NERVOUS SYSTEM

Dr. Kiran Kumar

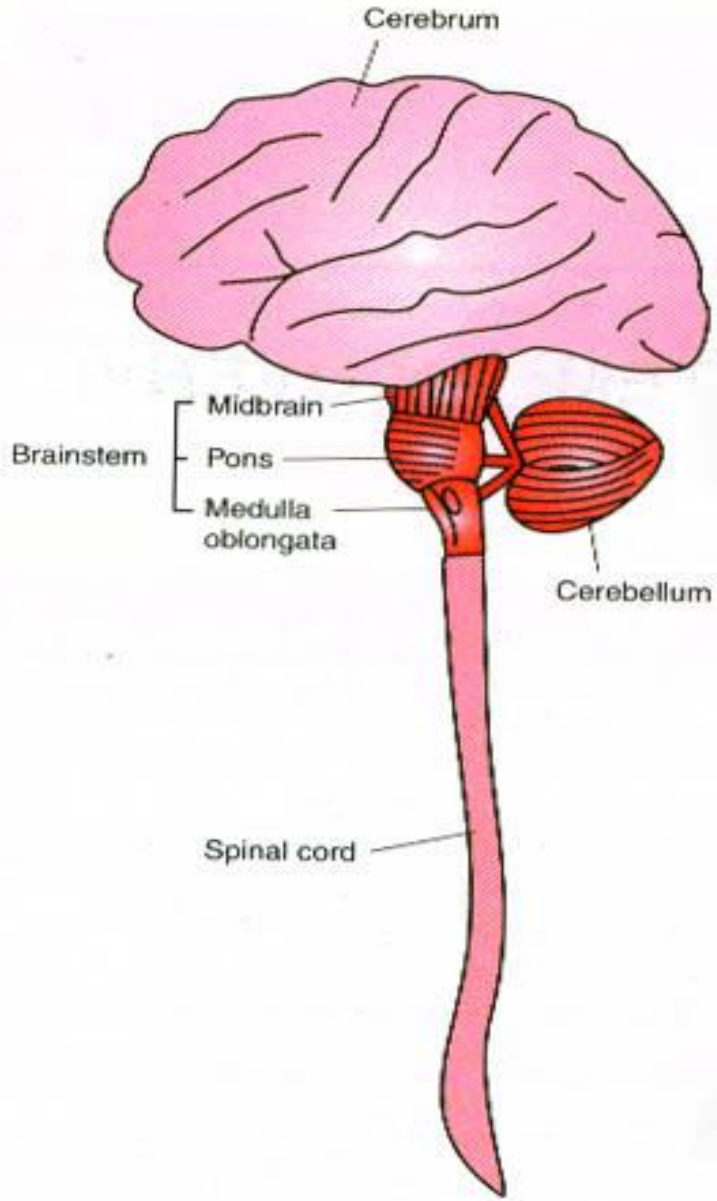
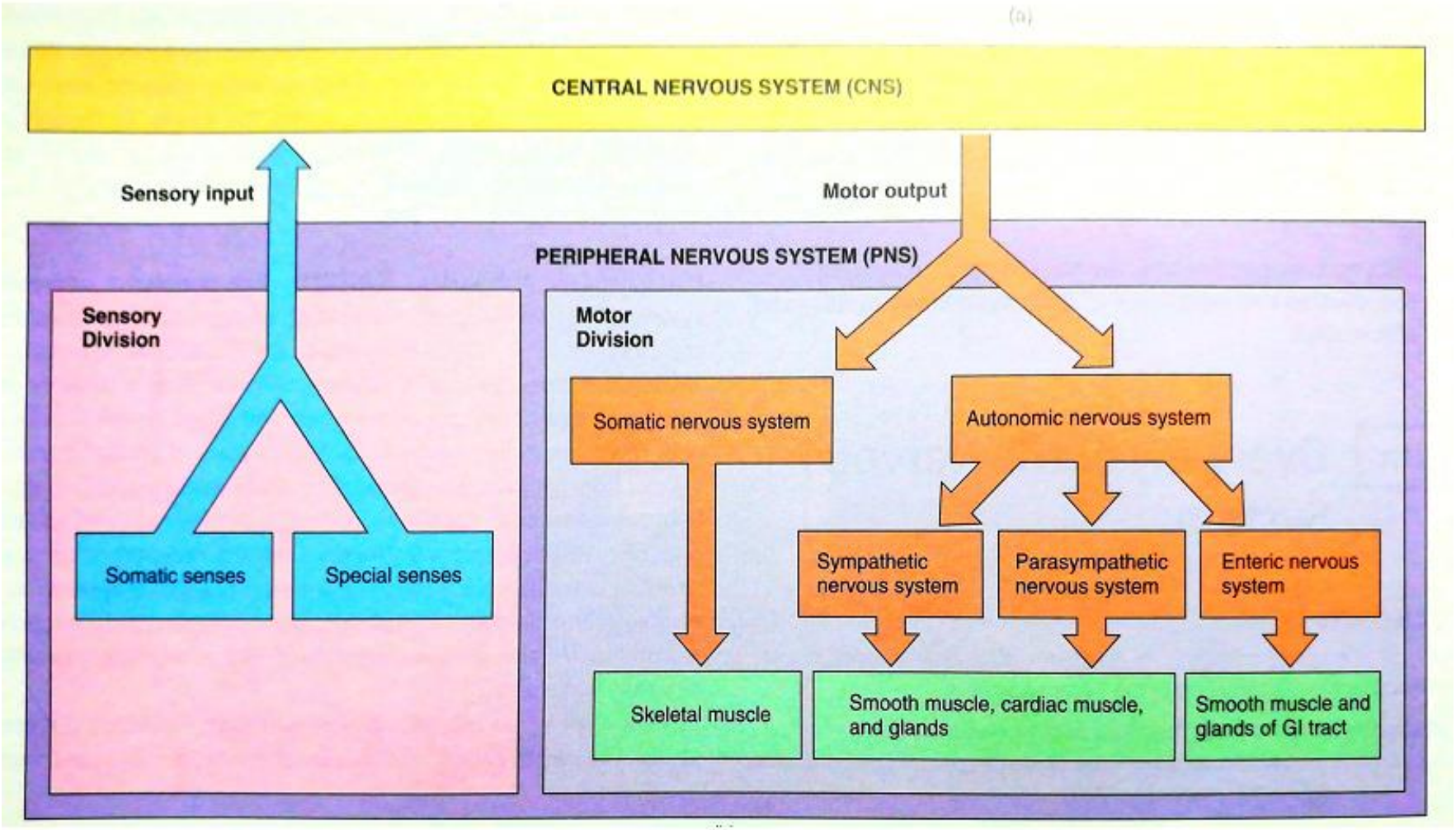


Fig. 6.1. The diagram showing parts of central nervous system. The diencephalon is not seen. The superior, middle and inferior cerebellar peduncles which connect the cerebellum with the midbrain, pons and medulla oblongata are shown schematically.


CENTRAL NERVOUS SYSTEM (CNS)
Sensory input
Motor output
PERIPHERAL NERVOUS SYSTEM (PNS)
Sensory Division
Somatic senses
Special senses
Motor Division
Somatic nervous system
Autonomic nervous system
Sympathetic nervous system
Parasympathetic nervous system
Enteric nervous system
Skeletal muscle
Smooth muscle, cardiac muscle, and glands
Smooth muscle and glands of GI tract

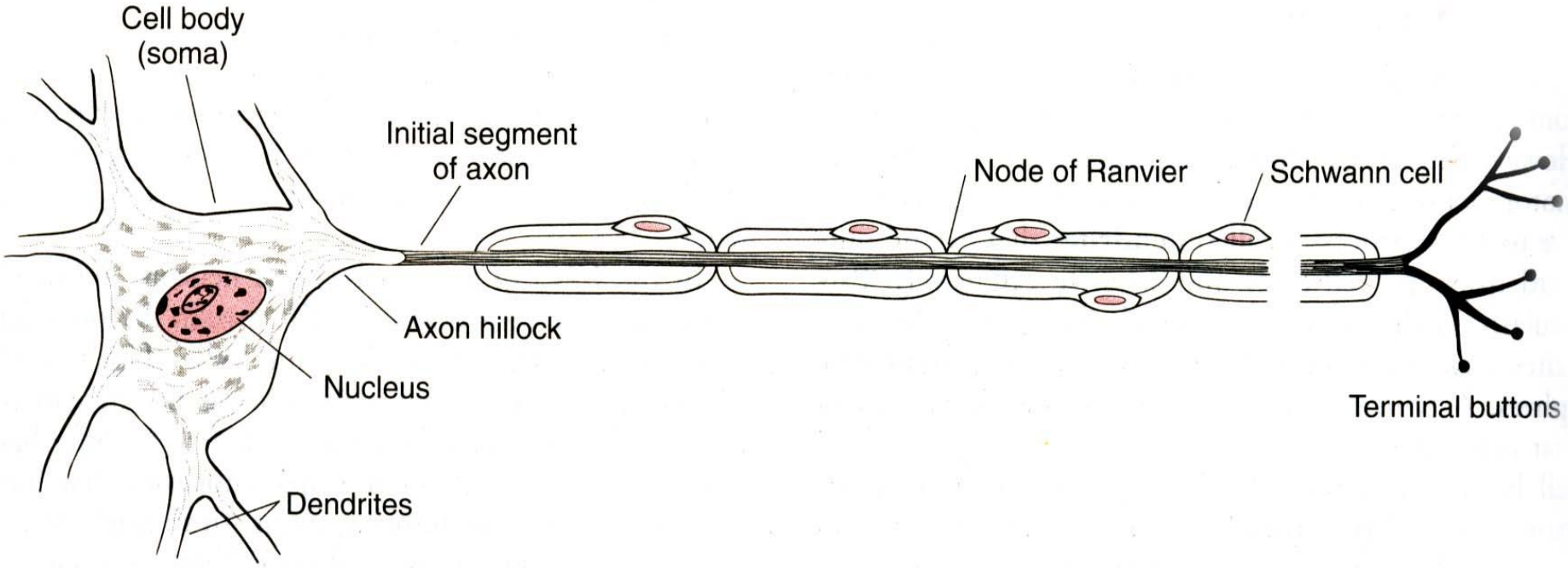


Figure 2–2. Motor neuron with myelinated axon.

SYNAPSE

- Definition
- Classification of synapses
- Structure of synapse
- Steps of synaptic transmission
- Properties of synapse

Definition;

It is a specialized junction between two neurons where there is no anatomical continuity but physiological contiguity.

Definition –

It is a junction between the **axon** or some other part of one neuron and the **dendrite, soma**(cell body), **axon** of another neuron or a **muscle** or a **gland** where there is physiological continuity but no anatomical continuity.

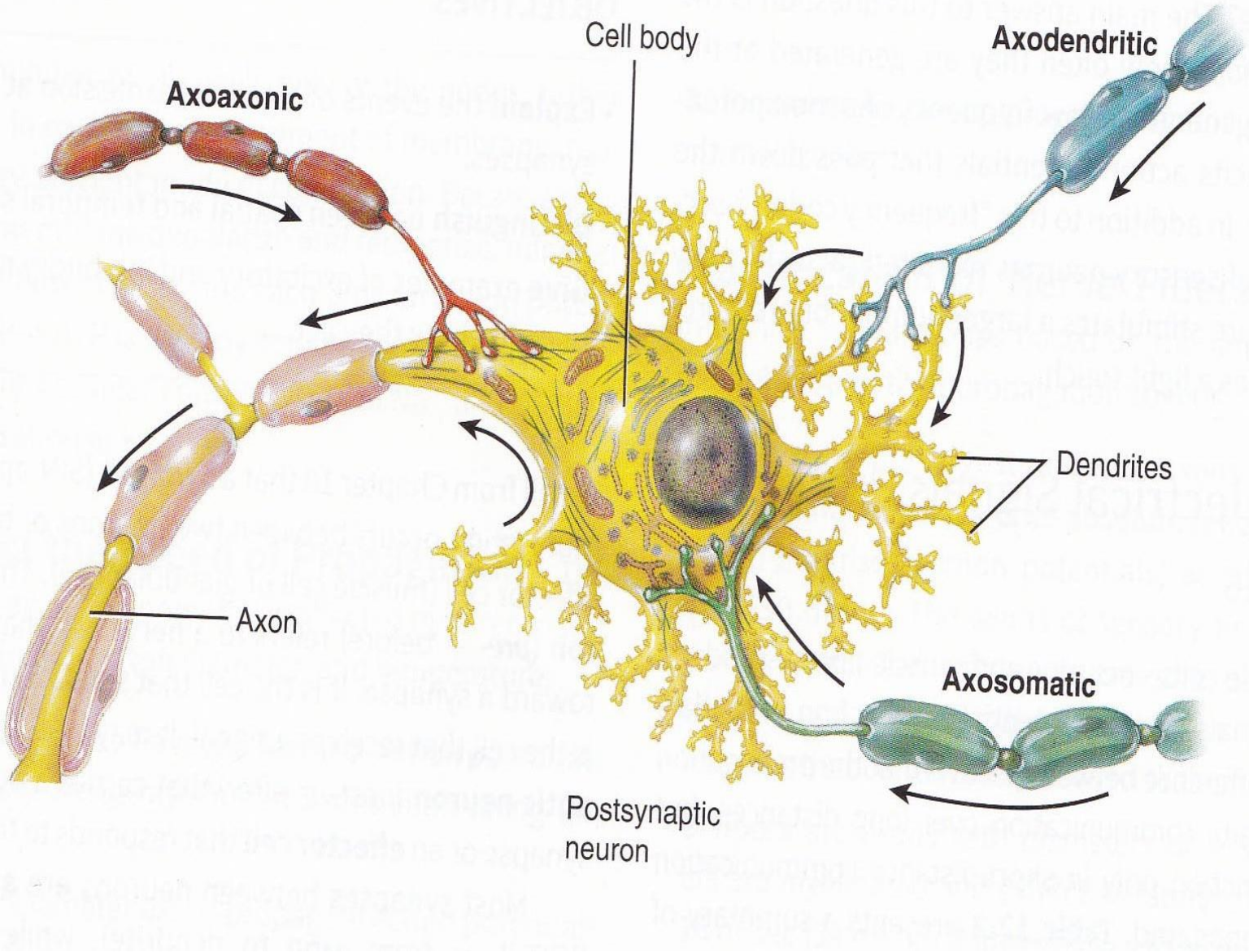
Classification of synapses

I. Anatomical classification

1. Axo-dendritic
2. Axo-somatic
3. Axo-axonal

II. Physiological classification

1. Chemical(99%)
2. Electrical(<1%)


Cell body
Axodendritic
Axoaxonic
Dendrites
Axon
Axosomatic
Postsynaptic neuron

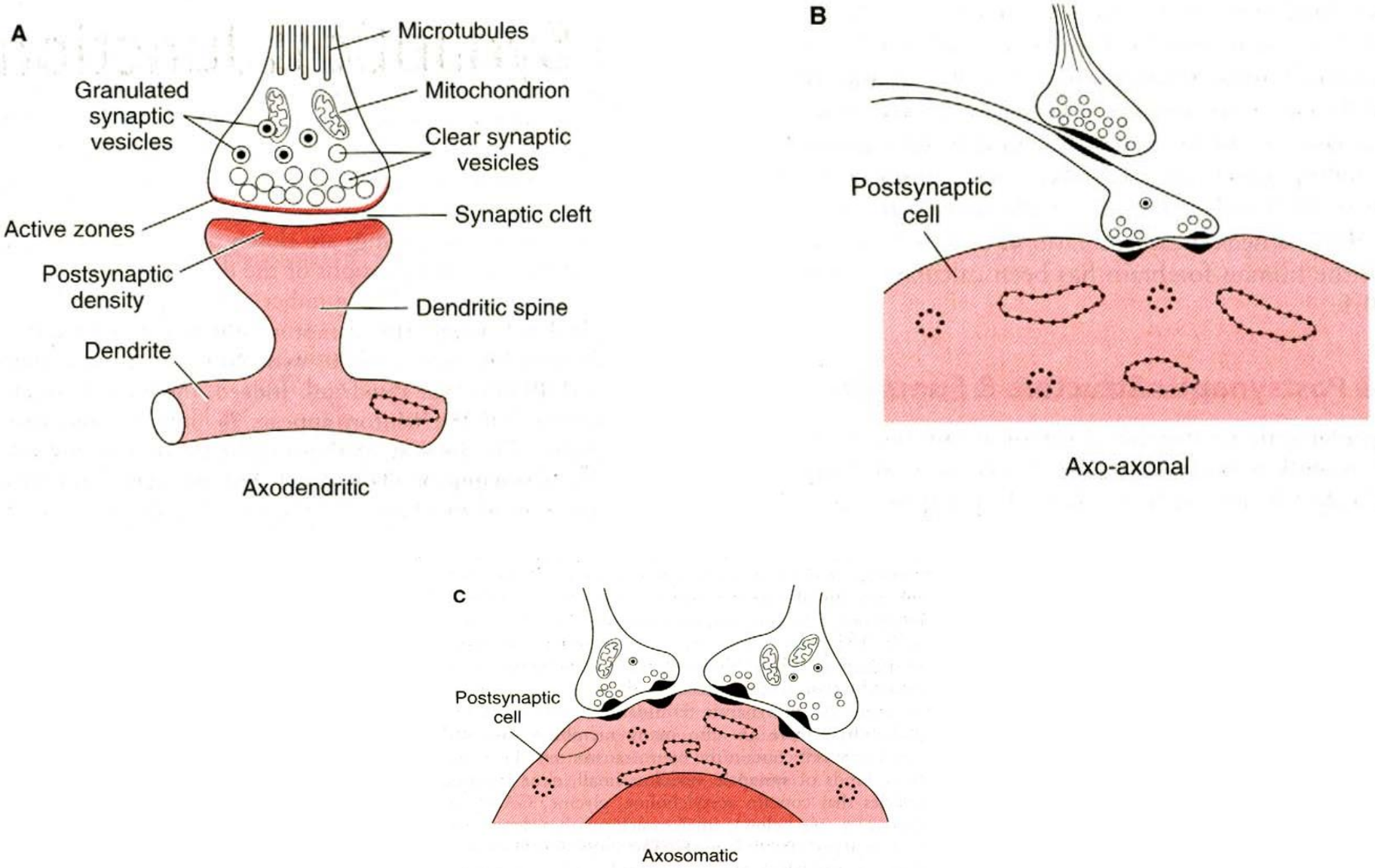
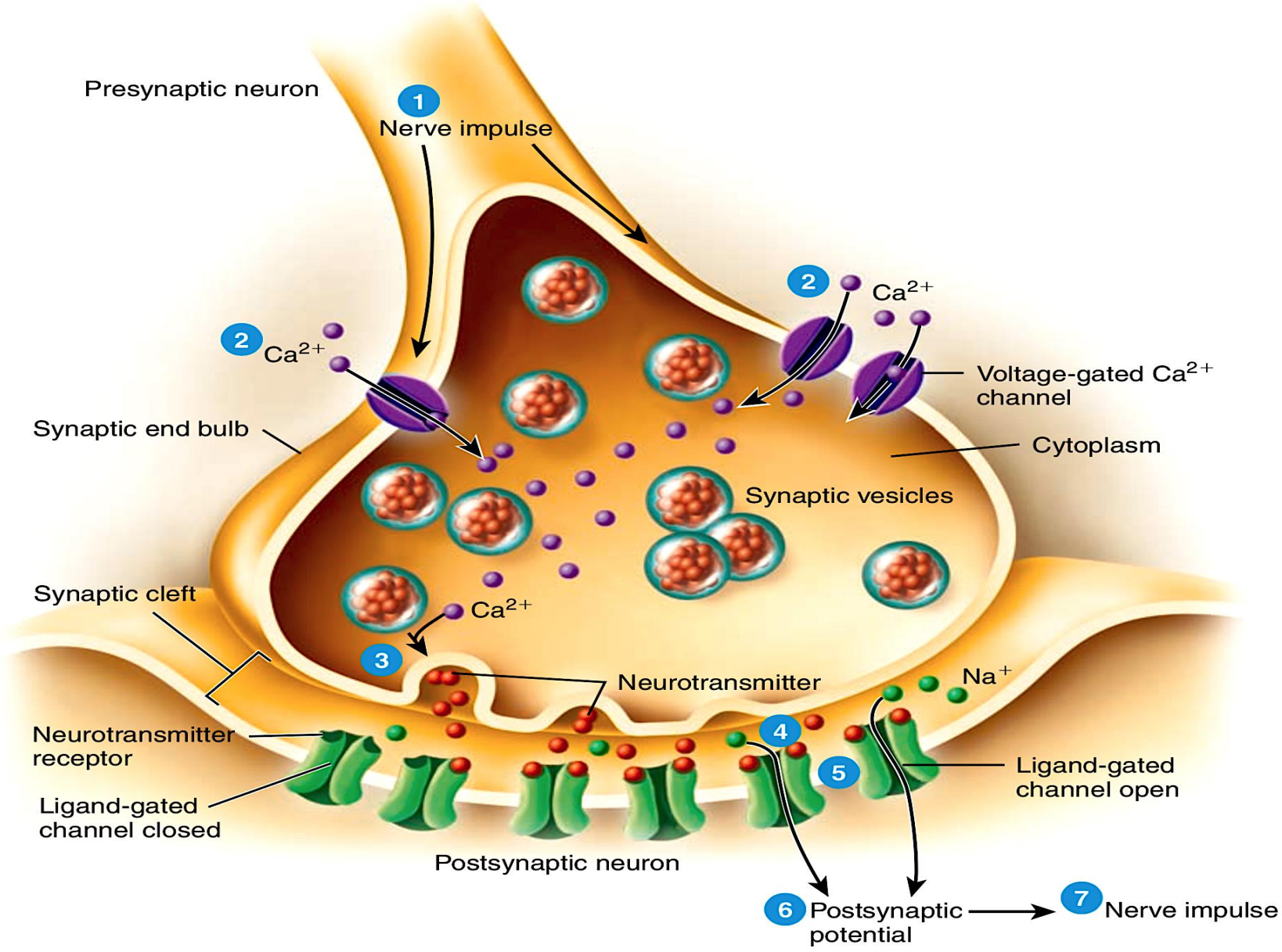
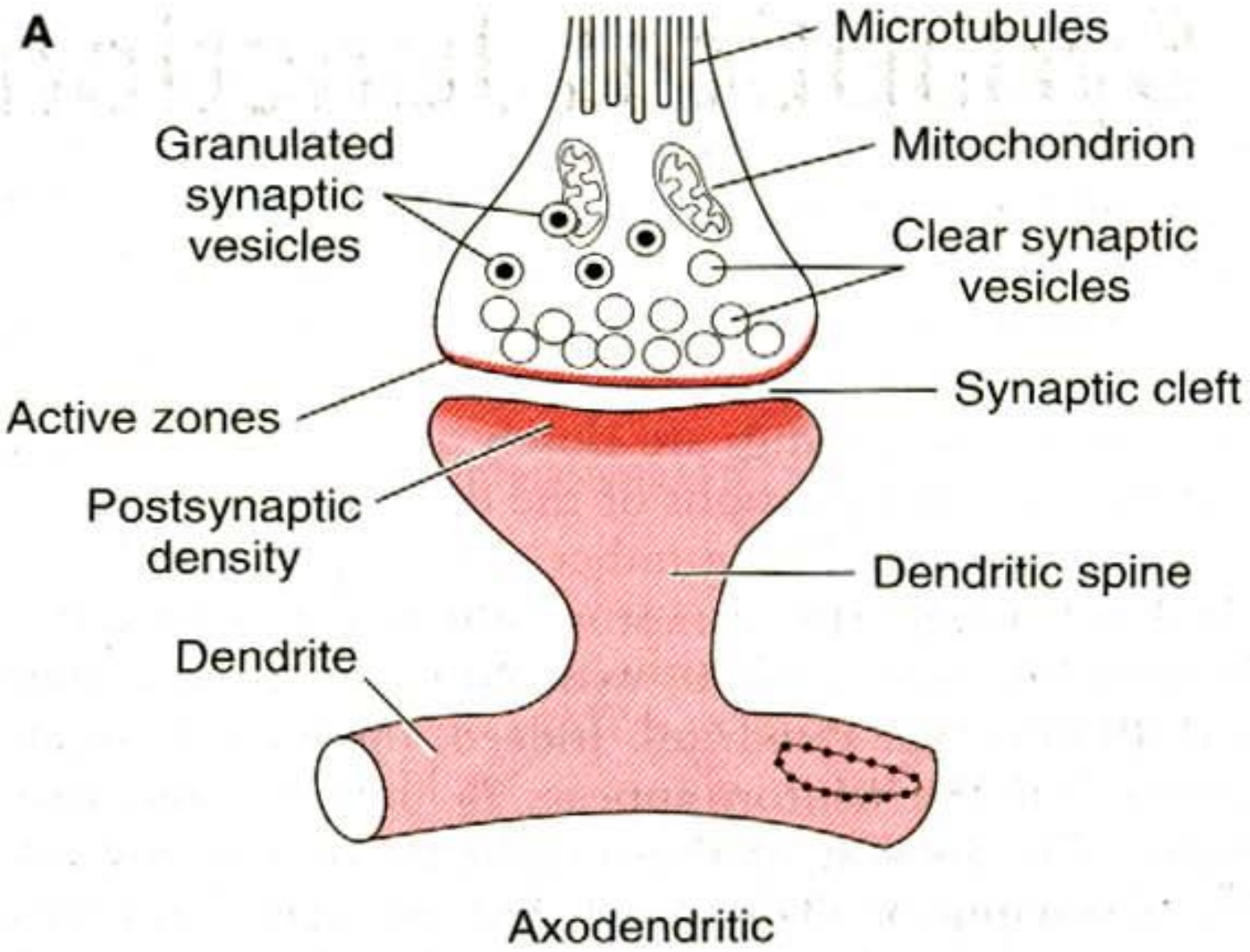
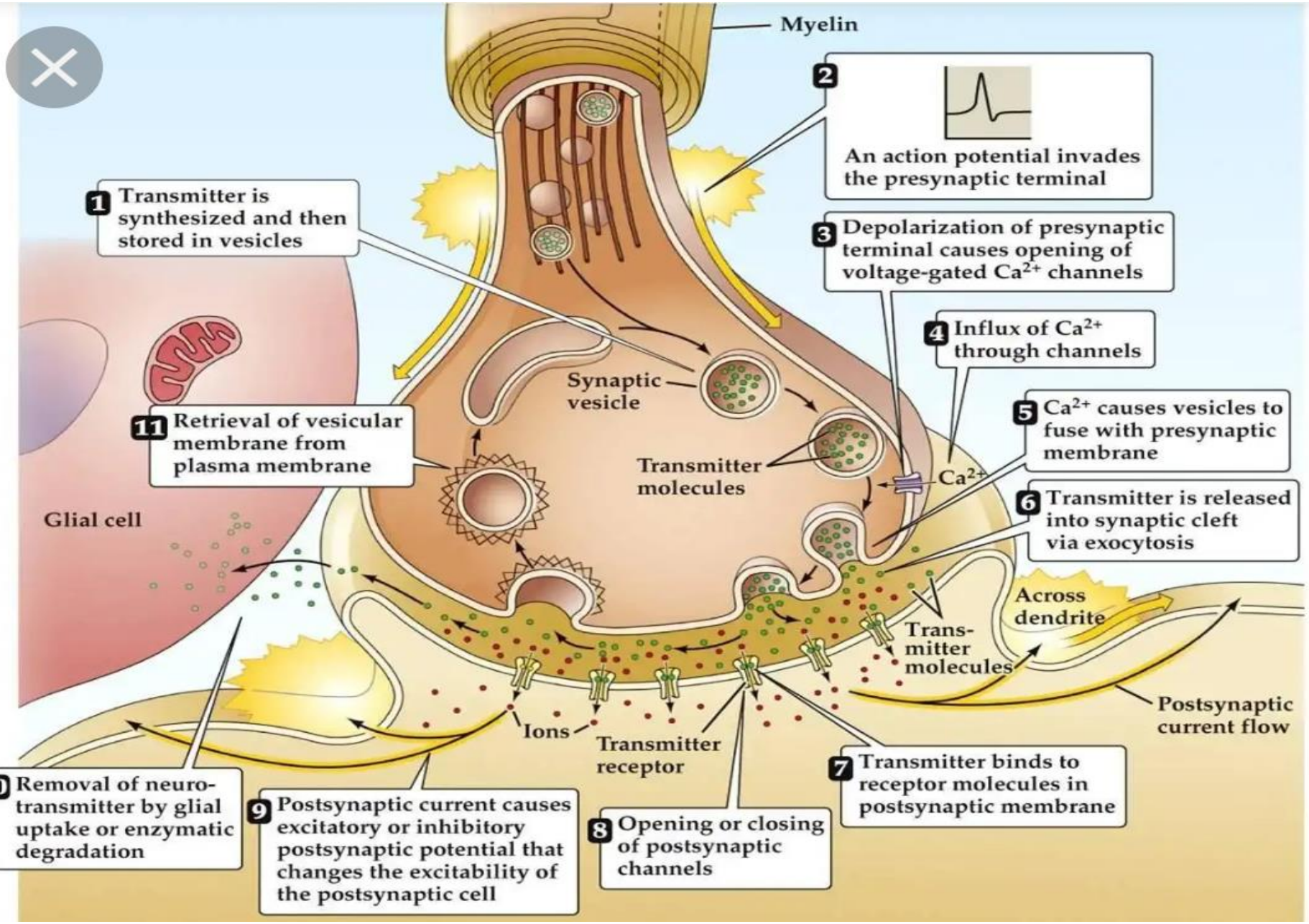


Figure 4–3. Axodendritic, axo-axonal, and axosomatic synapses. Many presynaptic neurons terminate on dendritic spines, as shown at the top, but some also end directly on the shafts of dendrites. Note the presence of clear and granulated synaptic vesicles in endings and clustering of clear vesicles at active zones, shown longitudinally in A and in cross section in B and C.


Presynaptic neuron
1
Nerve impulse
2
Ca2+
2
Ca2+
Voltage-gated Ca2+ channel
Synaptic end bulb
Cytoplasm
Synaptic vesicles
Synaptic cleft
Ca2+
3
Neurotransmitter
Na+
Neurotransmitter receptor
4
5
Ligand-gated channel open
Ligand-gated channel closed
Postsynaptic neuron
6
Postsynaptic potential
7
Nerve impulse


A
Microtubules
Granulated synaptic vesicles
Mitochondrion
Clear synaptic vesicles
Synaptic cleft
Active zones
Postsynaptic density
Dendritic spine
Dendrite
Axodendritic


Myelin
2
An action potential invades the presynaptic terminal
1 Transmitter is synthesized and then stored in vesicles
3 Depolarization of presynaptic terminal causes opening of voltage-gated Ca^{2+} channels
4 Influx of Ca^{2+} through channels
Synaptic vesicle
11 Retrieval of vesicular membrane from plasma membrane
5 Ca^{2+} causes vesicles to fuse with presynaptic membrane
Transmitter molecules
Ca^{2+}
6 Transmitter is released into synaptic cleft via exocytosis
Glial cell
Across dendrite
Trans-mitter molecules
Postsynaptic current flow
Ions
Transmitter receptor
7 Transmitter binds to receptor molecules in postsynaptic membrane
10 Removal of neuro-transmitter by glial uptake or enzymatic degradation
9 Postsynaptic current causes excitatory or inhibitory postsynaptic potential that changes the excitability of the postsynaptic cell
8 Opening or closing of postsynaptic channels

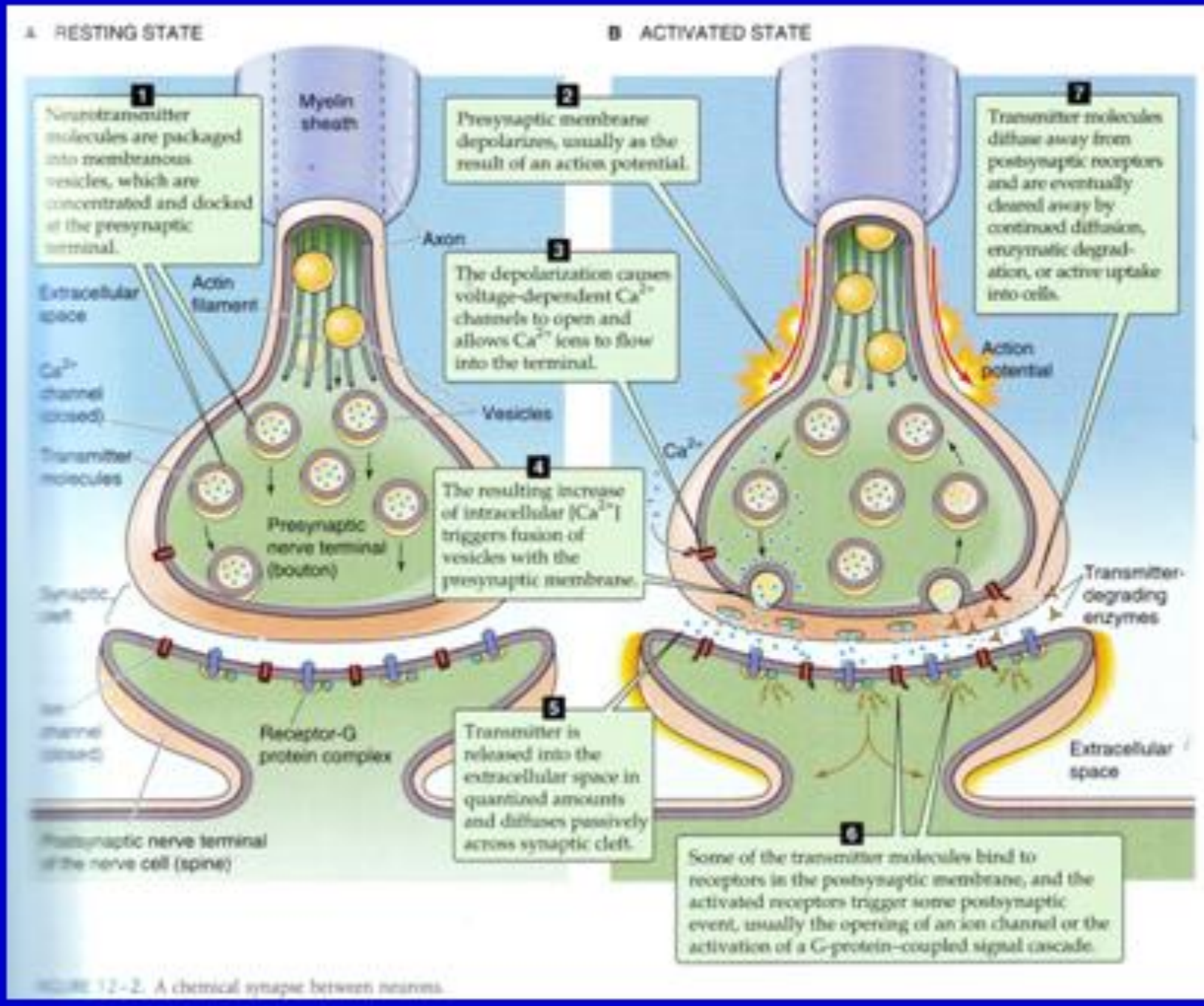


FIGURE 12-2. A chemical synapse between neurons.

