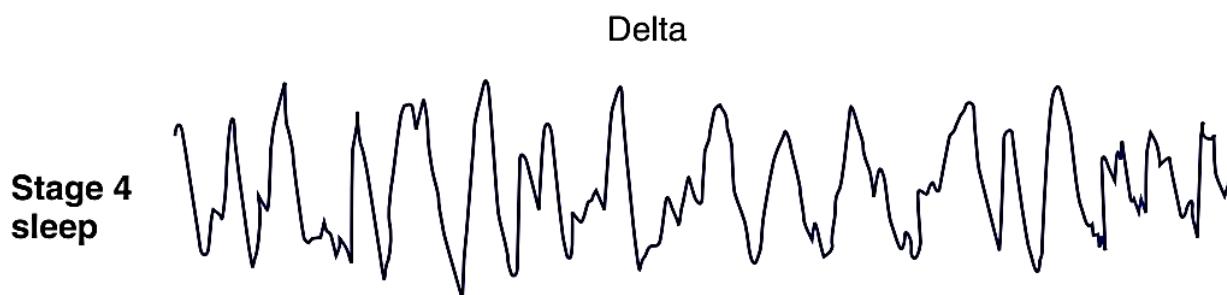
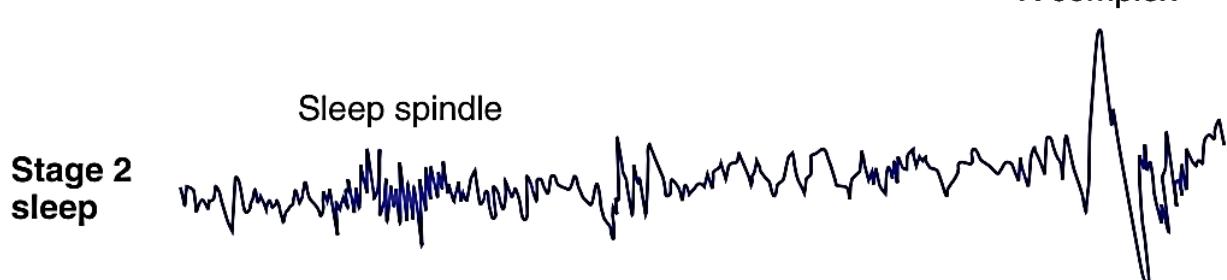
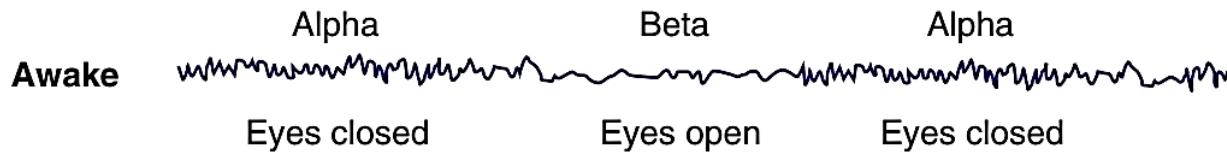
- dog is presented to food pellets, reflex salivation occurs (innate reflex)
- unconditioned stimulus
 - ↳ giving the food
 - ↳ natural
- conditioned stimulus
 - ↳ ringing the bell
 - ↳ learning stimulus
- conditioned stimulus, immediately followed by unconditioned stimulus
 - ↳ Pairing occurs (should be done repeatedly)
- Now salivation occurs even for conditioned stimulus alone

LESION OF HIPPOCAMPUS

- ↳ causes ANTEROGRADE AMNESIA
 - ↳ not able to form long term memory from that point onwards

Types Of Sleep

- **Slow Wave Sleep (non-REM)**
 - Stage 1 NREM
 - Stage 2 NREM
 - Stage 3 NREM
 - Stage 4 NREM
- **Rapid Eye Movement Sleep (REM)**

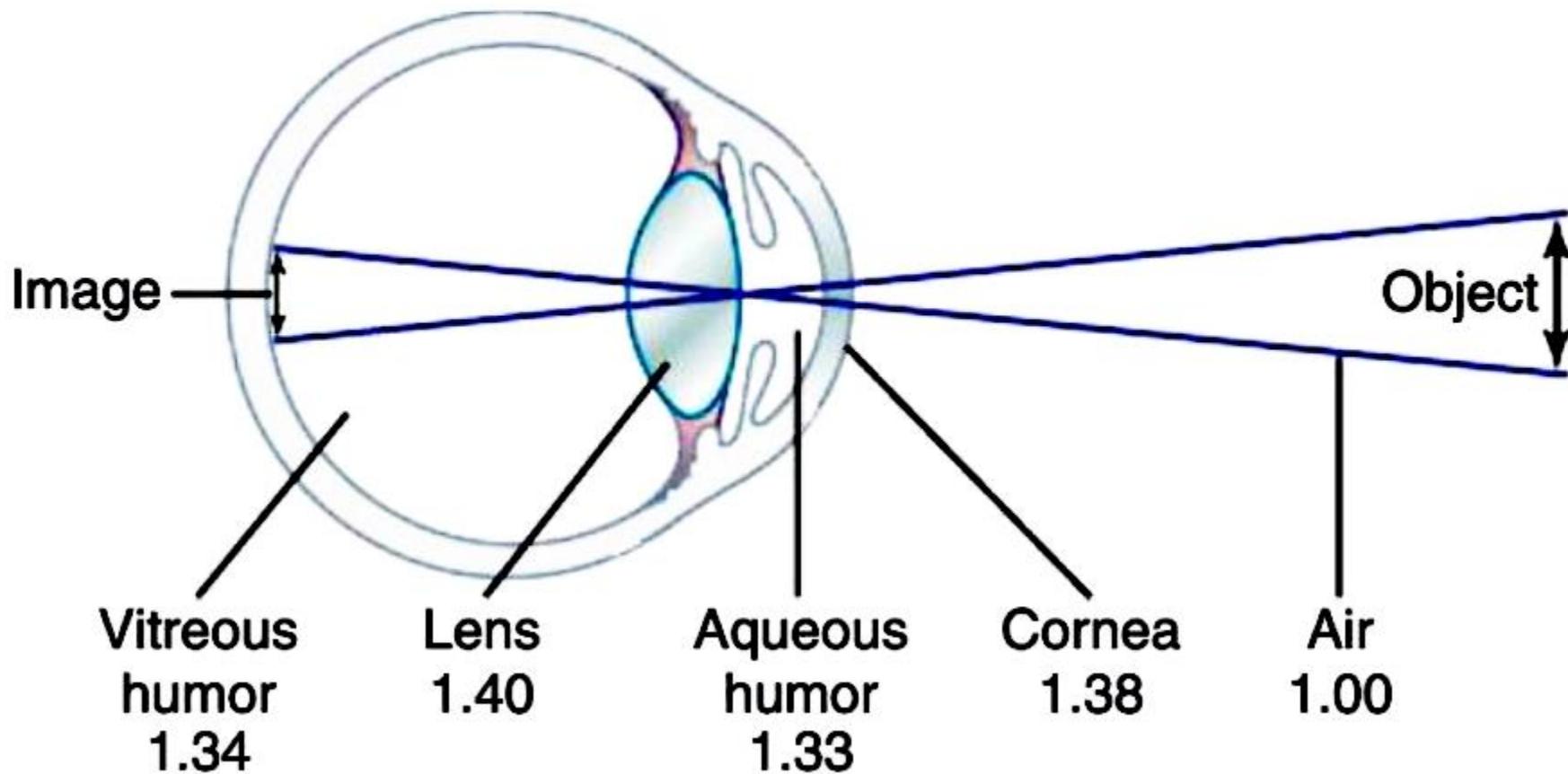


EEG wave	Frequency	Amplitude	Occurrence	site
Delta	0.5-4 Hz	2-4 times of other EEG waves	<ul style="list-style-type: none">• very deep sleep• characteristic of Stage 3 of NREM sleep• infancy• serious organic brain disease	cortex
Theta	4-7 Hz		<ul style="list-style-type: none">• Stage 1 NREM sleep• Emotional stress in adults• Children• Degenerative brain disorder	parietal and temporal regions
Alpha (Berger Rhythm)	8-13 Hz	50–100 µV	<ul style="list-style-type: none">• in almost all healthy adults in relaxed and awake state with eyes closed• Disappear during sleep	parietal and occipital lobes
Beta	13–30 Hz	Low voltage	<ul style="list-style-type: none">• mental activity or excitement• Arousal response (or α block),	parietal and frontal regions

Features	NREM Sleep (Non-Rapid Eye Movement Sleep)	REM Sleep (Rapid Eye Movement Sleep)
Duration	80–100 min/cycle	5–30 min
Type of sleep	Slow wave sleep	Paradoxical sleep/deepest sleep/dreamy sleep/fast wave sleep
Sequence	Followed by REM sleep	Follows NREM sleep
Eye movements	No eye movements	Saccadic eye movements
Autonomic changes	HR, BP, RR—low	HR, BP, RR—reduced and irregular, penile erection
Dream recall	Not Night terror	Recalled nightmare

NREM sleep disorder	
Sleepwalking (somnambulism)	Stage III and IV (stage N3) can occur during REM sleep also.
Somniloquy (sleep talking)	Stages I and II mainly but can possible in all.
Bruxism	Stages I and II mainly (stage II > I). May be in REM and stage III and IV
Nocturnal enuresis	All stages of NREM and REM except stage I (max during stage II)
Night terrors	Transition from stage III to stage IV (Stage N3)
REM sleep disorder	
Narcolepsy	REM sleep
Nightmare	REM sleep

Total refractive power = 59 diopters



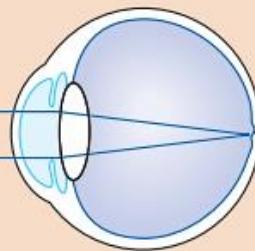
- ▶ Diopteric power of the eye:
Cornea 40-45 D (max refraction) Lens 15-20 D
Accommodation by lens +12 D

Errors of Refraction

- The refractive errors of the eye may be due to abnormality in the following:
 1. Axial length of the eye
 2. Refractive power
 - A. Curvature of the surface of the cornea or the lens
 - B. Refractive indices of the media
 - C. Position of the lens

Emmetropia

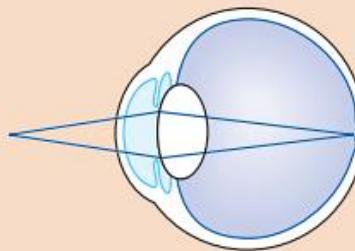
Distant object



Focused
on retina

No accommodation

Near object



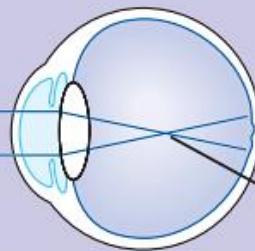
Focused
on retina

Accommodation

Myopia

(Lens of eye too strong for length of eyeball)

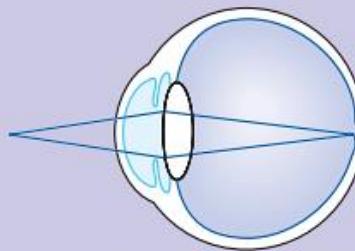
Distant object



Not focused
on retina

No accommodation

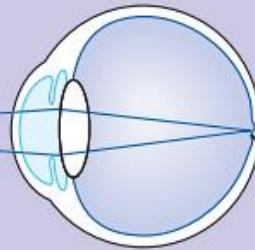
Near object



Focused
on retina

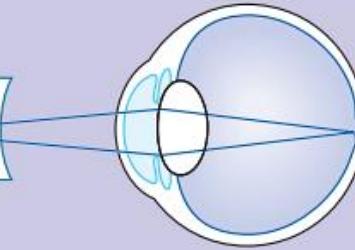
No accommodation

Myopia corrected
with concave lens
(which decreases
overall refractive
power)



Focused
on retina

No accommodation

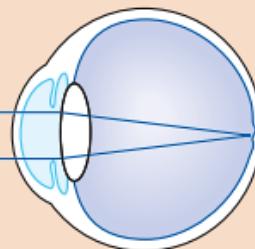


Focused
on retina

Accommodation

Emmetropia

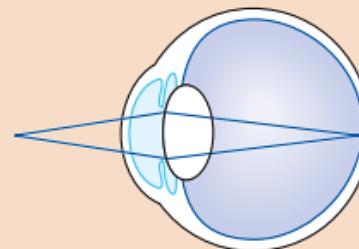
Distant object



Focused
on retina

No accommodation

Near object



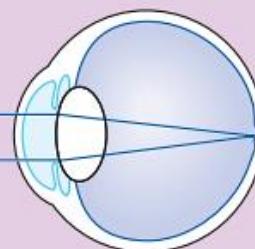
Focused
on retina

Accommodation

Hyperopia

(Lens of eye too weak for length of eyeball)

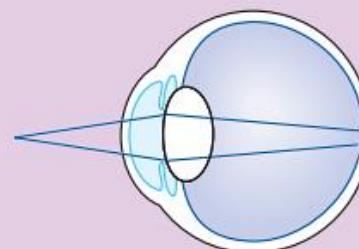
Distant object



Focused
on retina

Accommodation

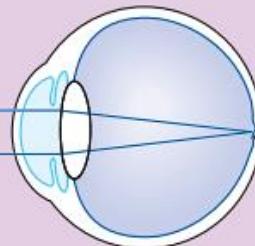
Near object



Not focused
on retina

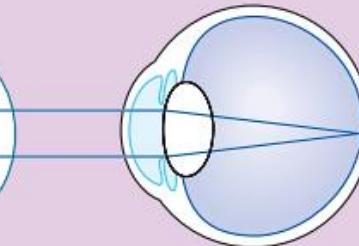
Accommodation

Hyperopia corrected with convex lens
(which increases overall refractive power)