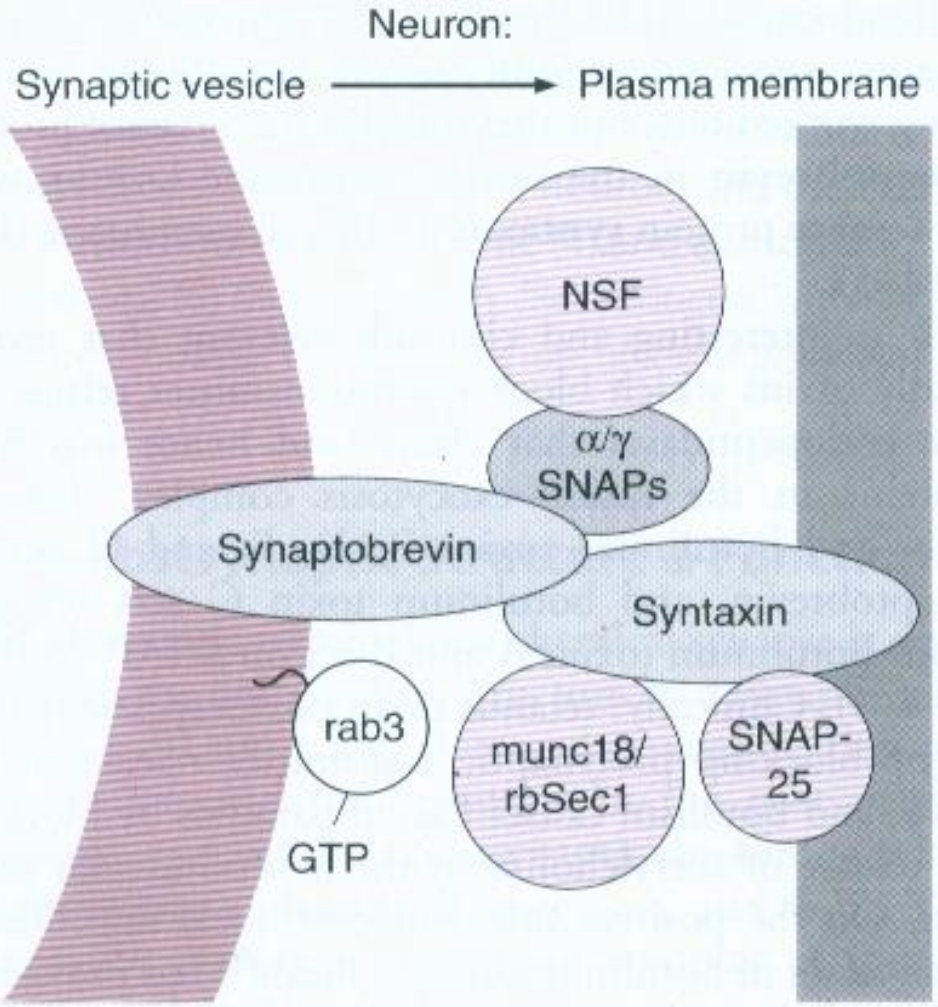


Figure 4–5. Main proteins that interact to produce synaptic vesicle docking and fusion in nerve endings. (Reproduced, with permission, from Ferro-Novick S, John R: Vesicle fusion from yeast to man. Nature 1994;370:191. Copyright © by Macmillan Magazines Ltd.)

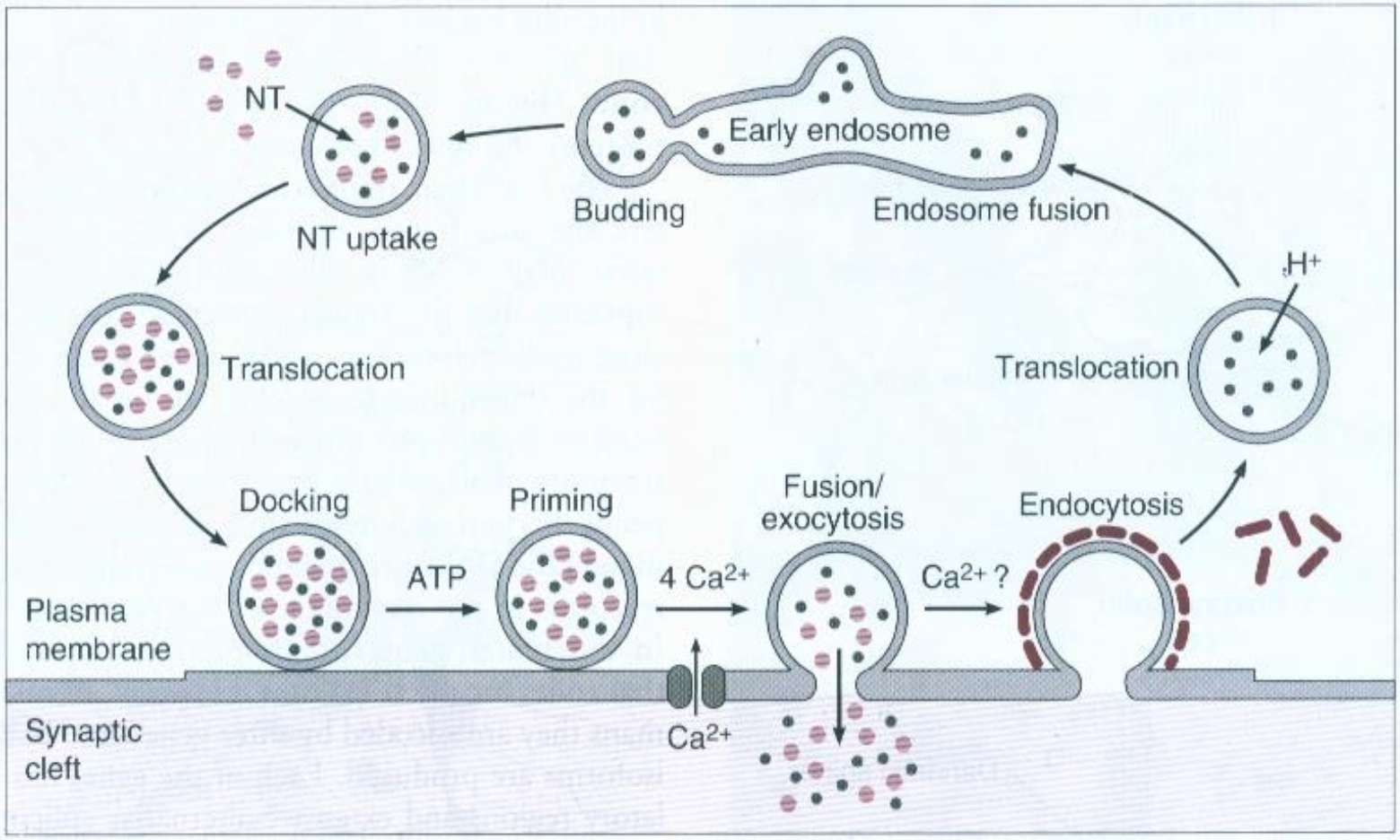


Figure 4–4. Small synaptic vesicle cycle in presynaptic nerve terminals. Vesicles bud off the early endosome and then fill with neurotransmitter (NT; top left). They then move to the plasma membrane, dock, and become primed. Upon arrival of an action potential at the ending, Ca^{2+} influx triggers fusion and exocytosis of the granule contents to the synaptic cleft. The vesicle wall is then coated with clathrin and taken up by endocytosis. In the cytoplasm, it fuses with the early endosome, and the cycle is ready to repeat. (Reproduced, with permission, from Südhof TC: The synaptic vesicle cycle: A cascade of protein–protein interactions. Nature 1995;375:645. Copyright © by Macmillan Magazines Ltd.)

Steps of synaptic transmission

Arrival of AP at pre synaptic knob

↓

Depolarization of pre synaptic membrane

↓

Opening of **voltage gated** calcium channels

↓

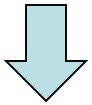
Influx of calcium into pre synaptic knob

↓

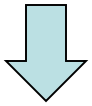
Fusion of vesicles with pre synaptic membrane

↓ (docking)

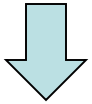
Release of NT into synaptic cleft by **exocytosis**

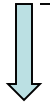


NT diffuses across synaptic cleft

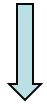


NT binds to receptors on the post synaptic density



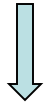


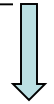
If NT is excitatory



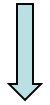
Ligand gated

Na^+ channels open



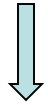


If NT is inhibitory



Ligand gated

Cl^- channels open



Influx of **Na^+** into Post synaptic membrane

⇩

Development of local depolarization – **EPSP**

Influx of **Cl^-** into post synaptic membrane

⇩

Development of local hyperpolarization – **IPSP**

NT acton is termianted by

1. Reuptake
2. Enzymatic degradation
3. Diffuse away or diluted in the ECF

clinical importance

- Tetanus toxin and botulinum toxin inactivate docking proteins synaptobrevin, syntaxin and SNAP-25
- Tetanus toxin inhibits exocytosis of NT leading to **spastic paralysis**
- Botulinum toxin inhbits Ach release at NMJ leading to **flaccid paralysis**
- Reuptake inhibitors are used in depression (SSRI)

Properties of synapse

1. One way conduction
2. Synaptic delay
3. Convergence
4. Divergence
5. EPSP *
6. IPSP *
7. Summation
8. Synaptic inhibition
9. Facilitation
10. Subliminal fringe
11. Occlusion
12. Synaptic plasticity –

    Habituation, sensitization,

    post-tetanic potentiation,

    long term potentiation

    long term depression

1. One way conduction

In a chemical synapse information is transferred always from pre synaptic neuron to post synaptic neuron.

Helps in orderly neuronal activity

2. Synaptic delay

There is a delay in the transmission of impulse from pre to post synaptic neuron. It is about **0.5ms.**

It represents the time required for steps of synaptic transmission.

3. Convergence

Many pre synaptic neurons ending on one post synaptic neuron.

4.Divergence

One pre synaptic neuron ending on many post synaptic neurons.

- Convergence and Divergence are the basis for summation, facilitation, subliminal fringe and occlusion.

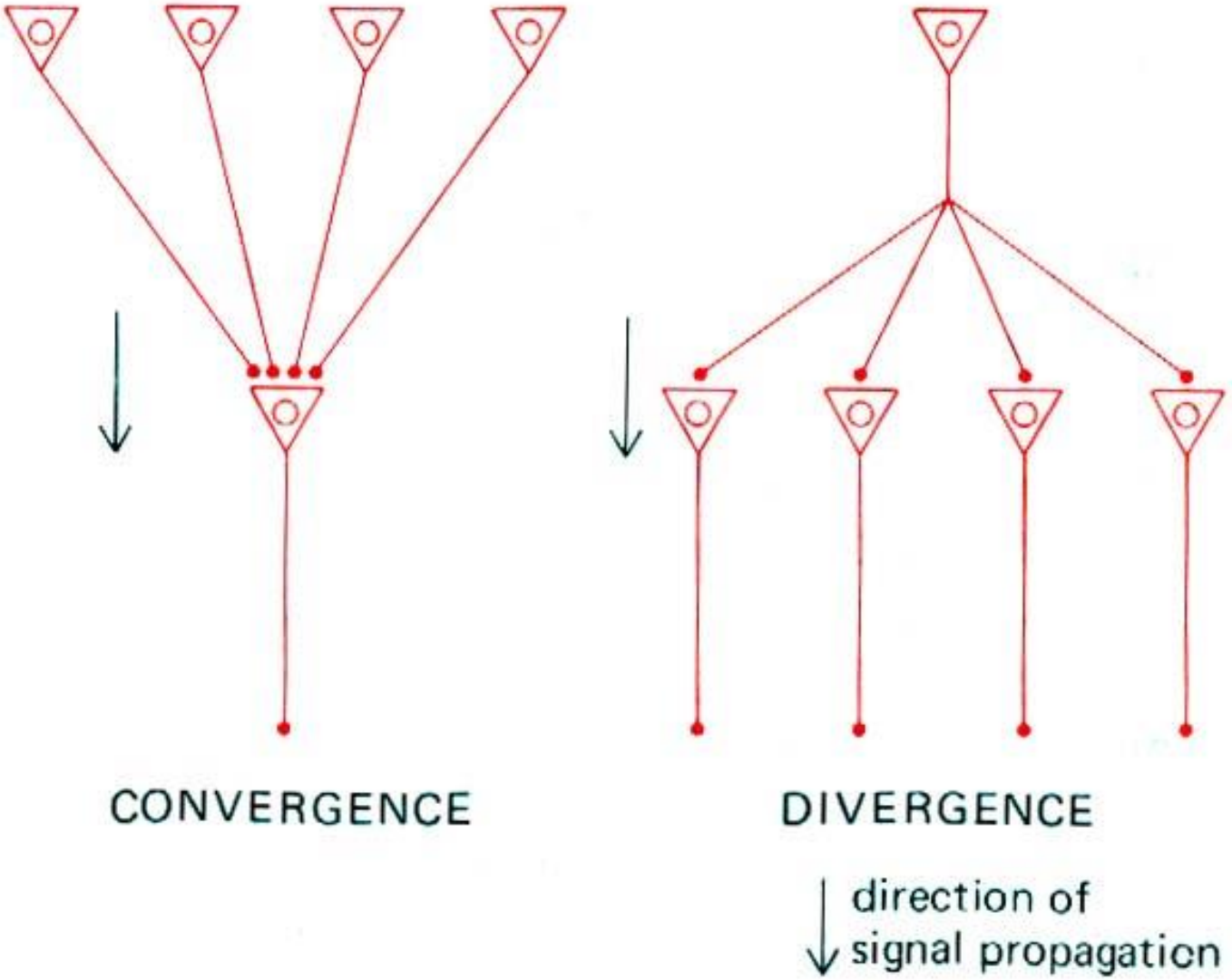


FIGURE 8-37. Convergence of neural input from many neurons onto a single neuron, and divergence of output from a single neuron onto many others.

5. EPSP (Excitatory Post Synaptic Potential)

It is the potential during which the excitability of post synaptic neuron to other stimuli is increased.

- It develops after a latency of 0.5ms.
- It reaches its peak in 1 – 1.5ms
- It decays exponentially with time.

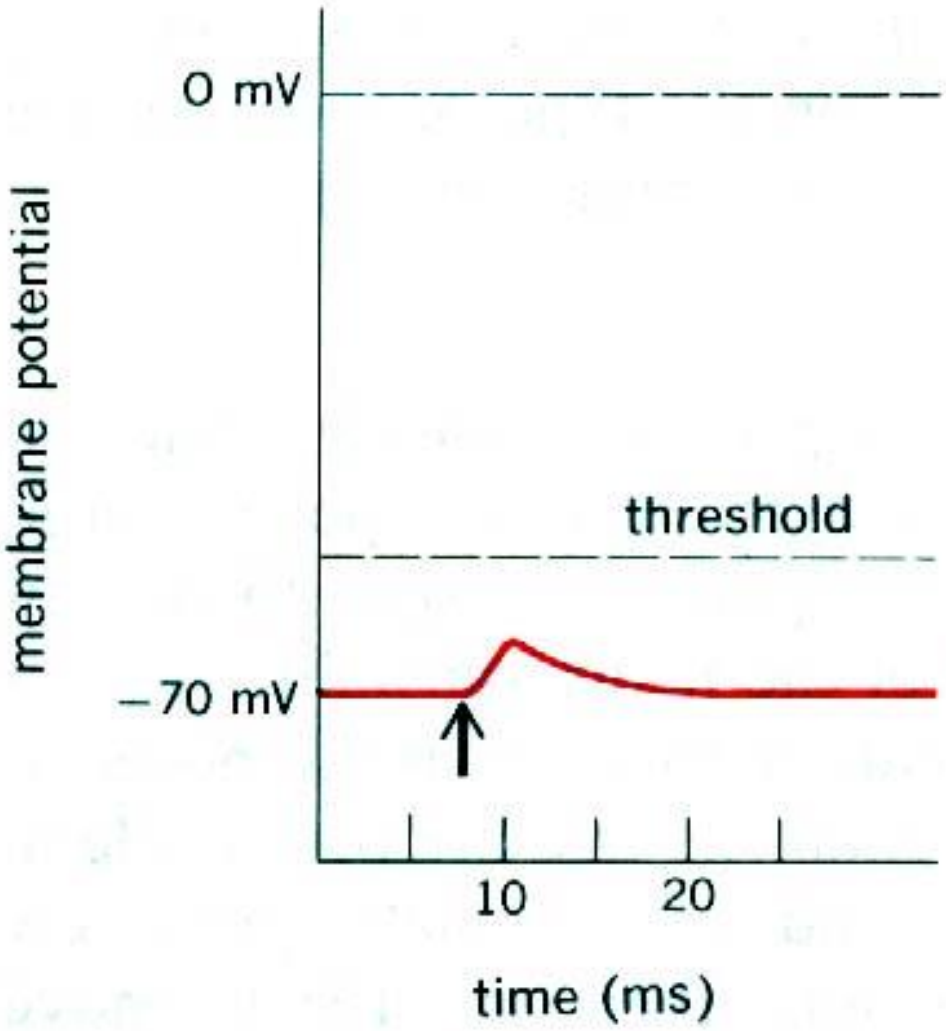


FIGURE 8-39. Excitatory postsynaptic potential (EPSP). Stimulation of the presynaptic neuron is marked by the arrow. Note the short synaptic delay before the postsynaptic cell responds.

Ionic basis of EPSP

EPSP develops in the post synaptic neuron when an excitatory NT is released from pre synaptic neuron . Ex. Glutamate.

Glutamate binds to receptors on the post synaptic membrane

↓

Opens ligand gated Na^+ channels and influx of Na^+

↓

Development of local, non propagated depolarization

EPSP

Properties of EPSP

- Local or graded potential
- Not self propagated
- Decay with time
- Do not obey All or none law
- Can be summated

6. IPSP (Inhibitory Post Synaptic Potential)

It is the potential during which the excitability of post synaptic neuron to other stimuli is decreased.

- It develops after a latency of 0.5ms.
- It reaches its peak in 1 – 1.5ms
- It decays exponentially with time(3ms)

Ionic basis of IPSP

IPSP develops in the post synaptic neuron when an inhibitory NT is released from pre synaptic neuron . Ex. GABA.

GABA binds to receptors on the post synaptic membrane

⇩

Opens ligand gated Cl^- channels and influx of Cl^-

⇩

Development of local, non propagated hyperpolarization

IPSP

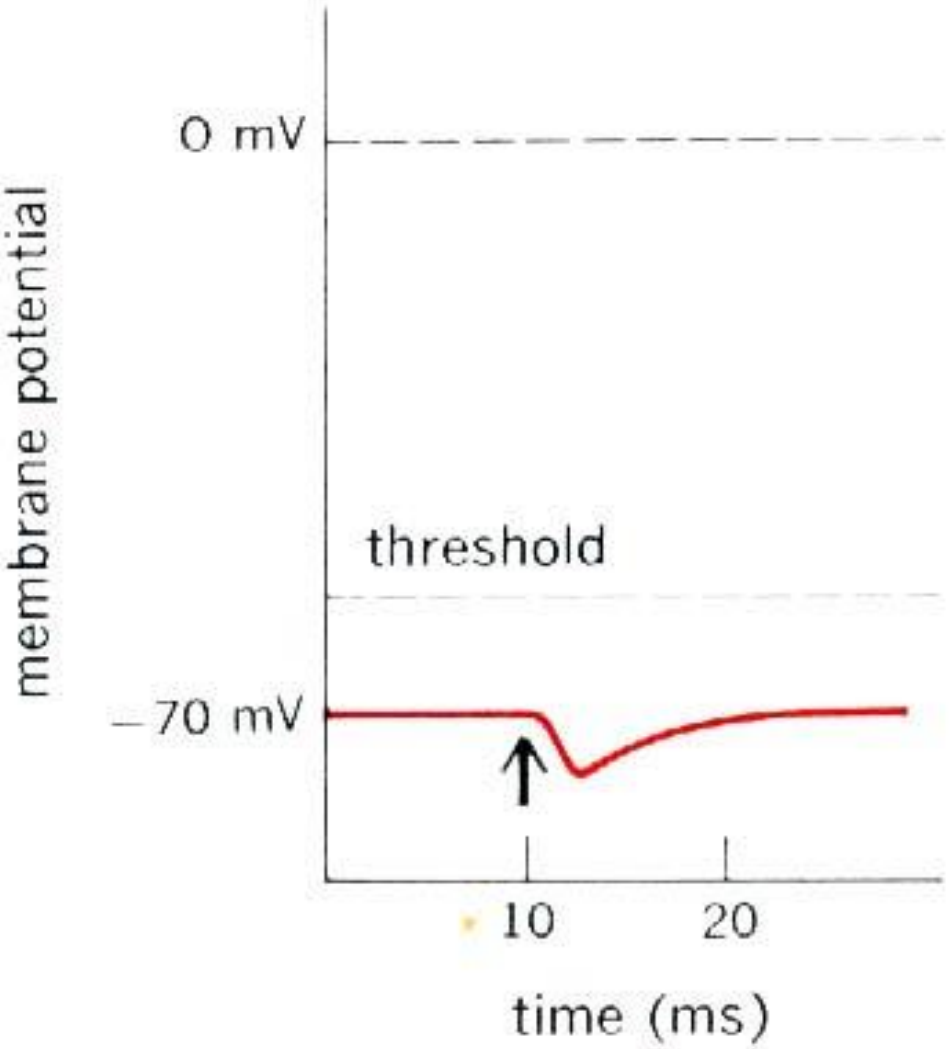


FIGURE 8-40. Inhibitory postsynaptic potential (IPSP). Stimulation of the presynaptic neuron is marked by the arrow. Note the short synaptic delay.

Properties of IPSP

- Local or graded potential
- Not self propagated
- Decay with time
- Do not obey All or none law
- Can be summated

7. Summation

- Spatial summation

When more than one pre synaptic neurons are stimulated and discharging almost simultaneously EPSPs summate.

- Temporal summation

When one pre synaptic neuron is repeatedly stimulated EPSPs summate.

8. Synaptic inhibition

- prevents over excitation of neurons
- Required for movement at joints
- prevents over shoot

Types of synaptic inhibition

- Post synaptic inhibition (direct)
- Pre synaptic inhibition (indirect)
- Renshaw cell inhibition (feed back)
- Feed forward inhibition

- **Postsynaptic inhibition**

  It is due to formation of IPSPs in the post synaptic neuron when an inhibitory NT is released from presynaptic neuron.

Ex: **Reciprocal innervation**

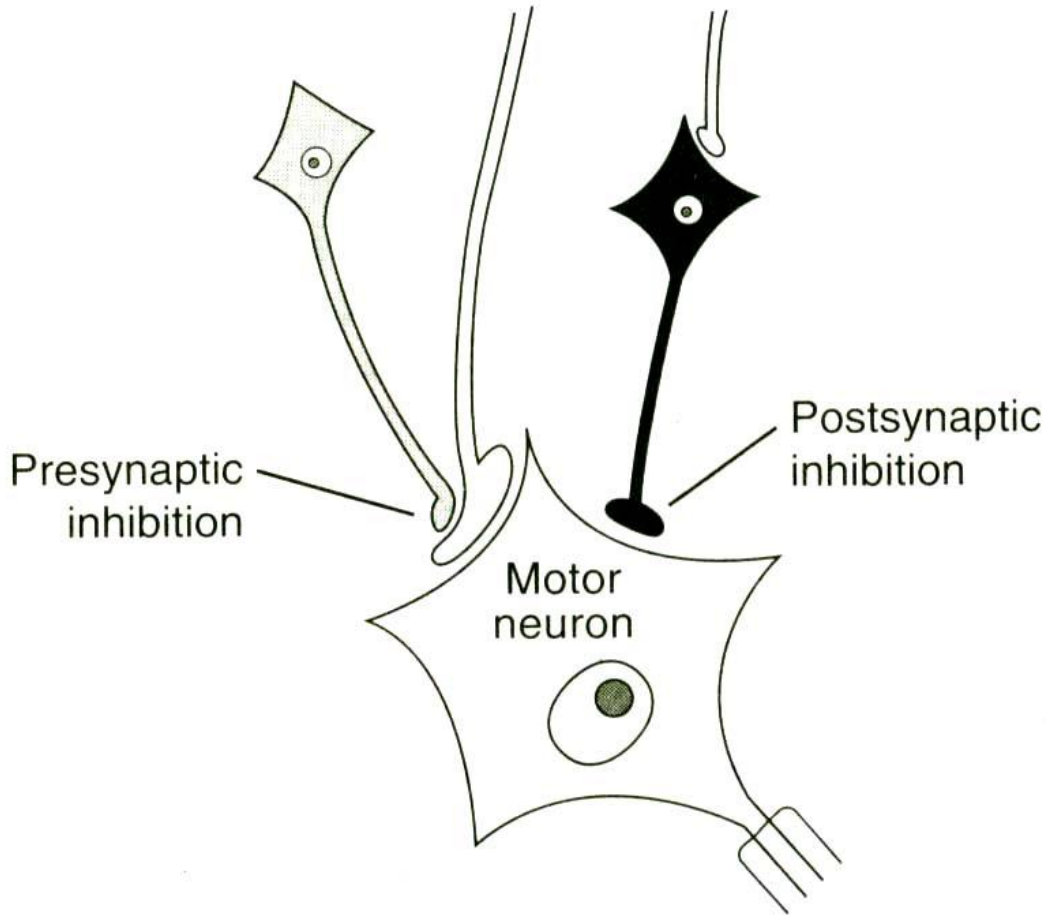


Figure 4–11. Arrangement of neurons producing presynaptic and postsynaptic inhibition. The neuron producing presynaptic inhibition is shown ending on an excitatory synaptic knob. Many of these neurons actually end higher up along the axon of the excitatory cell.

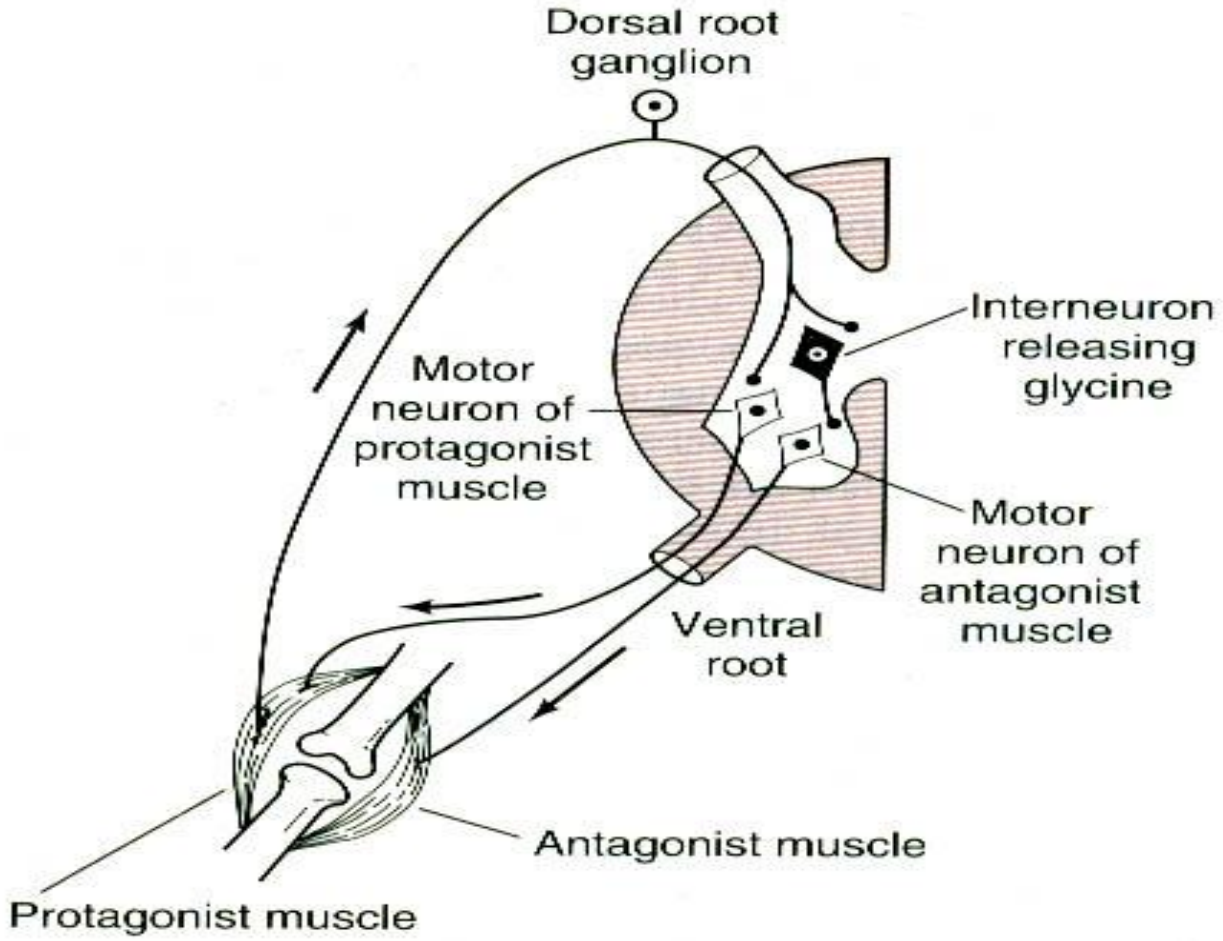


Figure 4–10. Diagram illustrating the anatomic connections responsible for inhibiting the antagonists to a muscle contracting in response to stretch. Activity is initiated in the spindle in the protagonist muscle. Impulses pass directly to the motor neurons supplying the same muscle and, via branches, to inhibitory interneurons that end on the motor neurons of the antagonist muscle.

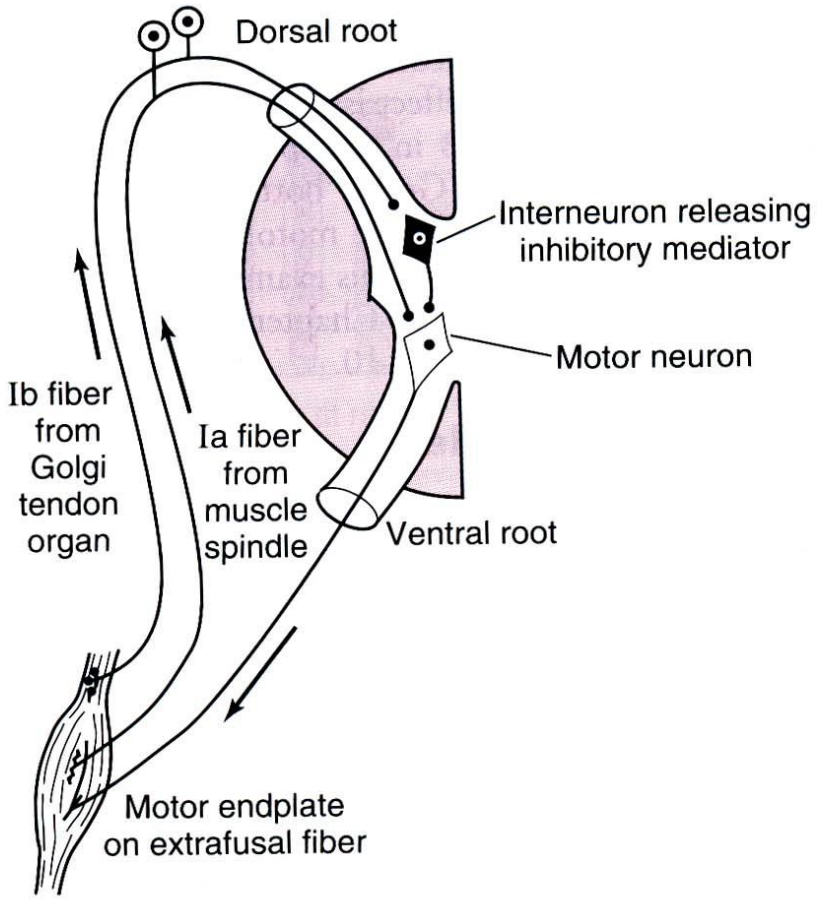


Figure 6–6. Diagram illustrating the pathways responsible for the stretch reflex and the inverse stretch reflex. Stretch stimulates the spindle, and impulses pass up the Ia fiber to excite the motor neuron. It also stimulates the Golgi tendon organ, and impulses passing up the Ib fiber activate the interneuron to release the inhibitory mediator glycine. With strong stretch, the resulting hyperpolarization of the motor neuron is so great that it stops discharging.

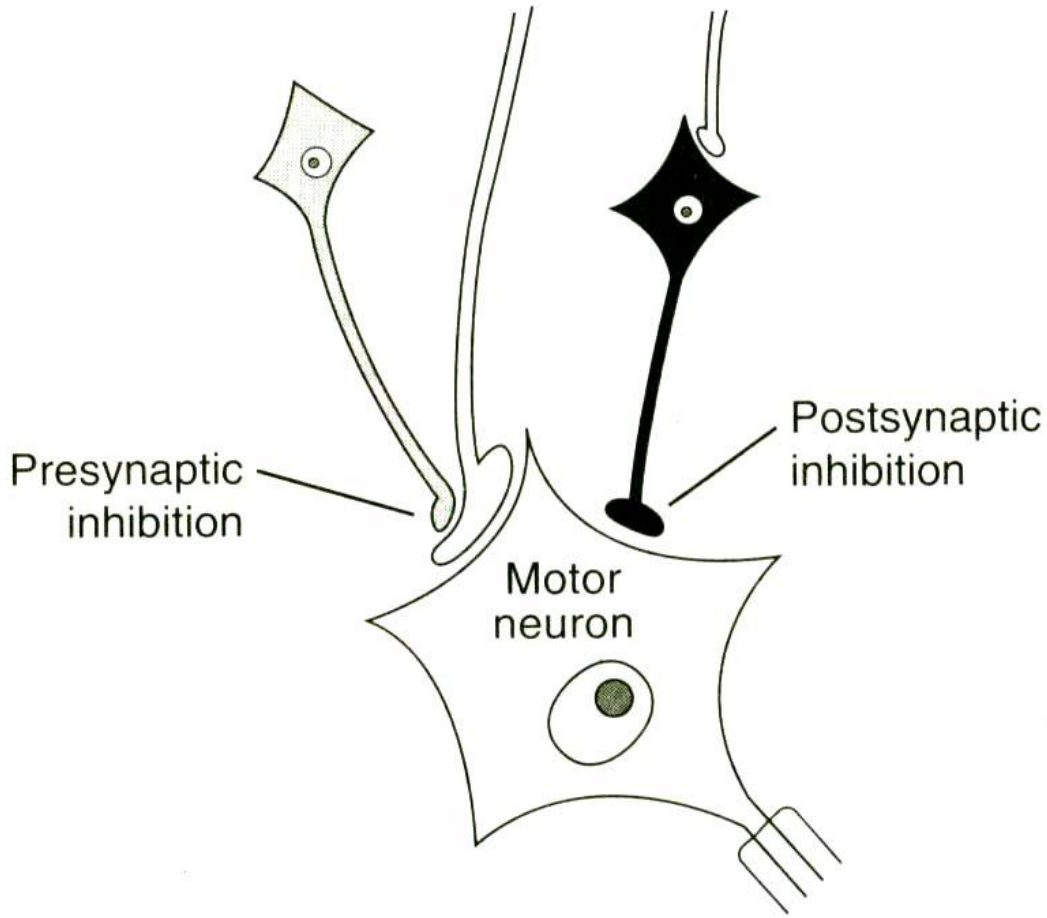


Figure 4–11. Arrangement of neurons producing presynaptic and postsynaptic inhibition. The neuron producing presynaptic inhibition is shown ending on an excitatory synaptic knob. Many of these neurons actually end higher up along the axon of the excitatory cell.

Pre synaptic inhibition

- Due to decreased release of NT from pre synaptic neuron
- Due to refractory period of previous discharge in the post synaptic neuron

Increased Cl^- influx in the pre synaptic neuron

increased K^+ efflux in the pre synaptic neuron

- **GABA**

- **Presynaptic facilitation**

It is produced when AP is prolonged and the calcium channels are open for longer period.

Aplysia

Serotonin – closure of K channels – slowing repolarization and prolonging AP

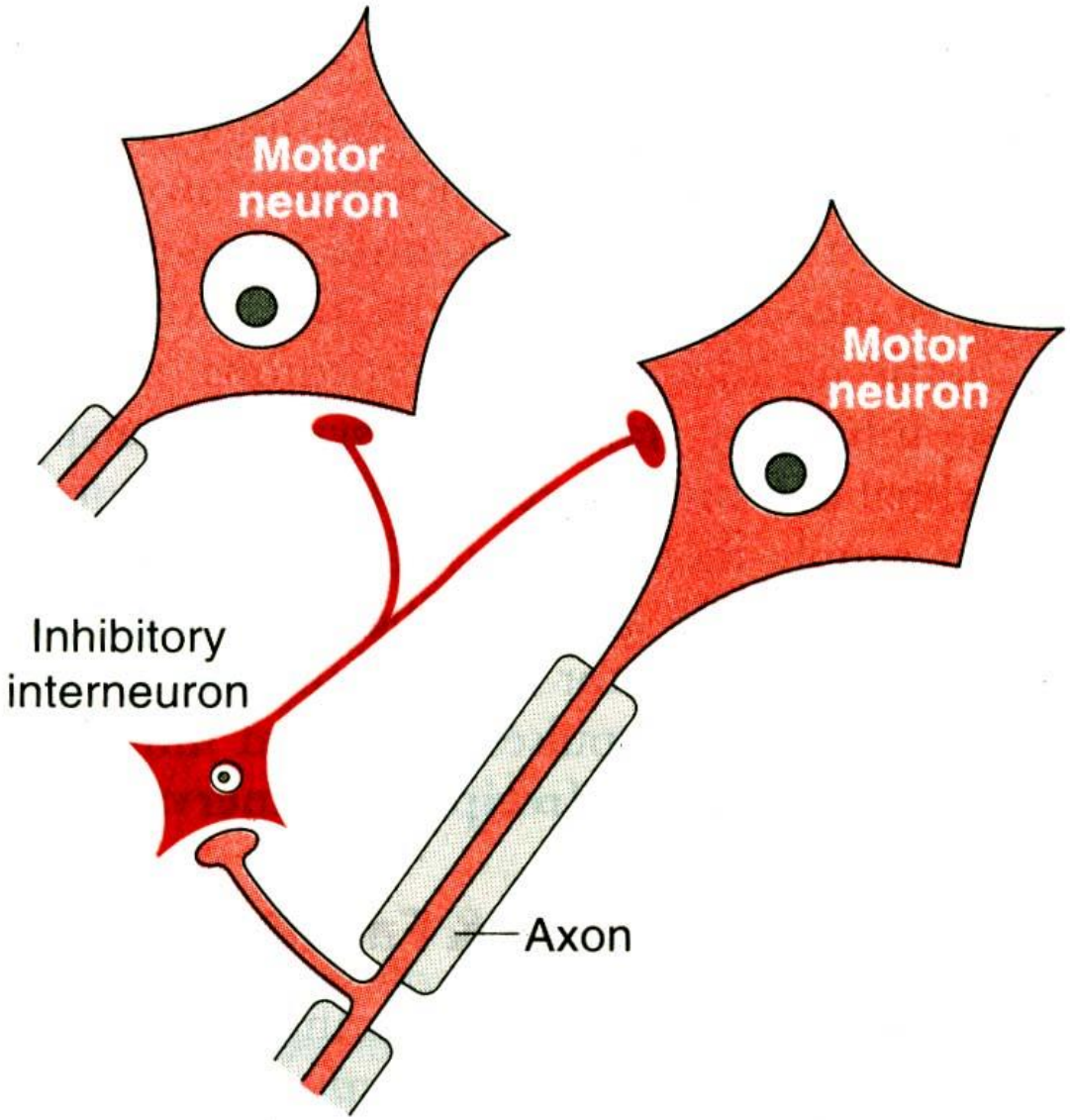
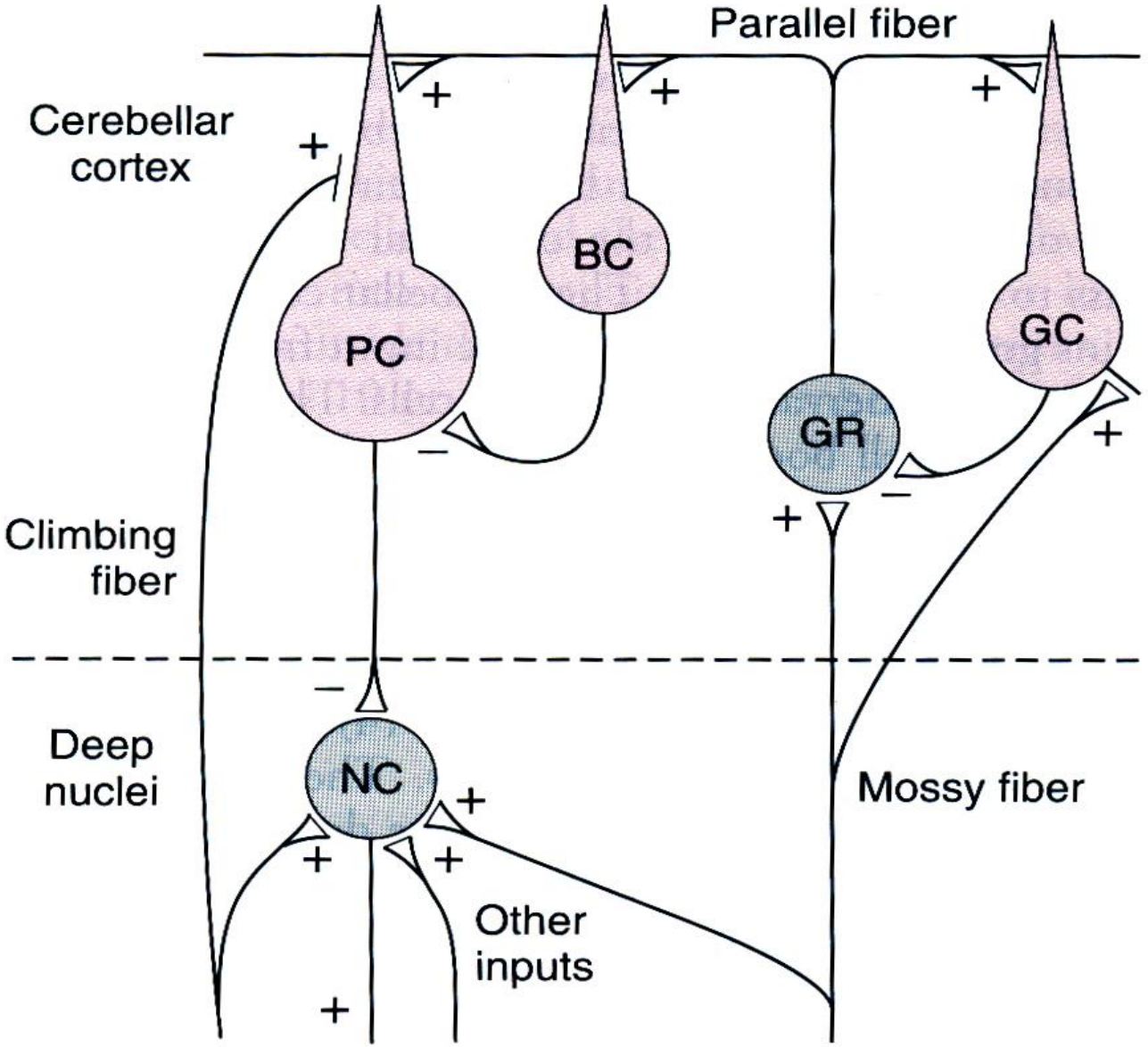


Figure 4–13. Negative feedback inhibition of a spinal motor neuron via an inhibitory interneuron (Renshaw cell).

Feed forward inhibition



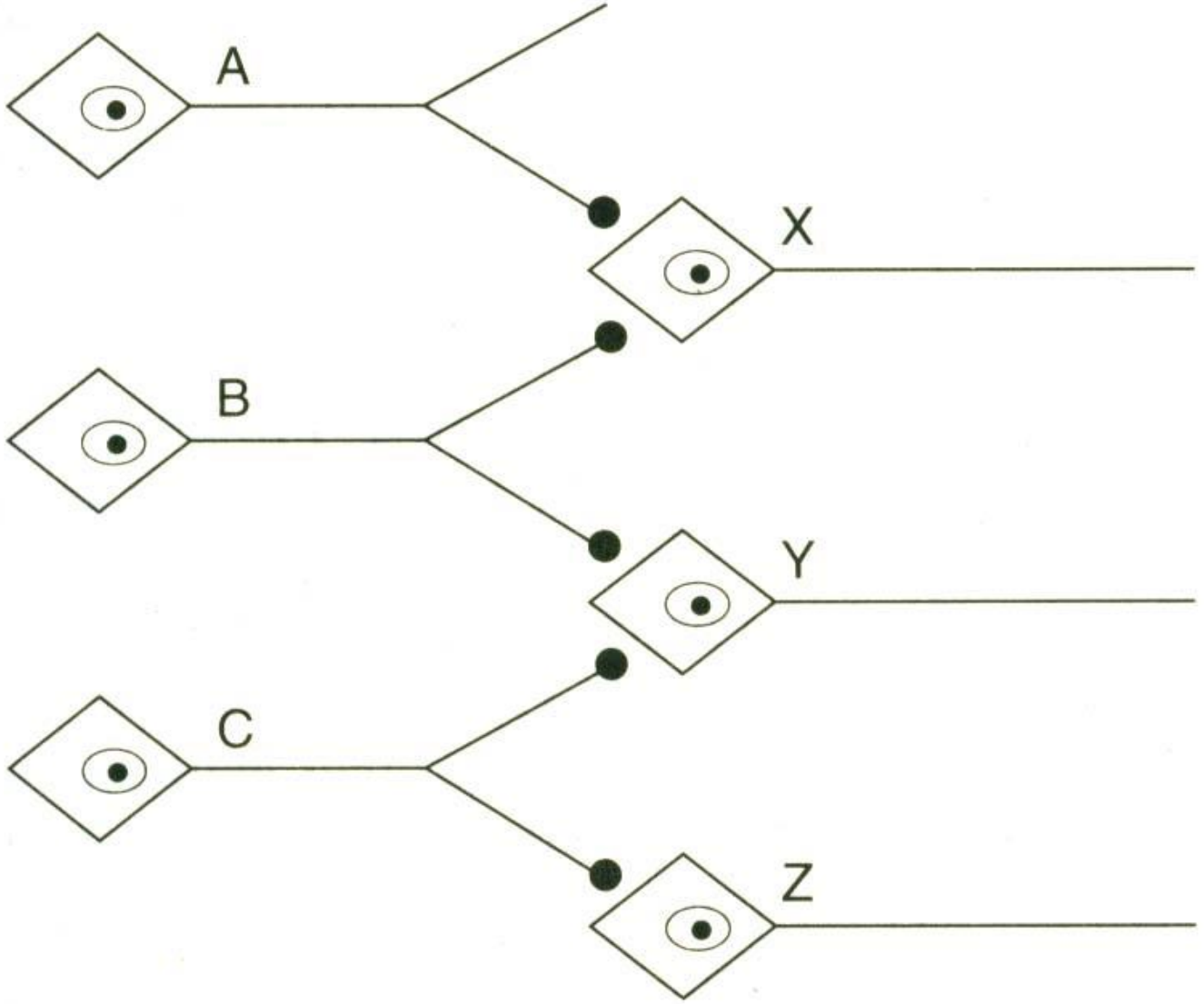


Figure 4–14. Simple nerve net. Neurons A, B, and C have excitatory endings on neurons X, Y, and Z.

9. Subliminal fringe

A neuron is said to be in subliminal fringe if it is not in discharge zone but its excitability is increased

10. Occlusion

It is the decrease in the expected response due to pre synaptic neurons sharing post synaptic neurons

11. Synaptic plasticity

- **Post tetanic potentiation**

- Production of enhanced post synaptic potentials in response to brief tetanizing stimuli.

- It is due to accumulation of calcium in the **presynaptic neuron**

- short lived (60s)

Habituation

- When a stimulus is benign and is repeated over and over, the response to the stimulus gradually disappears.

  - Gradual inactivation of Calcium channels
  - Decreased intracellular calcium
  - Decreased release of NT

- **Sensitization**

- If a stimulus to which habituation has developed is paired with a noxious stimulus,response is augmented

- It is due to presynaptic facilitation

- **Long-term potentiation**

  - It is a rapidly developing persistent enhanced response to a rapidly repeated stimulus for a brief period.

  - It is due to increase in intracellular **calcium** in the **postsynaptic neuron**

  - Last for several days

  - NMDA receptors

- **Long-term depression**

Neurotransmitters

Criteria

- Should be synthesized and stored in the pre synaptic neurons
- A NT should be released on stimulation of nerve.
- A NT should have specific receptors on the post synaptic membrane.
- A NT should be associated with an enzyme for its inactivation.
- A NT when applied extrinsically should mimic the effects of nerve stimulation.

Classification

A. Small molecule rapidly acting NTs

1. **Acetylcholine**
2. **amines**
    a. Catecholamines
    b. Serotonin
    c. Histamine
3. **Amino acids**
    a. Glutamate, Aspertate
    b. . GABA, Glycine

B. Large molecule slow acting NTs

Neuropeptides

- Releasing hormones- CRH, TRH, GnRH
- Posterior pituitary hormnes – ADH, Oxytocin

Somatostatin, Endorphins, Enkephalins, Substance P,

- **Purines** – Adenosine, ATP
- **Gases** – NO, CO

Physiological classification

- Excitatory NTs
  - Glutamate
  - Aspertate

- Inhibitory NTs
  - GABA
  - Glycine

- **Acetylcholine**

Cholinergic neurons

- Nerve endings at NMJ

- All Pre and Post ganglionic parasympathetic nerves

- Pre ganglionic sympathetic nerves

- Post ganglionic Sympathetic nerves to sweat glands and skeletal muscle blood vessels

-Betz cells

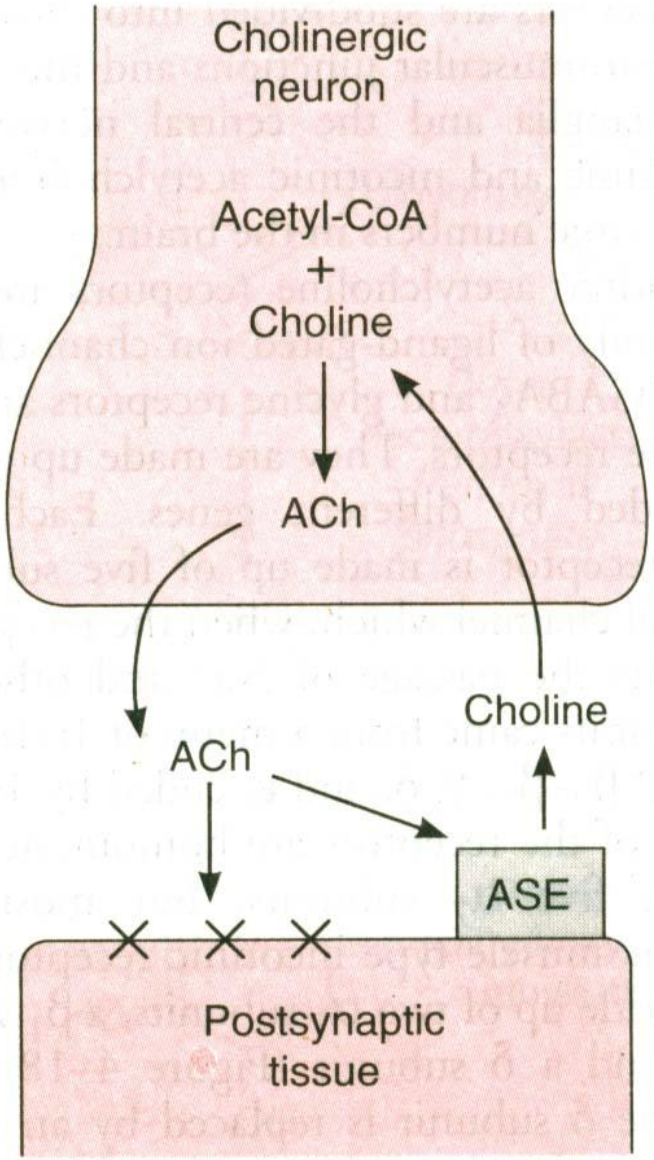


Figure 4–17. Biochemical events at cholinergic endings. ACh, acetylcholine; ASE, acetylcholinesterase; X, receptor. Compare with Figures 4–21 and 4–25.

Receptors

Nicotinic receptors

- All autonomic ganglia
- At NMJ

Muscarinic receptors

- Heart
- Smooth muscle
- Glands

Motivation, Cognition, Attention and Arousal, Learning and Memory, PGO spikes in REM sleep.

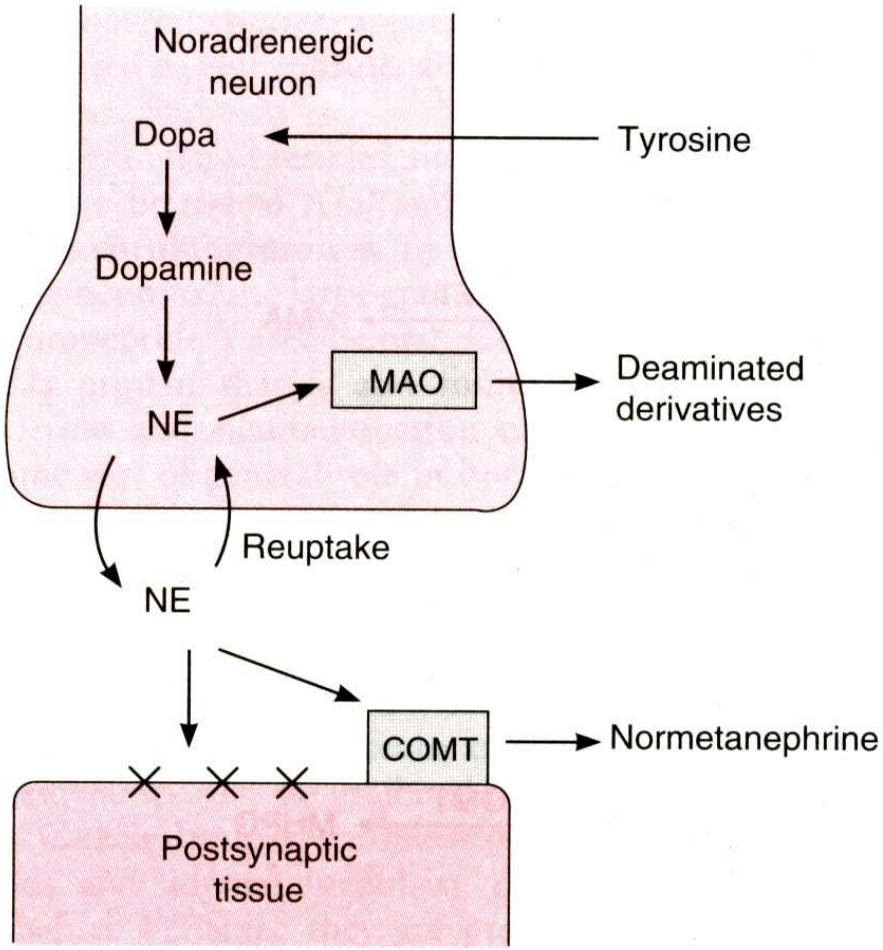


Figure 4–21. Biochemical events at noradrenergic endings. NE, norepinephrine; COMT, catechol-*O*-methyltransferase; MAO, monoamine oxidase; X, receptor. For clarity, the presynaptic receptors have been omitted. Note that MAO is intracellular, so that norepinephrine is being constantly deaminated in noradrenergic endings. COMT acts primarily on secreted norepinephrine. Compare with Figures 4–17 and 4–25.

- **Catecholamines (NE, EN, Dopamine**)

  Adrenergic neurons

  Receptors α_1, α_2 , $\beta_1 \beta_2 \beta_3$

  Sleep, arousal, regulation of posterior pituitary hormones and hypophysiotropic hormones, suppression of ACTH.

Dopamine (D1 and D2 Receptors)

Control of movement – Parkinsonism

Induction of vomiting

Inhibition of Prolactin

Schizophrenia

- **Serotonin**

CNS, ENS, Retina

Platelets, Mast cells

Endogenous analgesic system

Mood elevator

Hallucinations (LSD – Lysergic acid Diethylamide)

- SSRI (Citalopram, Fluoxetine)

- **Histamine**

CNS – Hypothalamus, Limbic system

Gastric mucosa, Mast cells.

H_1, H_2, H_3 receptors

- Controls Behavioural functions

-Smooth muscle contraction (Bronchospasm)

-HCl secretion

-Increases capillary permeability

-Arteriolar dilatation.

- **Amino Acids**

-Excitatory NTs

Glutamate, Aspartate

Metabotropic and Ionotropic receptors

NMDA, (N methyl D Aspartate)
AMPA (Alpha amino hydroxy methylisoxazole propionate)

-**Inhibitory NTs**

GABA, Glycine

GABAa , GABAb receptors

GAD- Stiff Man Syndrome .

Sensory system

- **Sensation:**

  It is the conscious or subconscious awareness of changes in the external or internal environment.

- **Perception:**

  It is the conscious interpretation of sensations – Function of cerebral cortex

- **Sensory modality**

Each unique type of sensation is called sensory modality – such as touch, pain, hearing.

- **Senses :**

- General Senses

(Somatic senses and Visceral senses **)**

- Special Senses – Vision**,** Audition, Gustation, Olfaction, Acceleration.

- **Somaesthetic senses**

  Touch, Pressure, Vibration, Itch, Tickle, Temperature (warm and cold), Pain and Proprioception.

- **Epicritic** – fine, well localized (Fine touch)
- **Protopathic** – crude, not well localized(pain and temperature)

- **Visceral senses**

Table 5–1. Principal sensory modalities.

Sensory Modality[a]	Receptor	Sense Organ
Vision	Rods and cones	Eye
Hearing	Hair cells	Ear (organ of Corti)
Smell	Olfactory neurons	Olfactory mucous membrane
Taste	Taste receptor cells	Taste bud
Rotational acceleration	Hair cells	Ear (semicircular canals)
Linear acceleration	Hair cells	Ear (utricle and saccule)
Touch-pressure	Nerve endings	Probably nerve endings
Warmth	Nerve endings	Probably nerve endings
Cold	Nerve endings	Probably nerve endings
Pain	Nerve endings	Probably nerve endings
Joint position and movement	Nerve endings	Various[b]

Muscle length	Nerve endings	Muscle spindle
Muscle tension	Nerve endings	Golgi tendon organ
Arterial blood pressure	Nerve endings	Stretch receptors in carotid sinus and aortic arch
Central venous pressure	Nerve endings	Stretch receptors in walls of great veins, atria
Inflation of lung	Nerve endings	Stretch receptors in lung parenchyma
Temperature of blood in head	Neurons in hypothalamus	
Arterial P_{O_2}	Glomus cells	Carotid and aortic bodies
pH of CSF	Receptors on ventral surface of medulla oblongata	
Osmotic pressure of plasma	Cells in OVLT and possibly other circumventricular organs in anterior hypothalamus	
Arteriovenous blood glucose difference	Cells in hypothalamus (glucostats)	

[a]The first 11 are conscious sensations.

[a]See text.

Components of Sensory system

- Sensory Receptors
- Ascending tracts
- Somatosensory Cortex

- **Sensory Receptor** :

  It is a modified nerve ending of peripheral division of a sensory nerve which functions as biological transducer that converts various forms of energy into Action Potentials**.**

Various forms of energy to which receptor responds are

Light, Sound, Thermal, Chemical, Mechanical.

Classification

- **Based on source of stimuli (Sherrington's)**
  - Exteroceptors – Ex; touch, pressure, pain
  - Interoceptors – Ex; chemoreceptors, Baro receptors, Osmoreceptors,
  - Teleceptors – Ex; Rods & Cones, Hair cells, Olfactory Receptors
  - Proprioceptors- Located in Muscles, Tendons and Joints

    Ex; Muscle Spindle, Golgi Tendon Organ

Based on type of stimuli to which receptor responds

- Mechanoreceptors – Touch and Pressure
- Chemoreceptors – Chemical composition
- Thermoreceptors – Changes in Temperature
- Nociceptors - Painful stimuli
- Photo Receptors - Light
- Osmoreceptors – Changes in Osmotic pressure

- **Based on Adaptation**

  - Phasic Receptors (Rapidly Adapting)

    Ex; Pacinian Corpuscle

  - Tonic Receptors (Slowly Adapting)

    Ex; Pain Receptors

Clinical or anatomical Classification

- Superficial receptors – Present in Skin and Mucous membrane
- Deep receptors- Prsent in Muscles, Tendons and Joints
- Visceral receptors – Present in the Visceral organs

- **Based on Microscopic Structure**

  - **Free Nerve endings**

    Ex ; Receptors for Pain, Temperature,

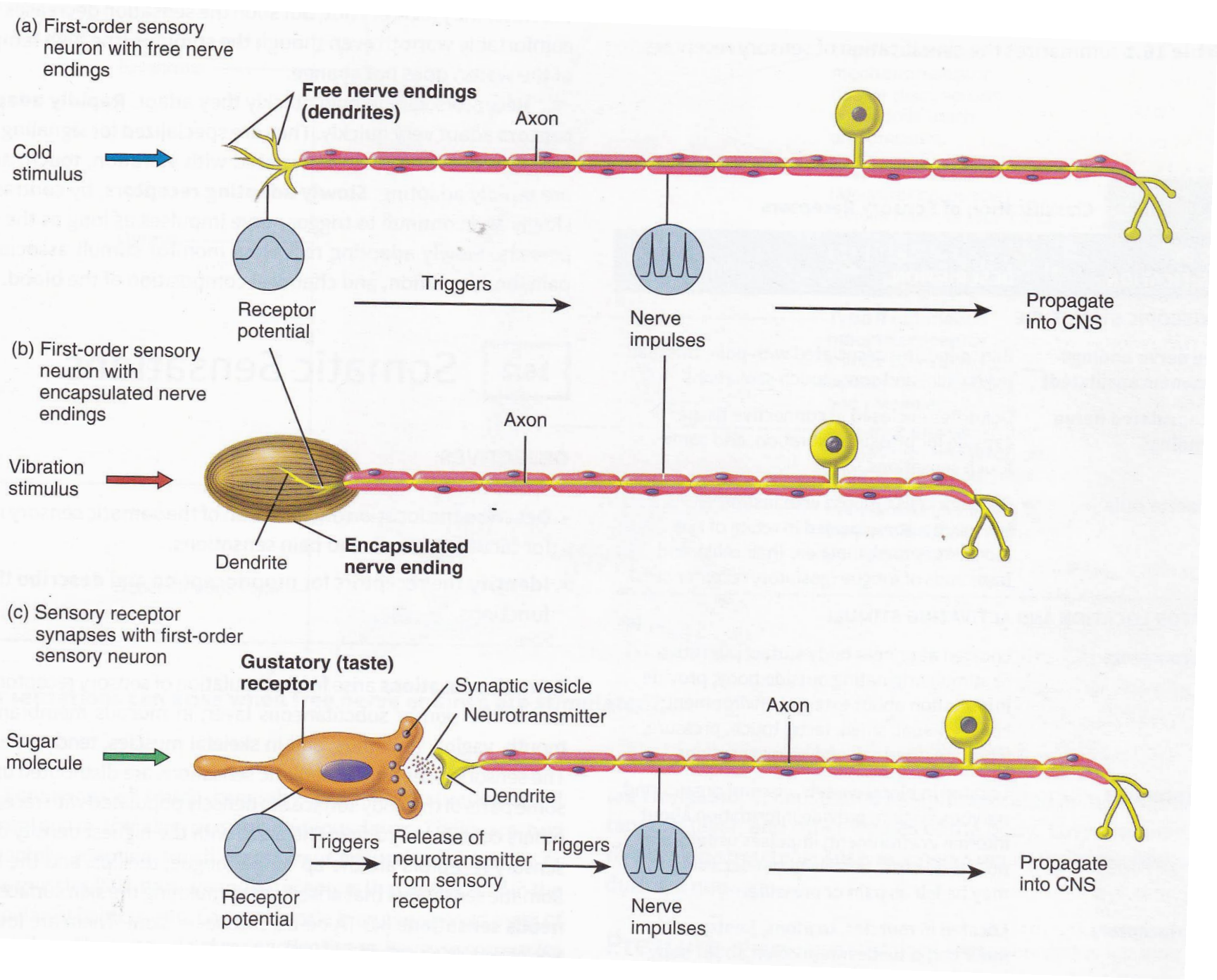
    Tickle, itch

  - Encapsulated Nerve endings

    Ex ; Receptors for Touch

  - Separate Cells

    Ex ; Receptors for Hearing (Hair cells)


(a) First-order sensory neuron with free nerve endings
Free nerve endings (dendrites)
Axon
Cold stimulus
Receptor potential
Triggers
Nerve impulses
Propagate into CNS
(b) First-order sensory neuron with encapsulated nerve endings
Axon
Vibration stimulus
Dendrite
Encapsulated nerve ending
(c) Sensory receptor synapses with first-order sensory neuron
Gustatory (taste) receptor
Synaptic vesicle
Neurotransmitter
Axon
Sugar molecule
Dendrite
Triggers
Release of neurotransmitter from sensory receptor
Triggers
Receptor potential
Nerve impulses
Propagate into CNS

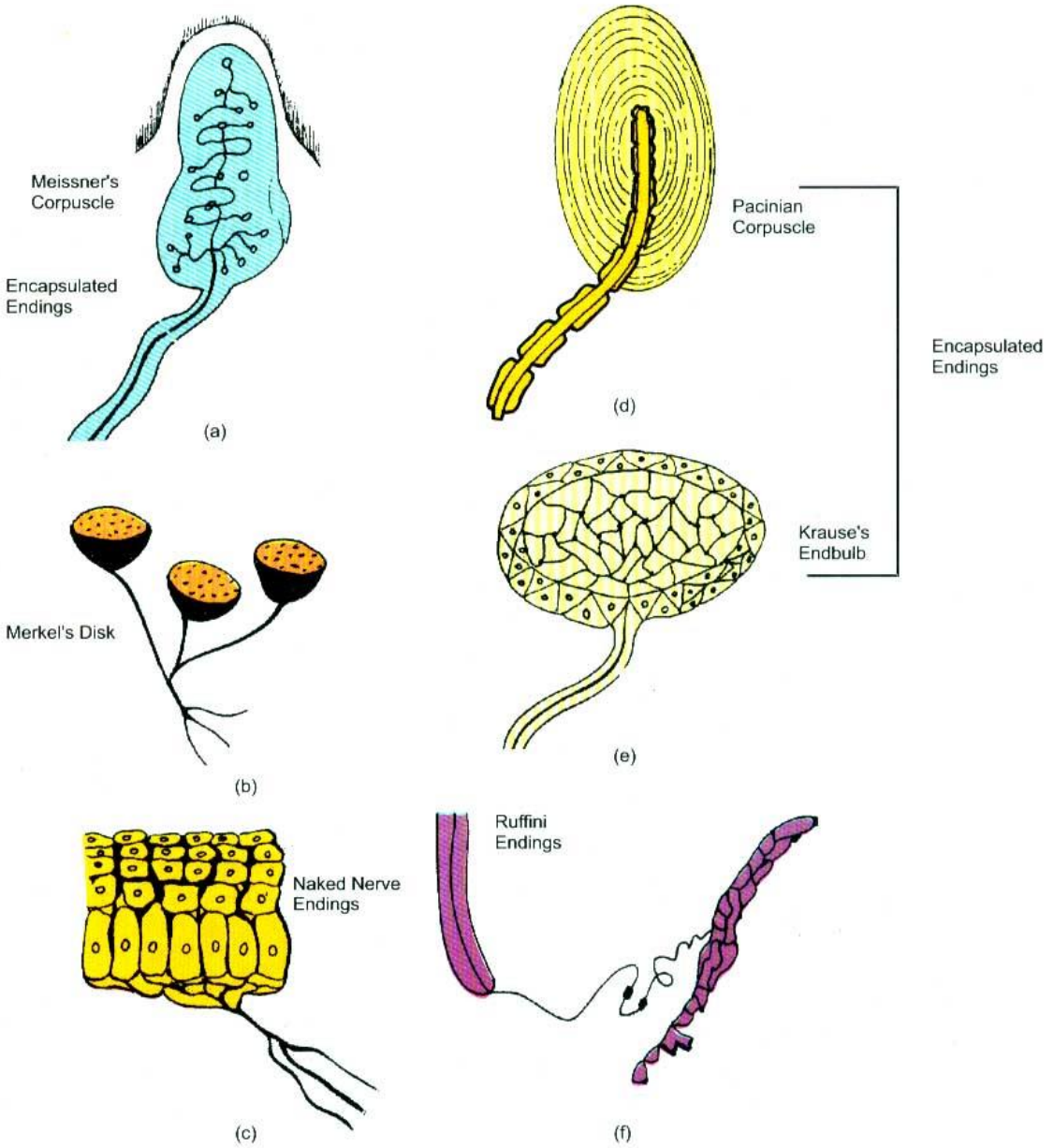


Fig. 8.15 : Sensory Receptors

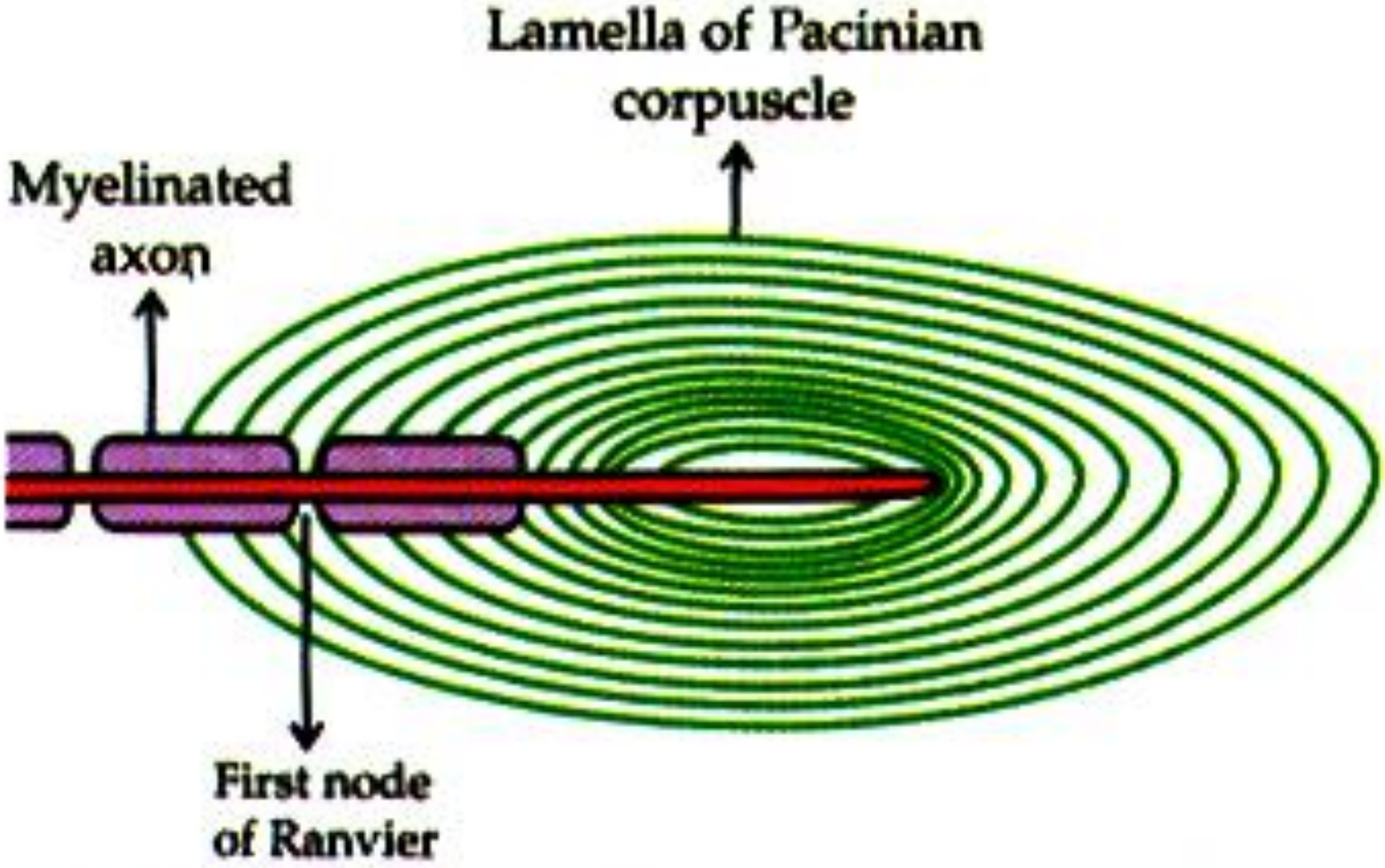


Figure 101.1 Pacinian corpuscle. Usually, first node of Ranvier remains within the lamella of the corpuscle.