Focused
on retina

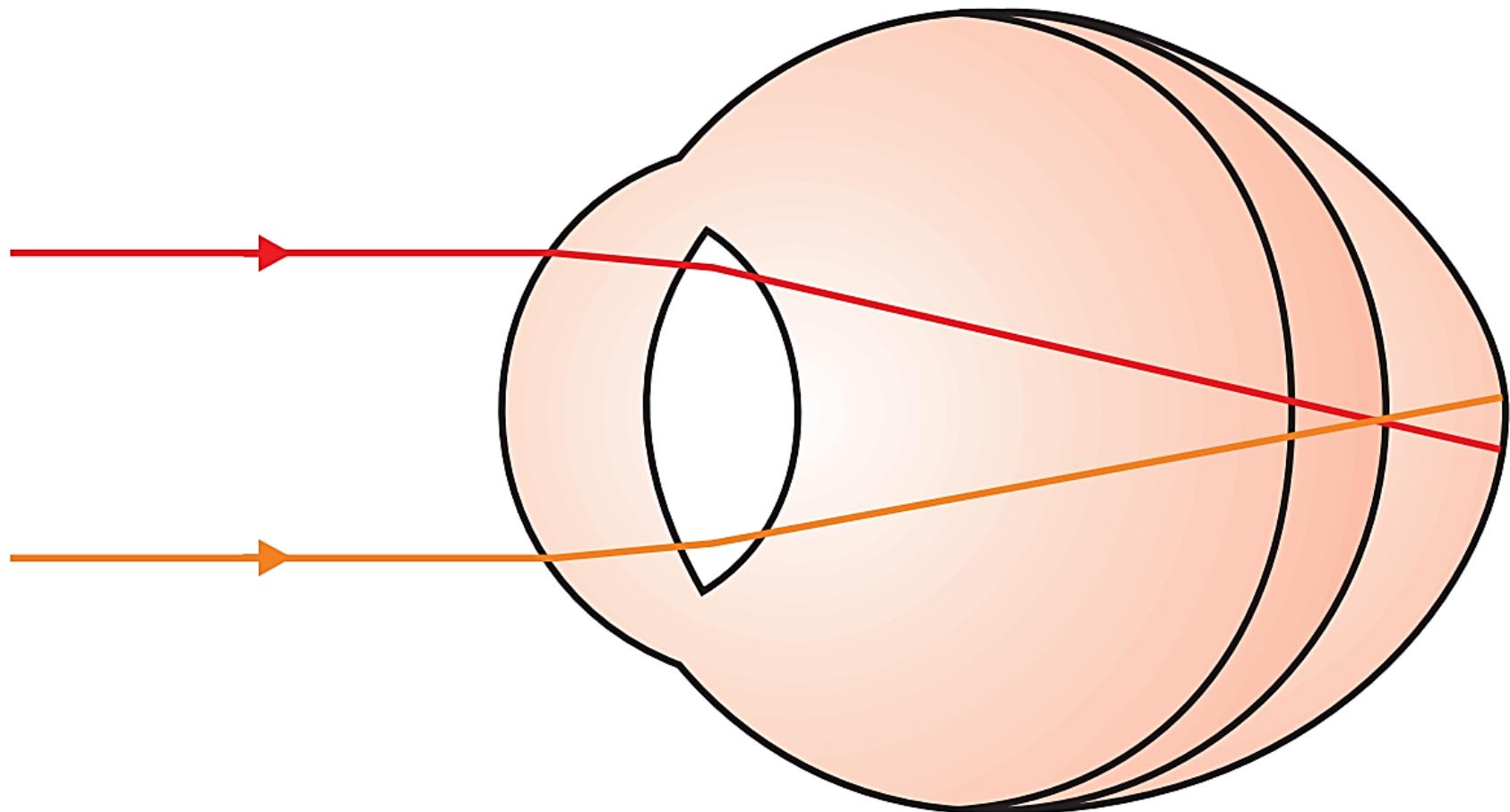
No accommodation



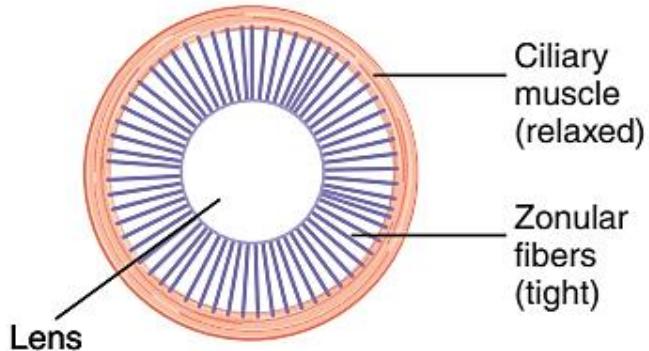
Focused
on retina

Accommodation

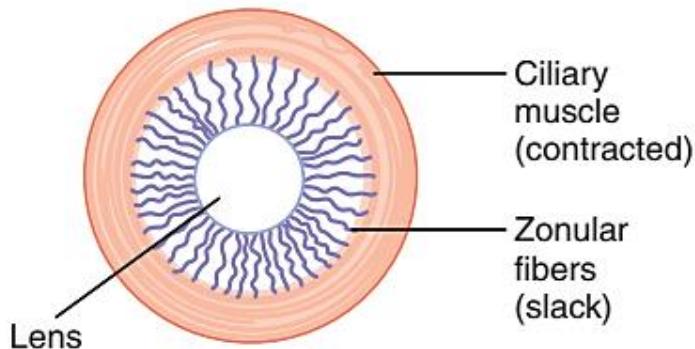
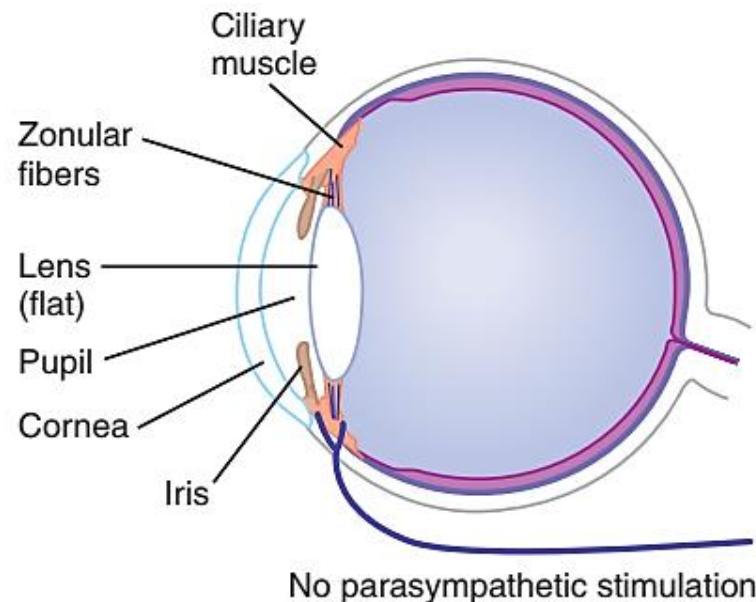
H E M



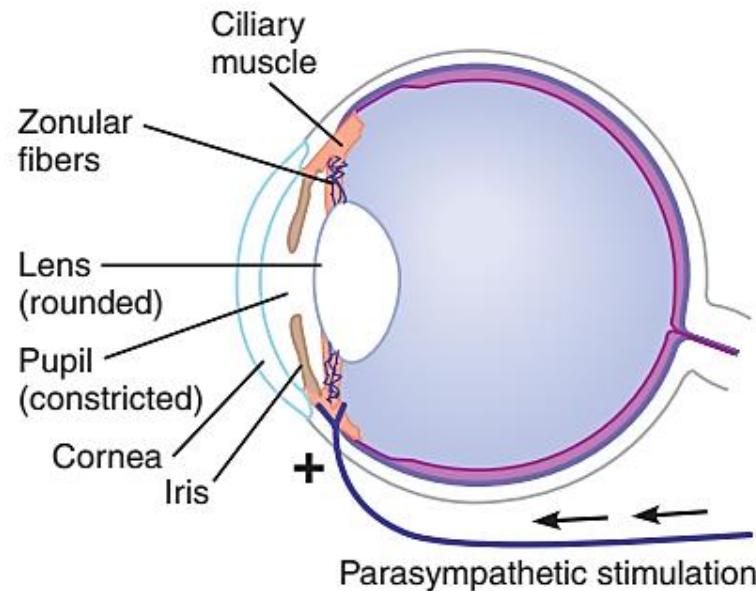
Emmetropia, hypermetropia and myopia. In emmetropia (E), parallel rays of light are focused upon the retina. In hypermetropia (H), the eye is relatively too short; in myopia (M), it is too long.



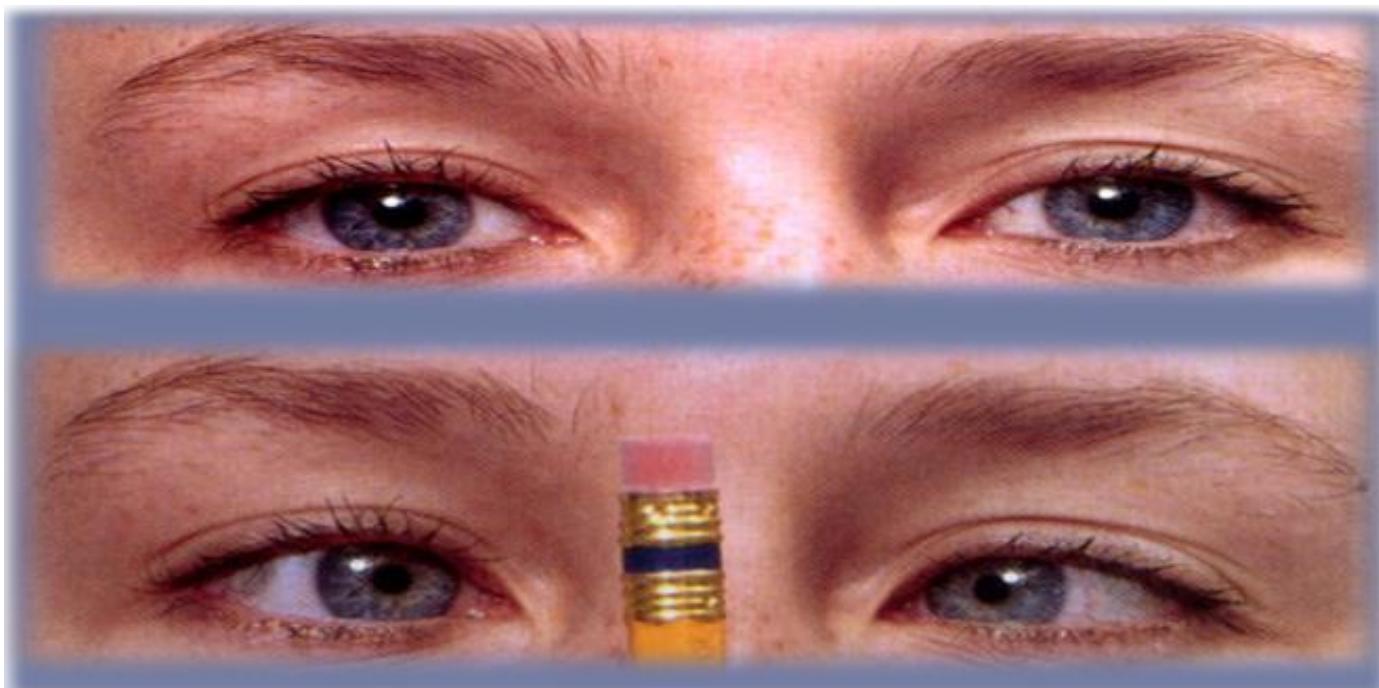
(a) Far vision of distant objects



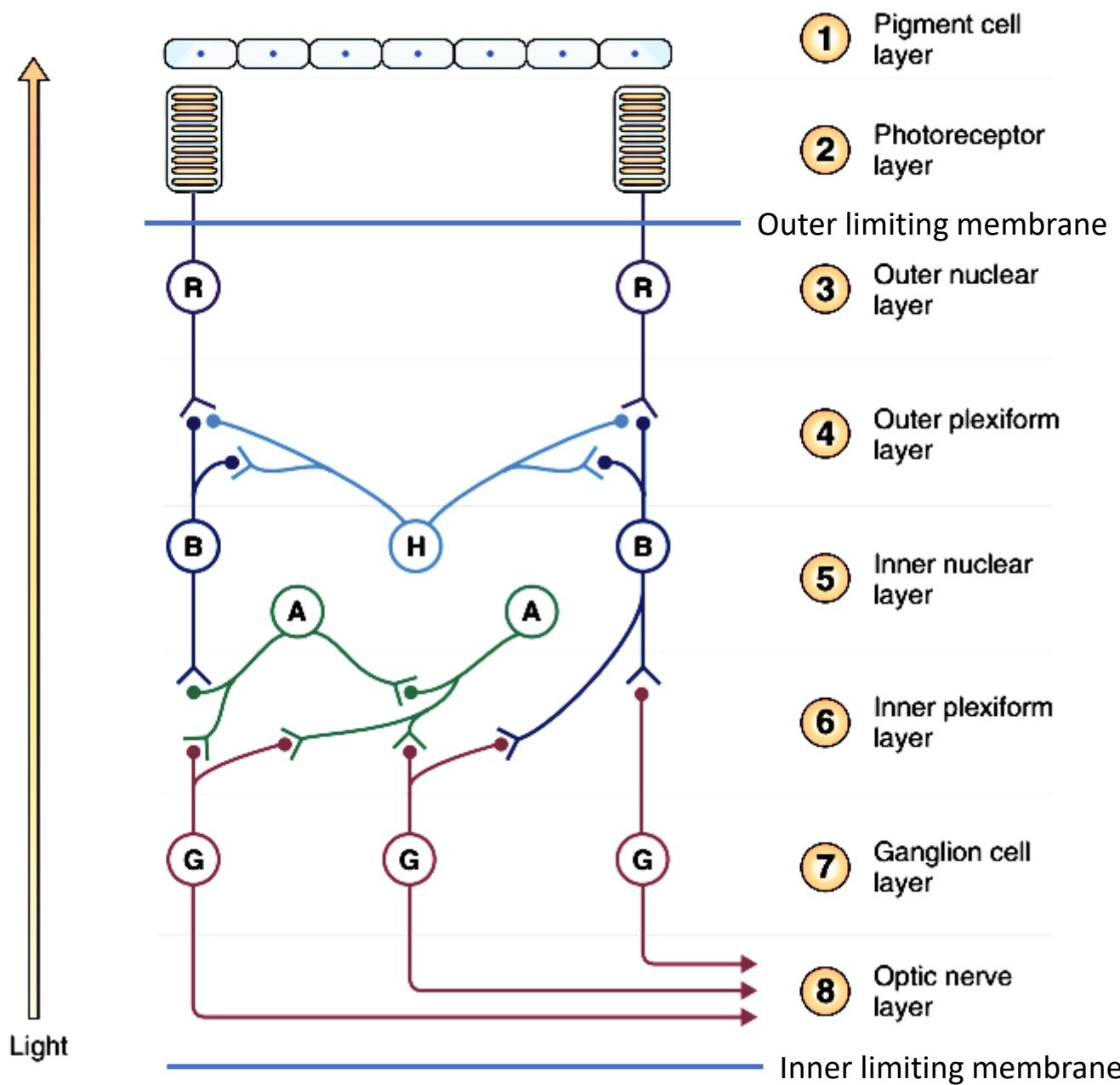
(b) Accommodation for near vision



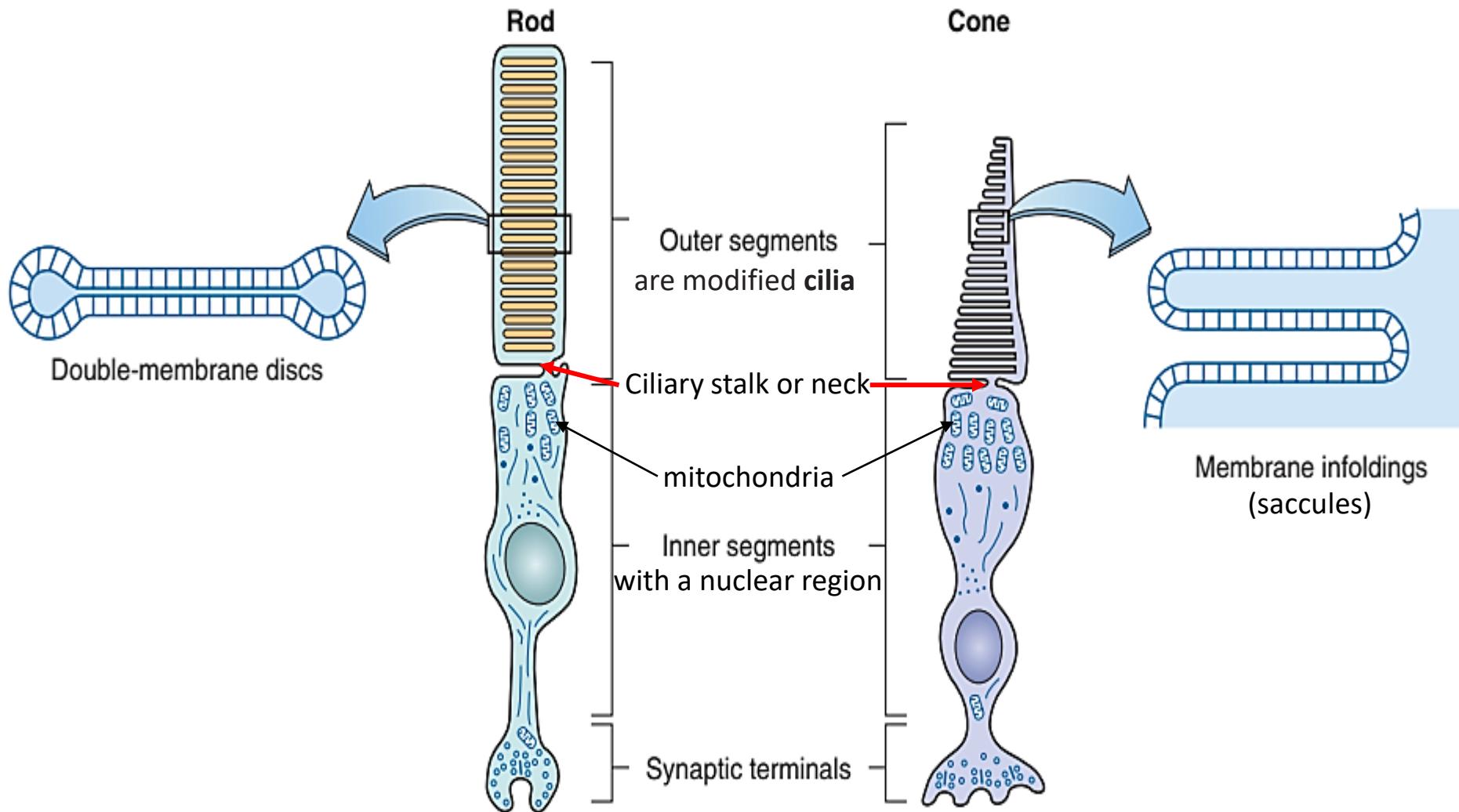
- In addition to accommodation, the visual axes converge and the pupil constricts when an individual looks at a near object.
- This three-part response—accommodation, convergence of the visual axes, and pupillary constriction—is called **the near response**