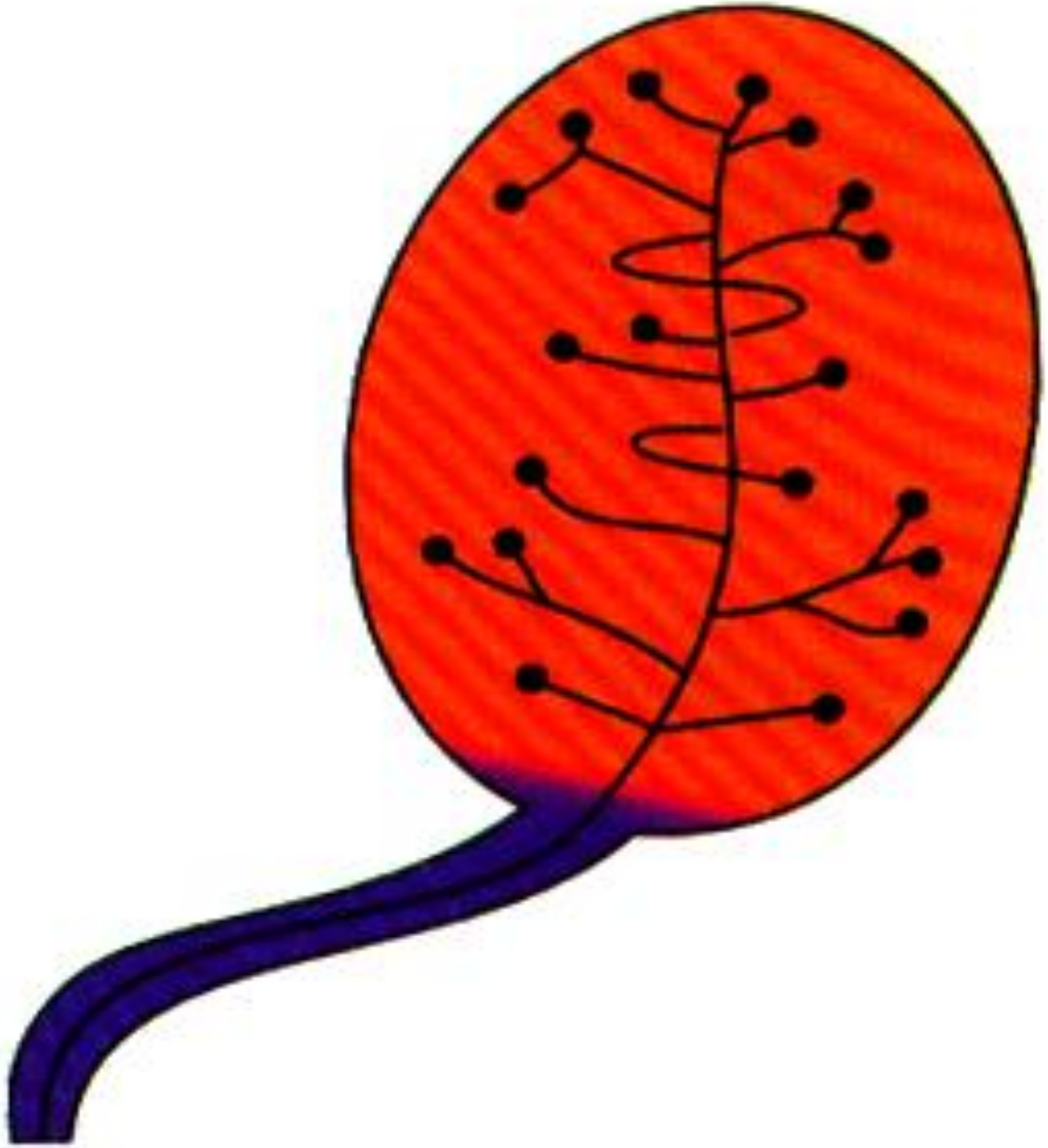


Figure 101.2 Meissner corpuscle.

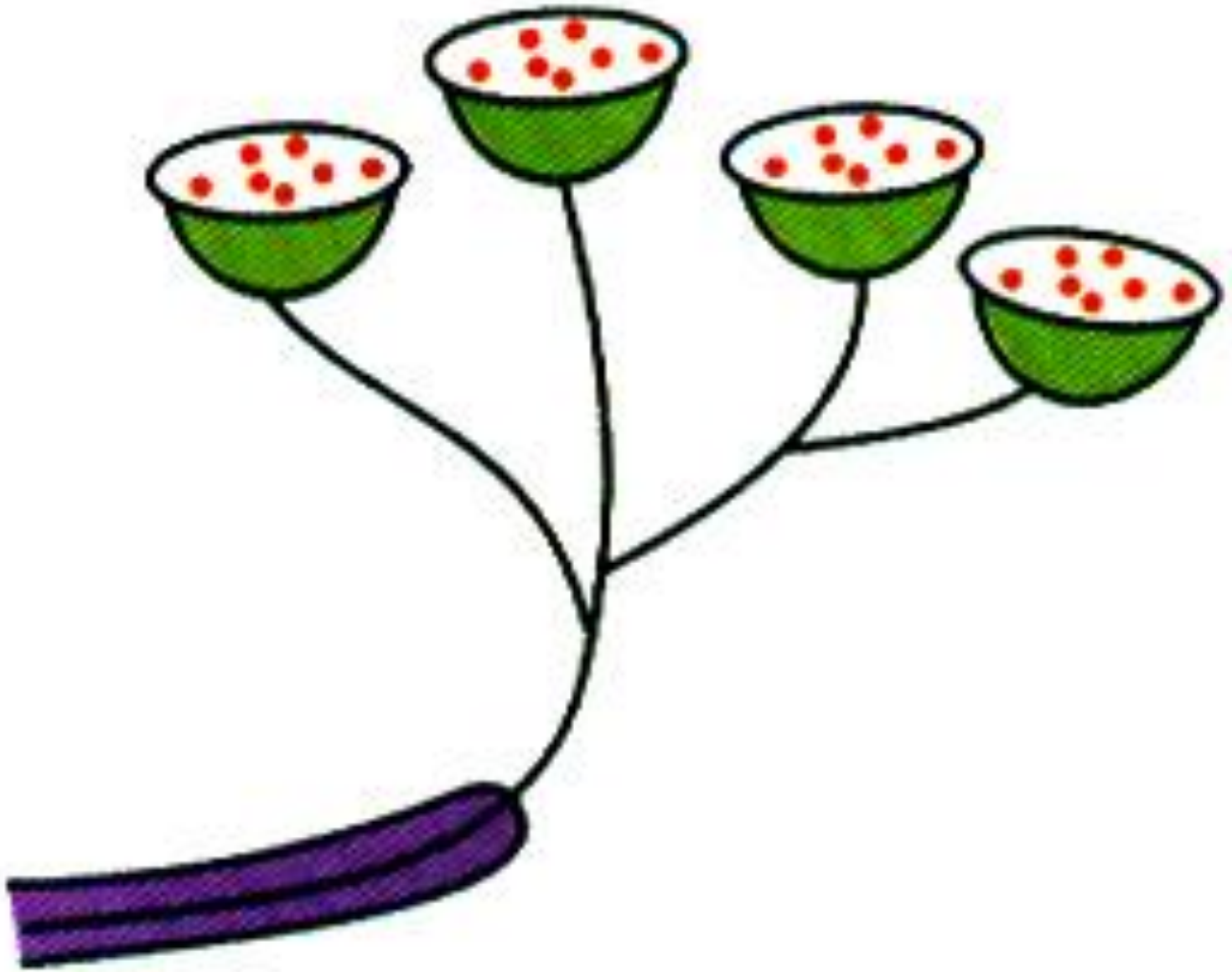


Figure 101.3 Merkel disk.

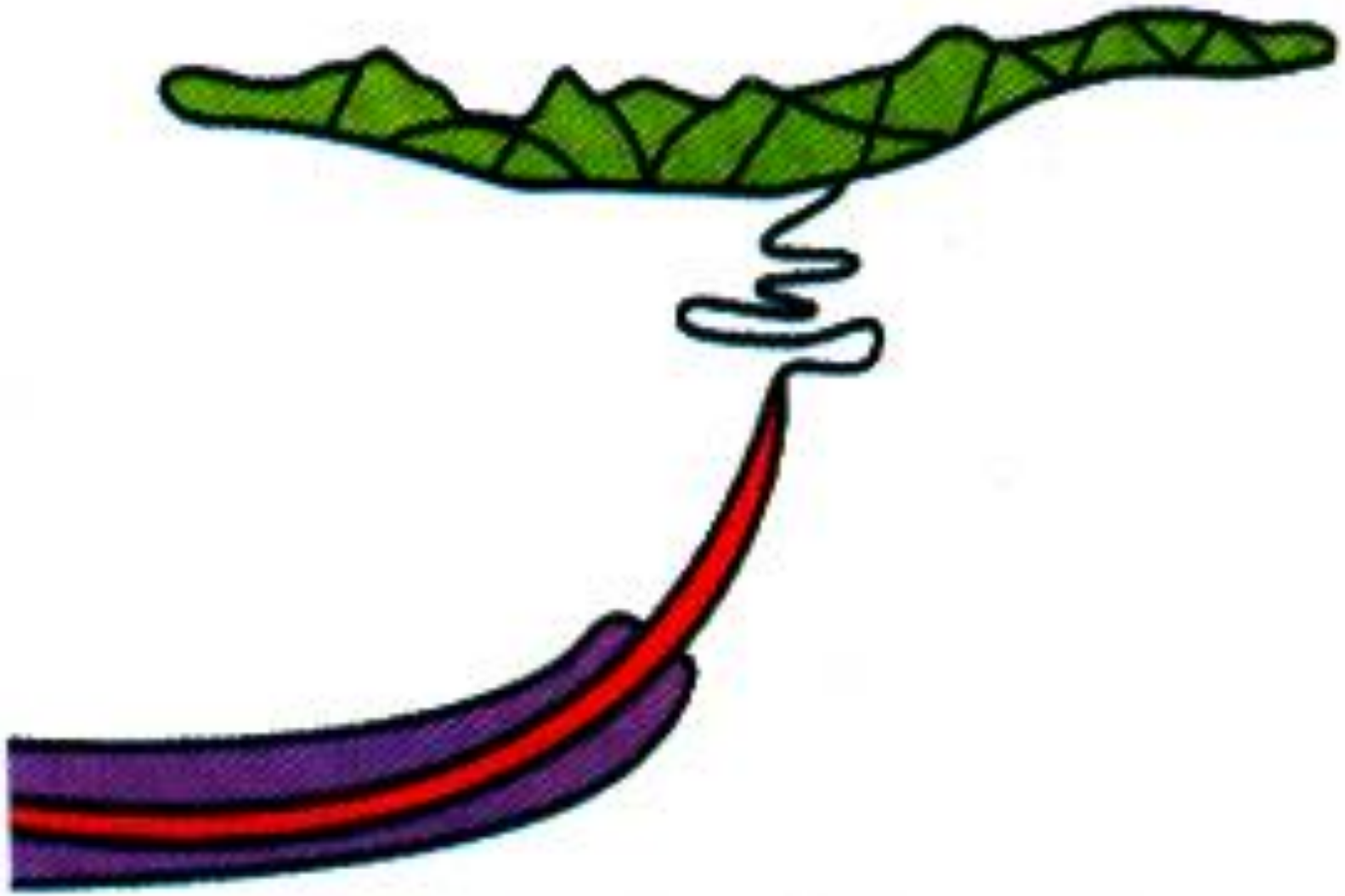


Figure 101.4 Ruffini ending.

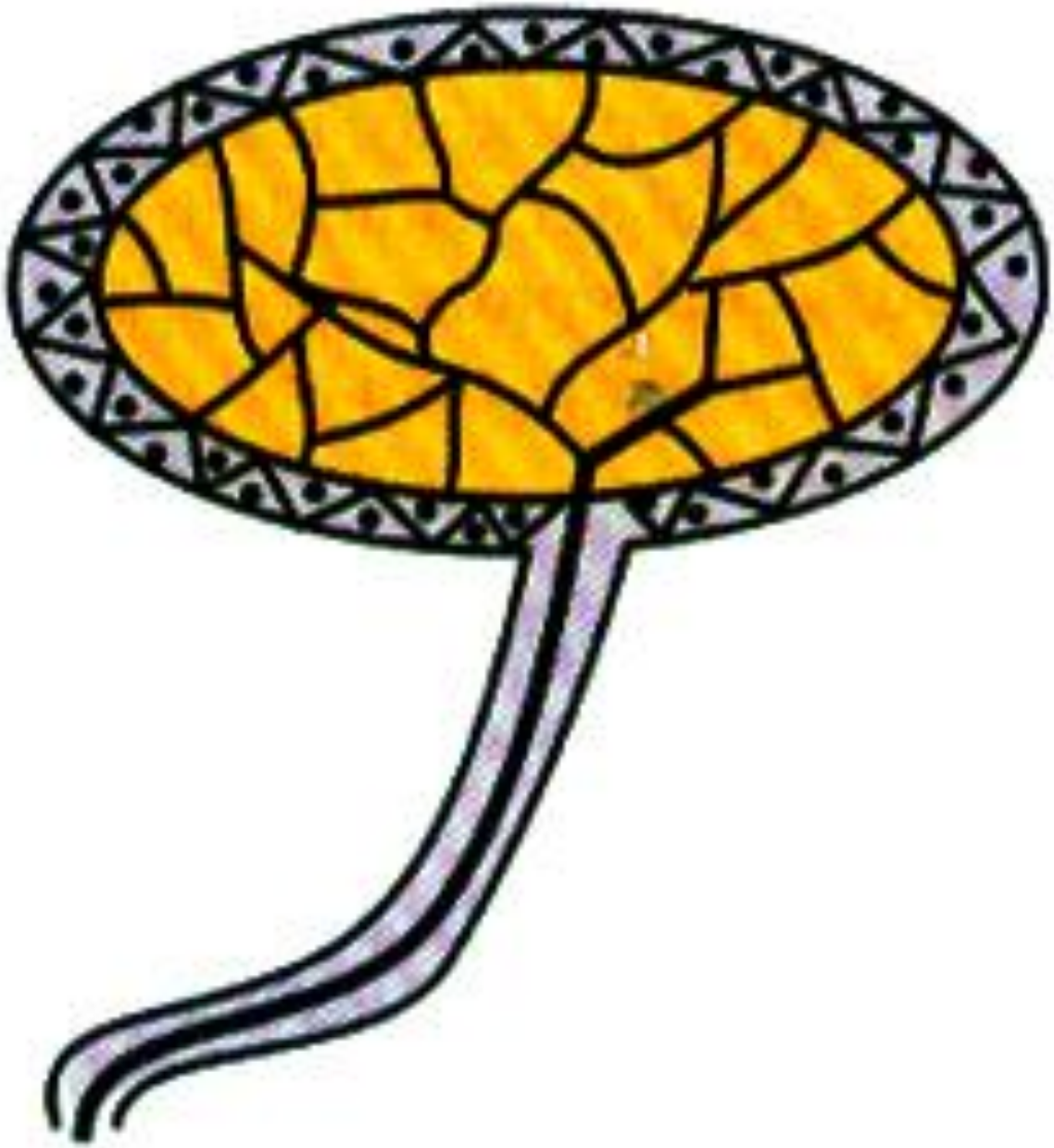


Figure 101.5 Krause end-bulb.

Properties of receptors

- Adequate stimulus
- Generator Potential
- Adaptation
- Labelled line principle
- Law of projection
- Weber- Fechner law

- **Adequate Stimulus**

  The particular form of energy to which a receptor is most sensitive is called adequate stimulus.

  Ex; Adequate stimulus for Rods&Cones is light

- **Generator Potential or Receptor Potential**

It is a nonpropagated depolarizing potential recorded in a receptor when a stimulus is applied.

- Best studied in Pacinian Corpuscle in the skin because of its large size,easily accessible .

- It is a touch receptor or mechanoreceptor

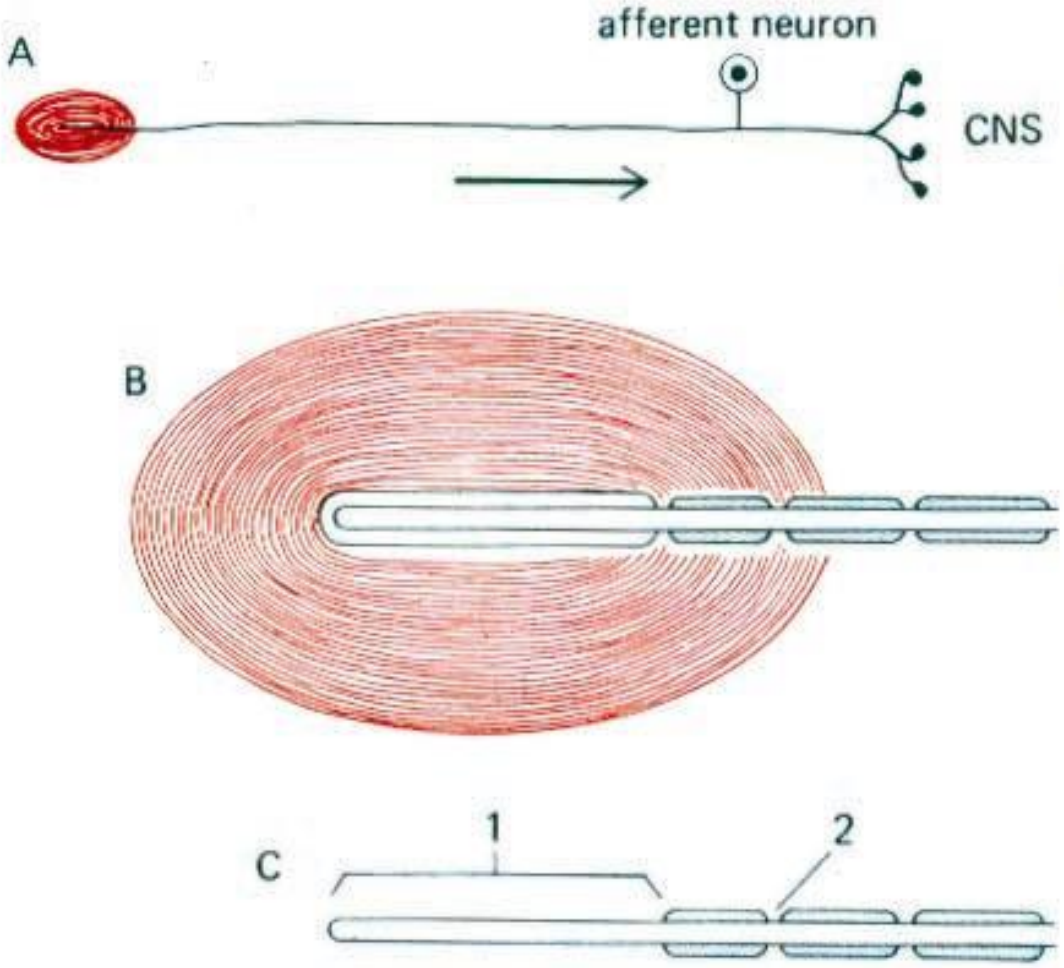


FIGURE 8-48. (A) An afferent neuron with a mechanoreceptor (pacinian corpuscle) ending. (B) A pacinian corpuscle showing the nerve ending modified by cellular structures. (C) The naked nerve ending of the same mechanoreceptor. The receptor potential arises at the nerve ending (1), and the action potential arises at the first node of the myelin sheath (2).

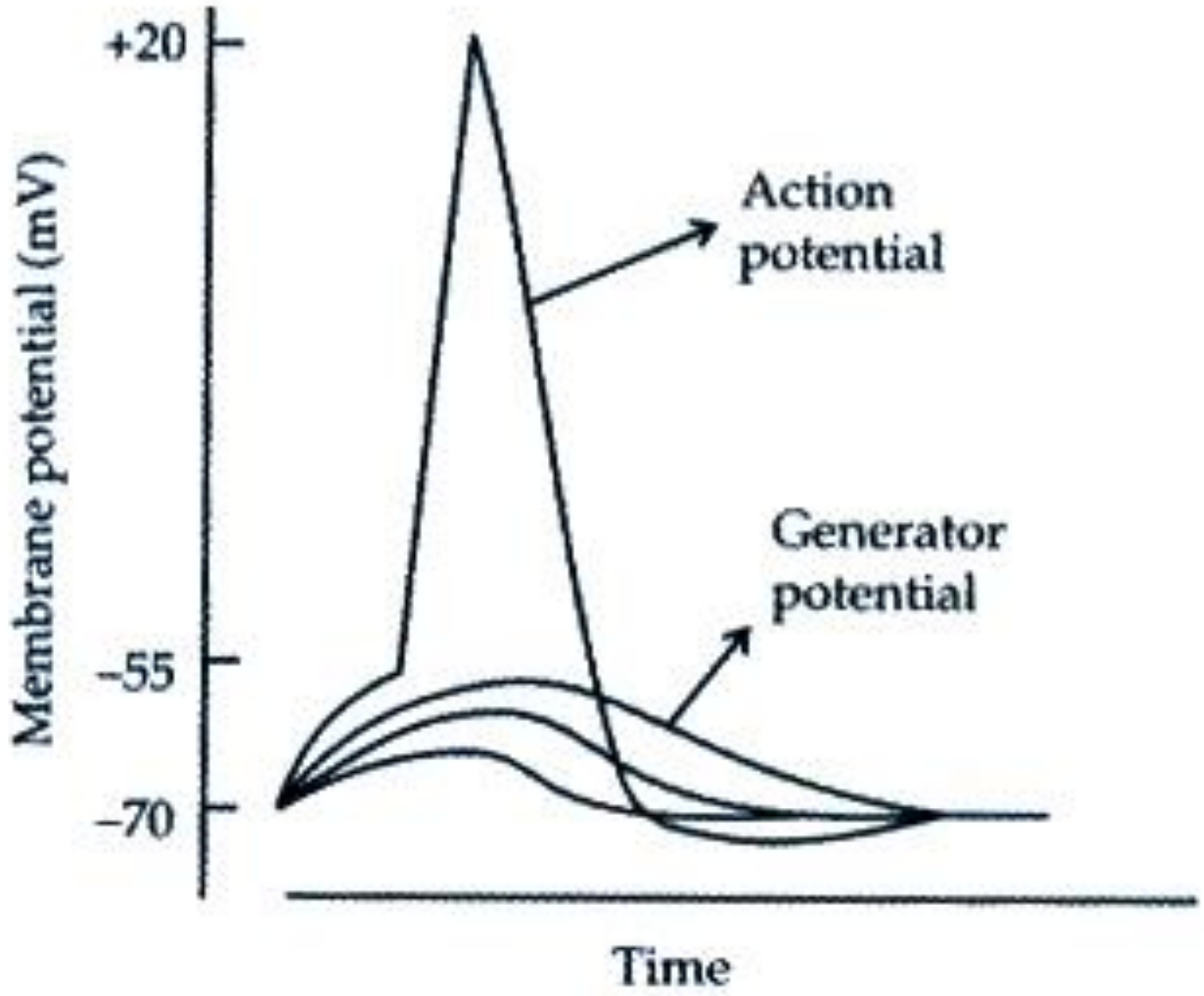


Figure 101.6 Receptor (generator) potential and action potential, formed in receptors.

- **Mechanism**

Pressure on the Pacinian corpuscle

↓

Mechanical Distortion of lamellas

↓

Opens Stretch Sensitve Na+ Ion channels in Unmyelinated nerve terminal

↓

Influx of Na+

↓

Receptor potential generated

- Features

  - Local or Graded Potential
  - No refractory period
  - Can be summated
  - Does not obey All or None law
  - Decremental conduction
  - Duration is more (5-10ms)

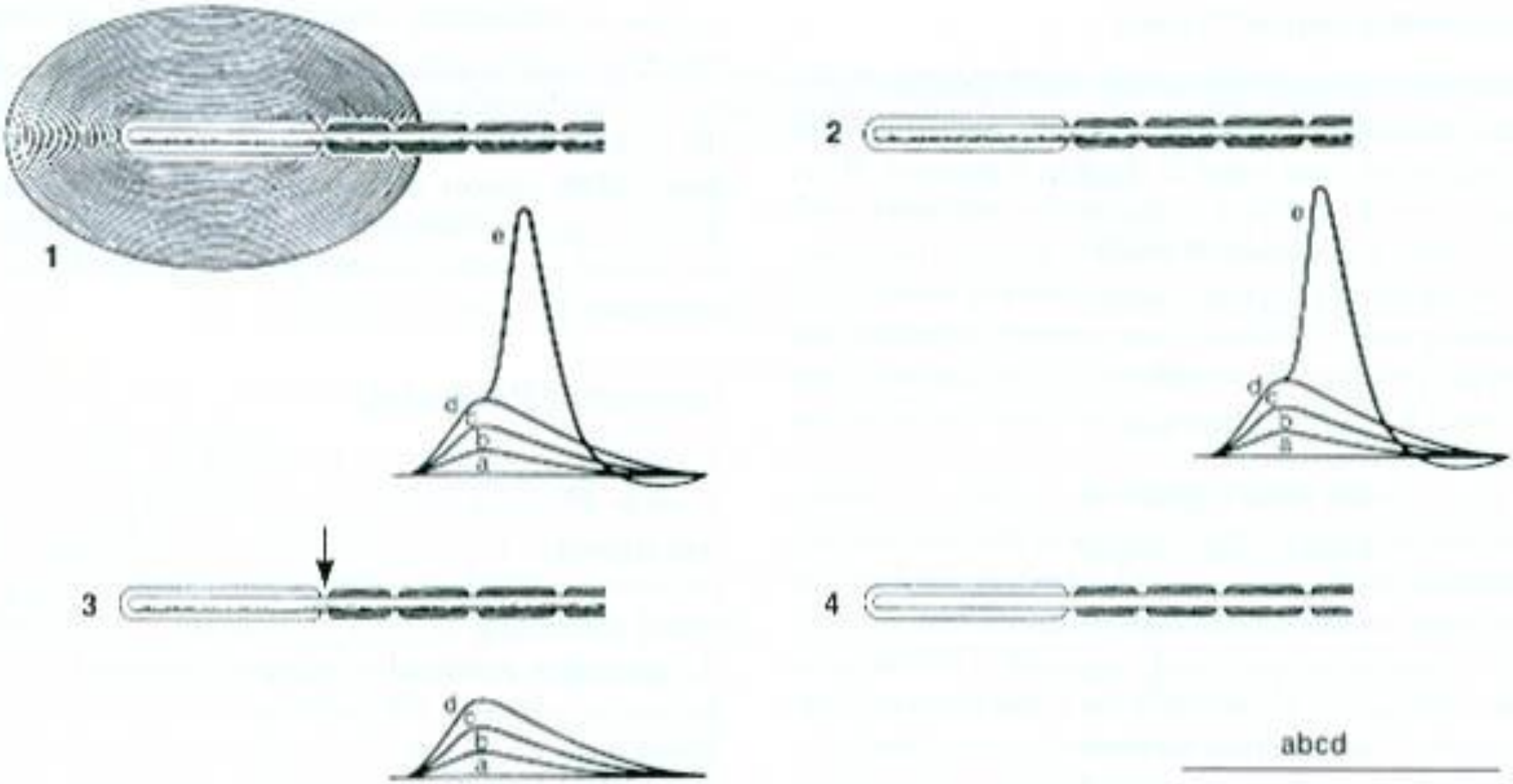


Figure 5–1. Demonstration that the generator potential in a pacinian corpuscle originates in the nonmyelinated nerve terminal. **1:** The electrical responses to a pressure of 1× (record a), 2× (b), 3× (c), and 4× (d) were recorded. The strongest stimulus produced an action potential in the sensory nerve (e). **2:** Similar responses persisted after removal of the connective tissue capsule, except that the responses were more prolonged because of partial loss of adaptation. **3:** The generator responses persisted but the action potential was absent when the first node of Ranvier was blocked by pressure or with narcotics (arrow). **4:** All responses disappeared when the sensory nerve was cut and allowed to degenerate before the experiment.

- **Adaptation**

  When a stimulus of constant strength is applied continuously to a receptor, the frequency of Acton potentials in its sensory nerve declines over a period of time.

  - Fast Adapting or Phasic receptors

    Ex; Pacinian corpuscle

    Advantageous

  - Slow Adapting or Tonic receptors

    Ex; Muscle spindle, Nociceptors

    Survival value

- **“Coding” of sensory information**

  - Stimulus Modality
  - Stimulus Localization
  - Stimulus Intensity

- **Labelled Line Principle or**

Muller's doctrine of specific nerve energies

The specific sensory pathways from the receptor to the cerebral cortex are discrete and are called **labelled lines**.

"No matter how or where a particular sensory pathway is stimulated along its course to the cerebral cortex, the sensation evoked is that for which the receptor is specialized".

- Different sensory modalities are encoded by this mechanism

- **Law of Projection**

"No matter where a particular sensory pathway is stimulated along its course to cerebral cortex, the sensation produced is referred to the location of receptors".