LAYERS OF THE RETINA



STRUCTURE OF PHOTORECEPTORS



Comparison of Rods and Cones

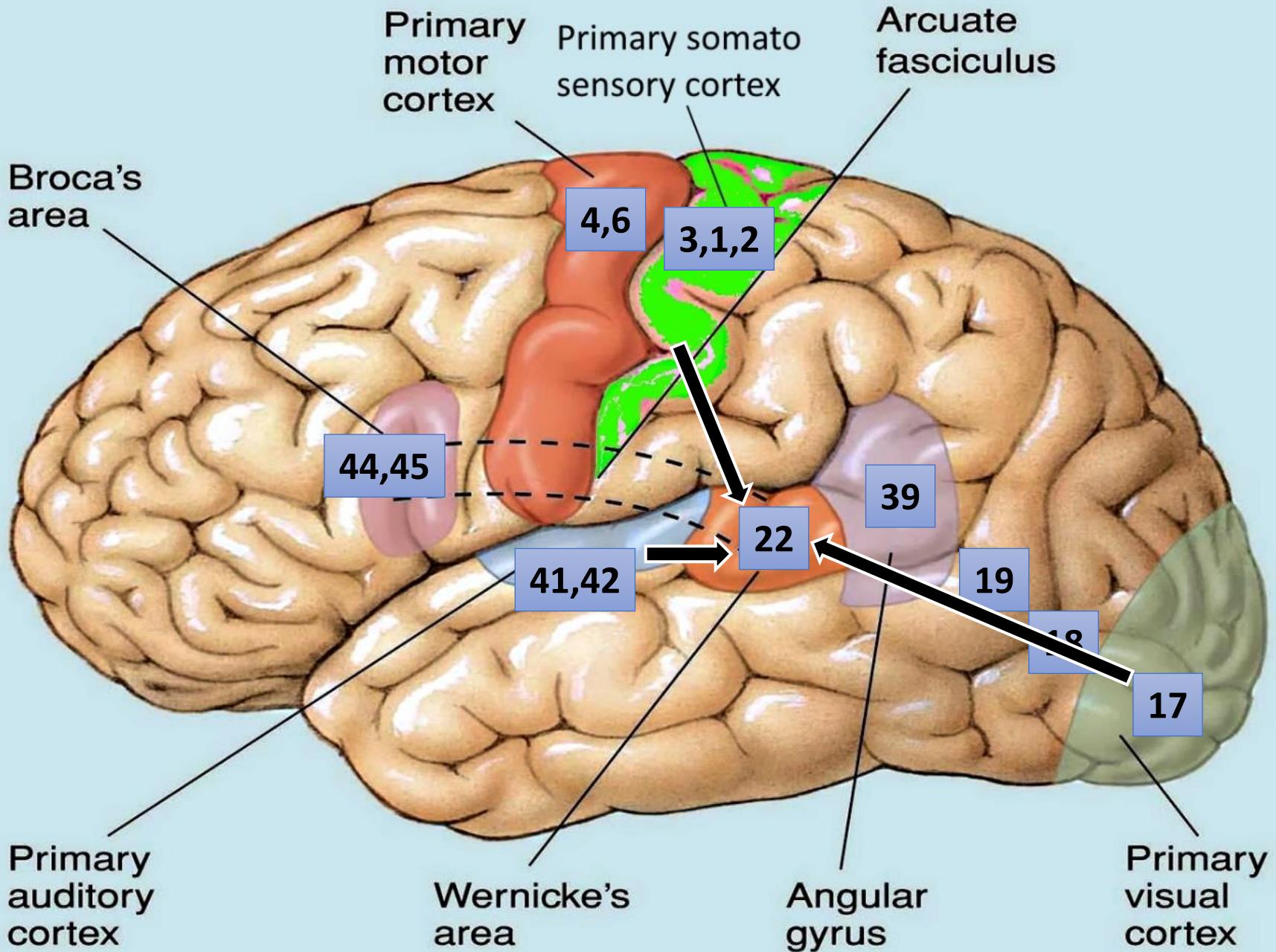
	Cones	Rods
Sensitivity to light stimulus	Low about 30 to 300 times less sensitive than the rods	High
Photosensitive pigments	Less abundant	More abundant
Response to light stimulus	Fast	Slow
Specialized for	Day vision	Night vision
Effects of damage	Loss of cones causes decreased visual acuity (legal blindness)	Loss of rods causes night blindness and loss of peripheral vision
Acuity of vision	Acuity of vision mediated by cones is high	Acuity of vision mediated by rods is low
Saturation	Saturate when light is very intense	Saturate in day light
Role in color vision	Mediate color vision (3 types of cone cells)	They are achromatic
Concentration in fovea	High	Absent in fovea
Relative numbers	Less numerous than rods 6 million per eye	More numerous than cones (20:1) 120 million per eye

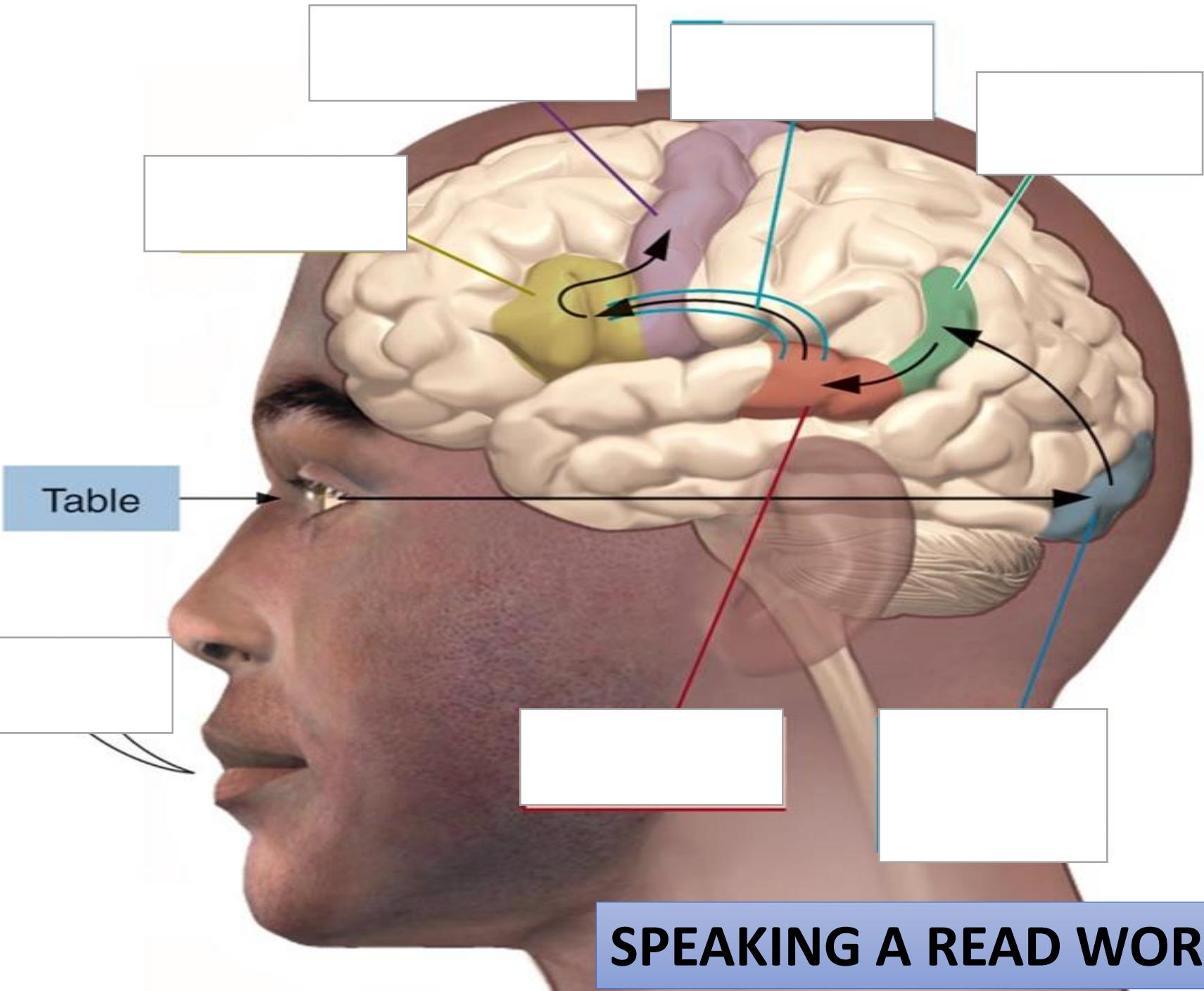
Photopigments = opsin + retinal

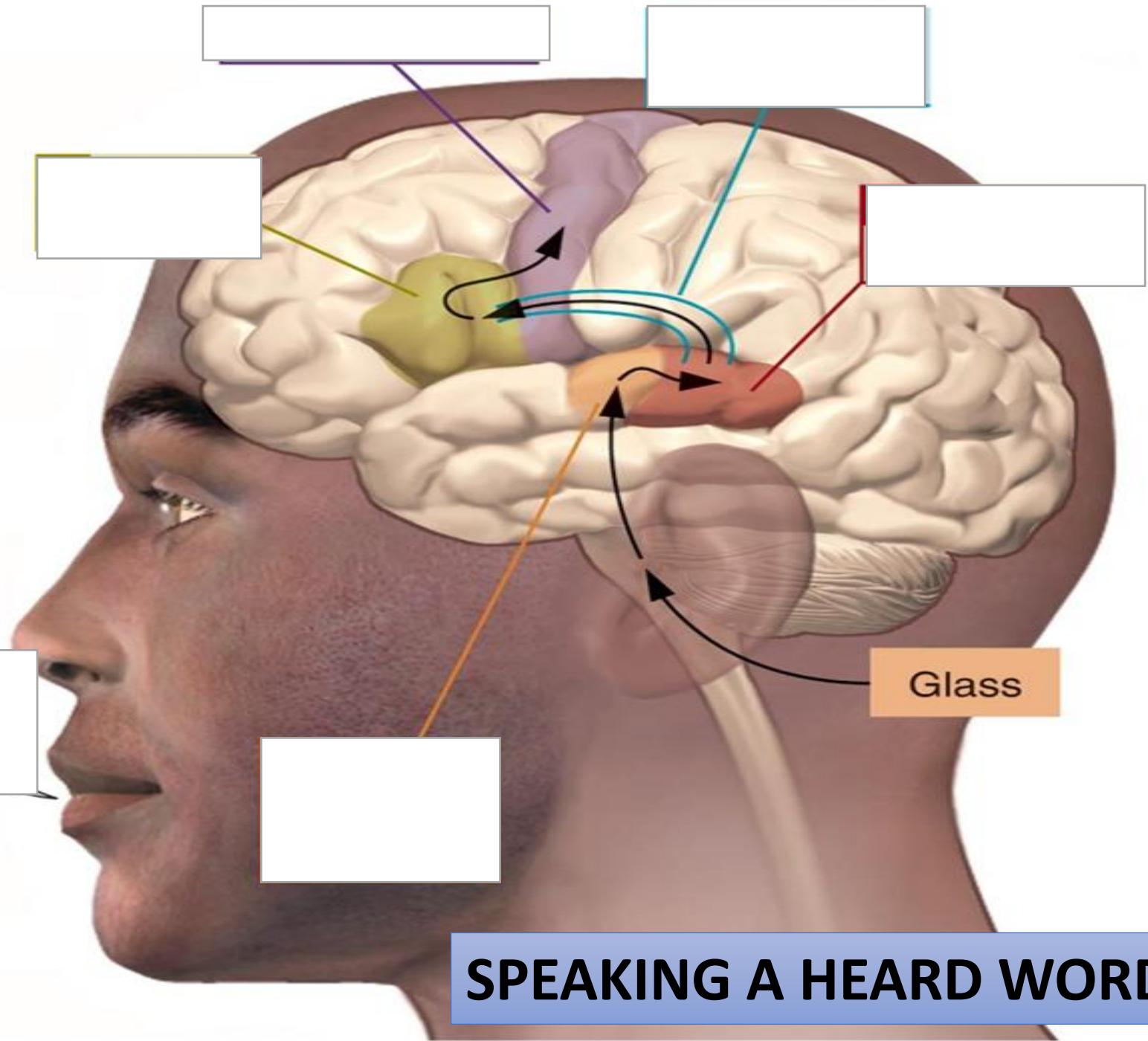
Scotopsin in rods

Photopsin in cones

- The retinal (retinene, retinaldehyde) portion is the same in all photopigments, but the kind of opsin present determines which light wavelengths are absorbed by a given photopigment
- The four photo-pigments include
 1. **Rhodopsin** (visual purple, blue-green sensitive 505 nm) in rods
 2. **LWS cone pigment** (erythrolabe, red sensitive 570 nm),
 3. **MWS cone pigment** (chlorolabe, green sensitive 535 nm),
 4. **SWS cone pigment** (cyanolabe, blue sensitive 445nm)



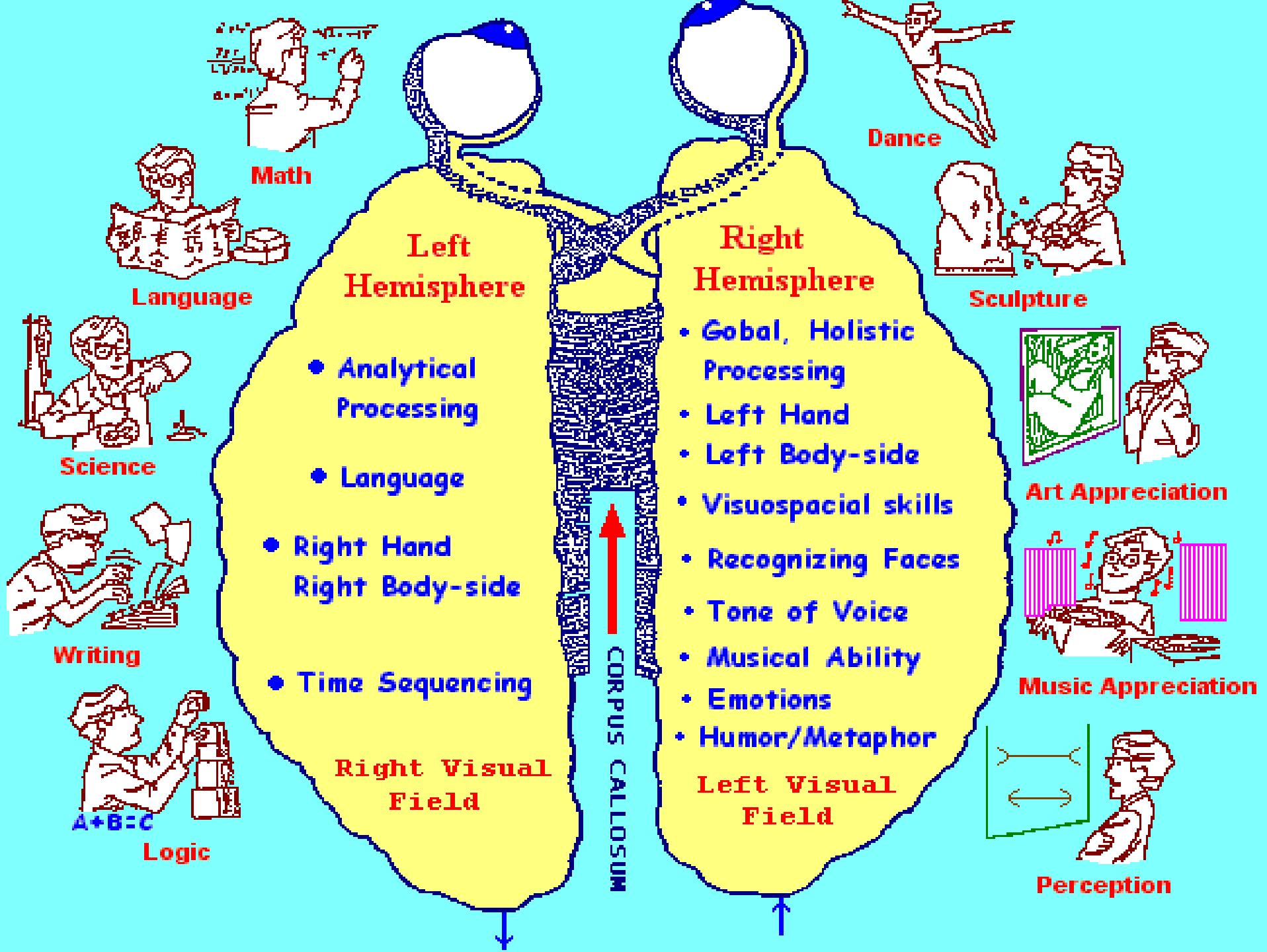




Type of Aphasia	Site of Brain Damage	Comprehension	Speech
Broca's	Inferior frontal gyrus	Preserved	Non fluent, agrammatical
Wernicke's	Posterior temporal lobe	Impaired	Fluent, meaningless
Conduction	Arcuate fasciculus	Preserved	Fluent

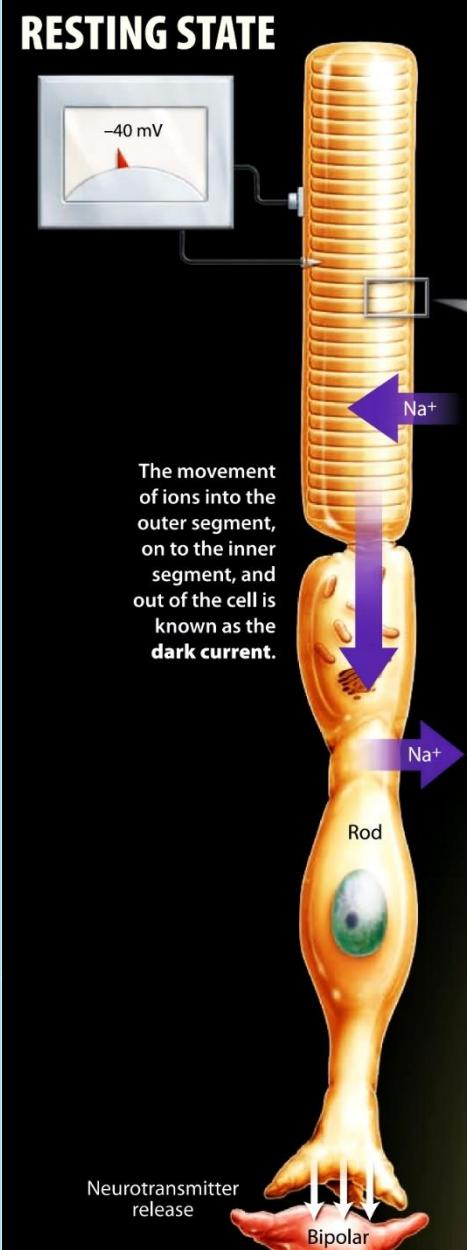
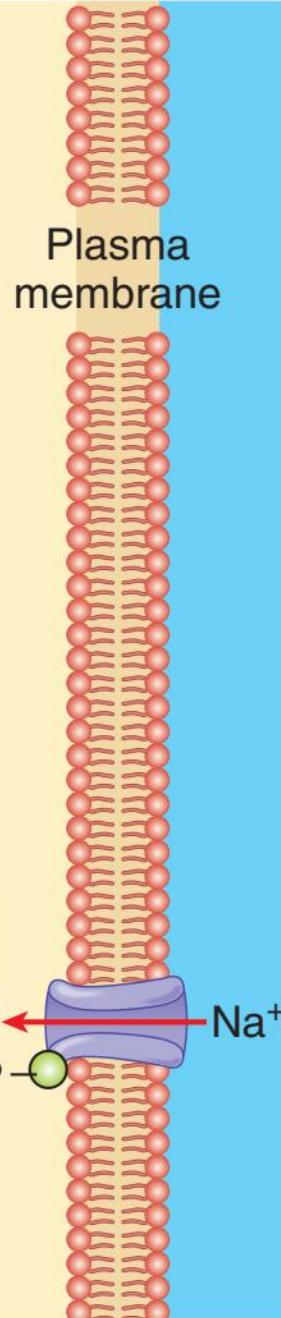
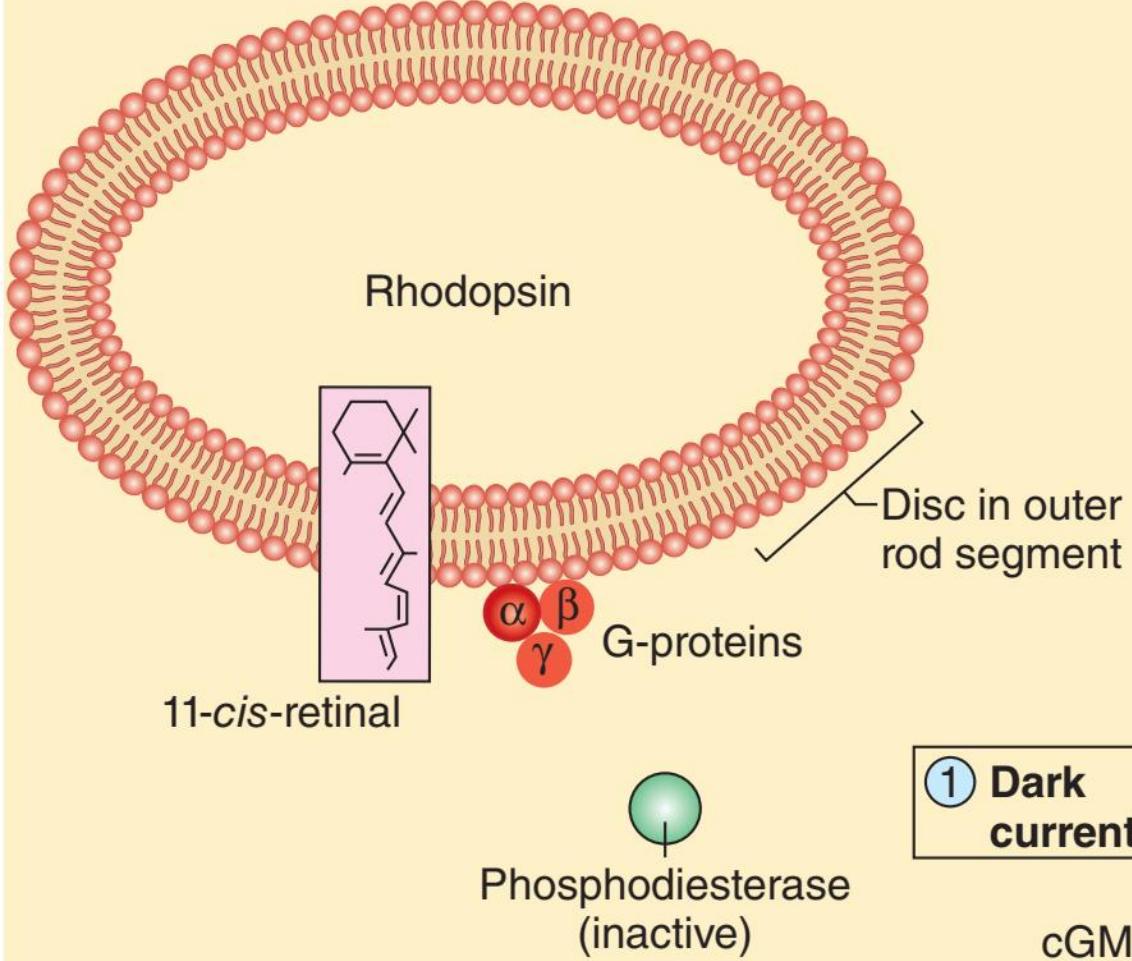
Lateralization in the Brain

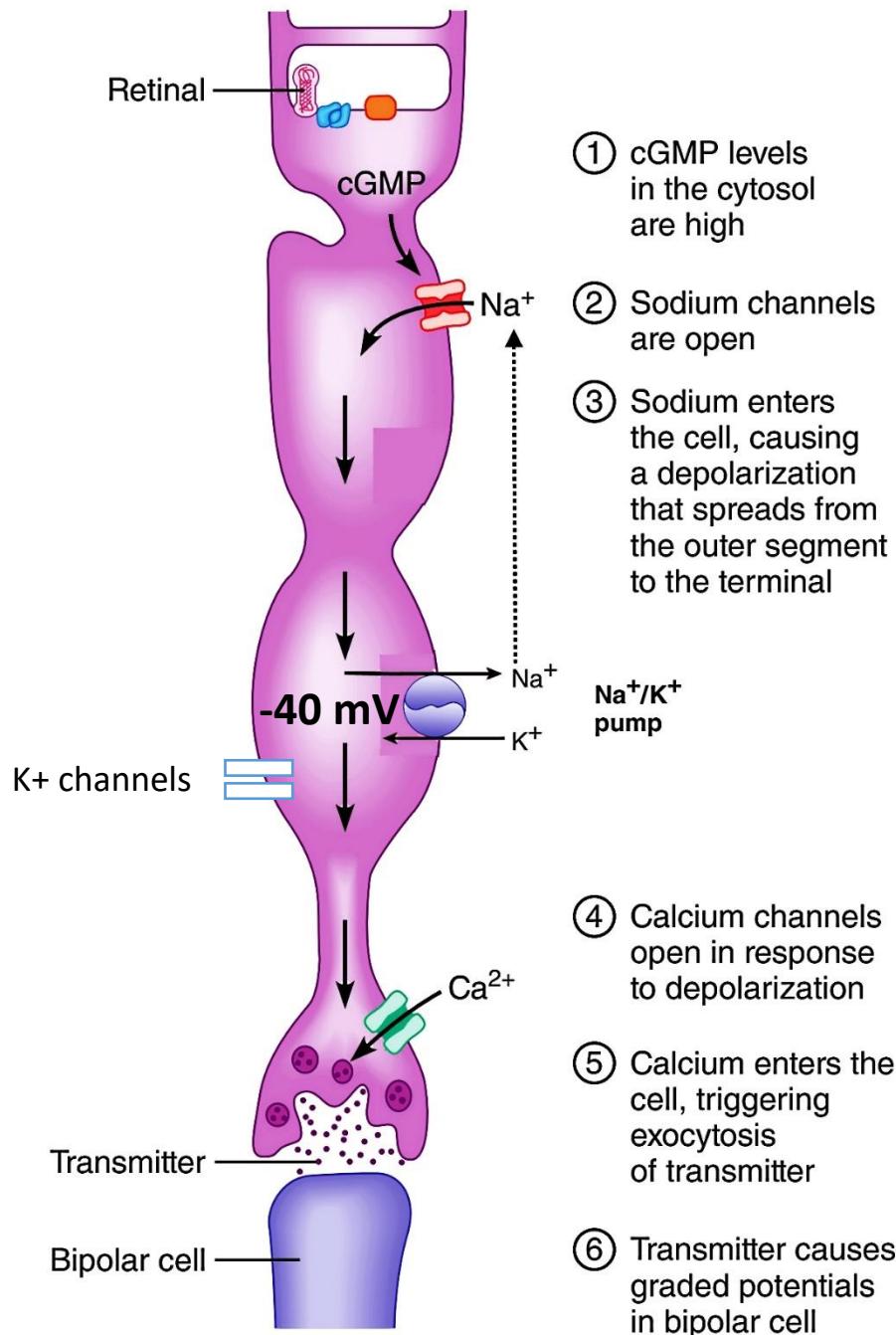
- According to current concept: hemispheres are CATEGORIAL (sequential analytic processes) and REPRESENTATIONAL (for visuospatial relations)