Stimulus localization is encoded by this mechanism

Ex; Phantom Limb

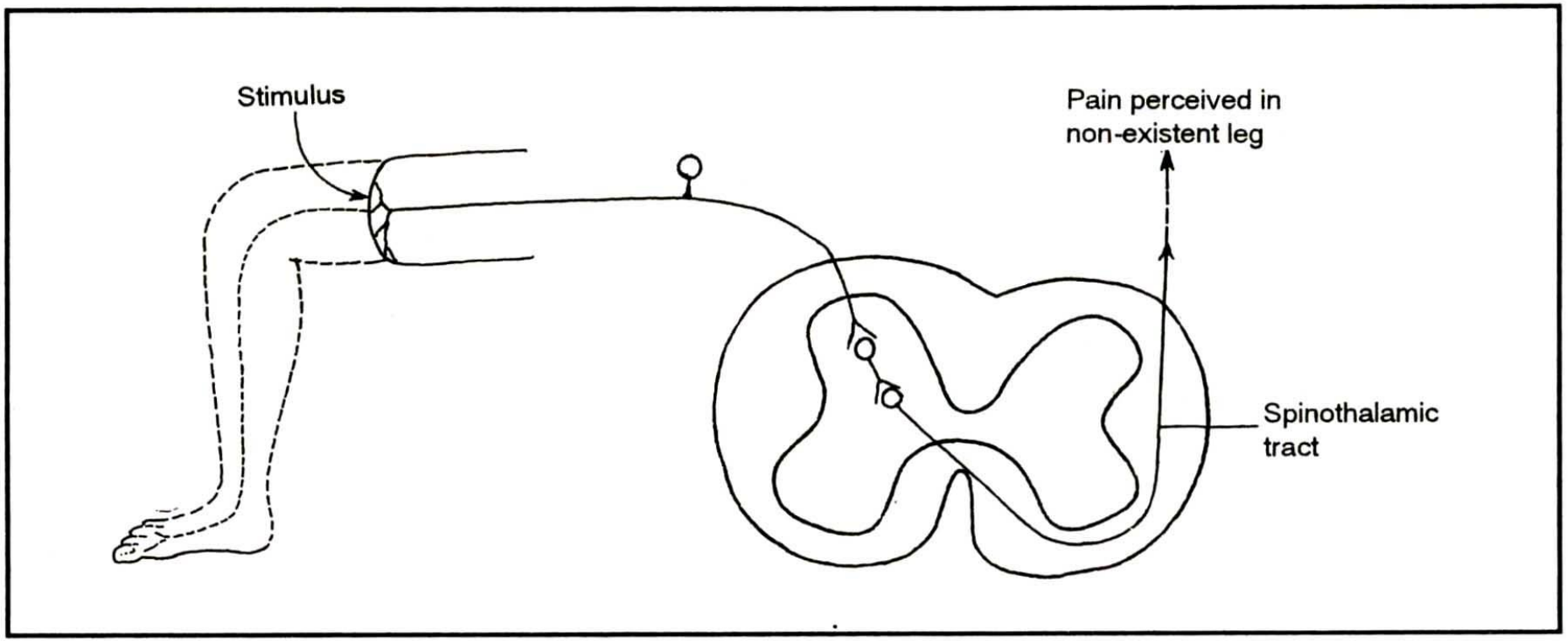


Fig. 12.7.9: *Explanation for the phenomenon of phantom pain. The stump may actually become hypersensitive because the cut nerve fibre may proliferate, forming a neuroma.*

- **Stimulus Intensity**

Intensity is discriminated by

- Frequency of Action Potentials
- Recruitment of Sensory Units

-Receptor associated with non neuronal cells that surround it – **Sense organ**

- A single sensory axon and all its peripheral branches – **Sensory Unit**
- The area from which a stimulus produces a response in the sensory unit – **Receptive field**

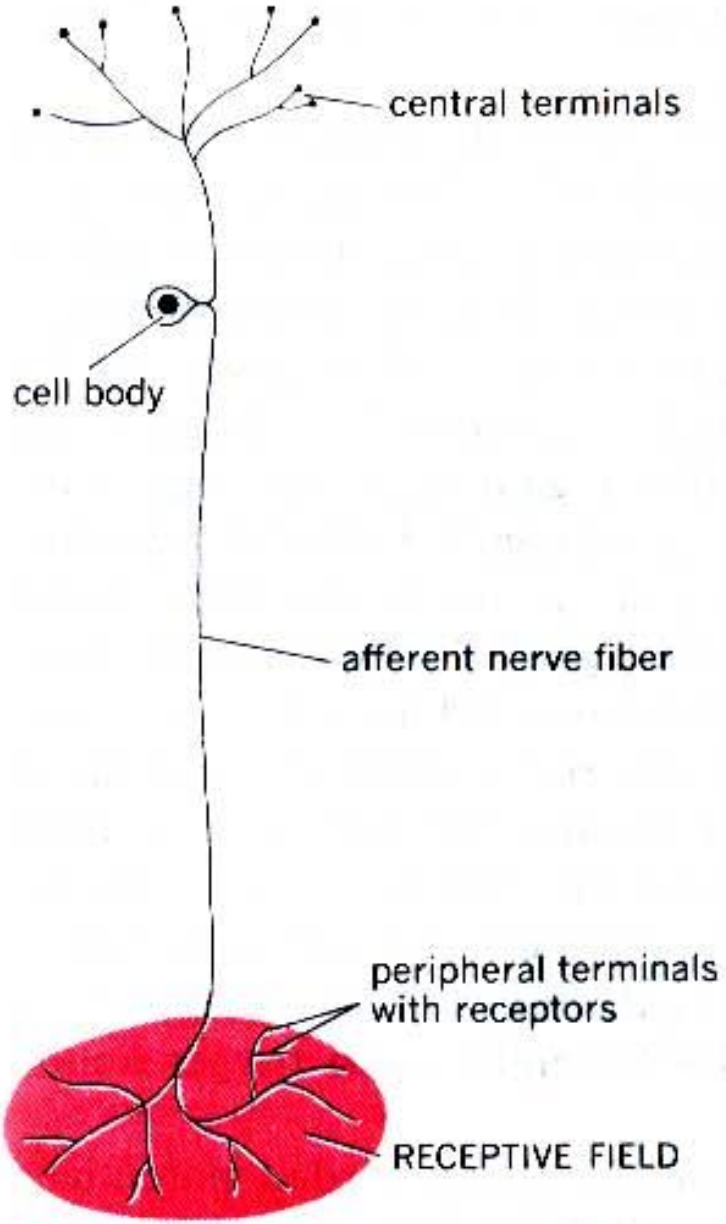


FIGURE 8-52. Sensory unit and receptive field.

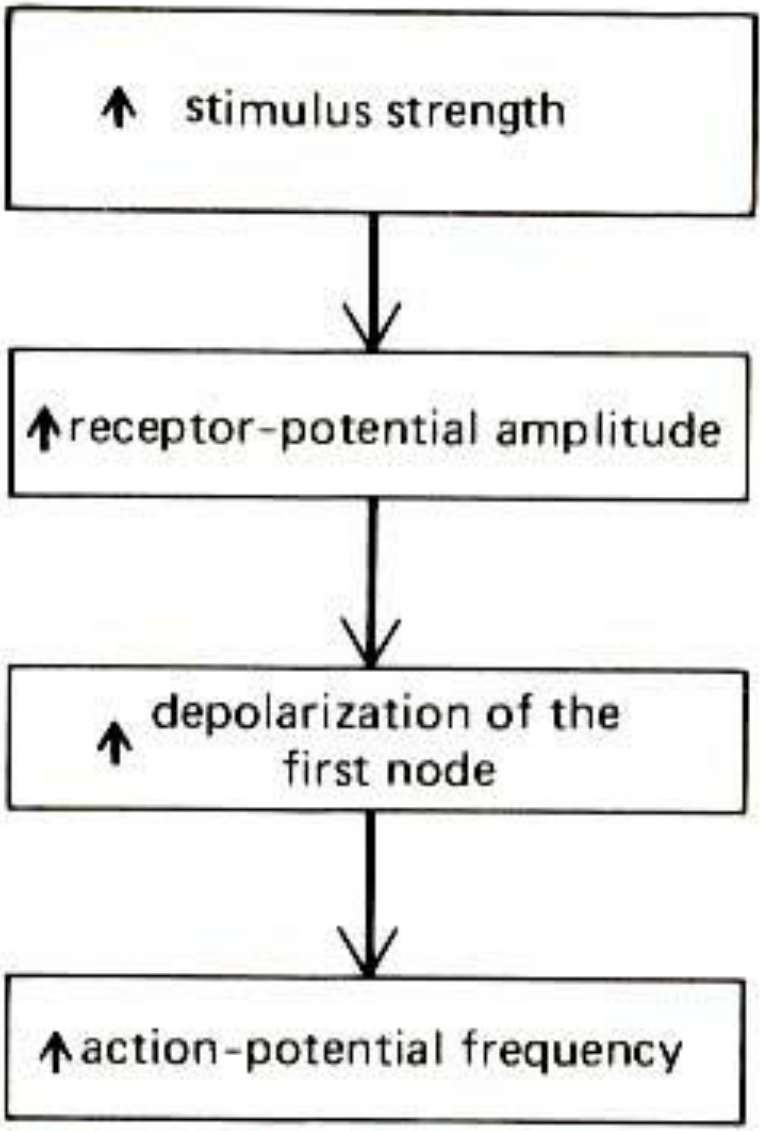


FIGURE 8-49. The effect of stimulus strength on action-potential frequency.

- **Weber-Fechner Law**

"The magnitude of sensation felt is proportionate to the log of intensity of stimulus".

$$R = KS^A$$

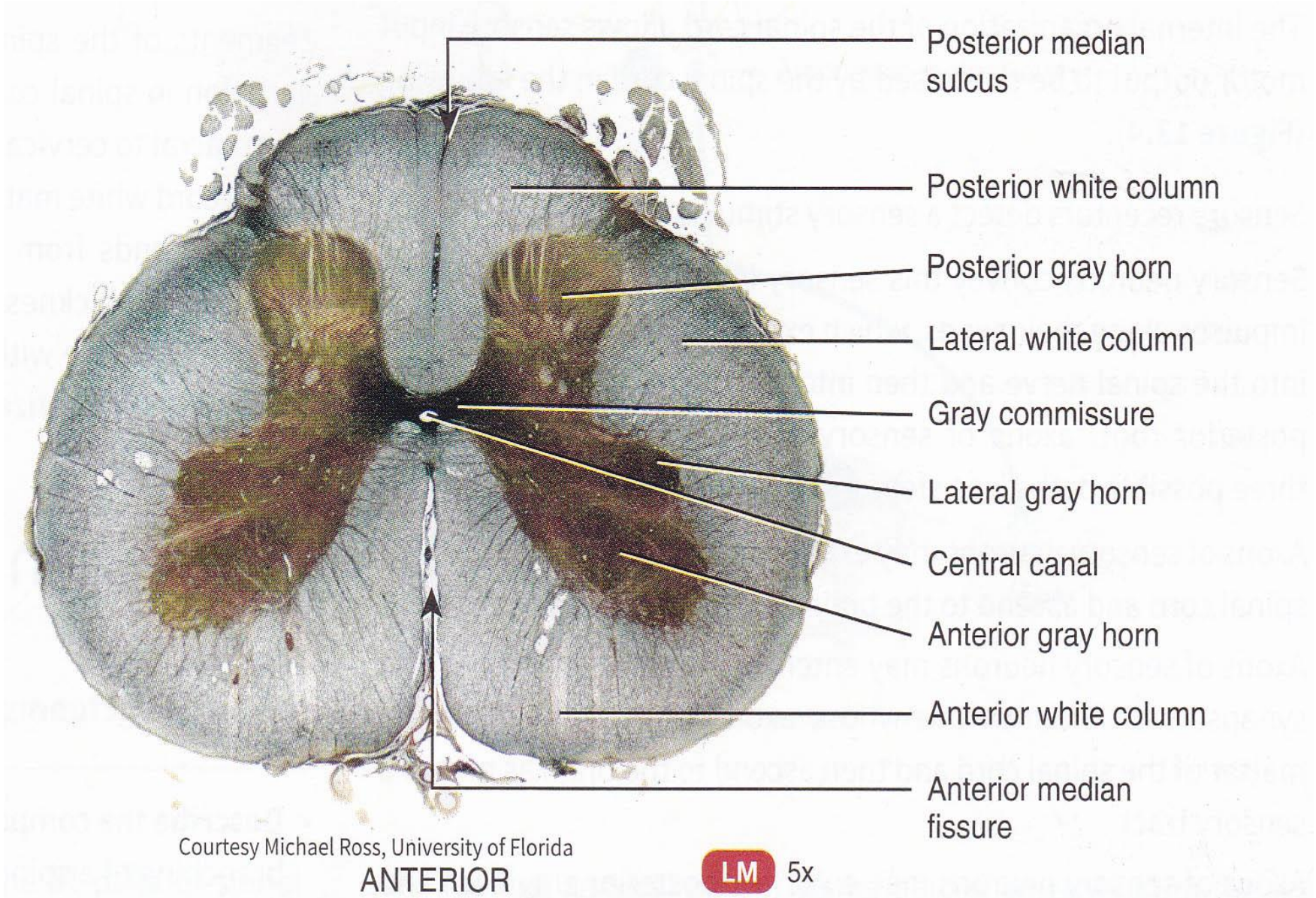
R - sensation felt

S - Intensity of stimulus

K & A – Constants

intensity of stimulus	-	Magnitude of sensation
10	-	1
100	**-**	**2**
1000	**-**	**3**

- safety mechanism



(b) Transverse section of lumbar spinal cord

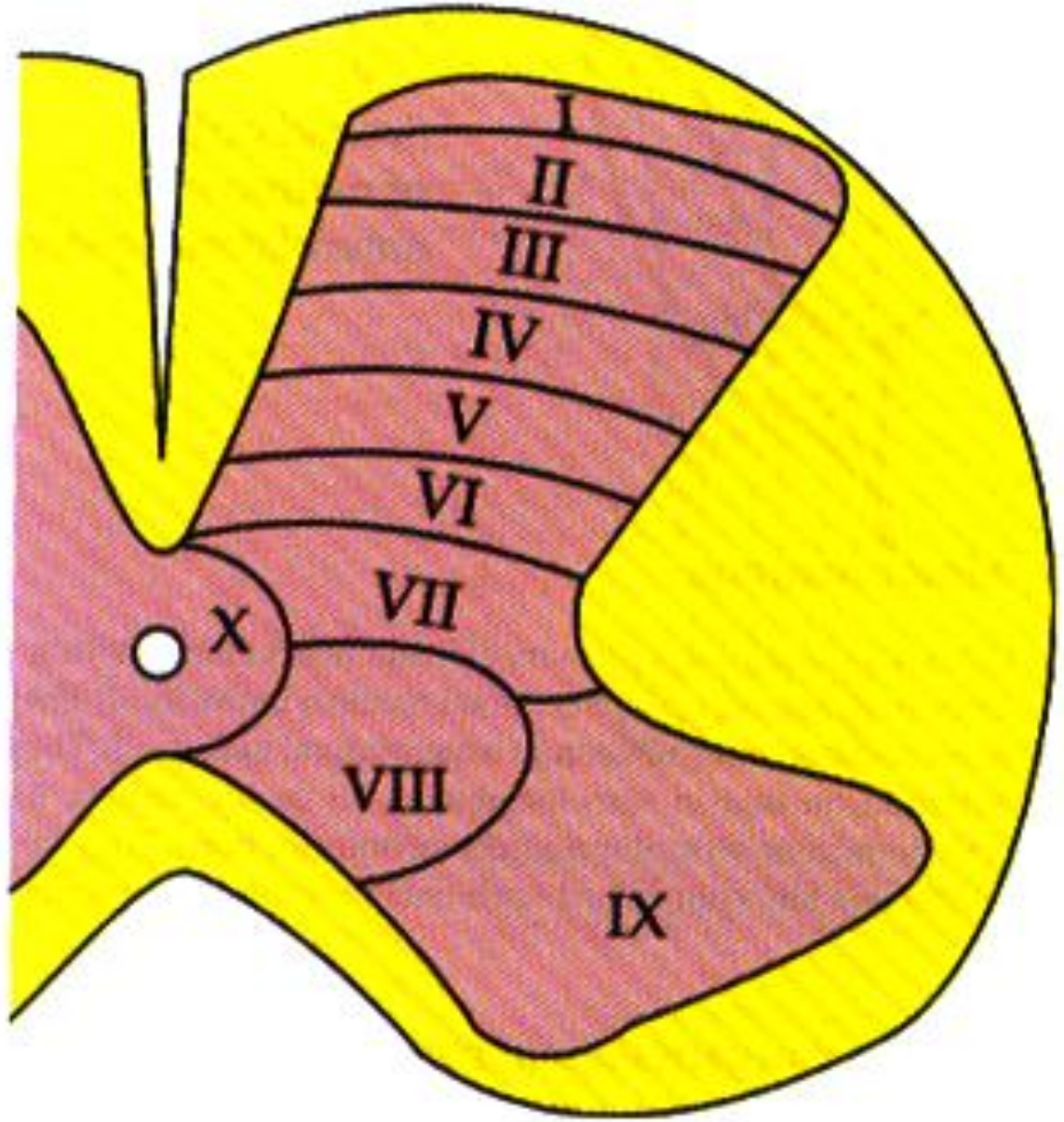


Figure 102.4 Spinal cord laminae. Note, laminae I to VI are dorsal horn laminae.

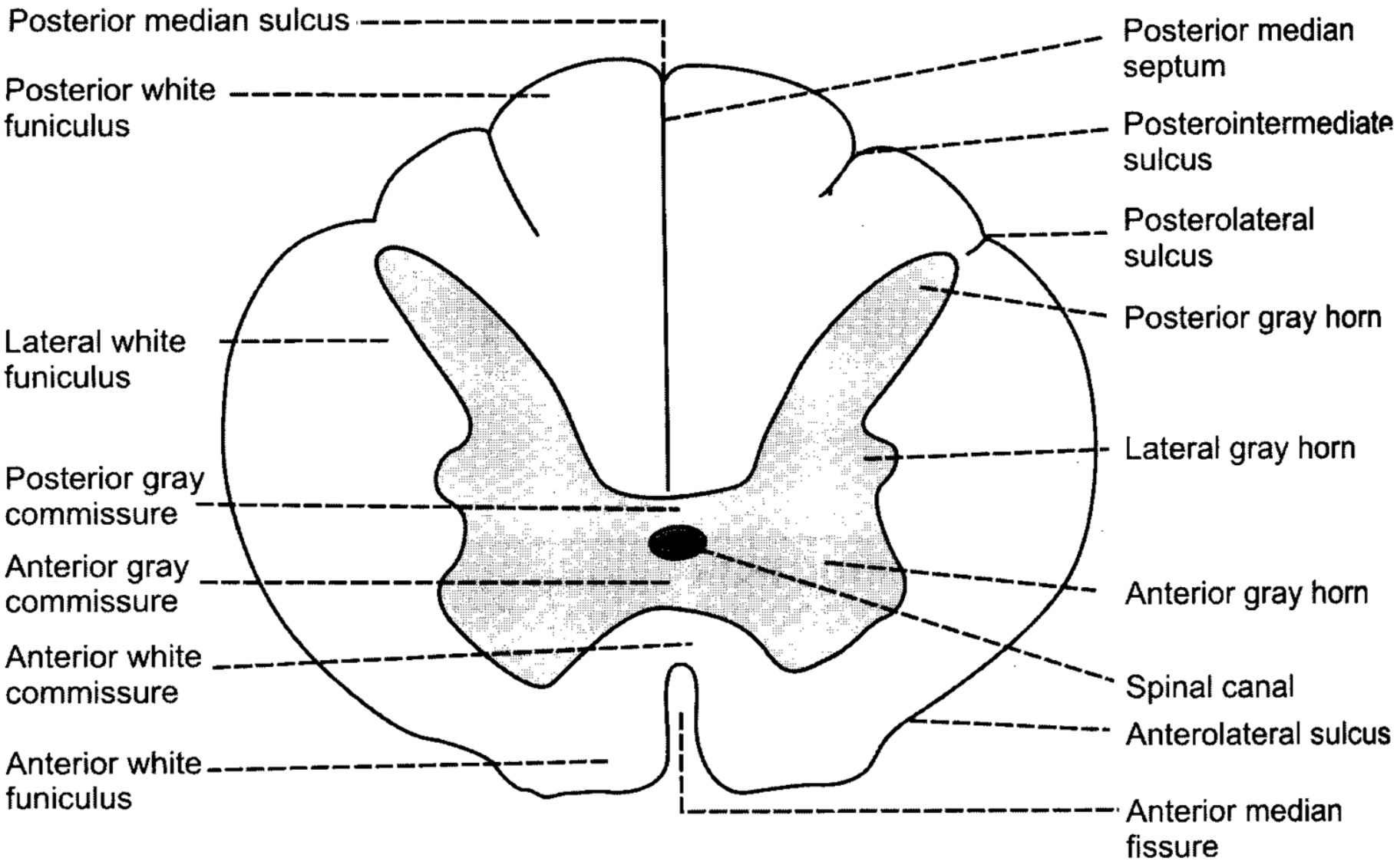


FIGURE 143-1: Section of spinal cord—thoracic segment

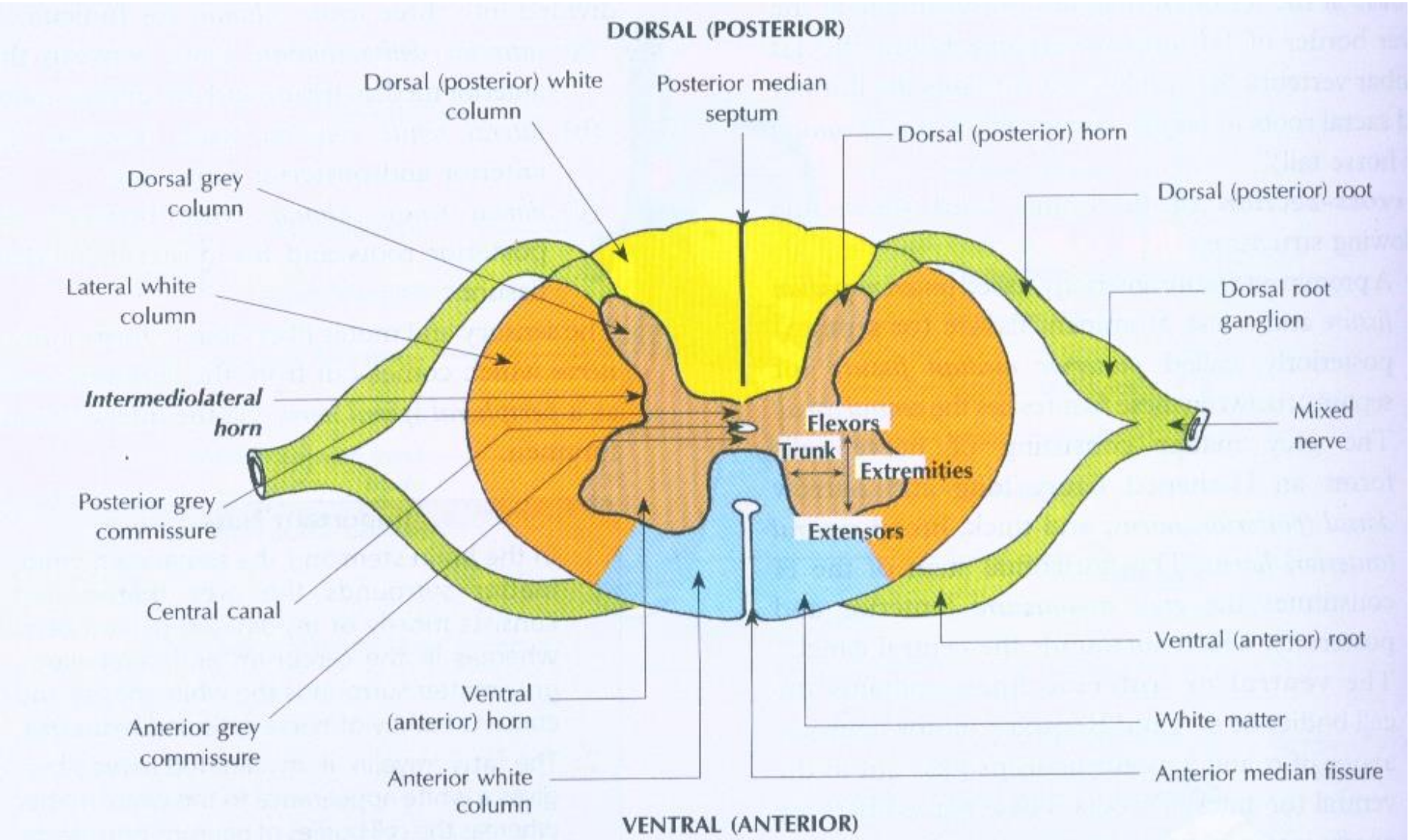
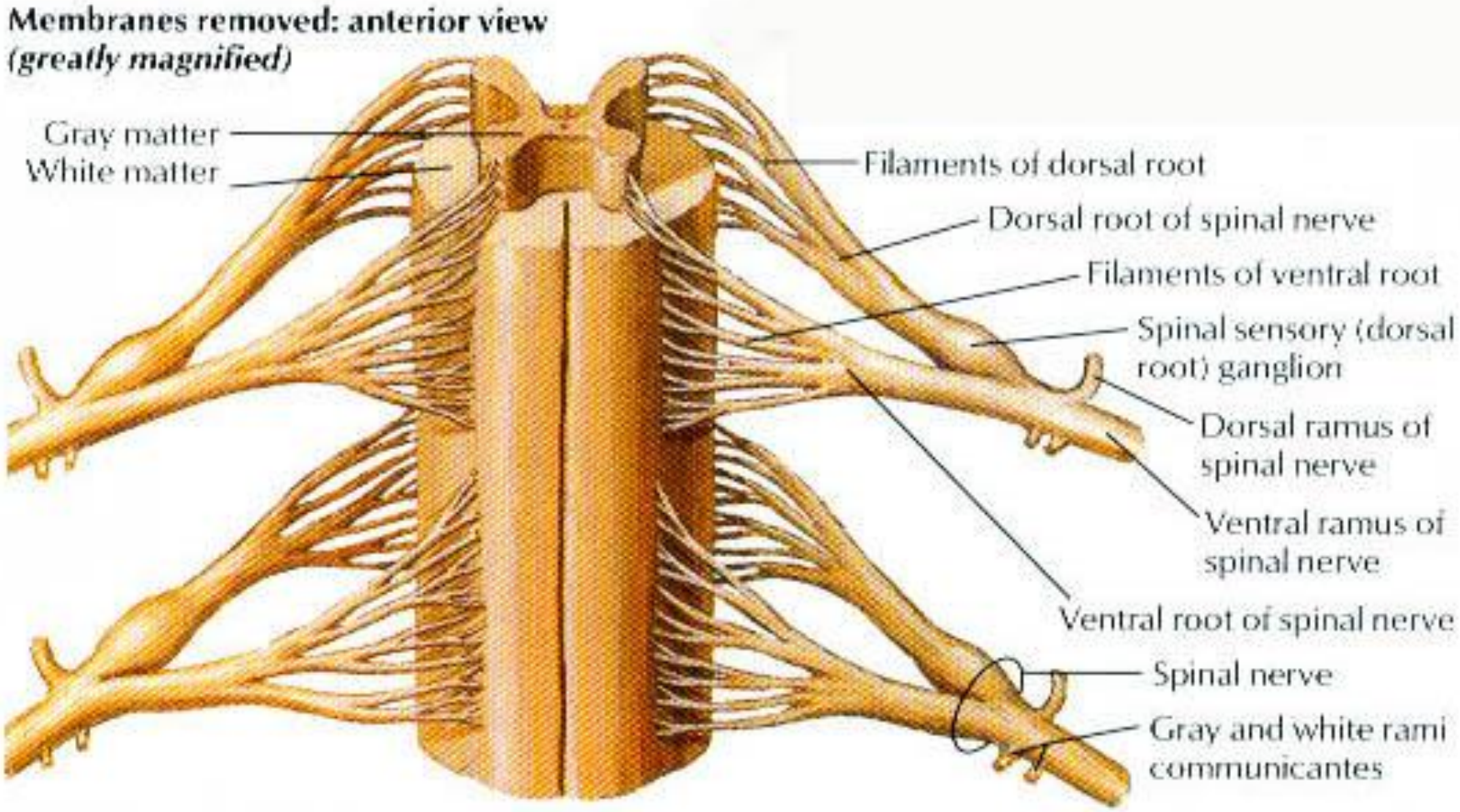
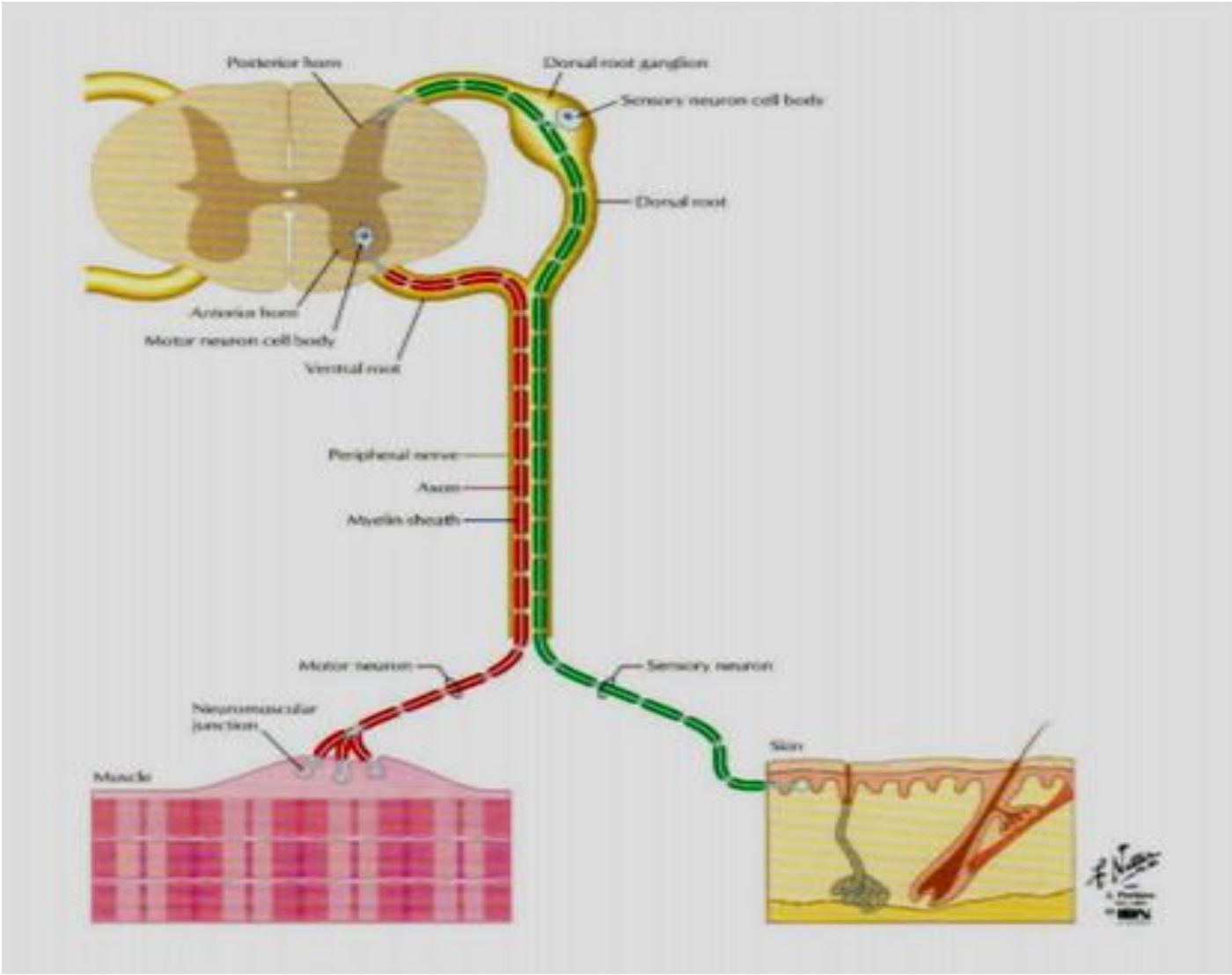
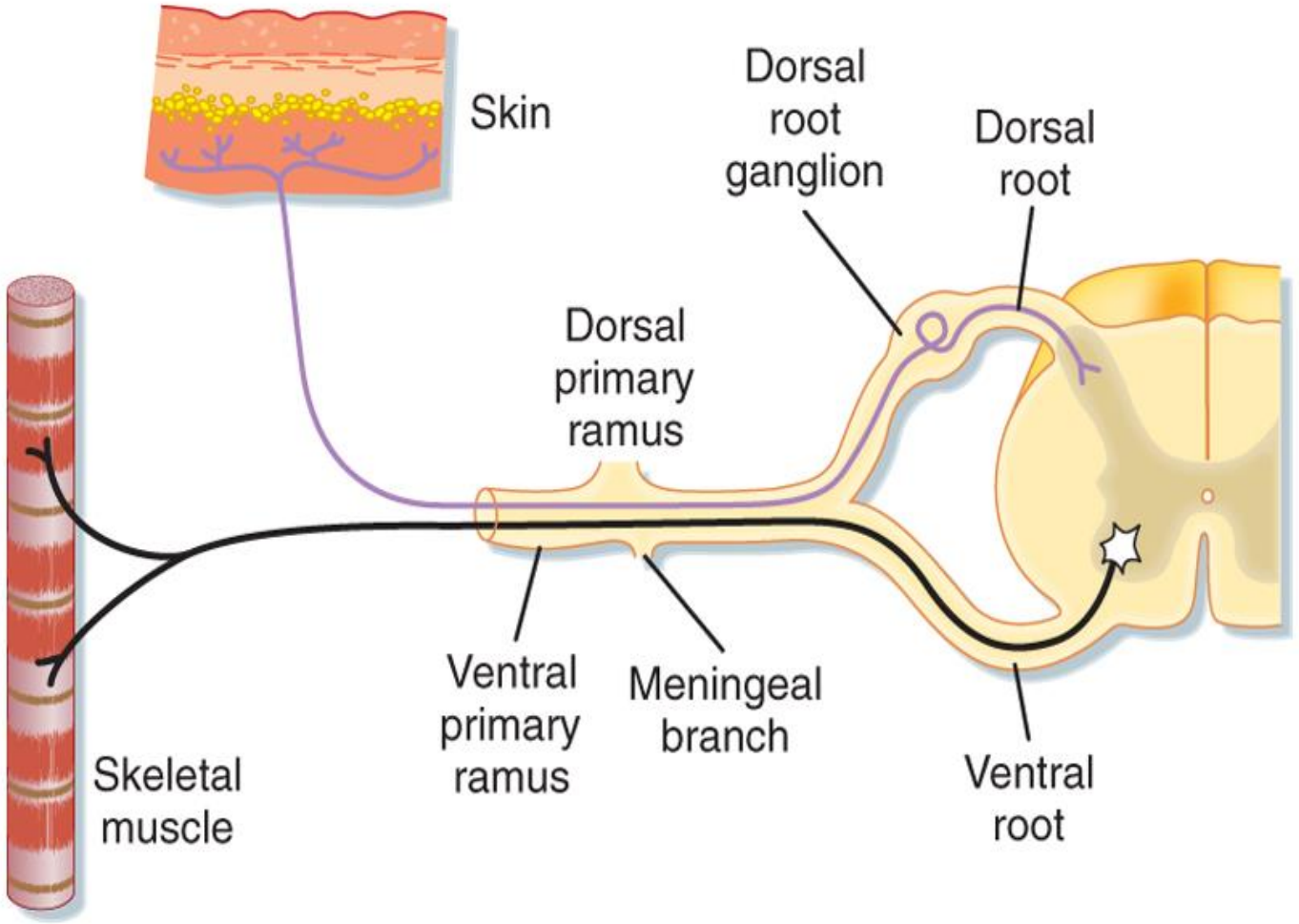


Fig. 11.87.3 Cross-section of the spinal cord



SPINAL MEMBRANES AND NERVE ROOTS

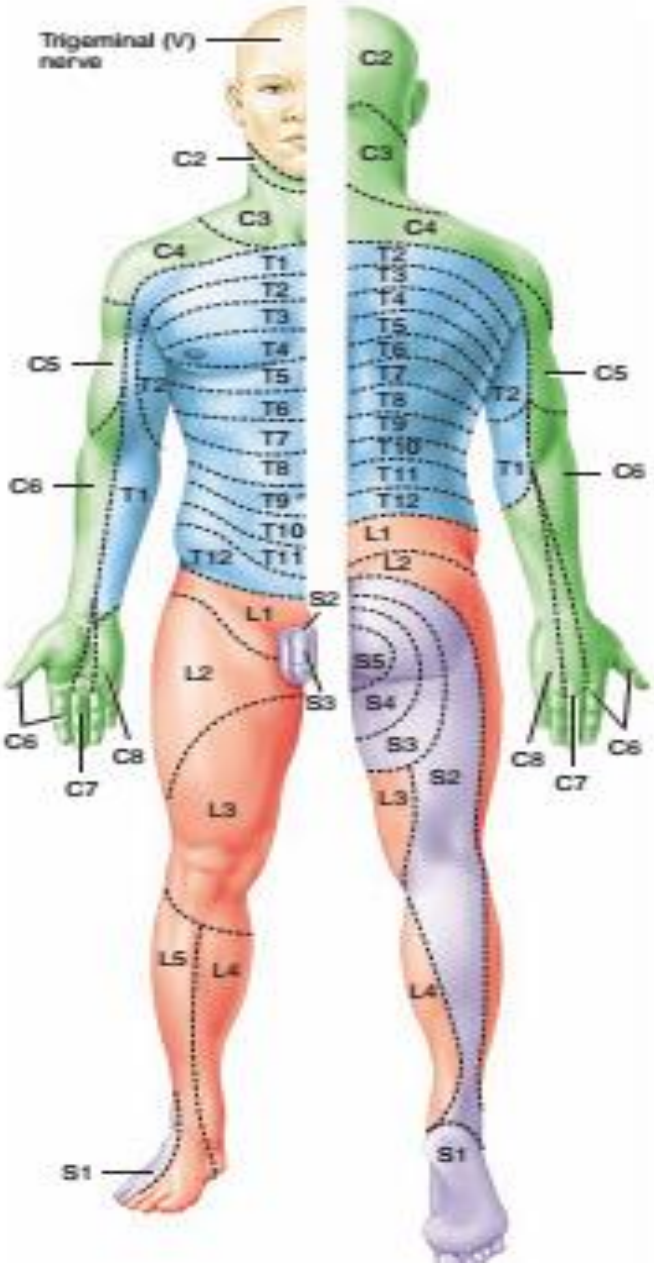

Posterior horn
Dorsal root ganglion
Sensory neuron cell body
Dorsal root
Anterior horn
Motor neuron cell body
Ventral root
Peripheral nerve
Axon
Myelin sheath
Motor neuron
Sensory neuron
Neuromuscular junction
Muscle
Skin


Skin
Dorsal root ganglion
Dorsal root
Dorsal primary ramus
Ventral primary ramus
Meningeal branch
Skeletal muscle
Ventral root

- DERMATOME :

“Area of the skin innervated by single dorsal root”

A dermatome is an area of skin that provides sensory input to the CNS via the posterior roots of one pair of spinal nerves or via the trigeminal (V) nerve.