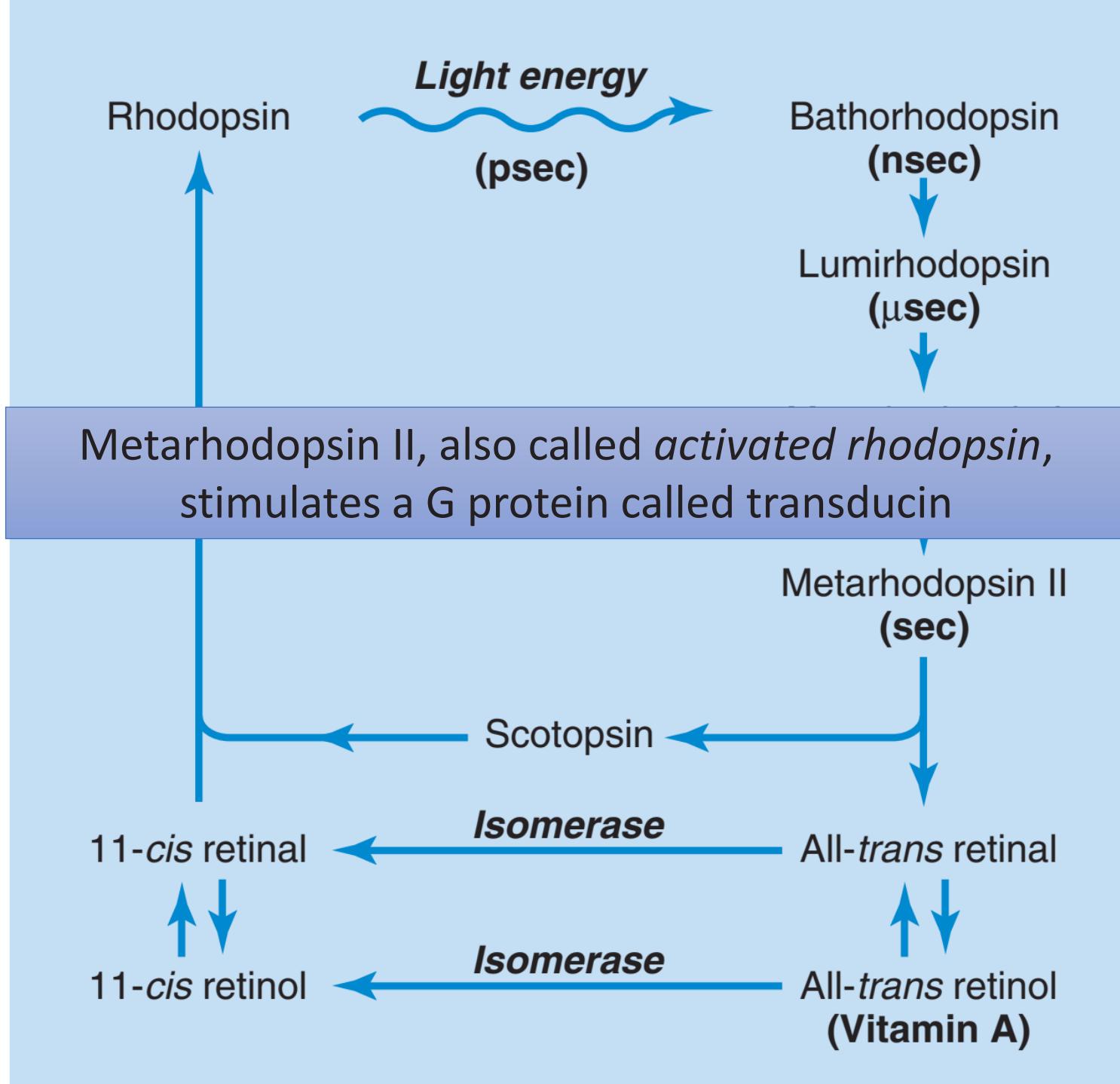
VISION

In the dark



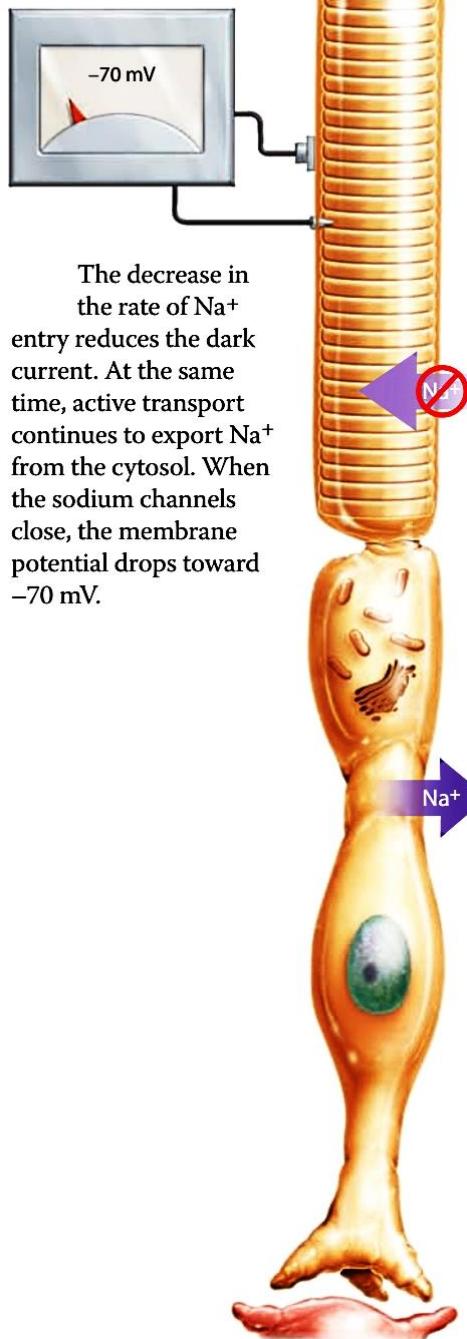
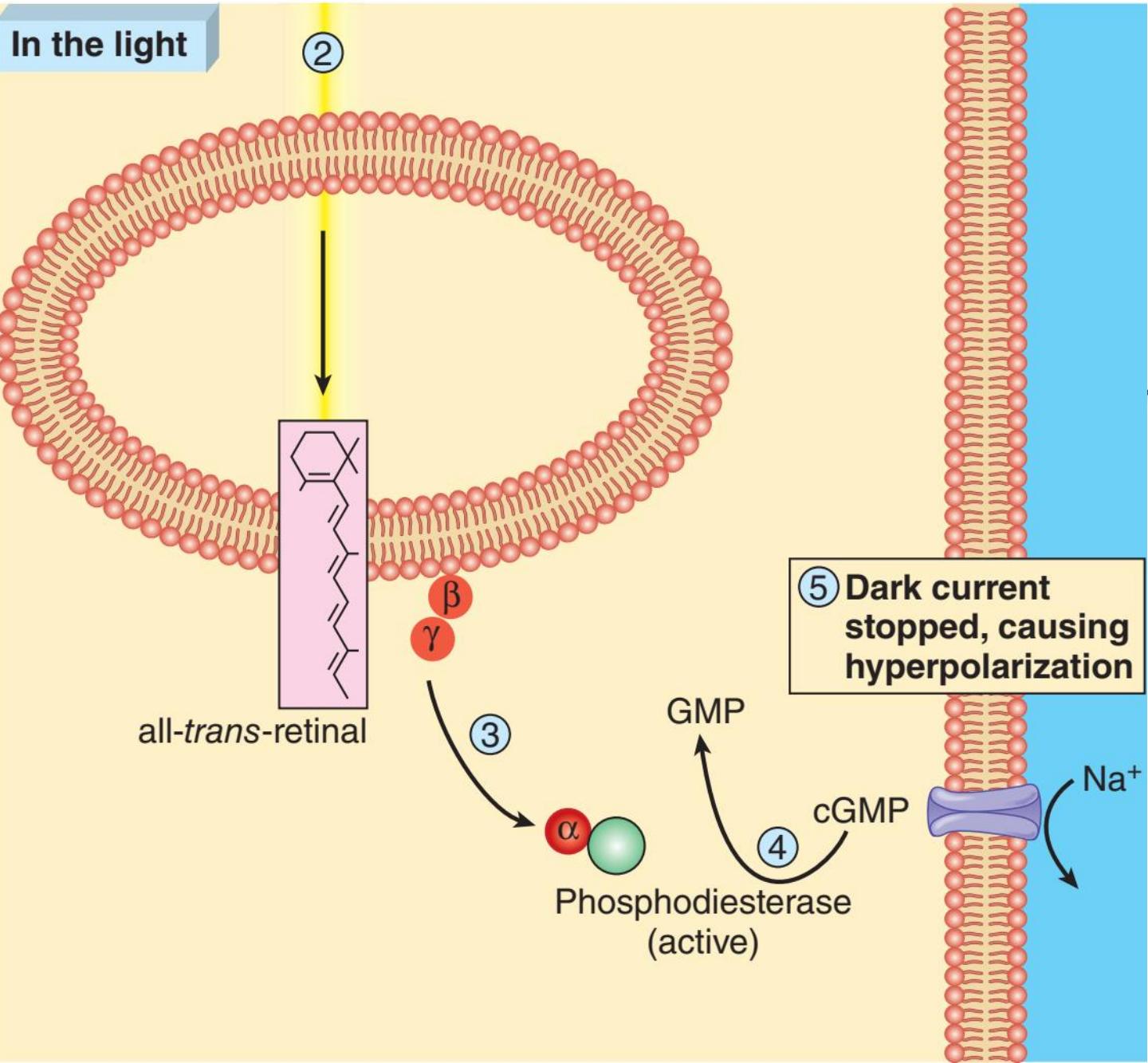


State of photoreceptor in the dark



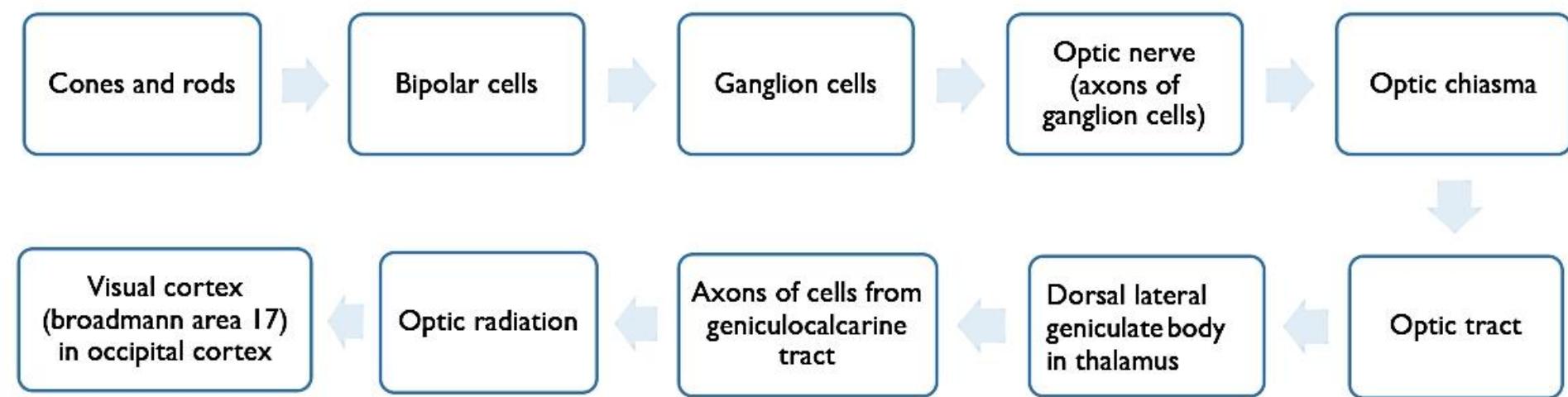
ACTIVE STATE

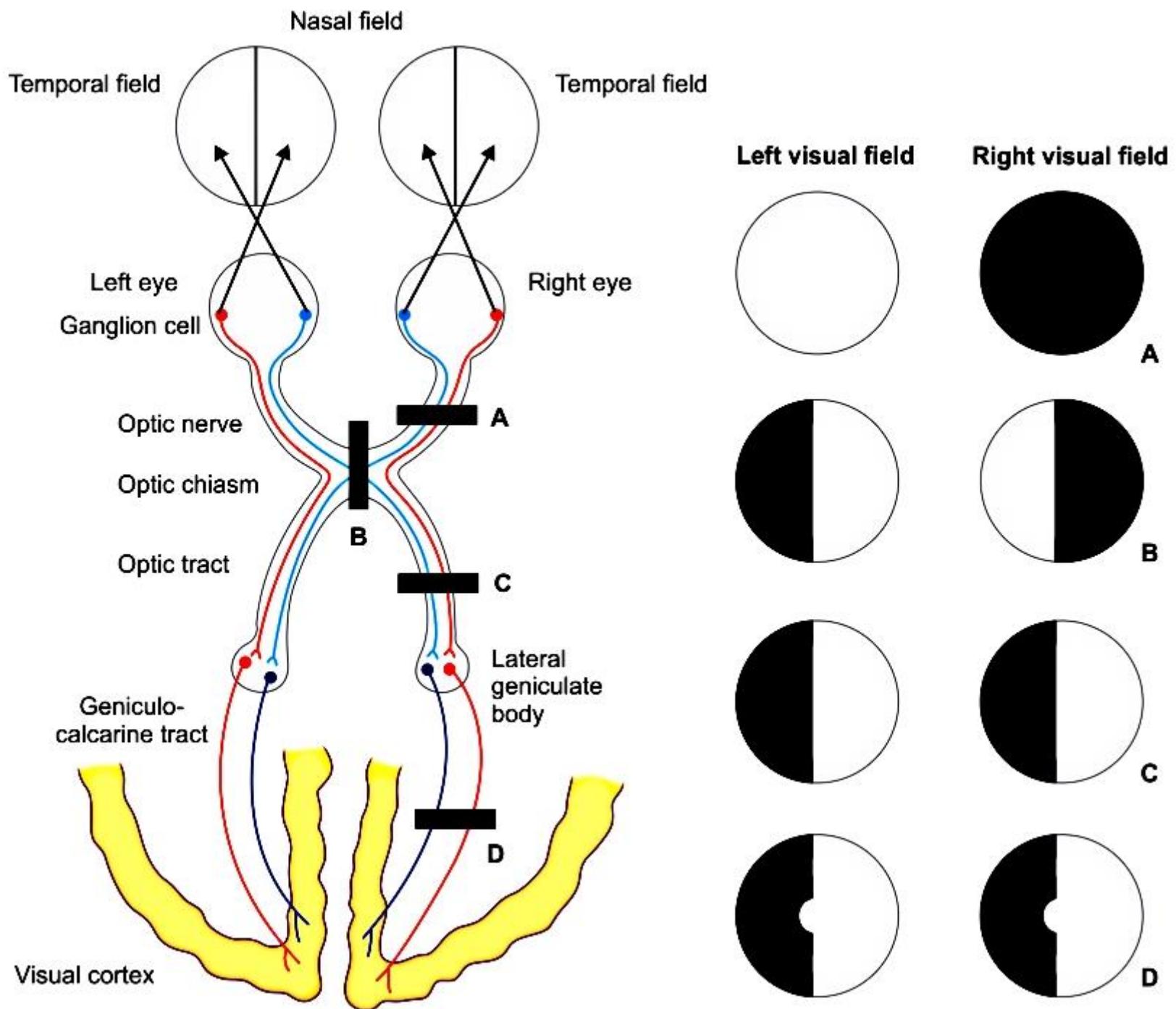
In the light

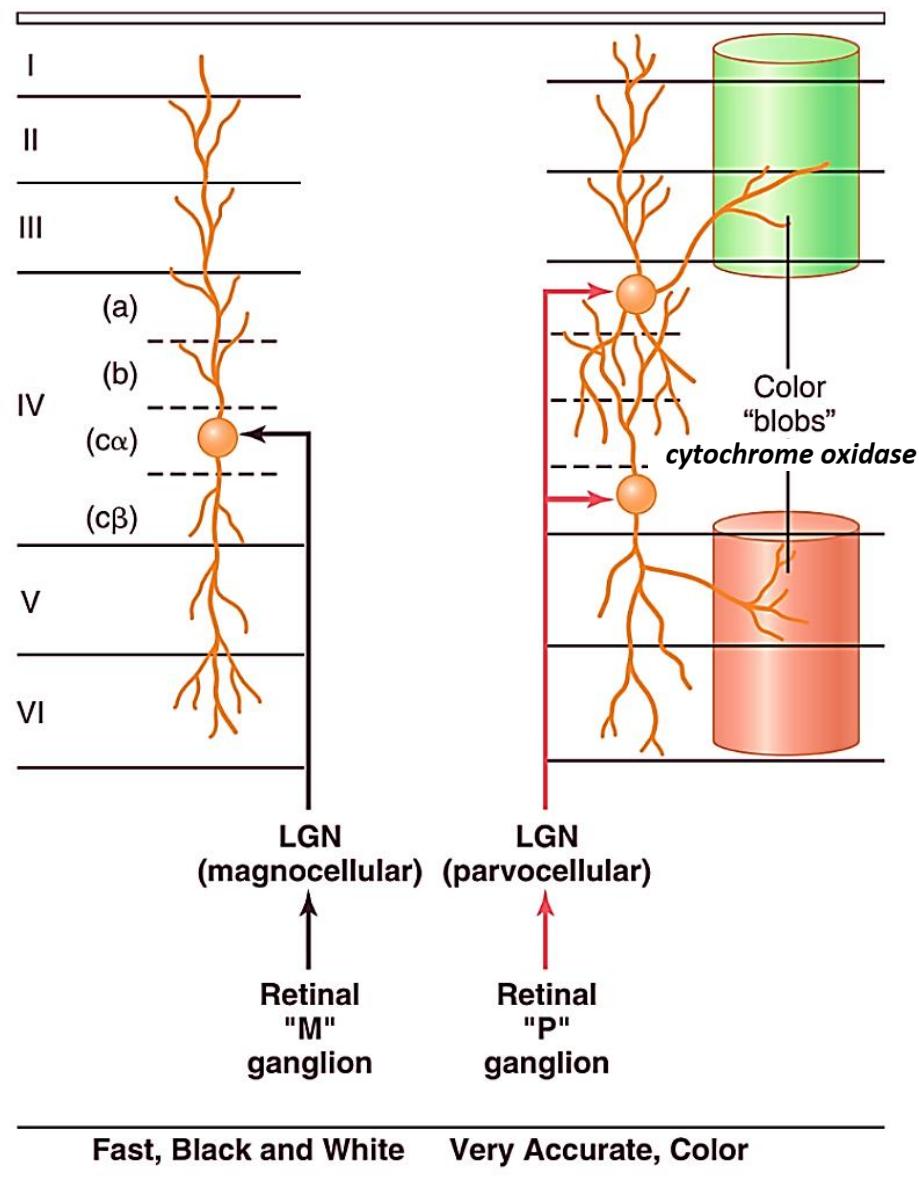
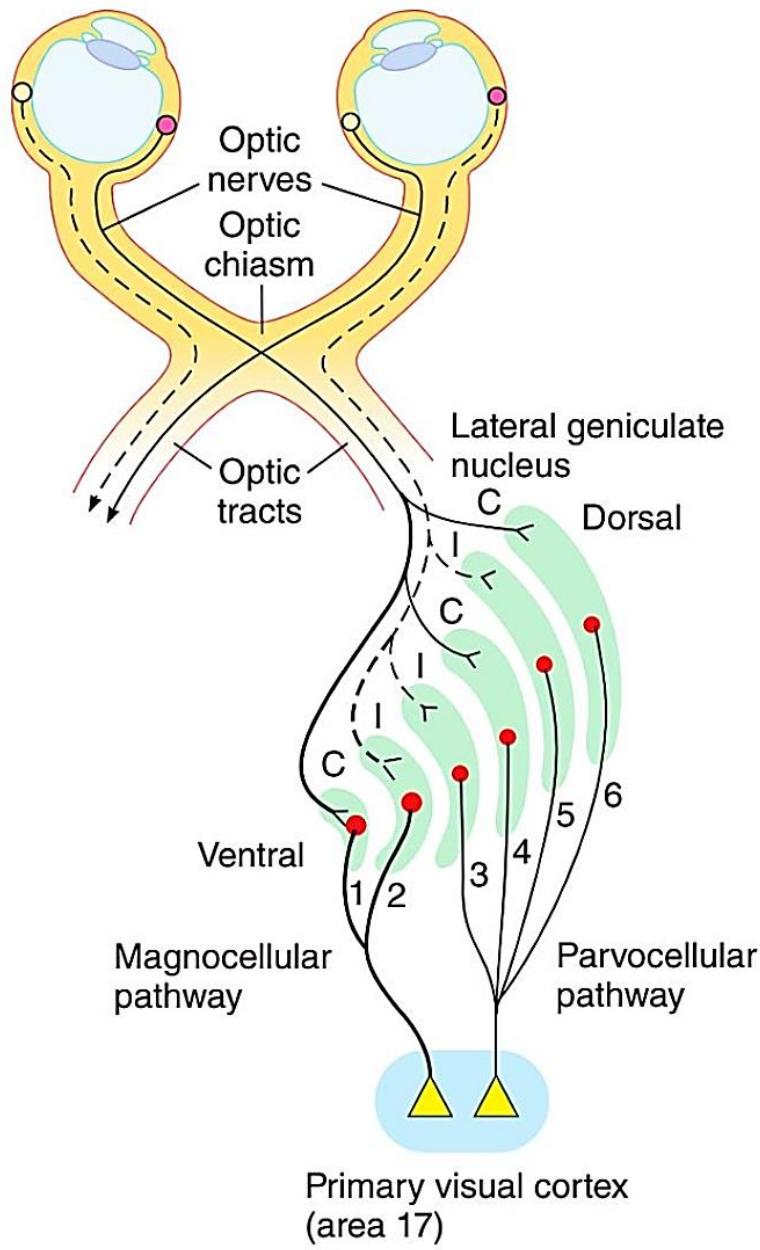


The decrease in the rate of Na^+ entry reduces the dark current. At the same time, active transport continues to export Na^+ from the cytosol. When the sodium channels close, the membrane potential drops toward -70 mV .

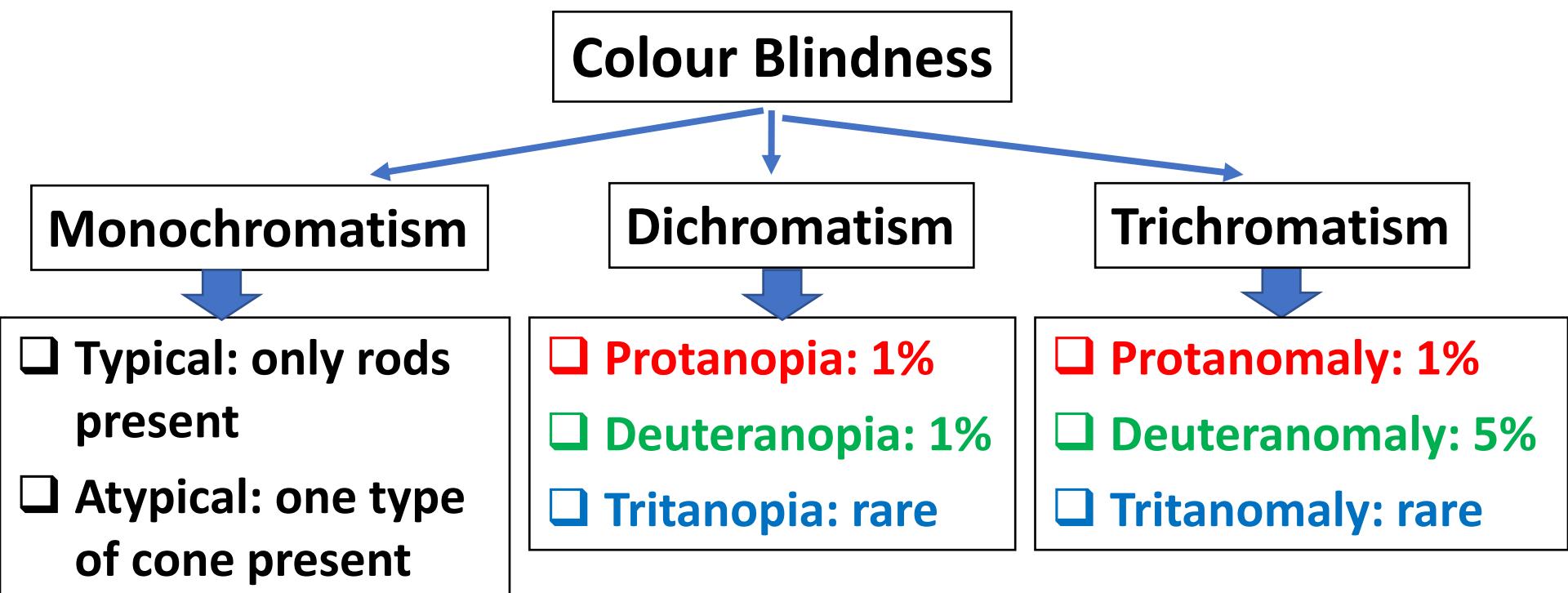
Visual Pathway



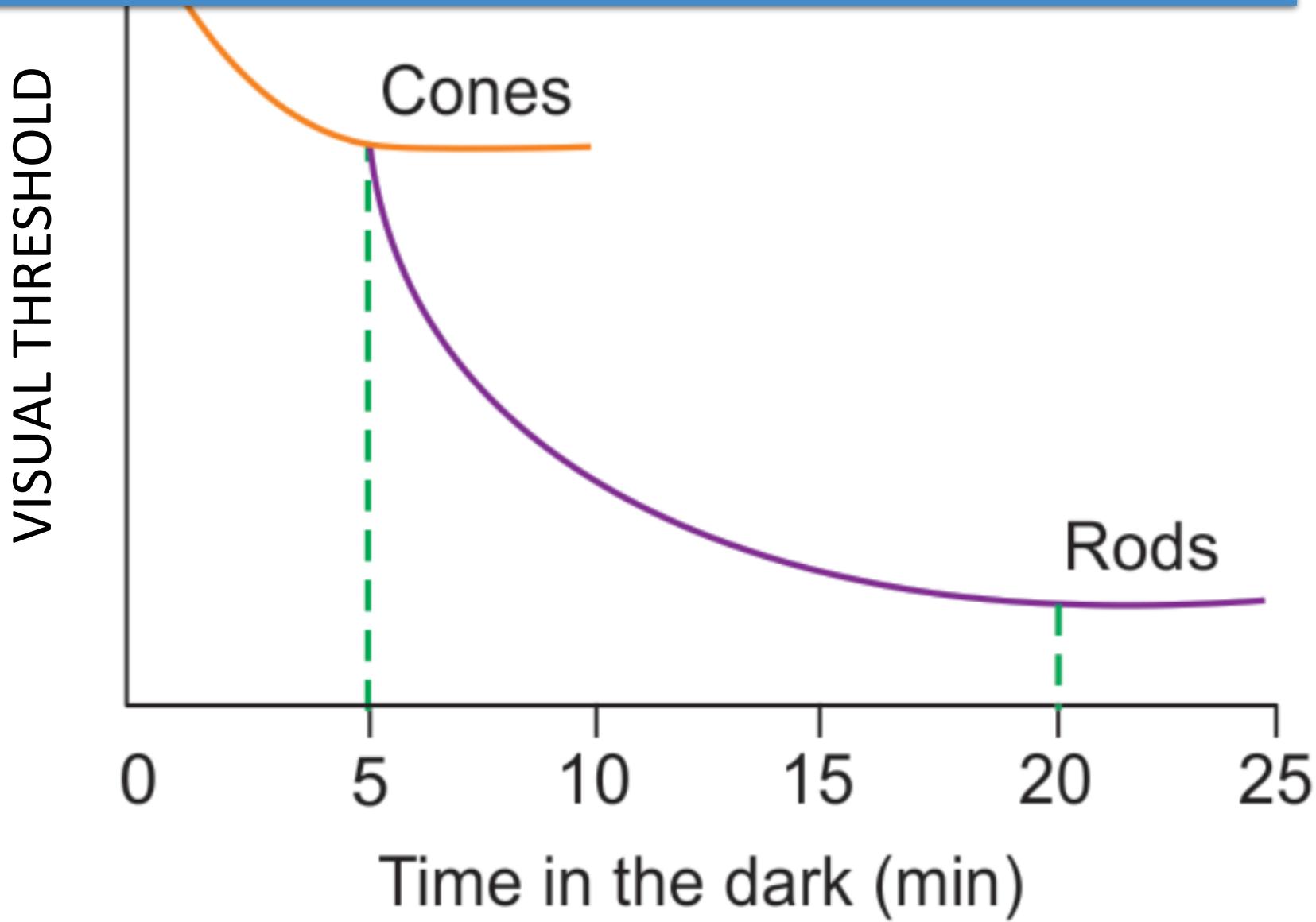




- Young–Helmholtz Trichromacy theory
- The gene for the *blue-sensitive* S cone pigment is on *chromosome 7*
- The genes for the *red-sensitive* and the *green-sensitive* cone pigments are on the *X-chromosome*