Afferent fibers mediating cutaneous sensation

- Large myelinated **Aα** and **Aβ** fibers
  - mechanceptors
- Small myelinated **Aδ** fibers
  - Nociceptrs, cold receptors, some mechanoceptors
- Small unmyelinated **C** fibers

  Nociceptors, thermoreceptors, mechanoceptors

- **First order neurons**

  Sensory receptors → Brain stem or spinal cord

- **Second order neurons**

  Brain stem or spinal cord → Thalamus

  Decussation

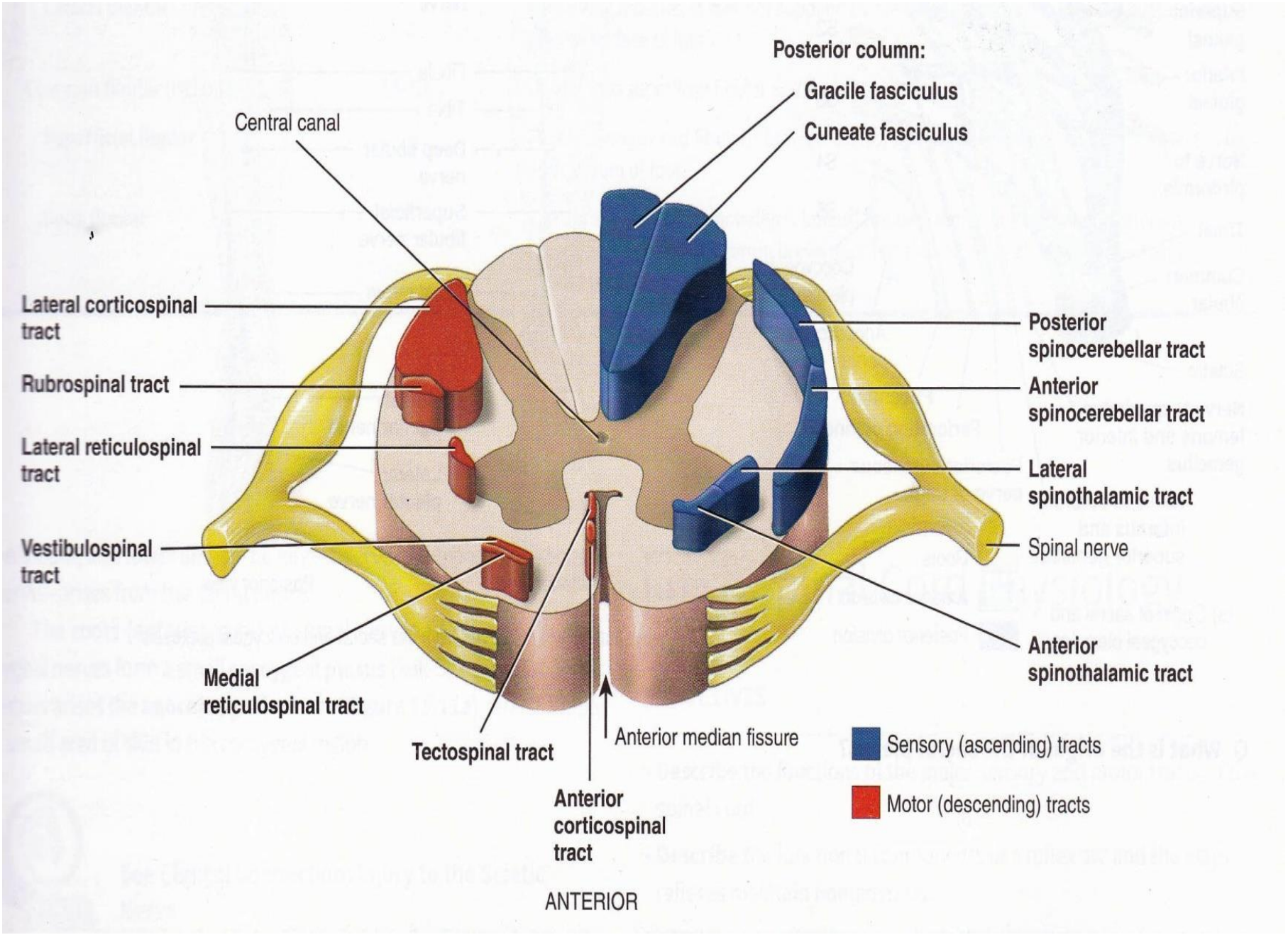
- **Third order neurons**

  Thalamus → Primary somatosensory area

Sensory tracts or Ascending tracts

- **Dorsal column medial lemniscal system**
- **Anterolateral spinothalamic tract**

- Dorsal & Ventral spinocerebellar tracts
- Spino - olivary tract
- Spinotectal tract
- Spinoreticular tract


Posterior column:
Gracile fasciculus
Cuneate fasciculus
Central canal
Lateral corticospinal tract
Rubrospinal tract
Lateral reticulospinal tract
Vestibulospinal tract
Posterior spinocerebellar tract
Anterior spinocerebellar tract
Lateral spinothalamic tract
Spinal nerve
Anterior spinothalamic tract
Medial reticulospinal tract
Tectospinal tract
Anterior median fissure
Anterior corticospinal tract
Sensory (ascending) tracts
Motor (descending) tracts
ANTERIOR

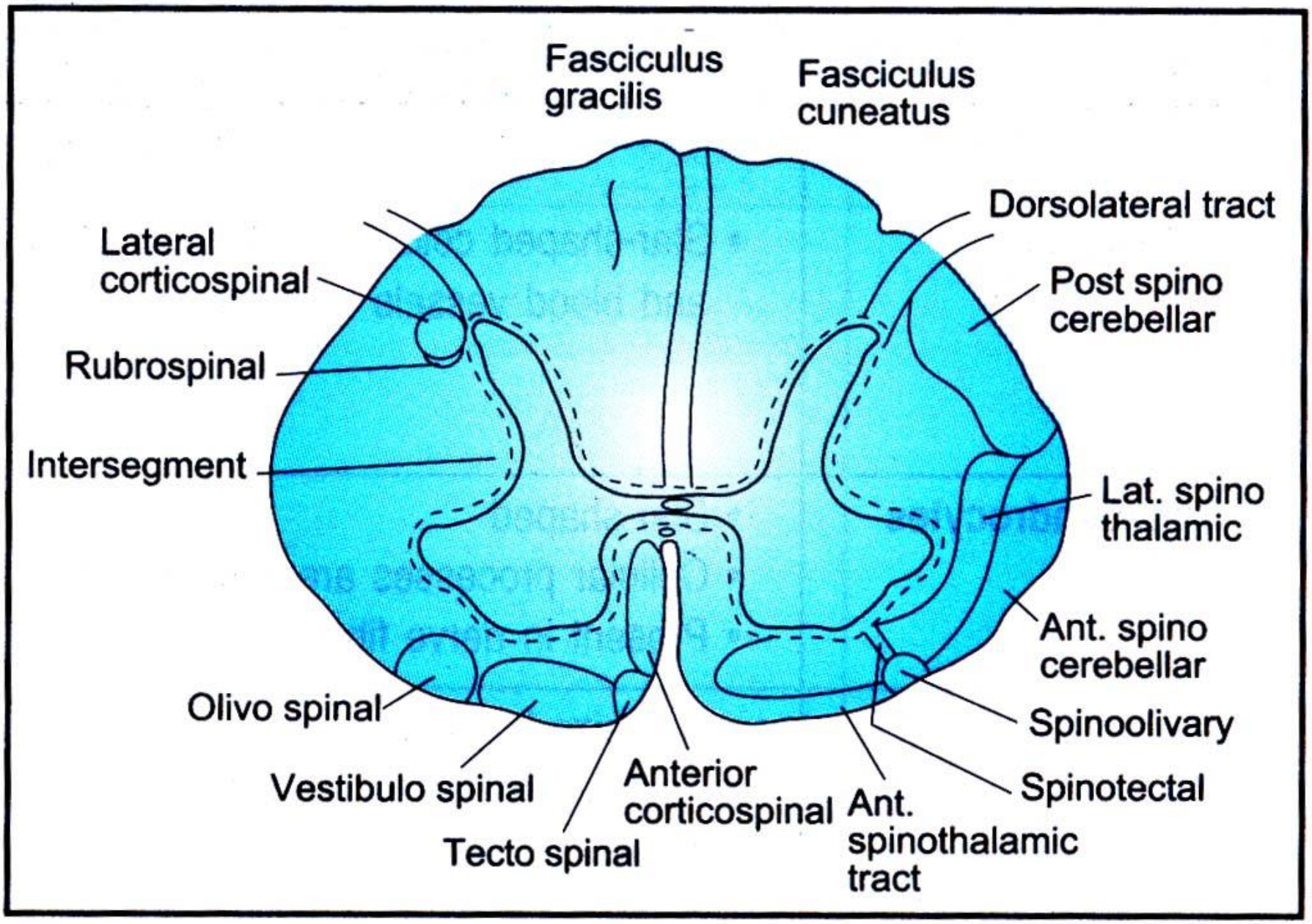


Fig. 11.7 : General arrangement of tracts

- **Dorsal column medial lemniscal system**

-fasciculus gracillie – tract of Goll

-fasciculus cuneatus – tract of Burdach

-Posterior columns or dorsal columns

carries : Fine touch

Tactile localization

Tactile or two point discrimination

Proprioception

Vibration sense

Stereognosis

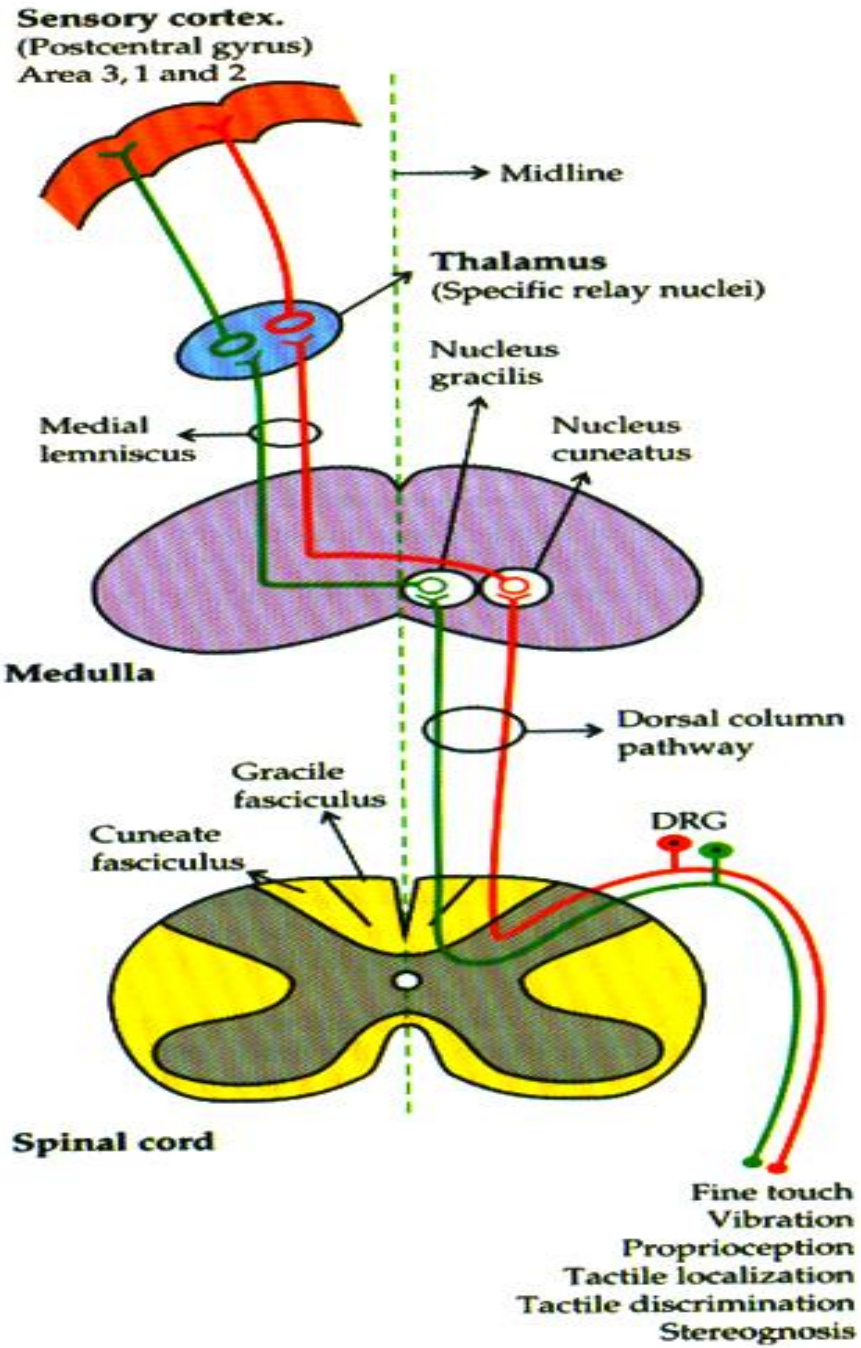


Figure 103.1 Dorsal column pathways.

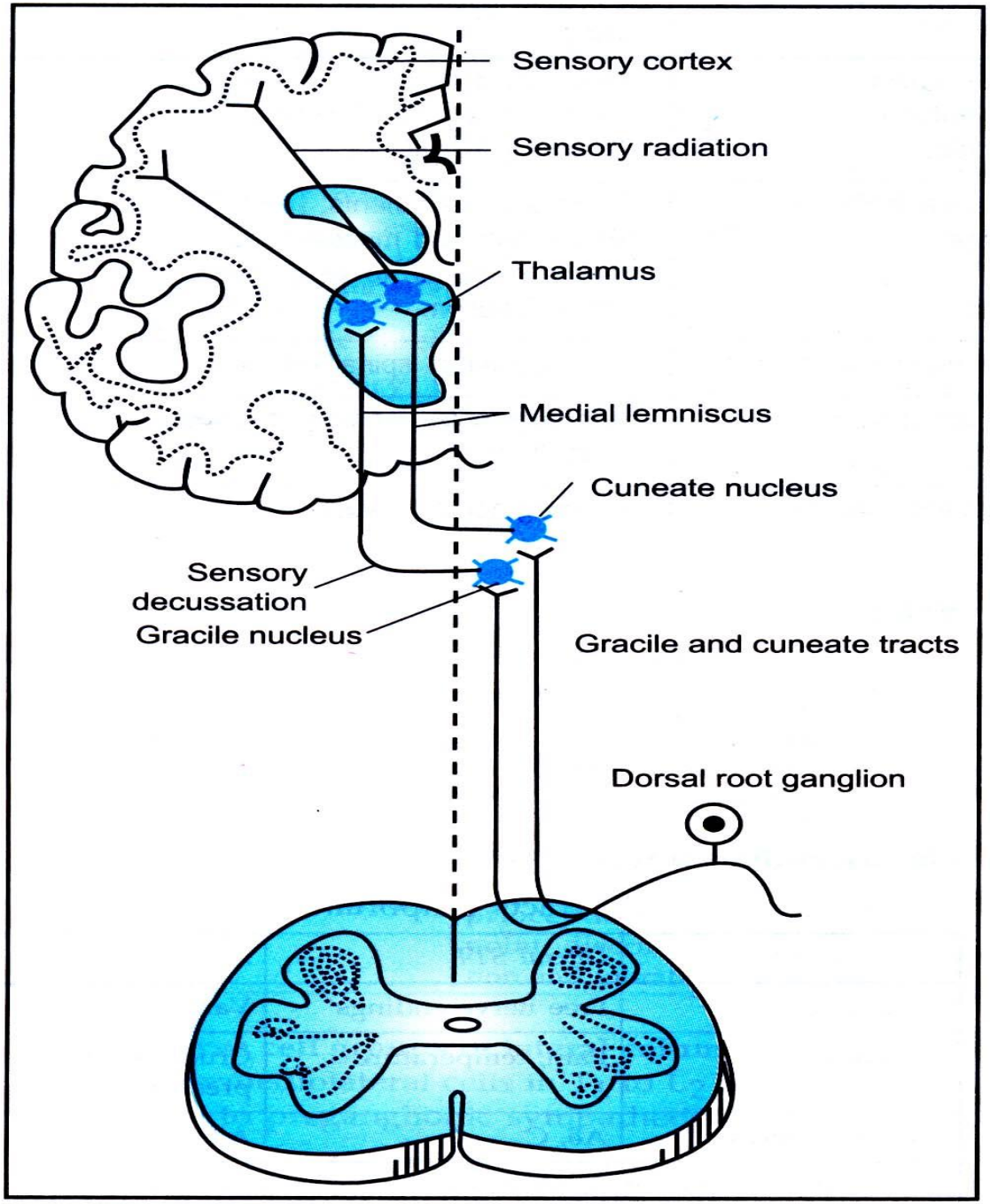
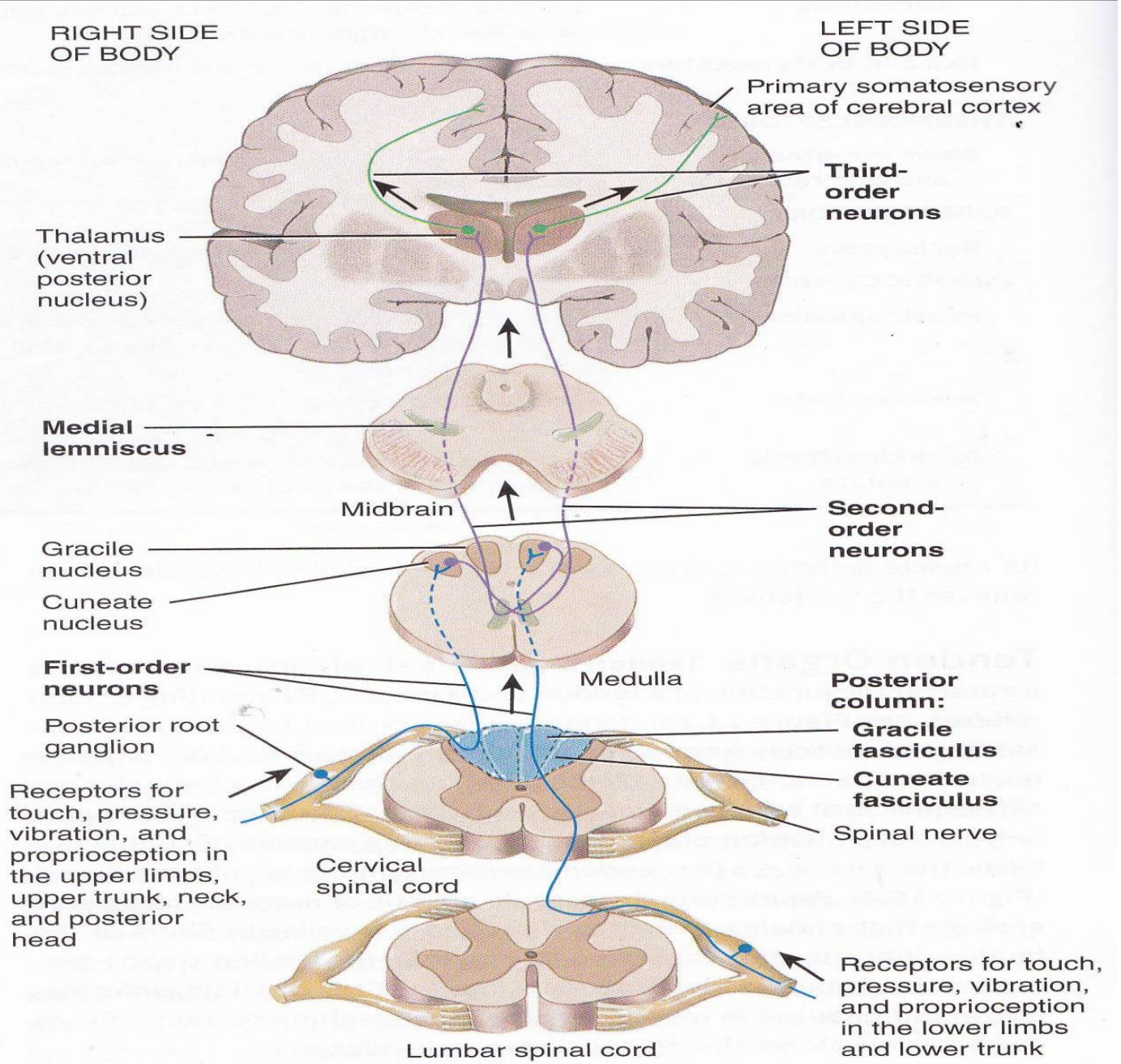


Fig. 11.8 : Scheme of posterior column pathway



Posterior column–medial lemniscus pathway

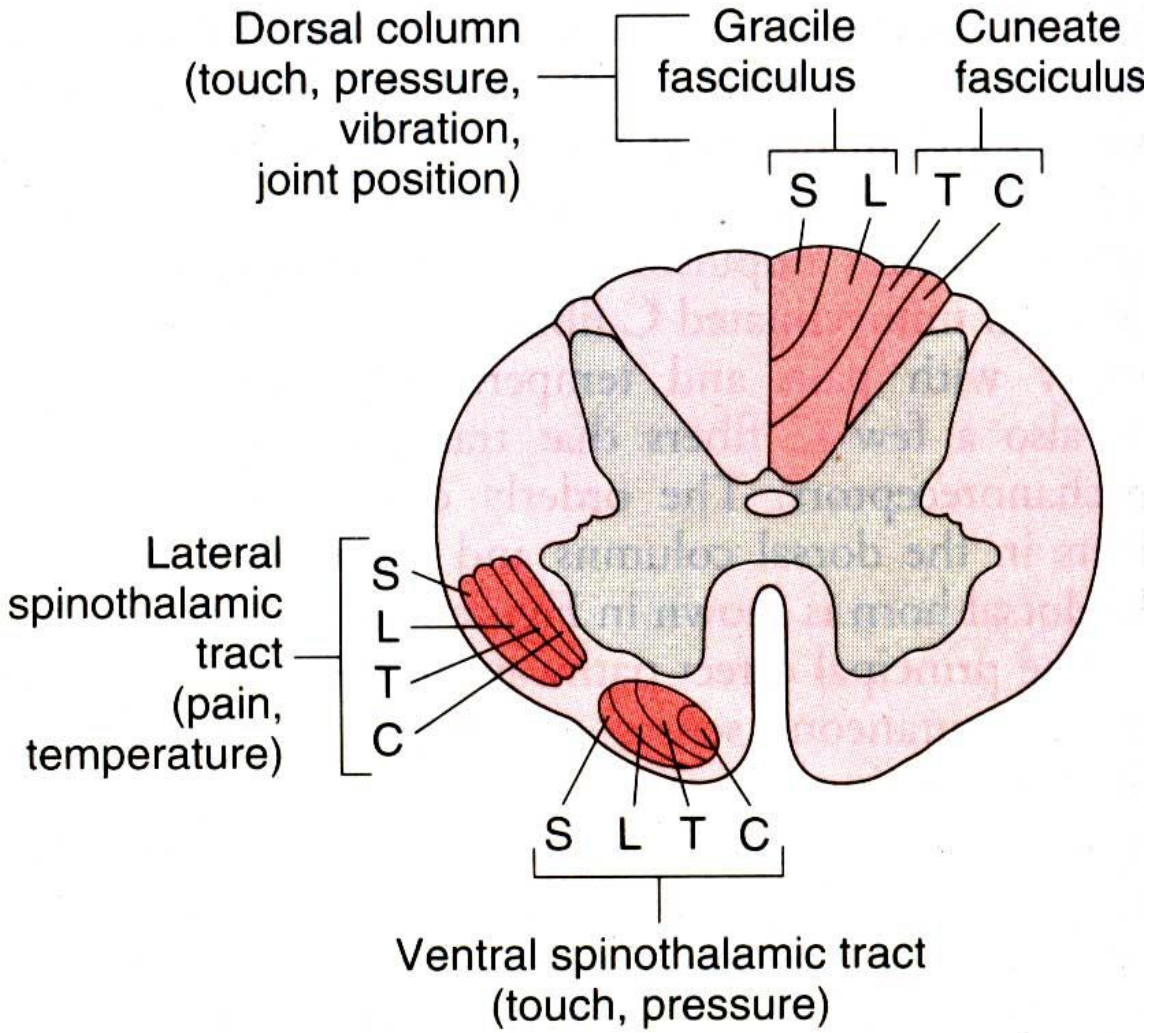


Figure 7–3. Cross section of spinal cord, showing location of ascending sensory pathways. Note that eacl is laminated. S, sacral; L, lumbar; T, thoracic; C, cervical.

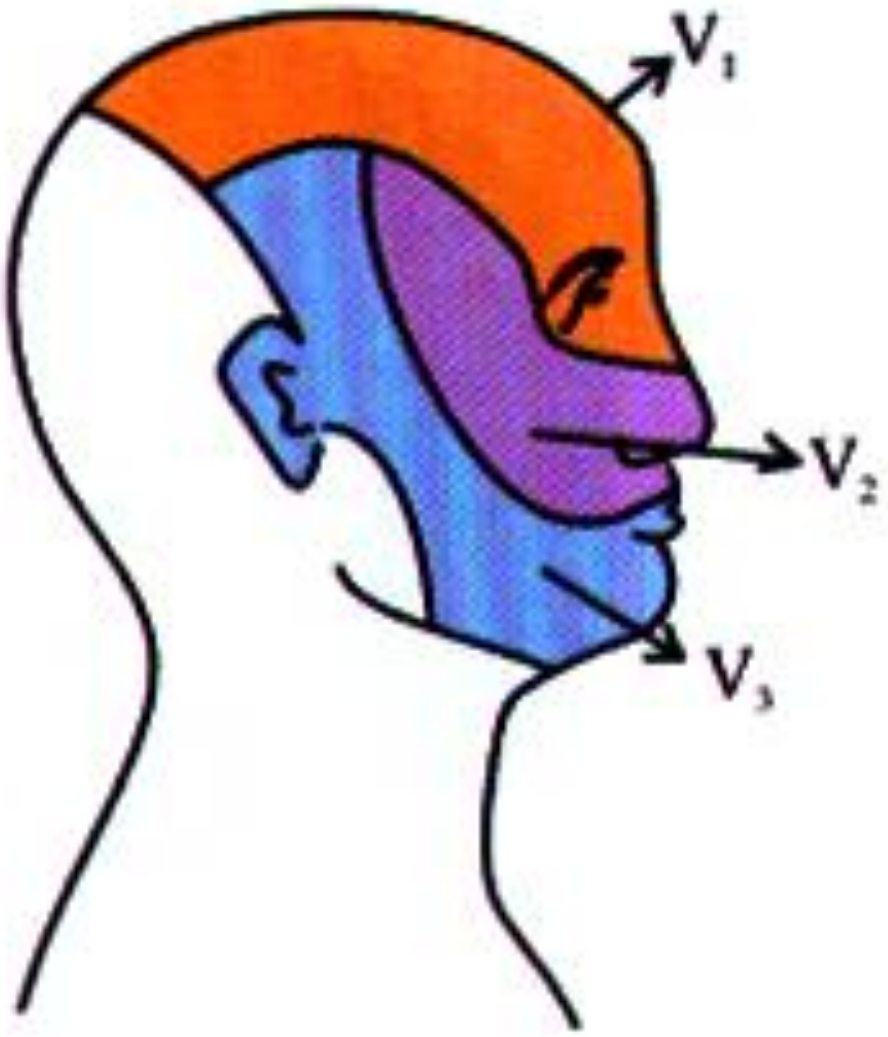
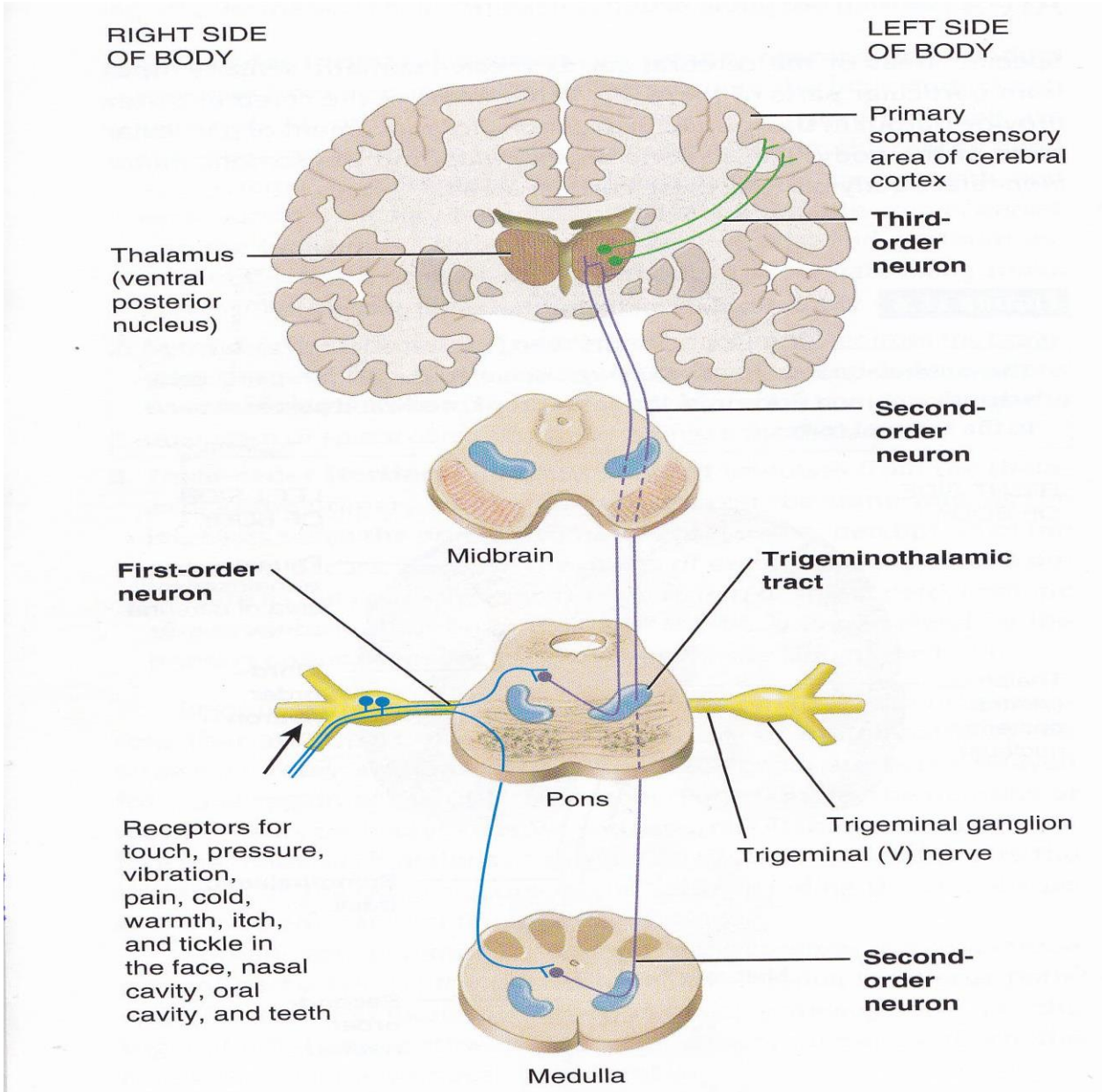


Figure 105.1 Innervation of face ophthalmic, maxillary and mandibular divisions of trigeminal nerve.