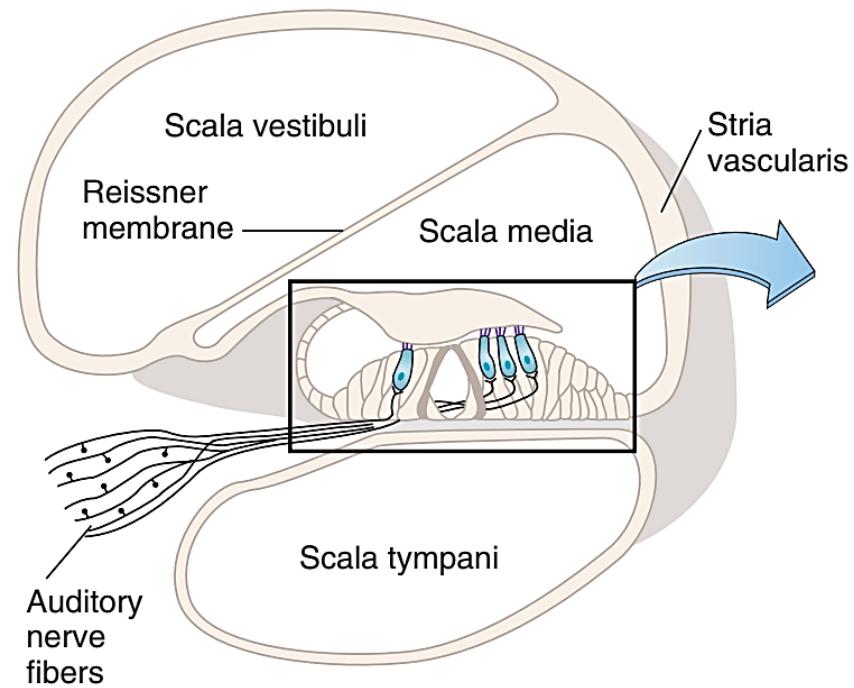


Dark adaptation: 20 min approx

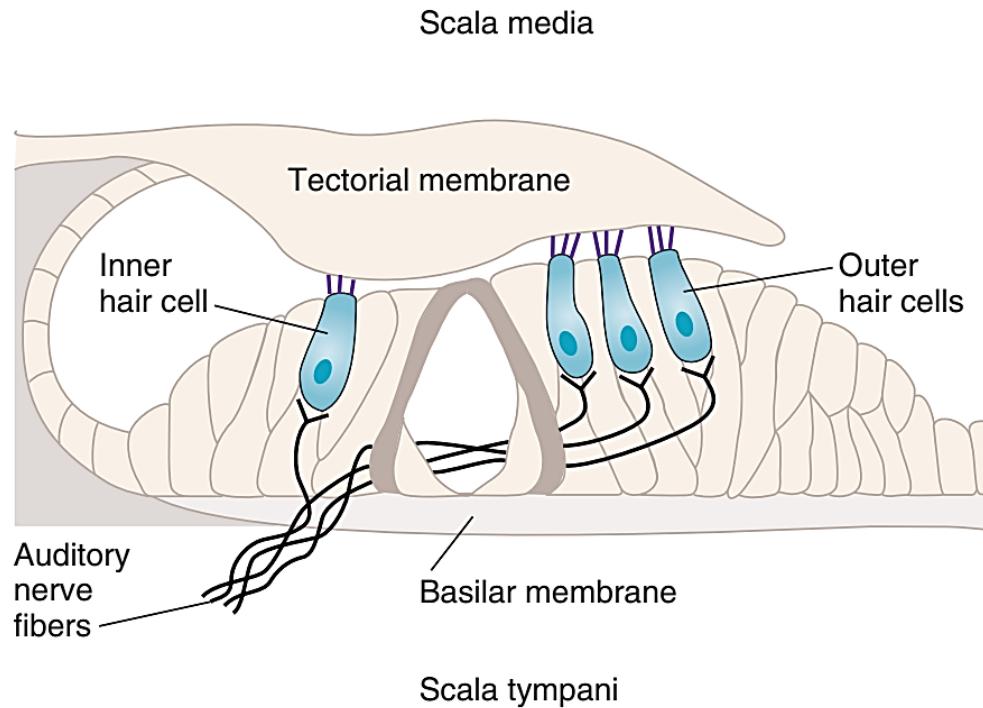


Hearing

Cross-section of cochlea

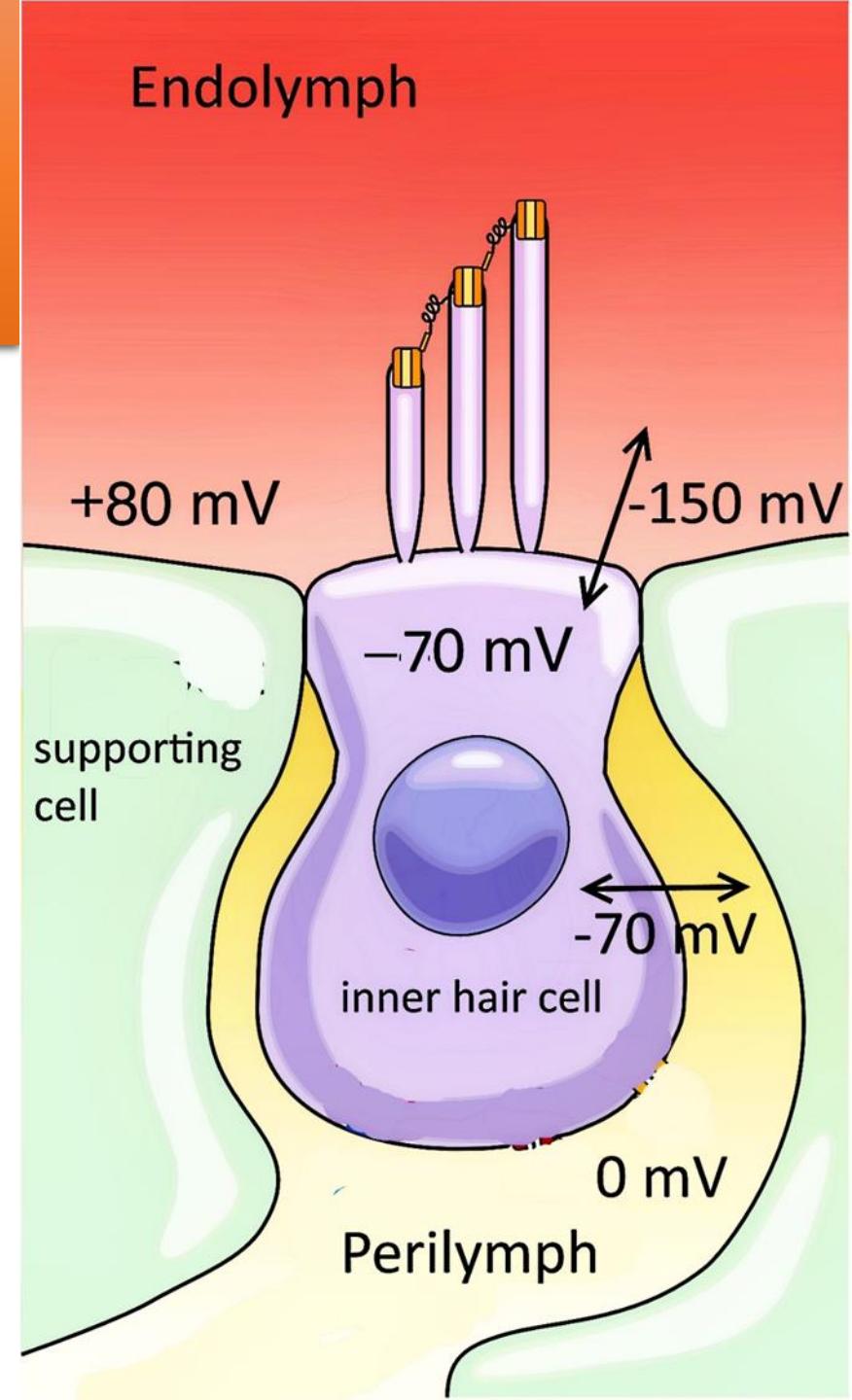


Organ of Corti

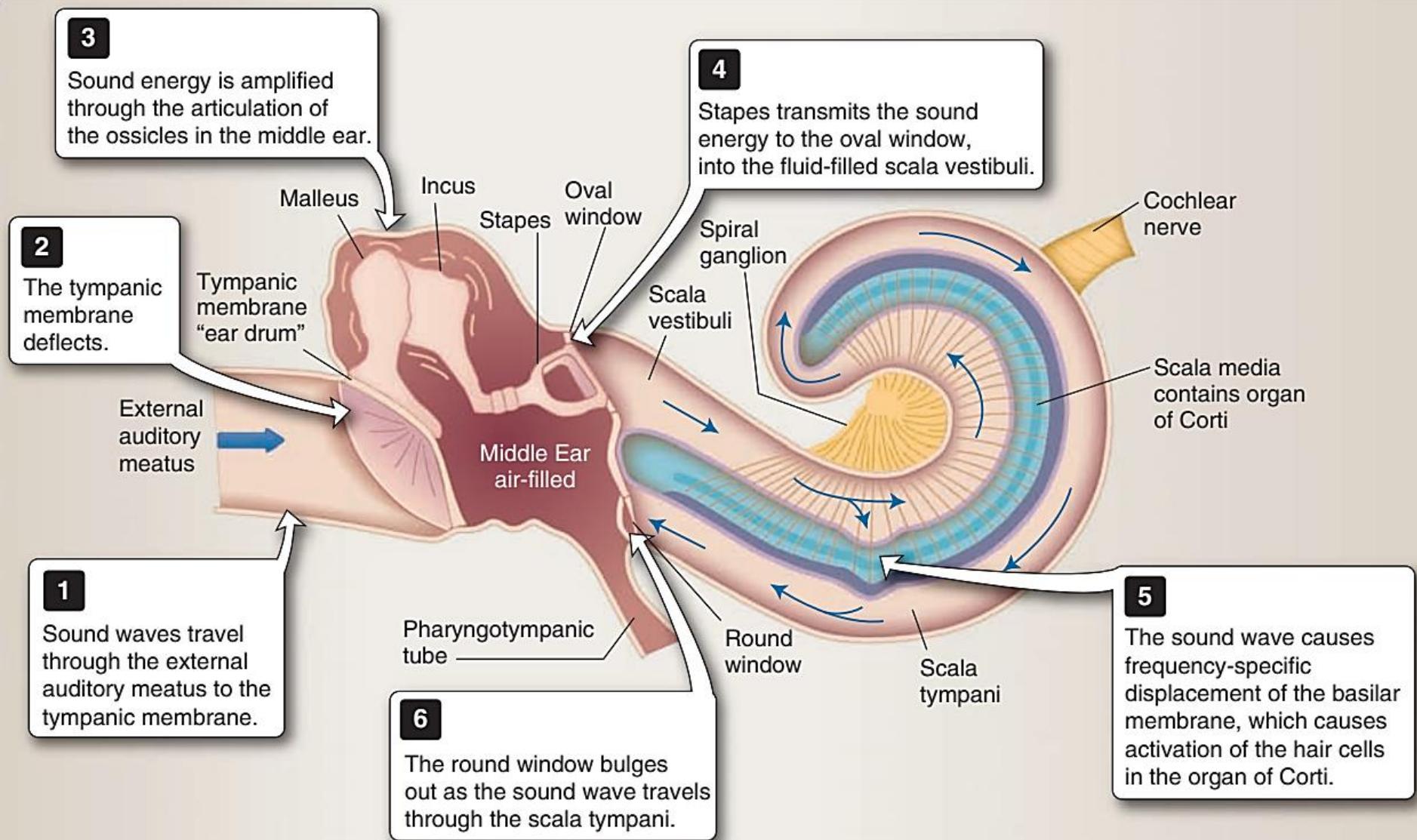


Endolymphatic or Endocochlear Potential

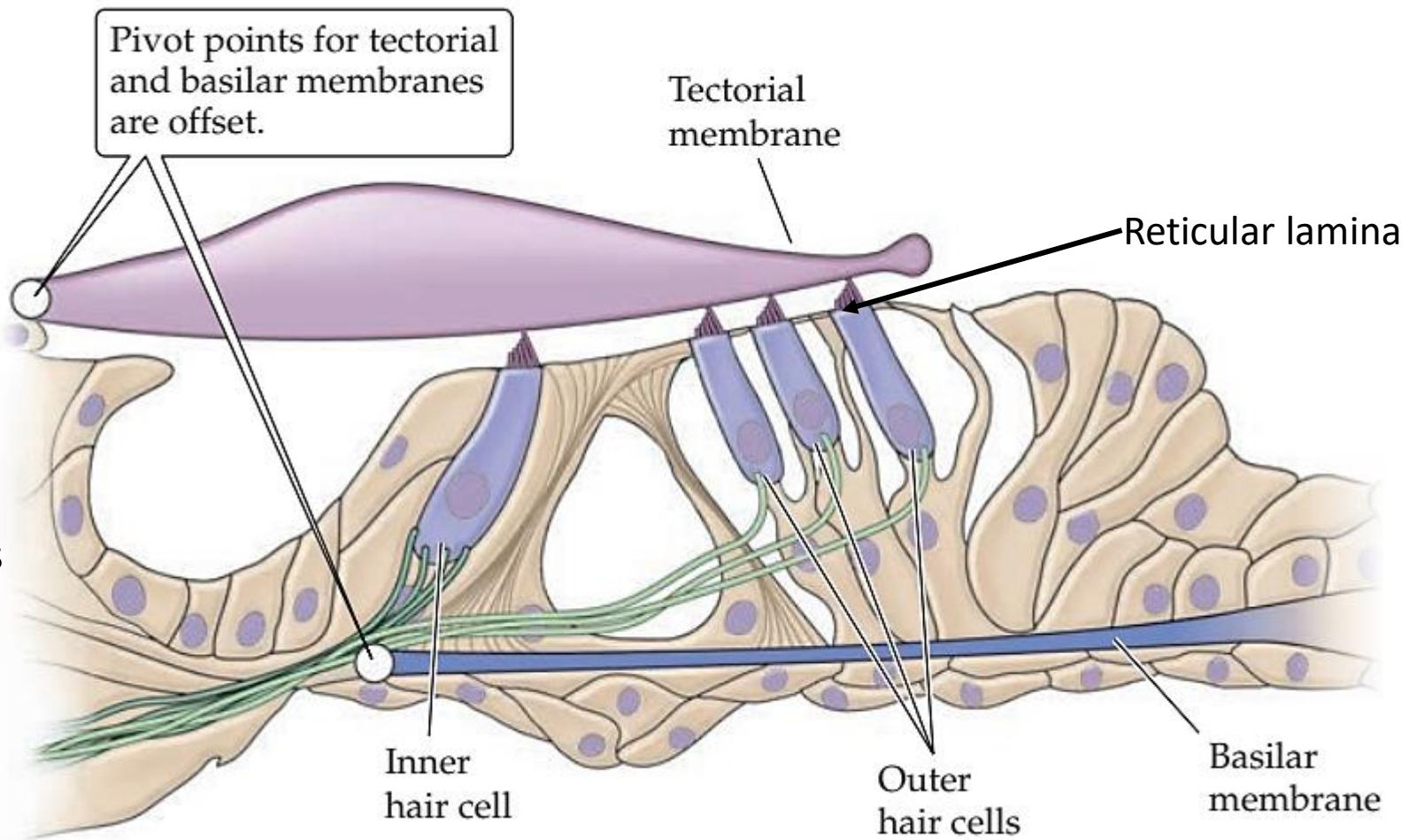
- About +80 mV of endocochlear potential exists between endolymph and perilymph, due to high K⁺ secreted by stria vascularis
- Hair cells have a RMP of -70 mV with respect to the perilymph but -150 mV [-70-(+80)] with respect to the endolymph



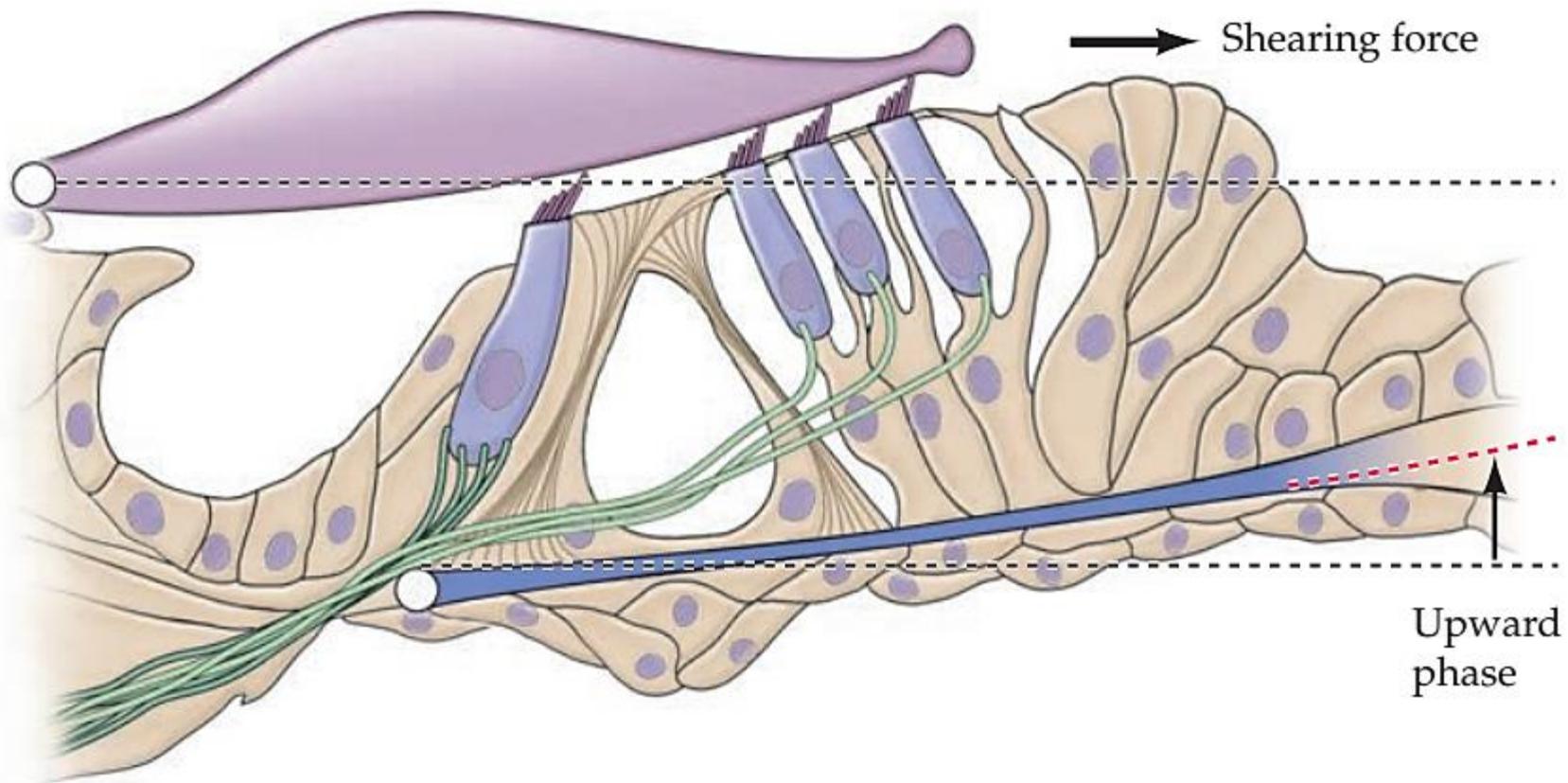
Mechanotransduction



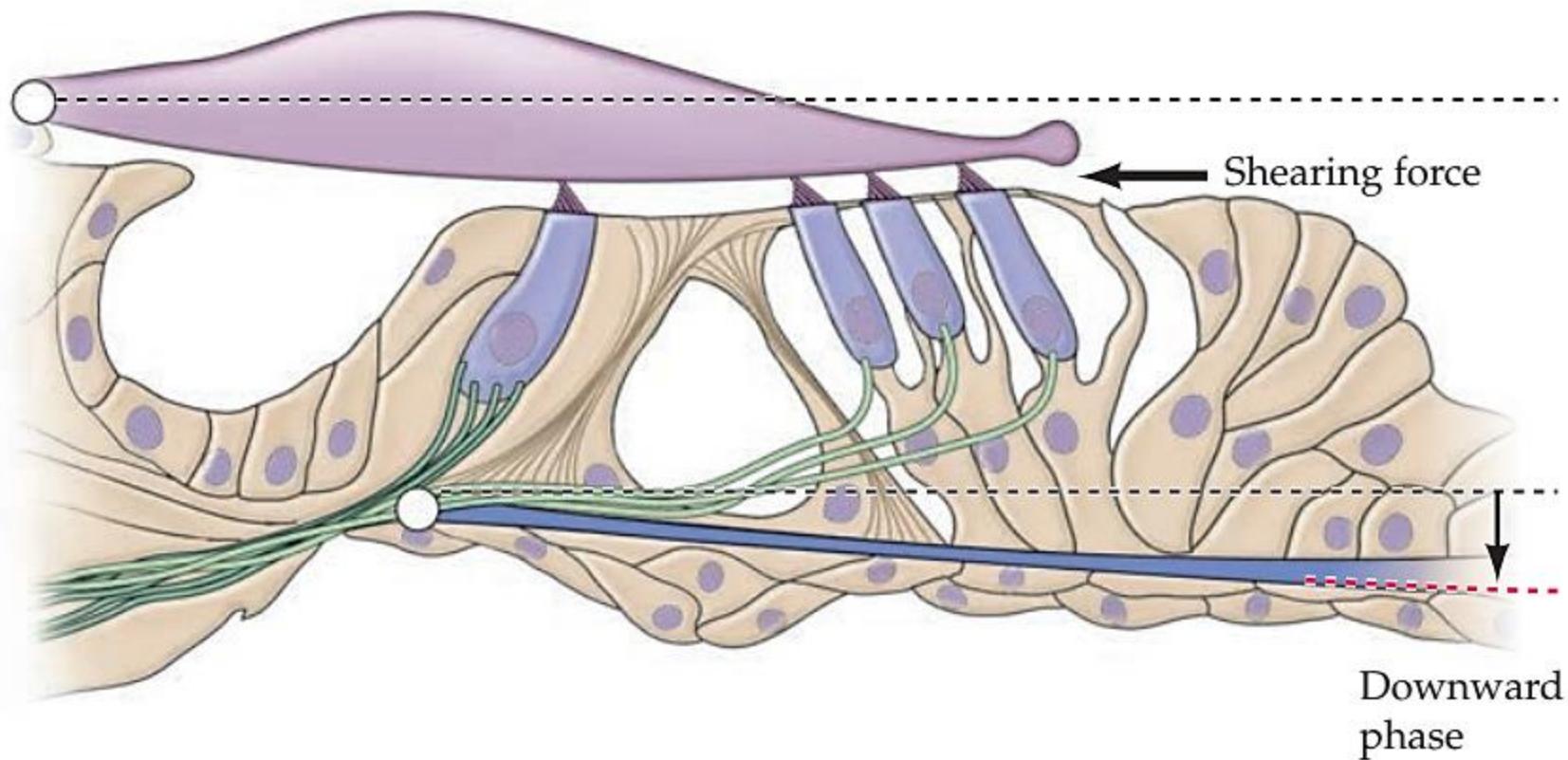
Resting position



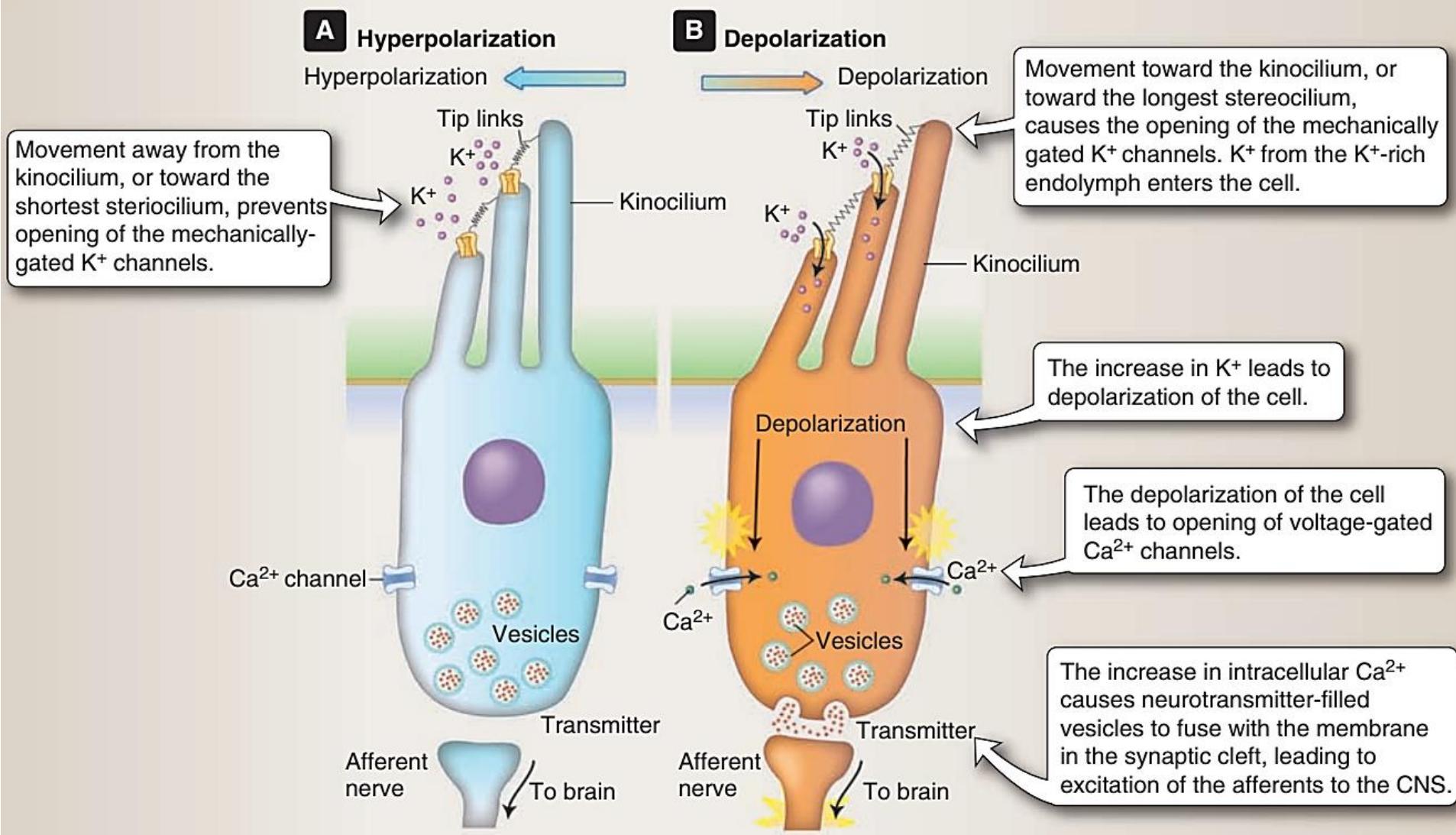
Sound-induced vibration

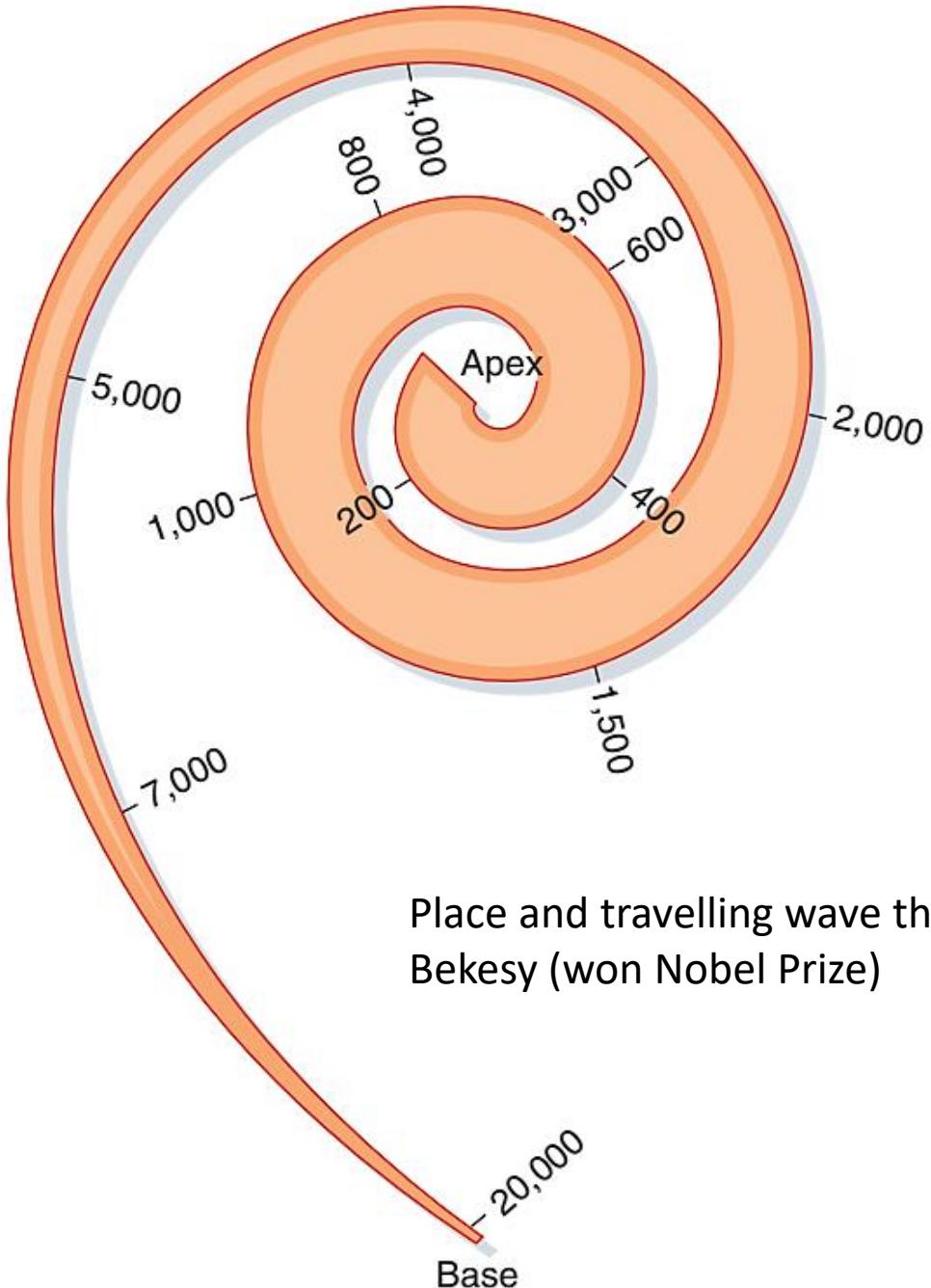


Depolarisation



Hyperpolarisation





Place and travelling wave theory by Georg von
Bekesy (won Nobel Prize)

The tonotopic map of the cochlea.

AUDITORY PATHWAY

e → eighth cranial nerve

c → Cochlear nuclei

o → Superior olivary nucleus

L → Lateral Lemniscus

i → Inferior Colliculi

m → medial geniculate body (Thalamic nucleus)

A → Auditory cortex [Brodmann Area - 41]



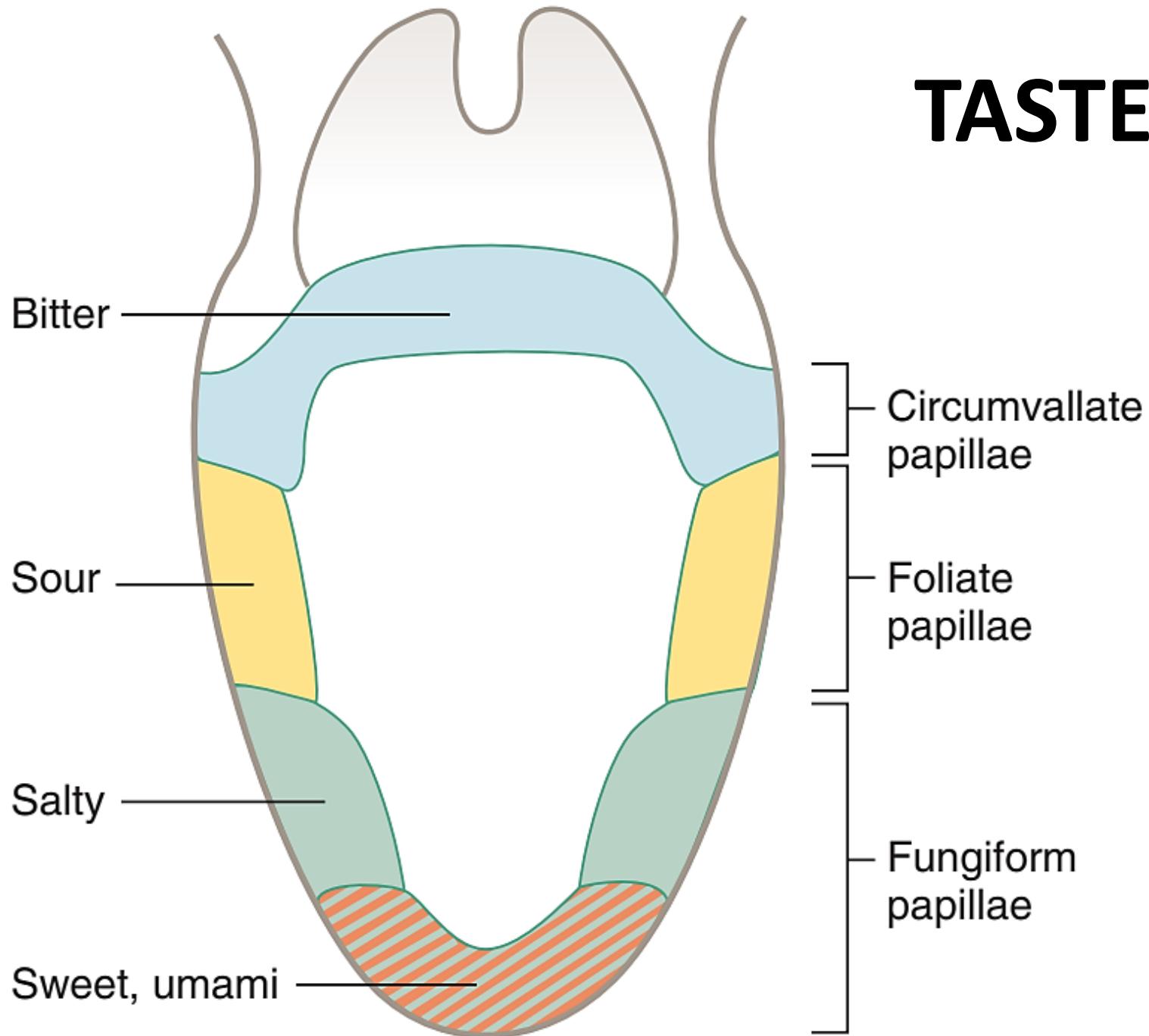
Present in the Superior Temporal Gyrus

VESTIBULAR APPARATUS (organ for Equilibrium)

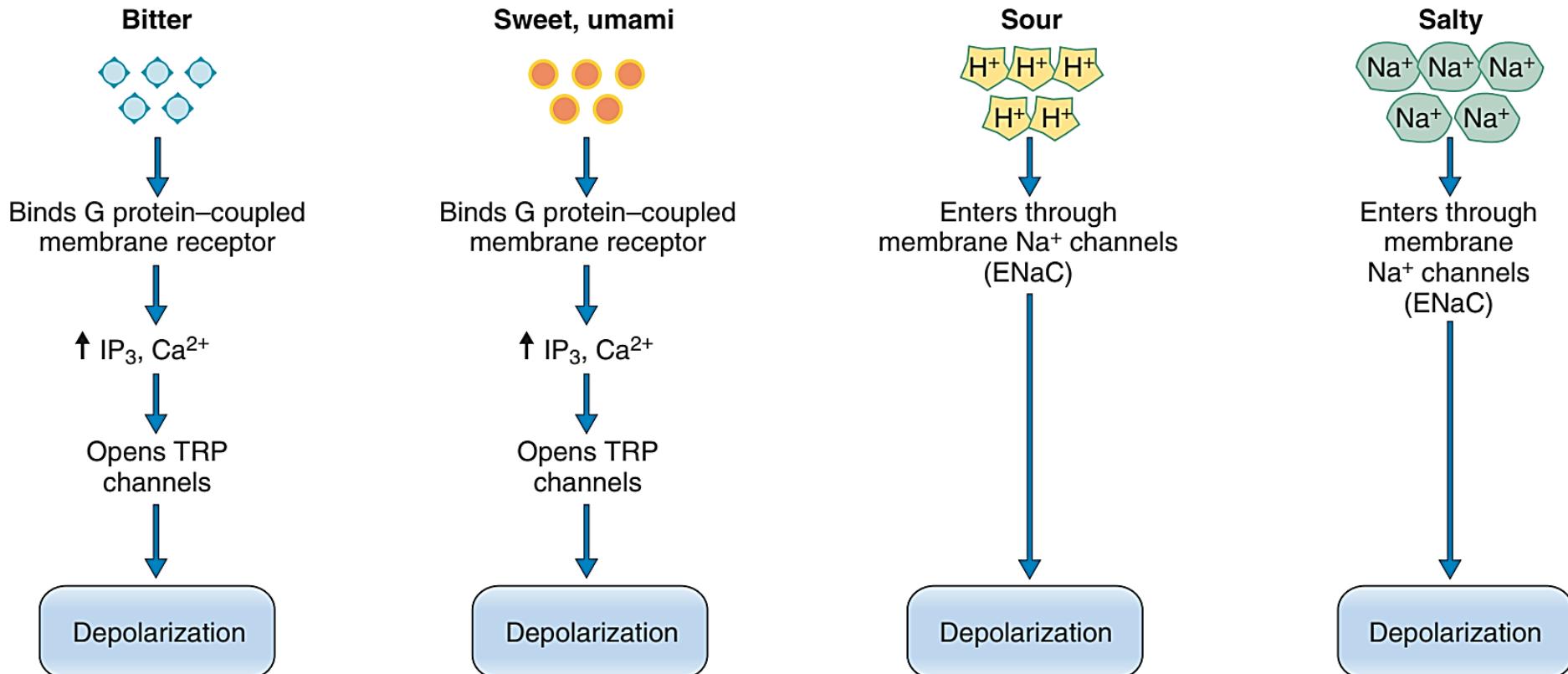
- a. UTRICLE
- b SACCULE
- c. 3 SCC

- Utricle & Saccule have MACULA
 - ↳ responsible for stationary equilibrium & linear acceleration
- Semi circular canal
 - ↳ responsible for angular acceleration
 - ↳ one end of each canal is dilated → AMPULLA
 - ↳ CRISTA AMPULLARIS → crest like elevation inside ampulla
 - contains hair cells

TASTE

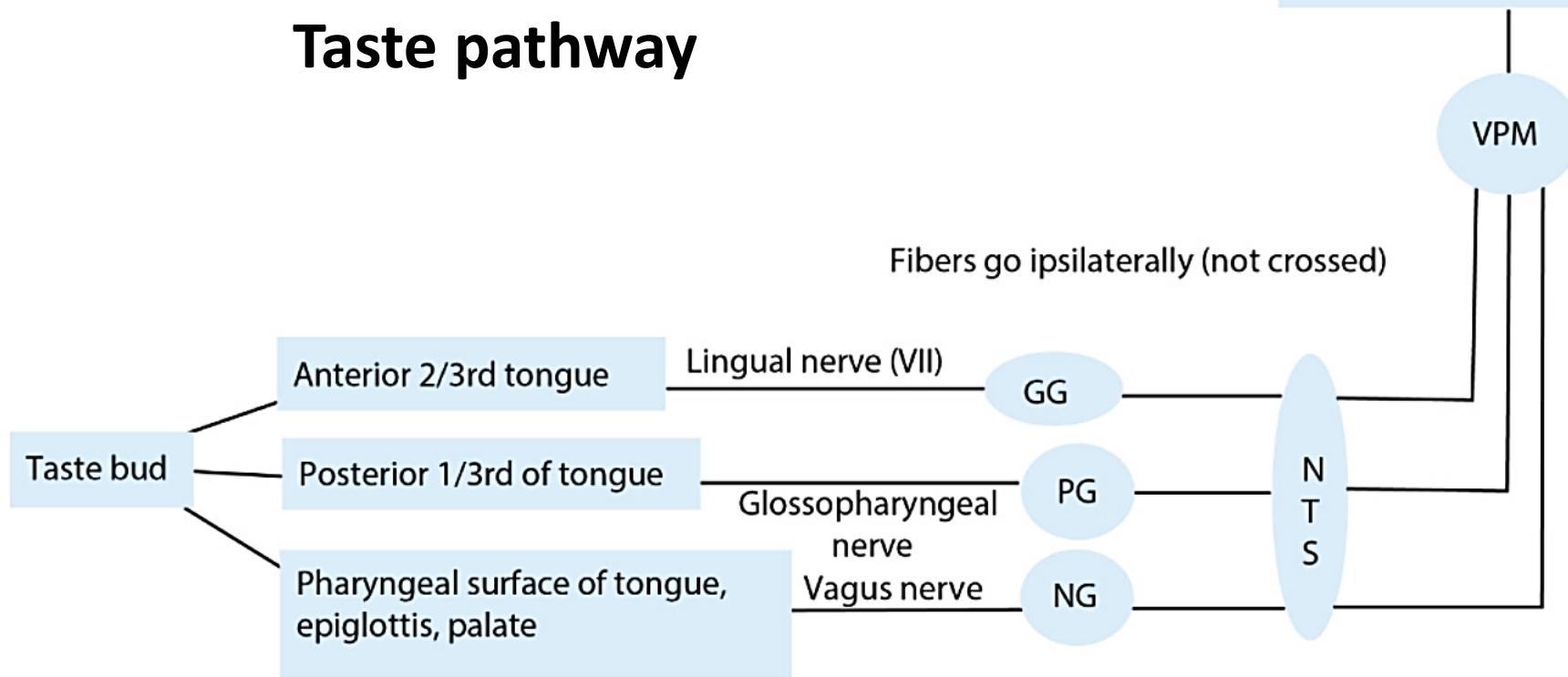


MECHANISMS OF TASTE TRANSDUCTION



Ipsilateral gustatory cortex
(insula, frontal operculum)

Taste pathway



GG, Geniculate ganglion; NG, nodose ganglia; NTS, nucleus of tractus solitarius;
PG, petrosal ganglion; VPM, ventro-medial nucleus of thalamus