RIGHT SIDE OF BODY
LEFT SIDE OF BODY
Primary somatosensory area of cerebral cortex
Third-order neuron
Thalamus (ventral posterior nucleus)
Second-order neuron
Midbrain
First-order neuron
Trigeminothalamic tract
Pons
Receptors for touch, pressure, vibration, pain, cold, warmth, itch, and tickle in the face, nasal cavity, oral cavity, and teeth
Trigeminal ganglion
Trigeminal (V) nerve
Second-order neuron
Medulla

- Astereognosis

- Sensory ataxia (Romberg sign)

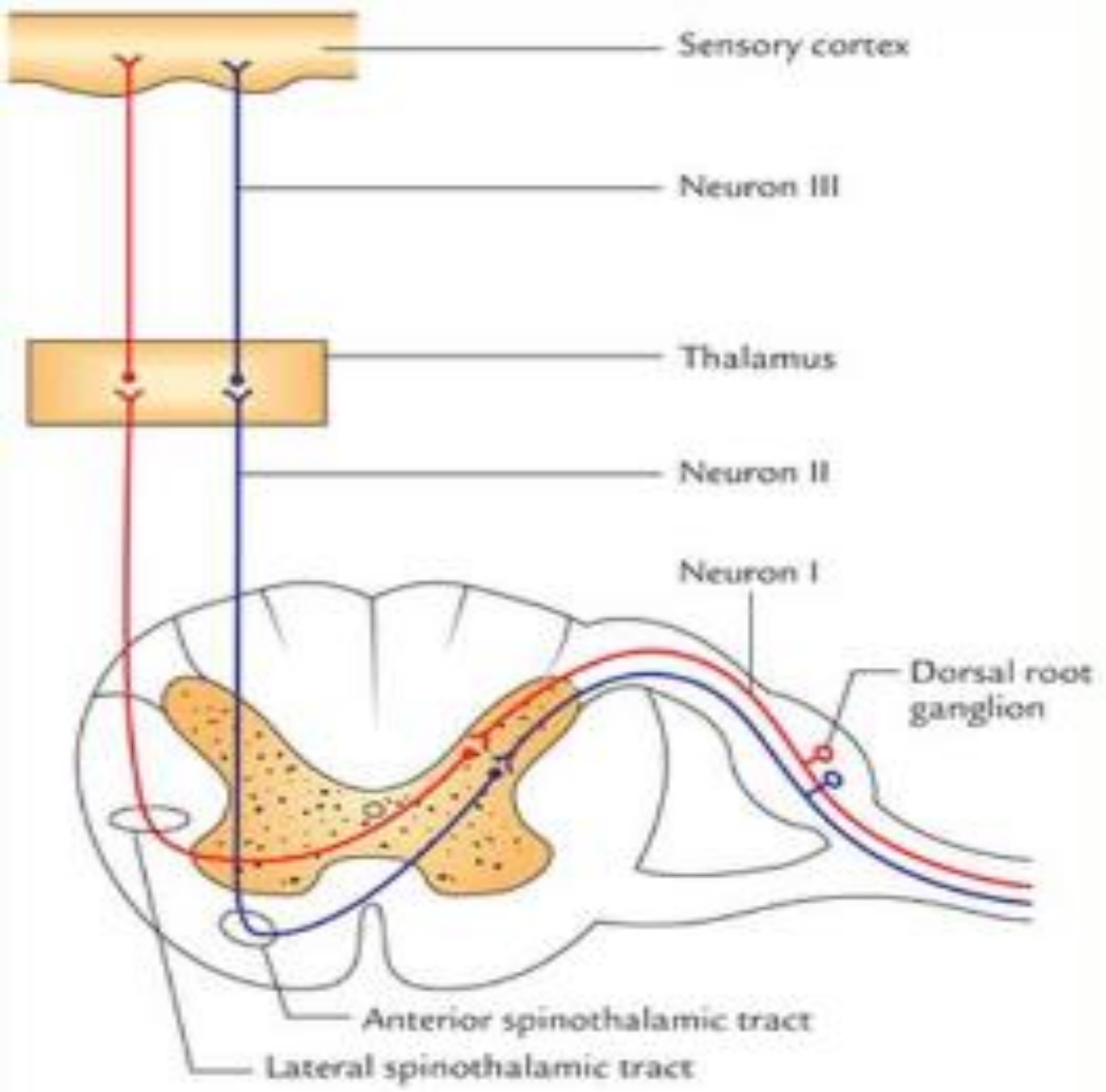
ANTEROLATERAL SYSTEM

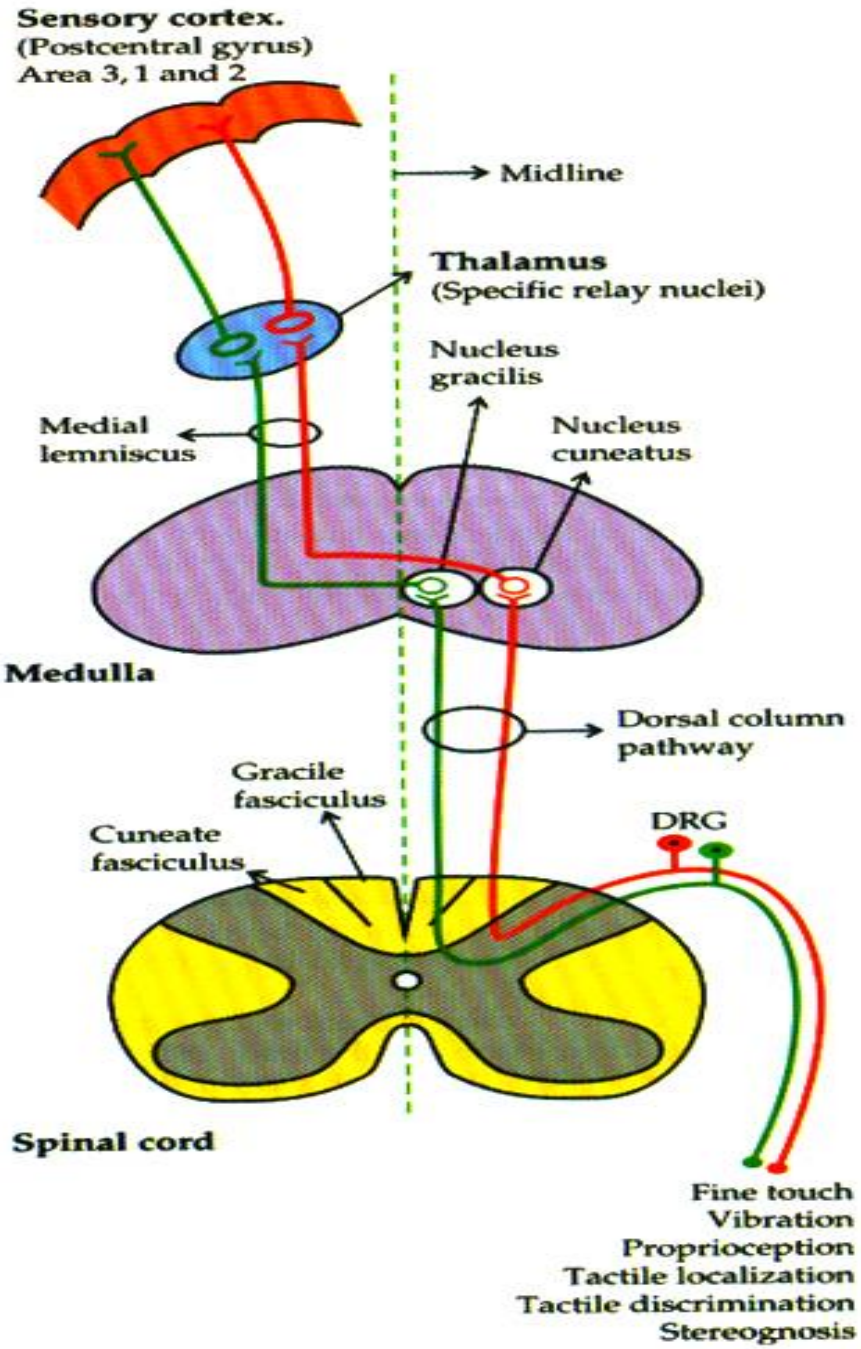
- **Anterior or ventral spinothalamic tract**

  carries: crude touch & pressure

- **Lateral spinothalamic tract**

  carries: pain & temperature


Sensory cortex
Neuron III
Thalamus
Neuron II
Neuron I
Dorsal root ganglion
Anterior spinothalamic tract
Lateral spinothalamic tract



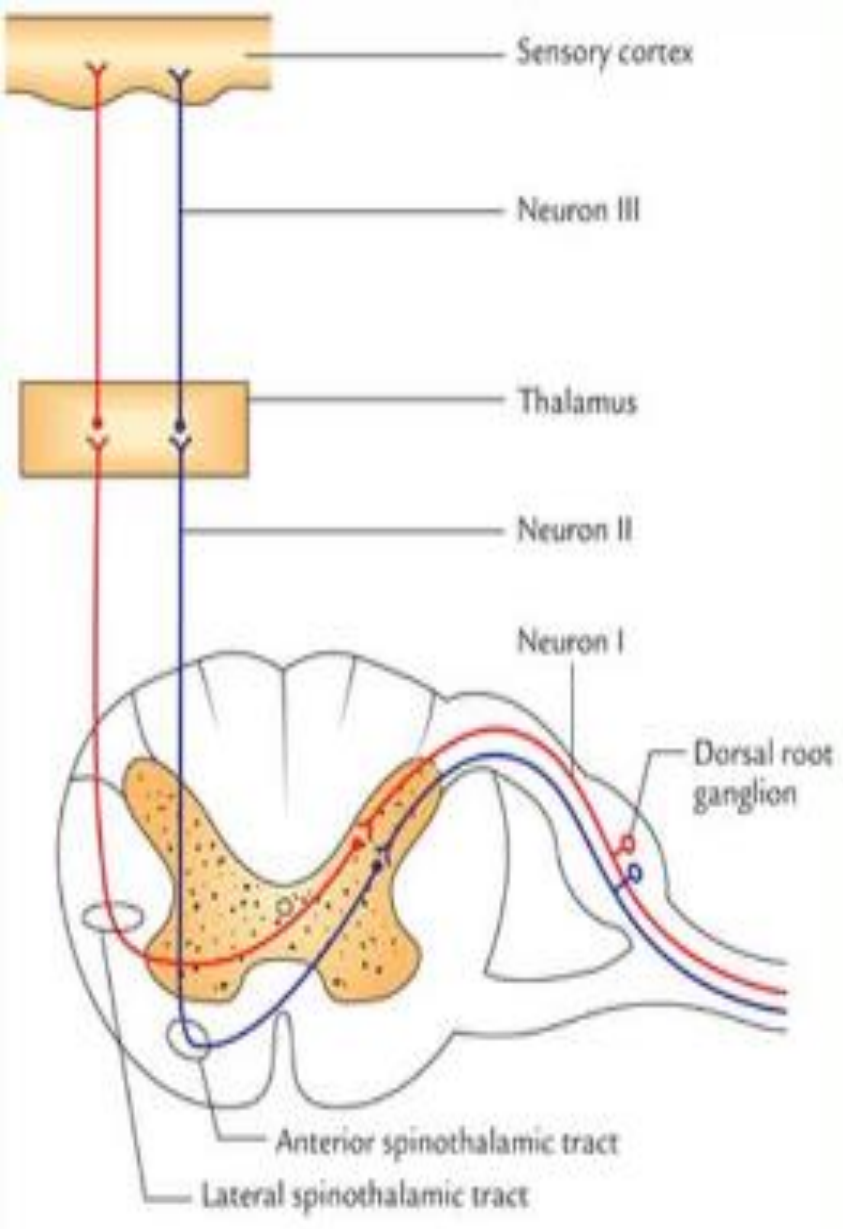
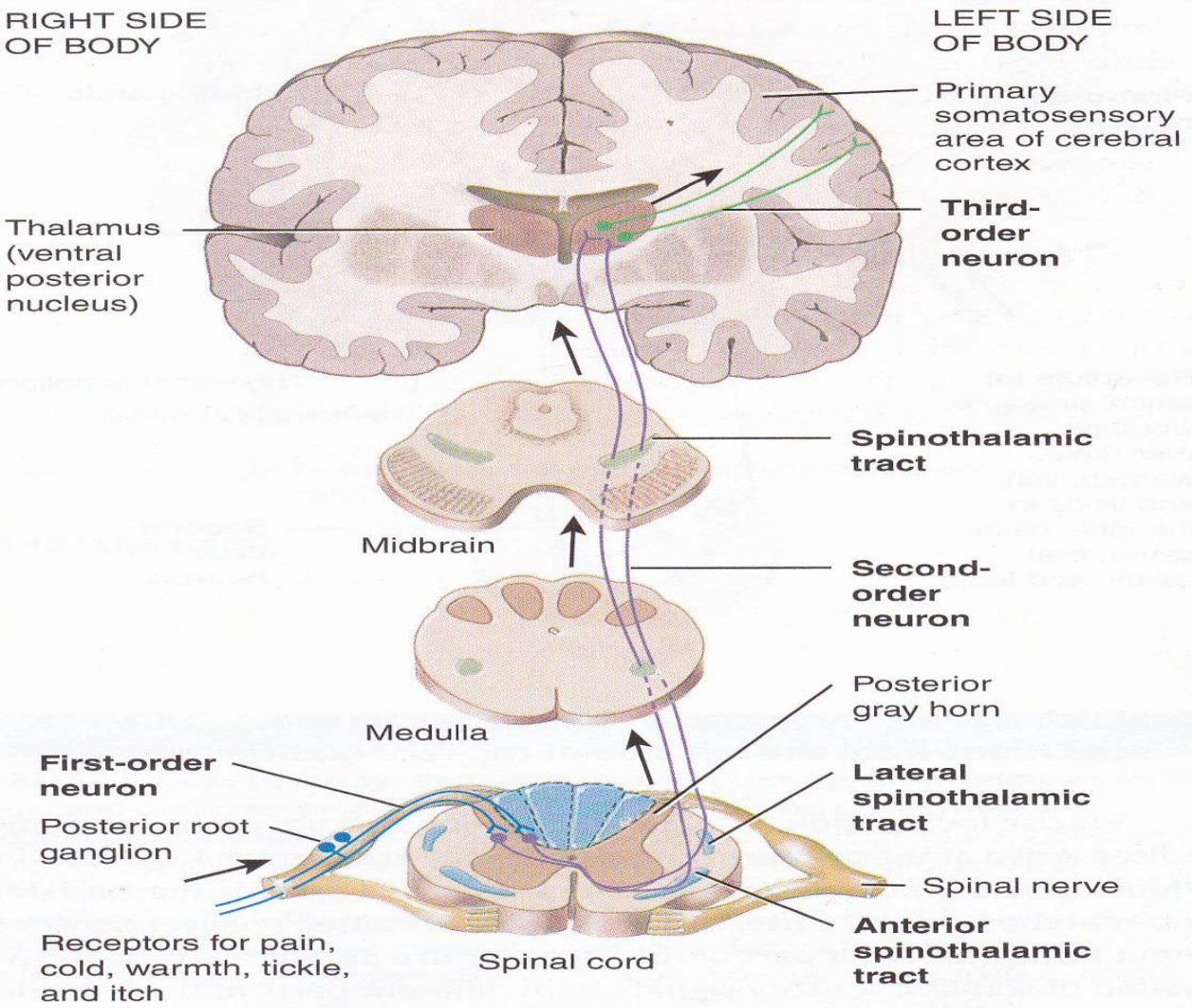


Figure 103.1 Dorsal column pathways.

FIGURE 16.6 **The anterolateral (spinothalamic) pathway.**

The anterolateral pathway conveys nerve impulses for pain, cold, warmth, itch, and tickle from the limbs, trunk, neck, and posterior head to the cerebral cortex.



Anterolateral (spinothalamic) pathways

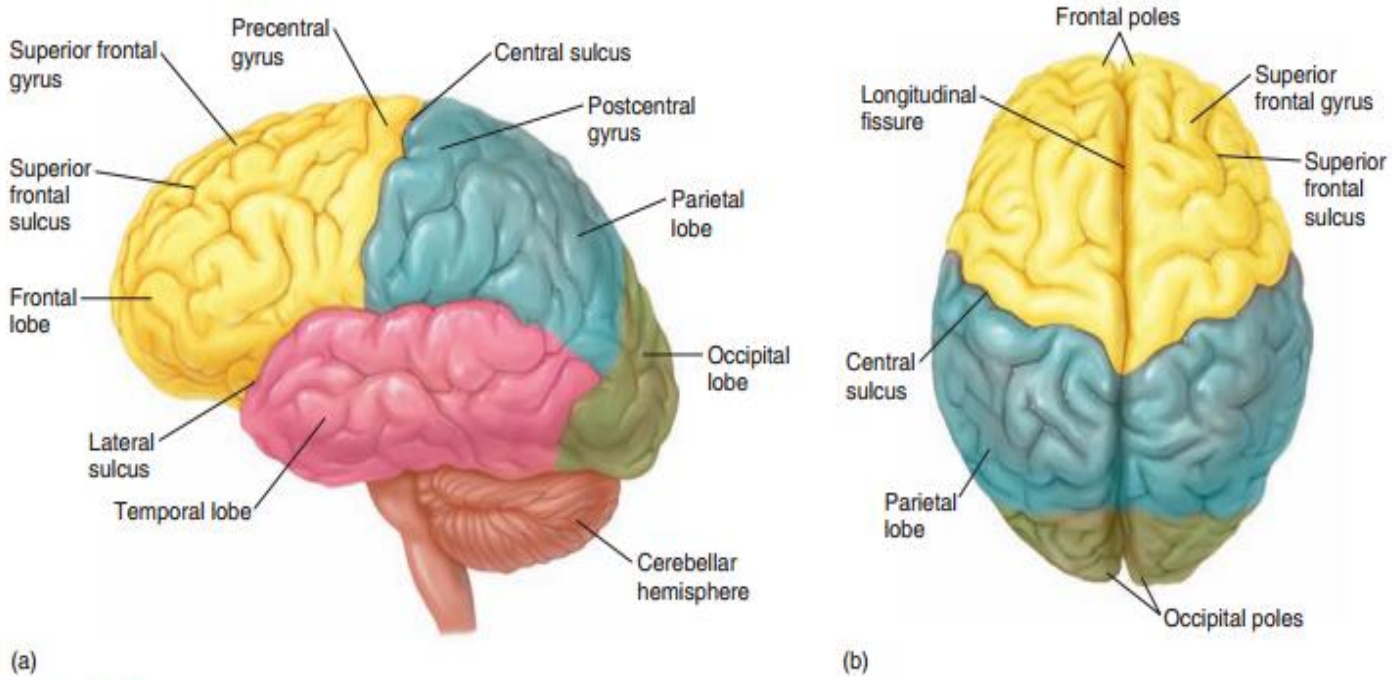


Figure 8.5 **The cerebrum.** (*a*) A lateral view and (*b*) a superior view.

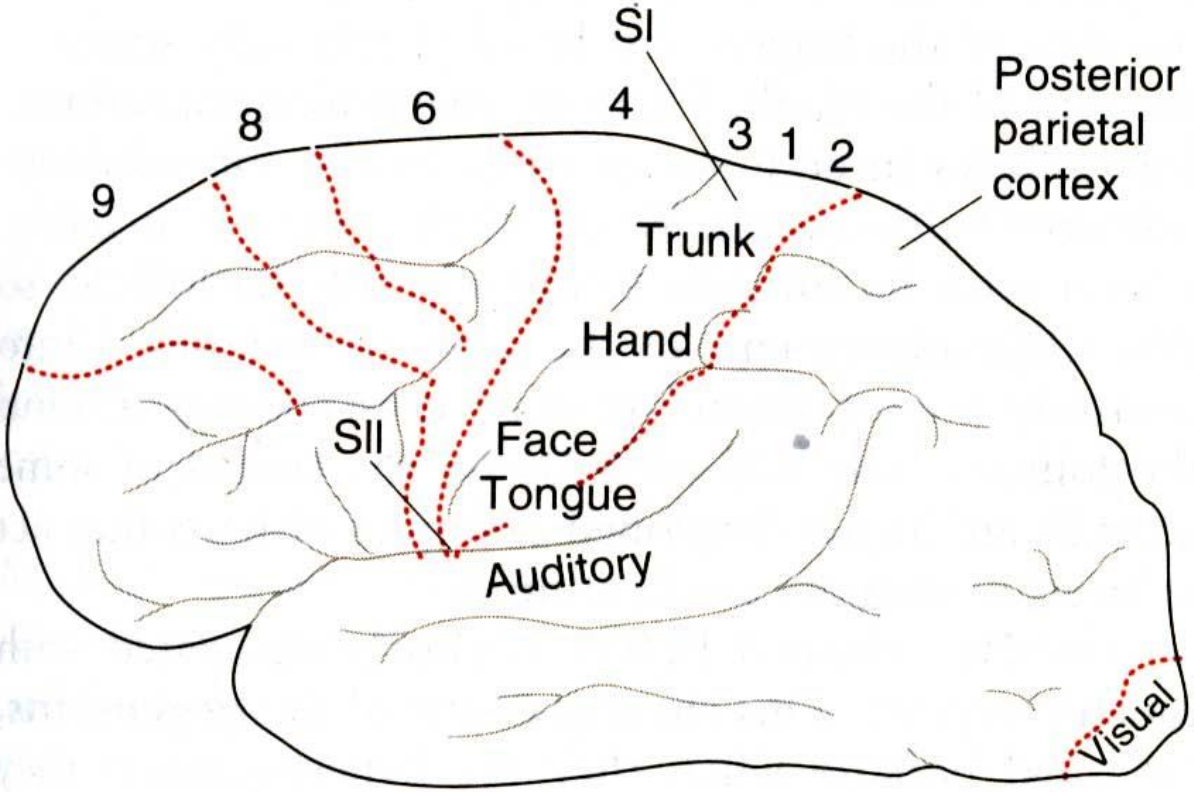
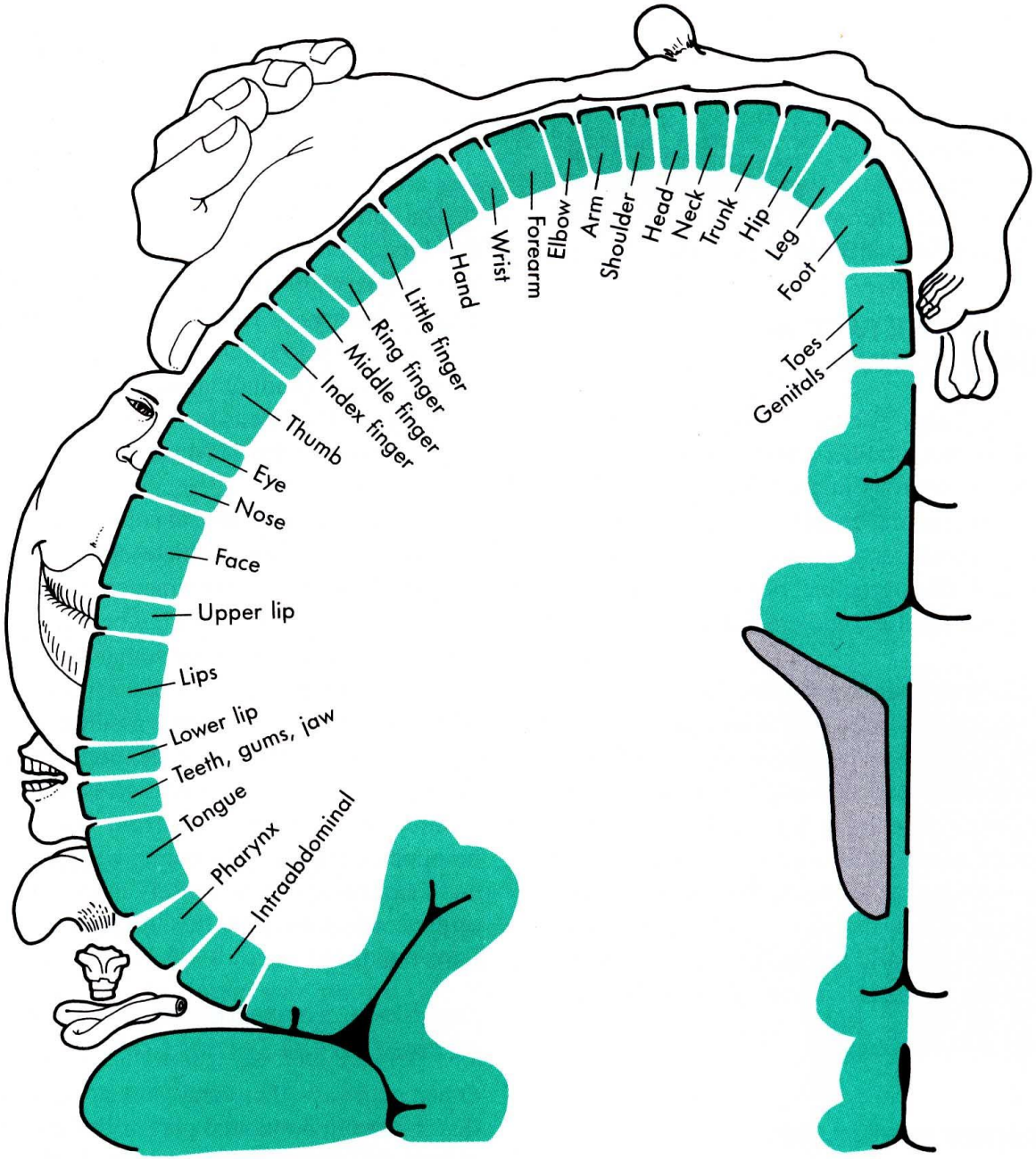


Figure 7–4. Brain areas concerned with somatic sensation, and some of the cortical receiving areas for other sensory modalities in the human brain. The numbers are those of Brodmann's cortical areas. The primary auditory area is actually located in the sylvian fissure on the top of the superior temporal gyrus and is not normally visible.



■ **Fig. 7-8** Sensory homunculus.

PAIN

"The physical adjunct of an imperative protective reflex" - Sherrington

- **Somatic pain**
  - Superficial - surface of the body
  - Deep - muscles, tendons, joints
- **Visceral pain** - visceral organs

- **Receptors –** naked nerve endings (nociceptors)
  - **Polymodal receptors**
  - Tonic receptors

Pain: Fast pain, Slow pain

- **Fast or first pain**
  - occurs within 0.1 second
  - bright, sharp, localized
  - carried by **Aδ fibers** (12 - 30 m/s)
  - NT is **Glutamate**
  - **Neo spinothalamic tract**
  - first order neurons end on lamina 1 & 5

- **Slow or second pain**

  -Begins after 1 second, continues for minutes
  - Dull, intense, diffuse, unpleasant feeling
  - Carried by **C fibers** (0.5 – 2 m/s)
  - NT is **Substance “P”**
  - **Paleo Spinothalamic tract**
  - First order neurons end on lamina 1&2

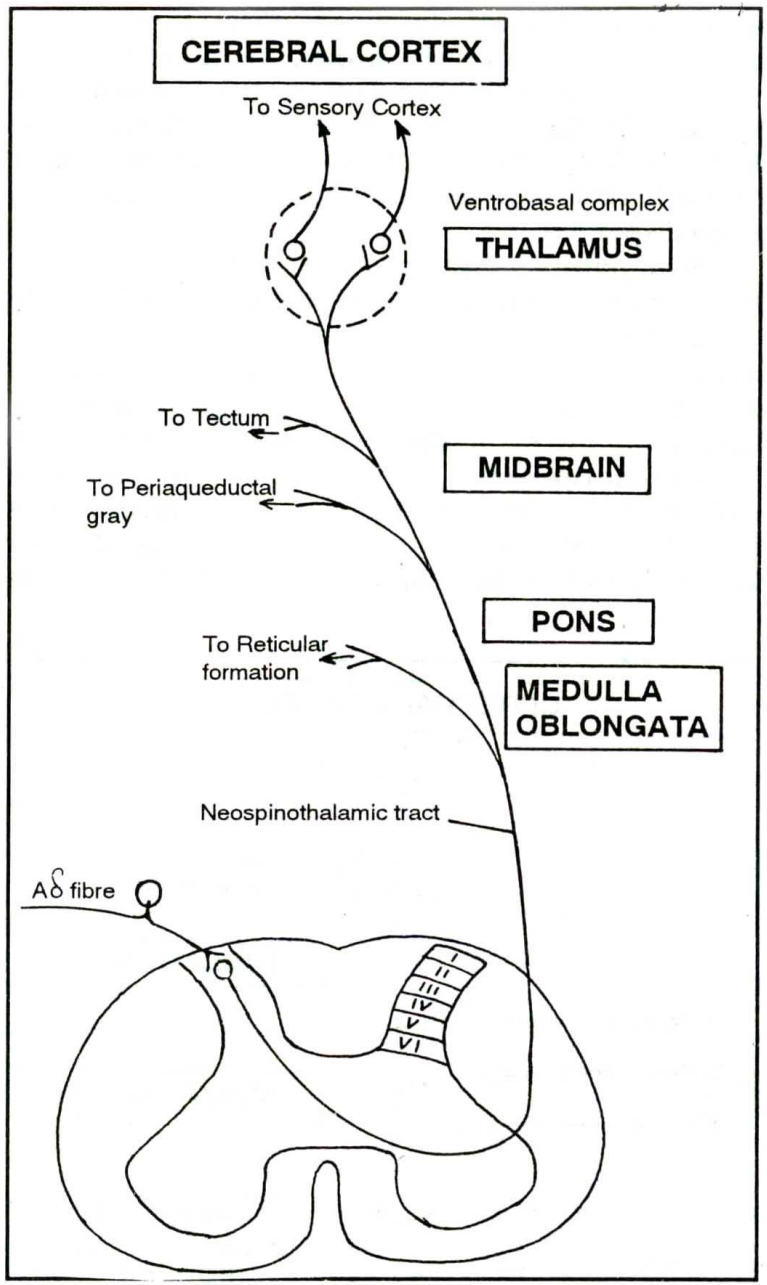


Fig. 12.7.1: *Pathway by which fast pain is conveyed to the central nervous system. I-VI, laminae of the dorsal horn of the spinal cord.*

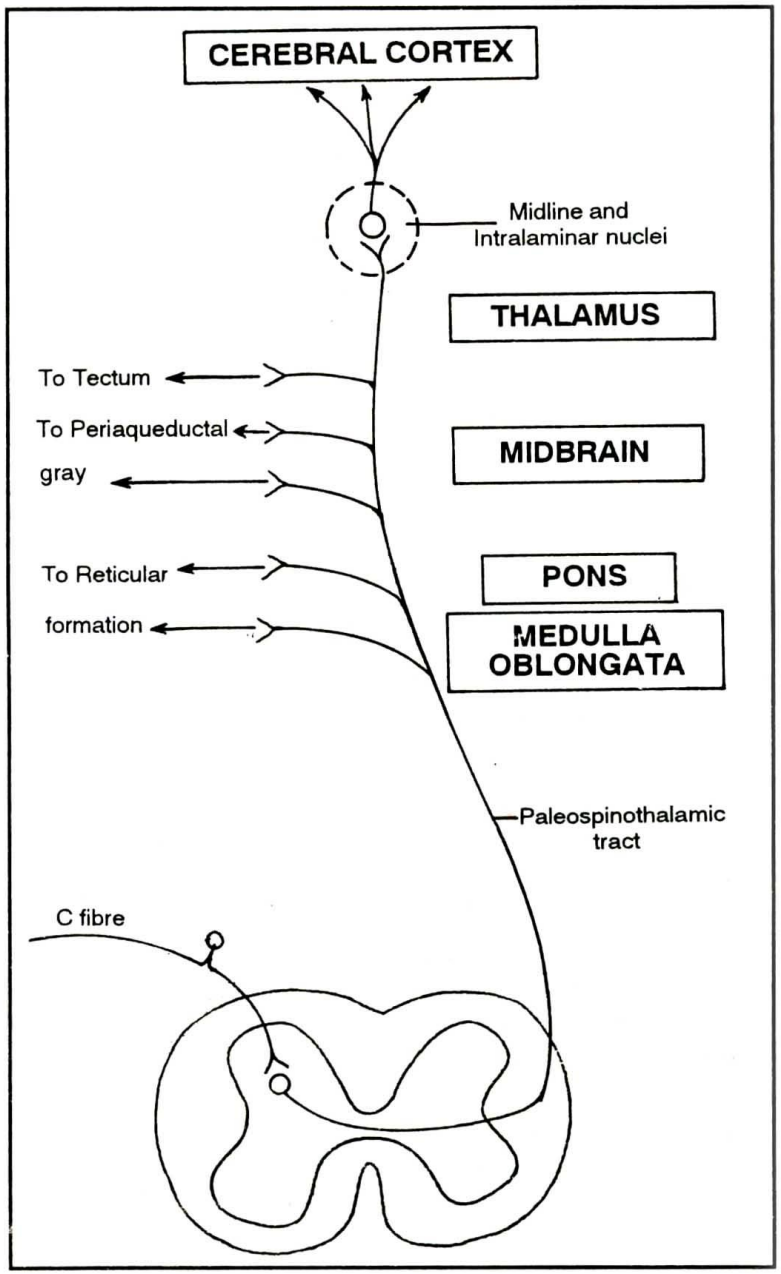


Fig. 12.7.2: *Pathway by which slow pain is conveyed to the central nervous system.*

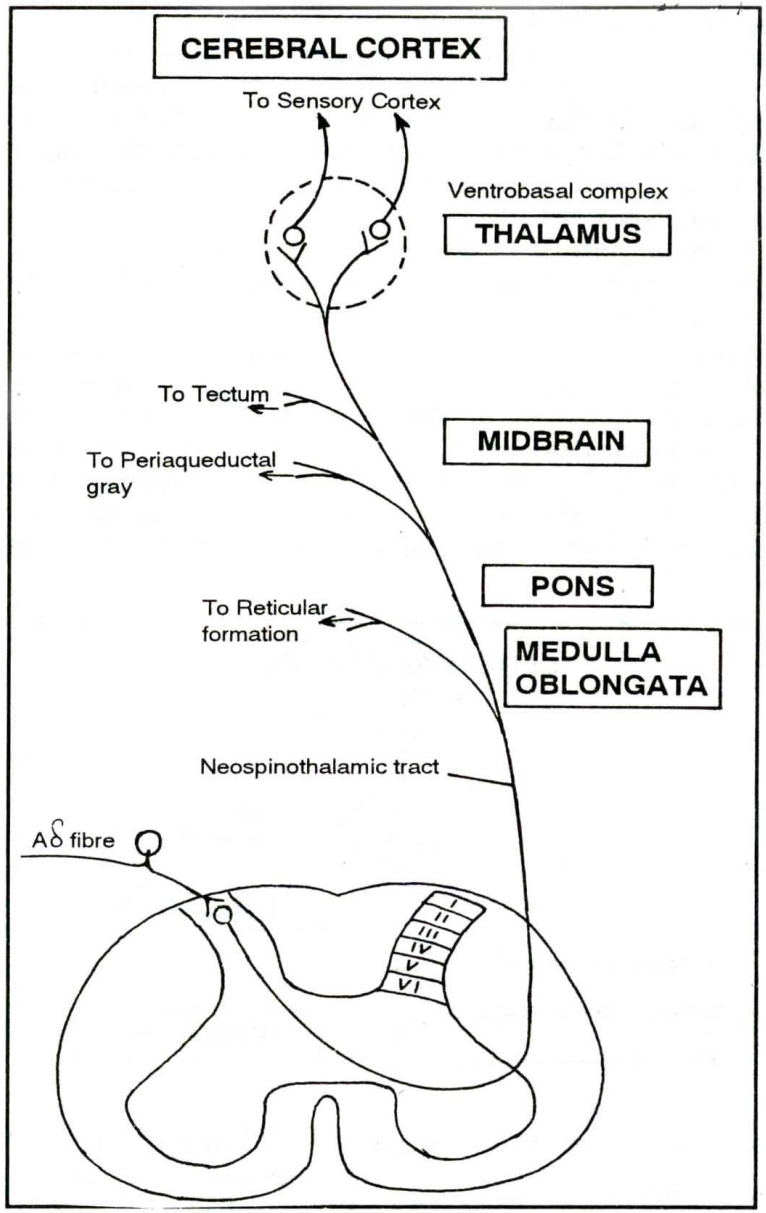


Fig. 12.7.1: *Pathway by which fast pain is conveyed to the central nervous system. I-VI, laminae of the dorsal horn of the spinal cord.*

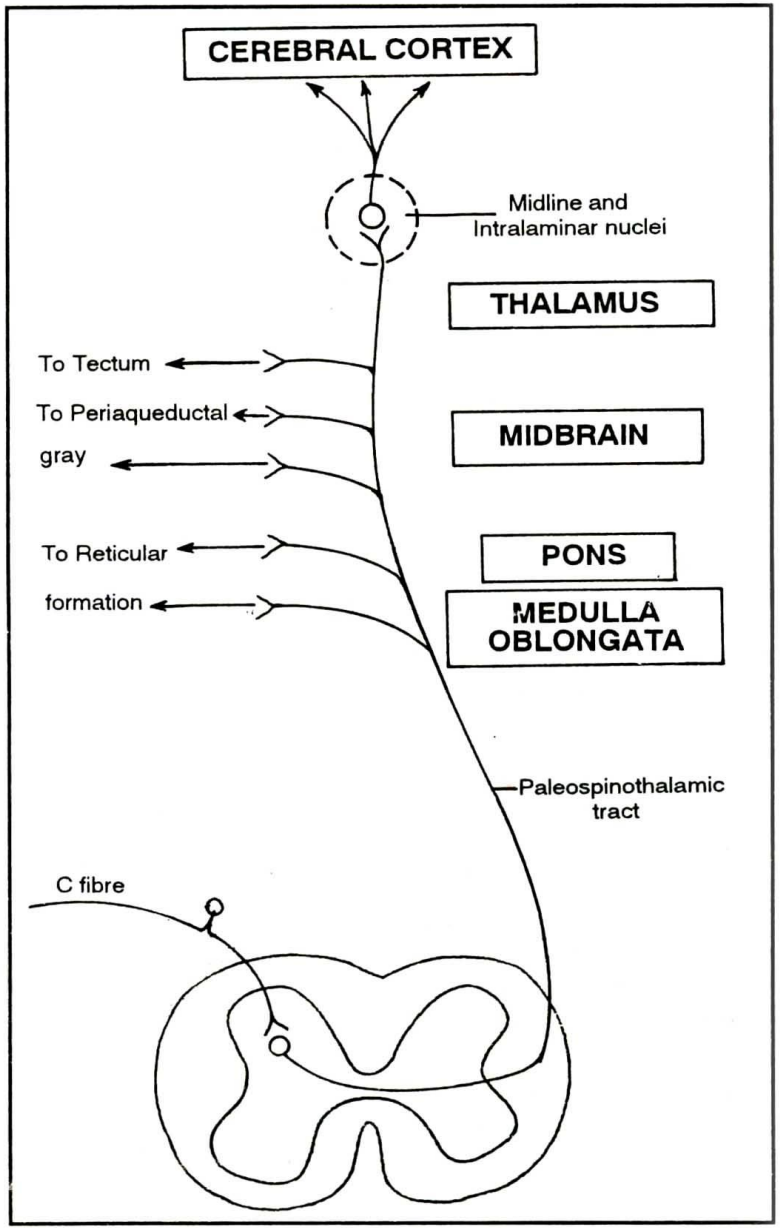


Fig. 12.7.2: *Pathway by which slow pain is conveyed to the central nervous system.*

- **Inflammatory pain**
    - Hyperalgesia
    - Allodynia
- **Neuropathic Pain**

- **Visceral pain**

Dull, continuous, poorly localized, associated with autonomic changes and often radiates or referred to somatic structures.

- Viseral pain sensatins are carried along autonomic nerves
- Stimulus : Distension, inflammation, ischemia, irritation

- **Referred pain**

  The pain produced in the visceral organ is not felt in the viscera but in somatic strucures that are away from the viscera.

Ex; - Cardiac pain referred to left arm

- Irritation of diaphragm – tip of shoulder
- Ureteric pain - testis

Theories to explain referred pain

1. **Dermatomal rule**
2. **Convergence theory**
3. **Facilitation theory**

1. Dermatomal rule

The **visceral organ** in which pain is produced and the **somatic structure** where pain is referred or felt, have developed from same embryonic segment or **dermatome**

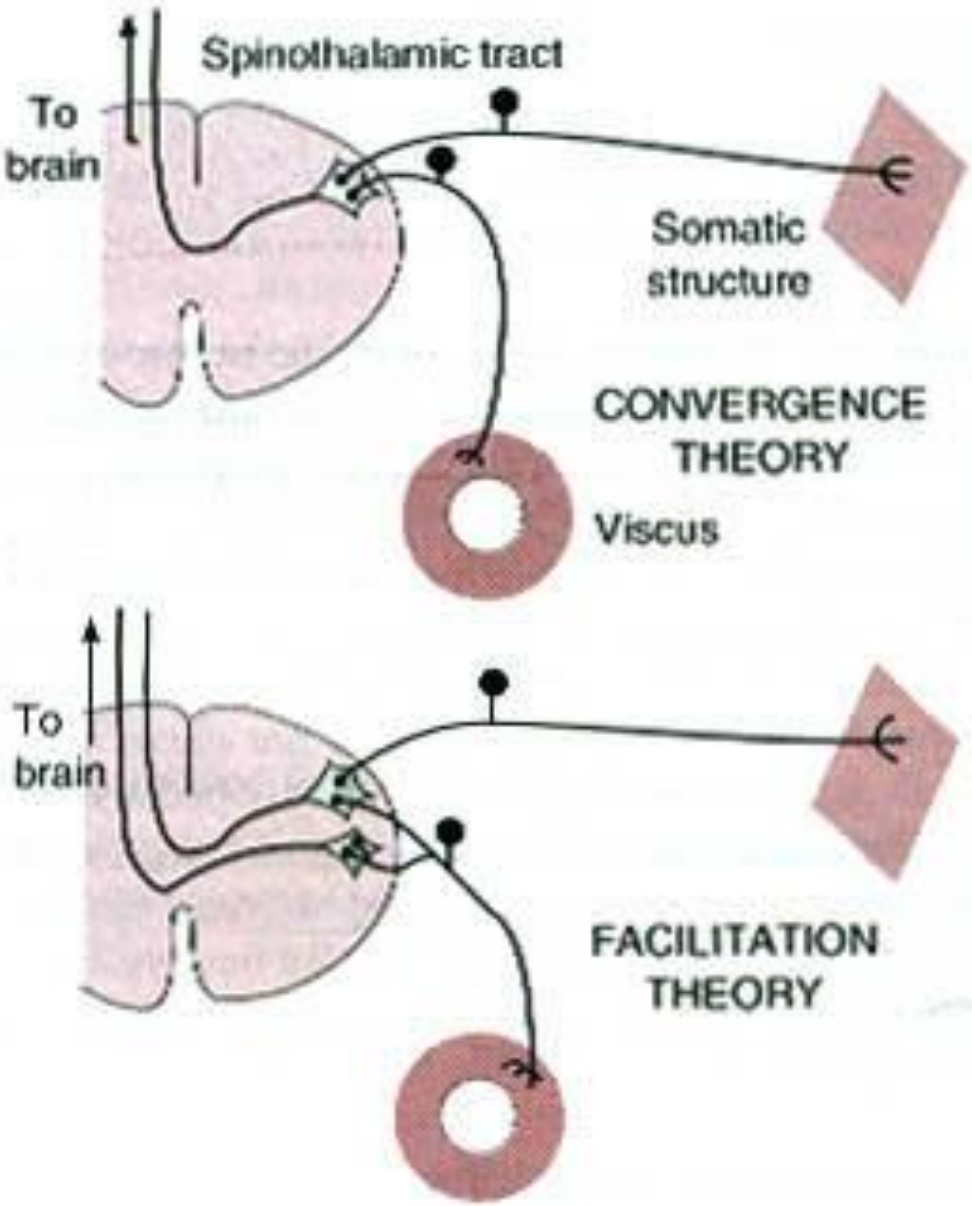


Figure 7–8. Diagram of convergence and facilitation theories of referred pain.

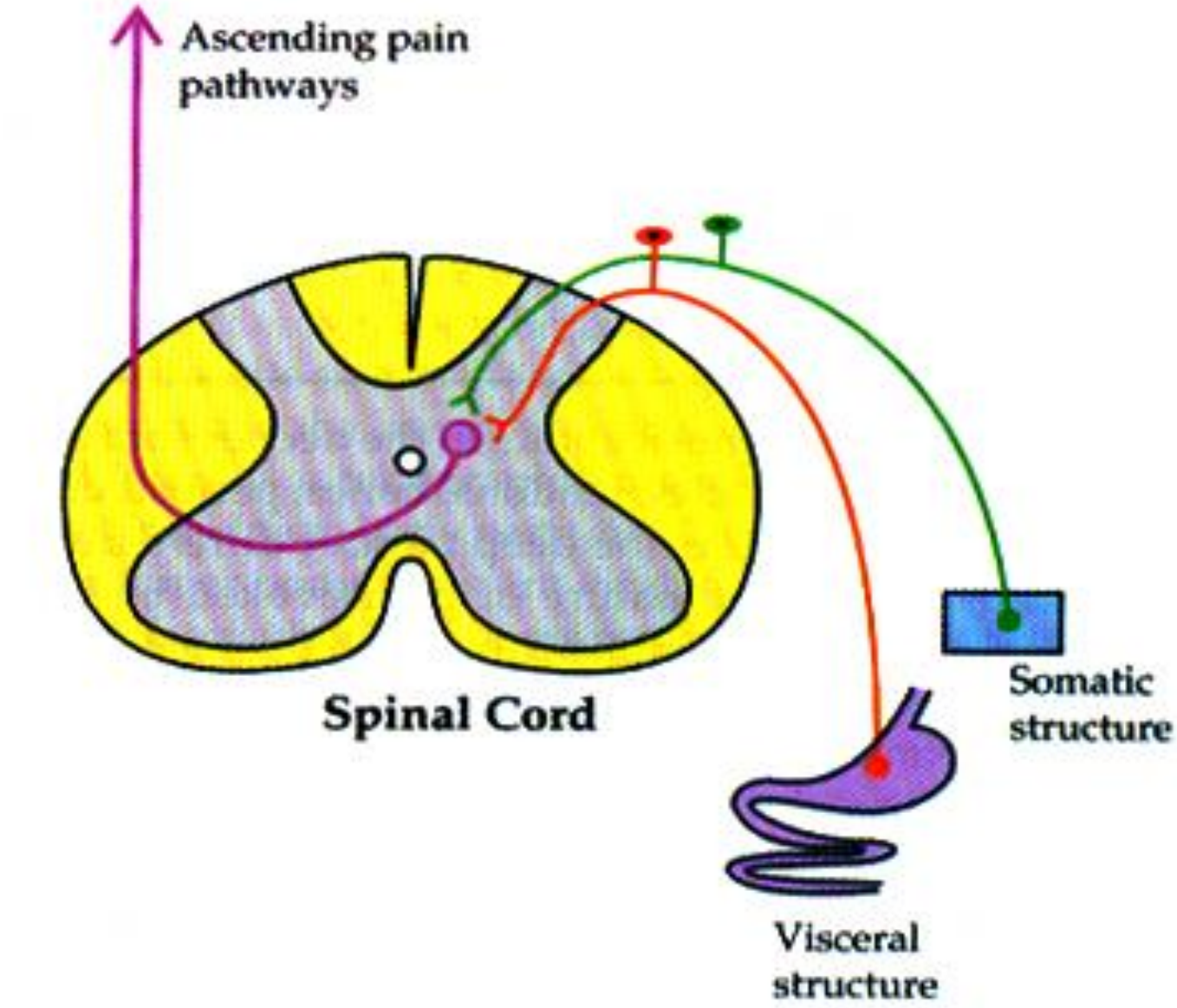


Figure 104.4 Convergence theory of referred pain. Note, due to convergence of fibers from somatic and visceral structures on a single second order neuron, fibers transmitting pain sensation from the somatic structure also carry the pain sensation arising from the visceral structures.

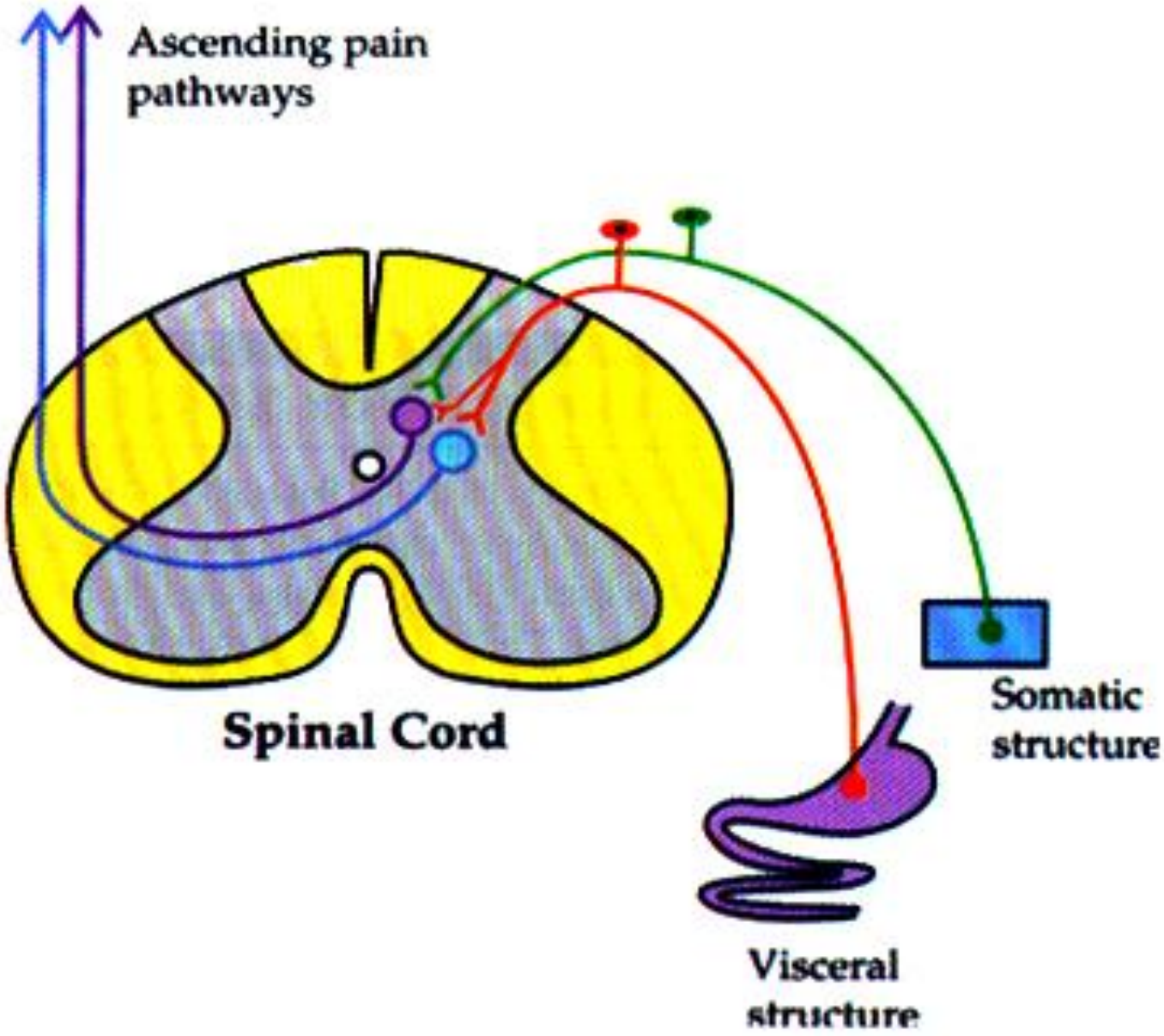


Figure 104.5 Facilitation theory of referred pain.

Endogenous pain inhibiting systems

- Gate control theory (Melzack and wall)
- Endogenous Enkephalinergic ,descending analgesic system
- Endogenous opioid system

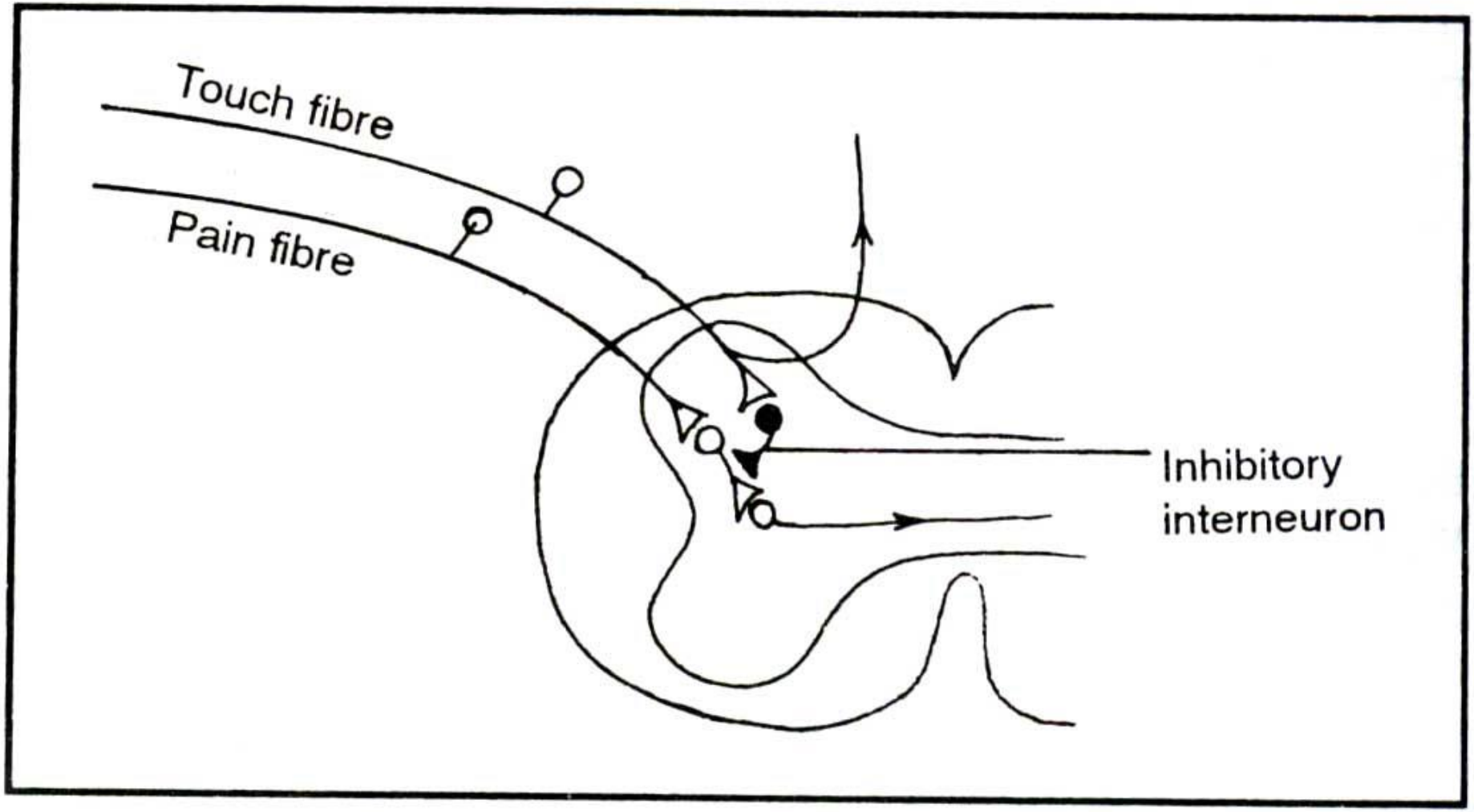


Fig. 12.7.3: *Gate control theory. Activation of the inhibitory interneuron in the substantia gelatinosa 'closes the gate' through which the sensation of pain may be conveyed.*

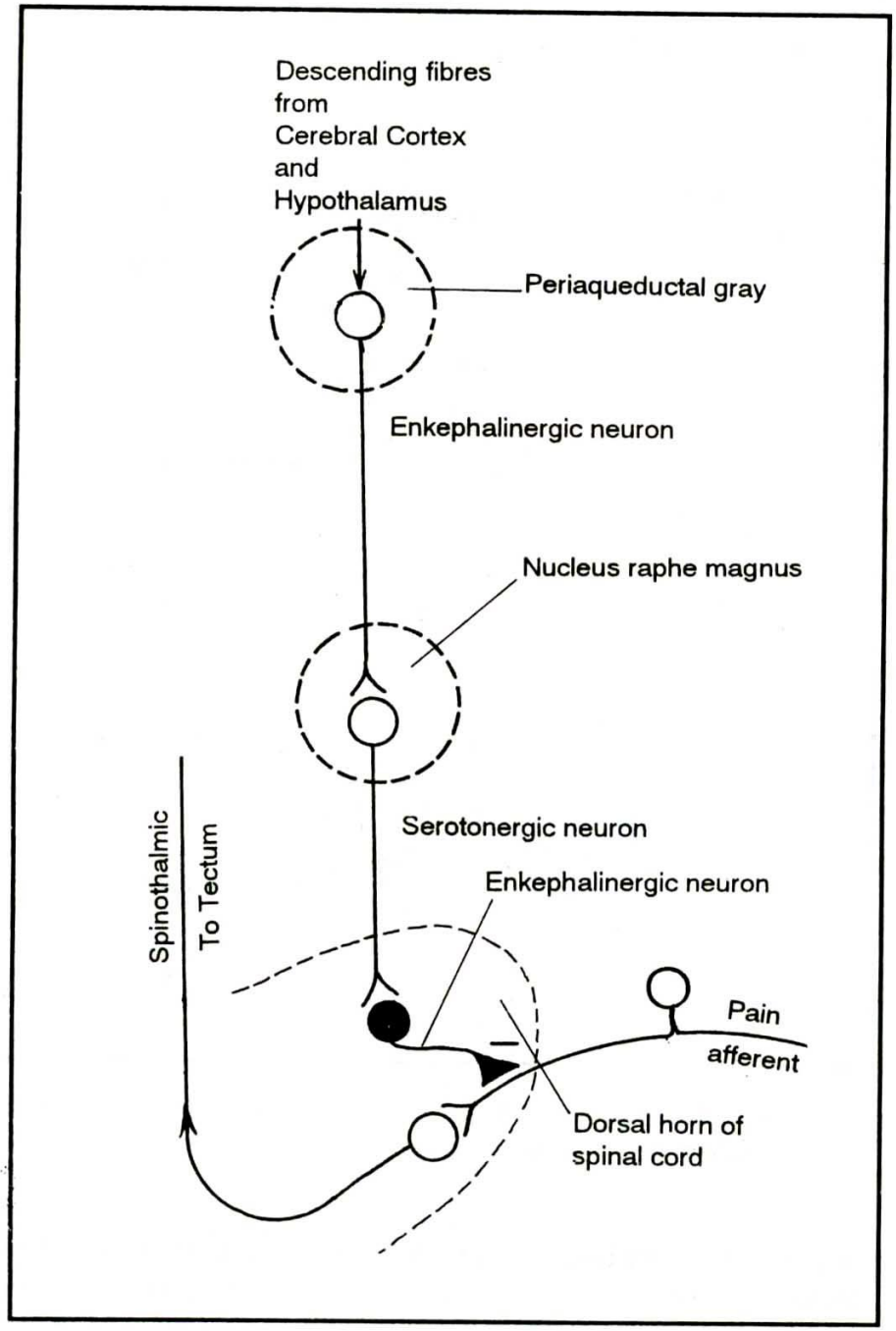
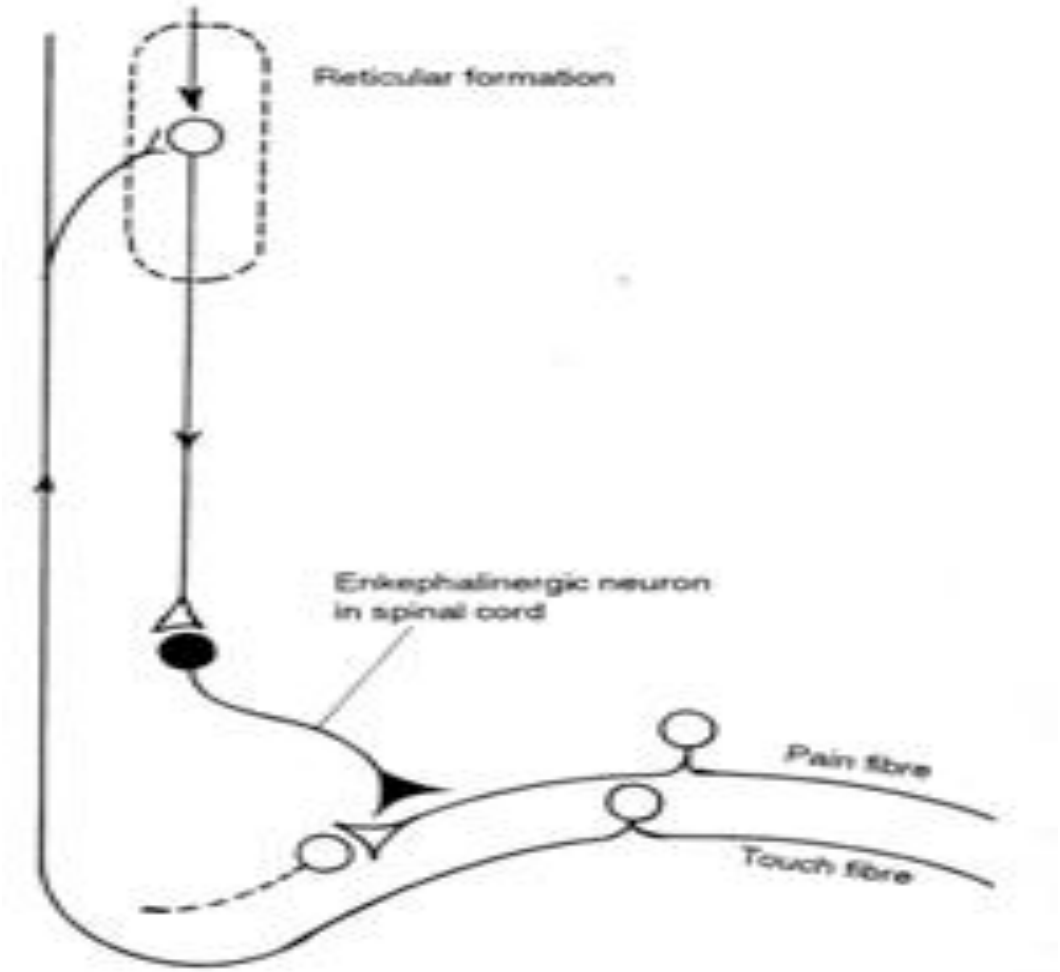


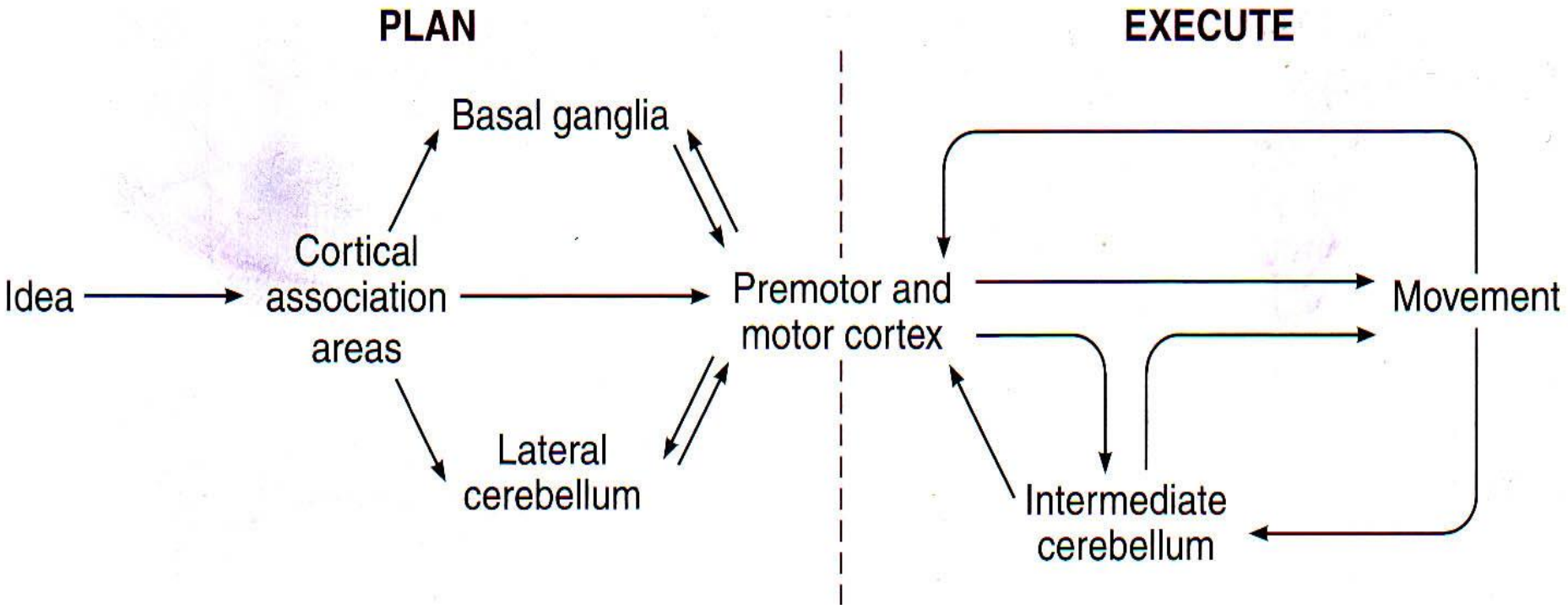
Fig. 12.7.5: *Endogenous pain relief system. Details in text.*



. **12.7.6:** *Revaluation of the gate control theory. Th*
ly simplified neuronal circuitry in this diagram show
stimulation of a touch fibre might indirectly activate th
bitory enkephalinergic interneuron in the spinal cord

- Anaesthesia
- Dissociateted anaesthesia
- Analgesia
- Hypoesthesia
- Paresthesia
- Ataxia (Romberg sign)
- Agraphesthesia

MOTOR SYSTEM



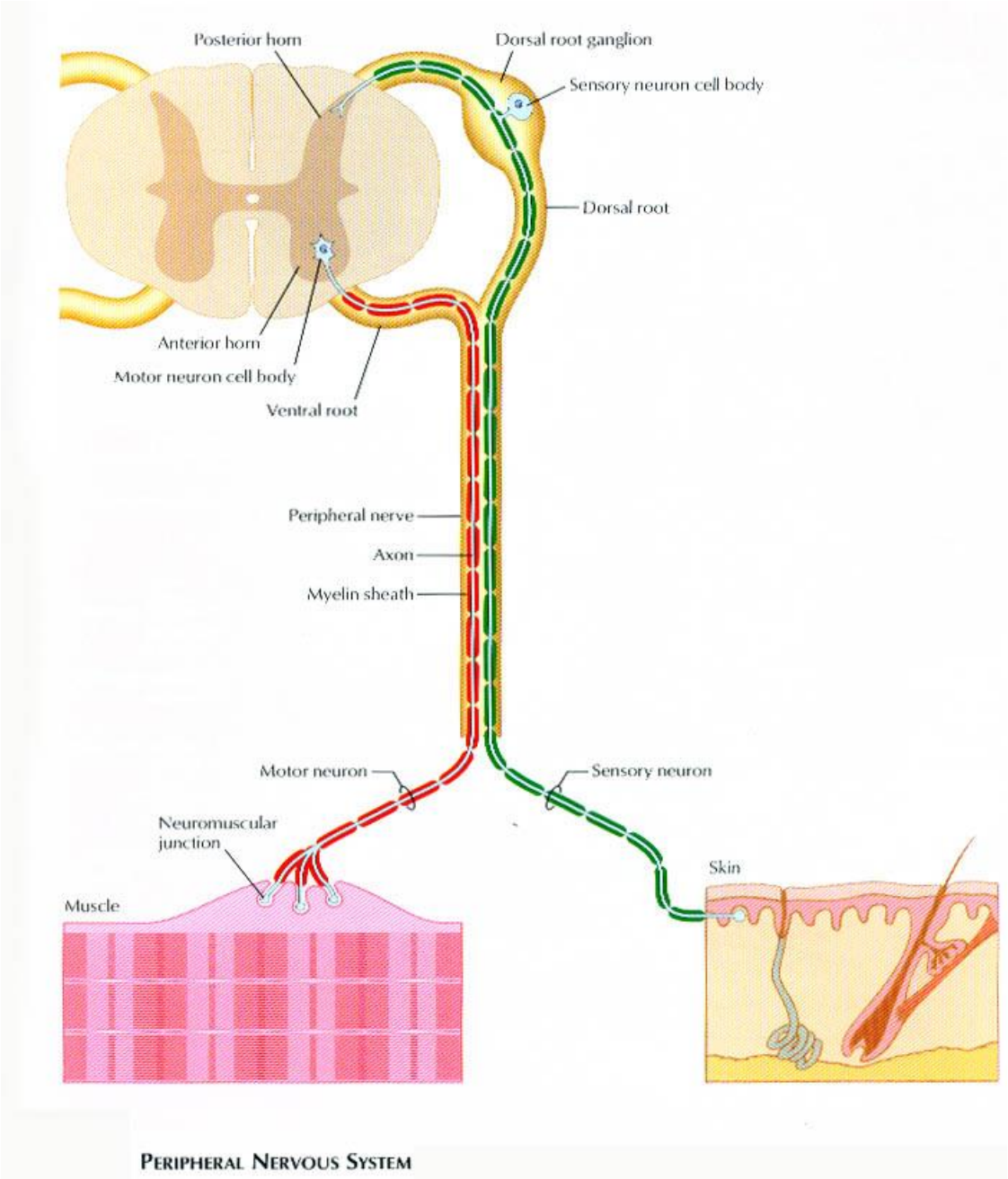
- **Skeletal muscles**

  - **Proximal Muscles**

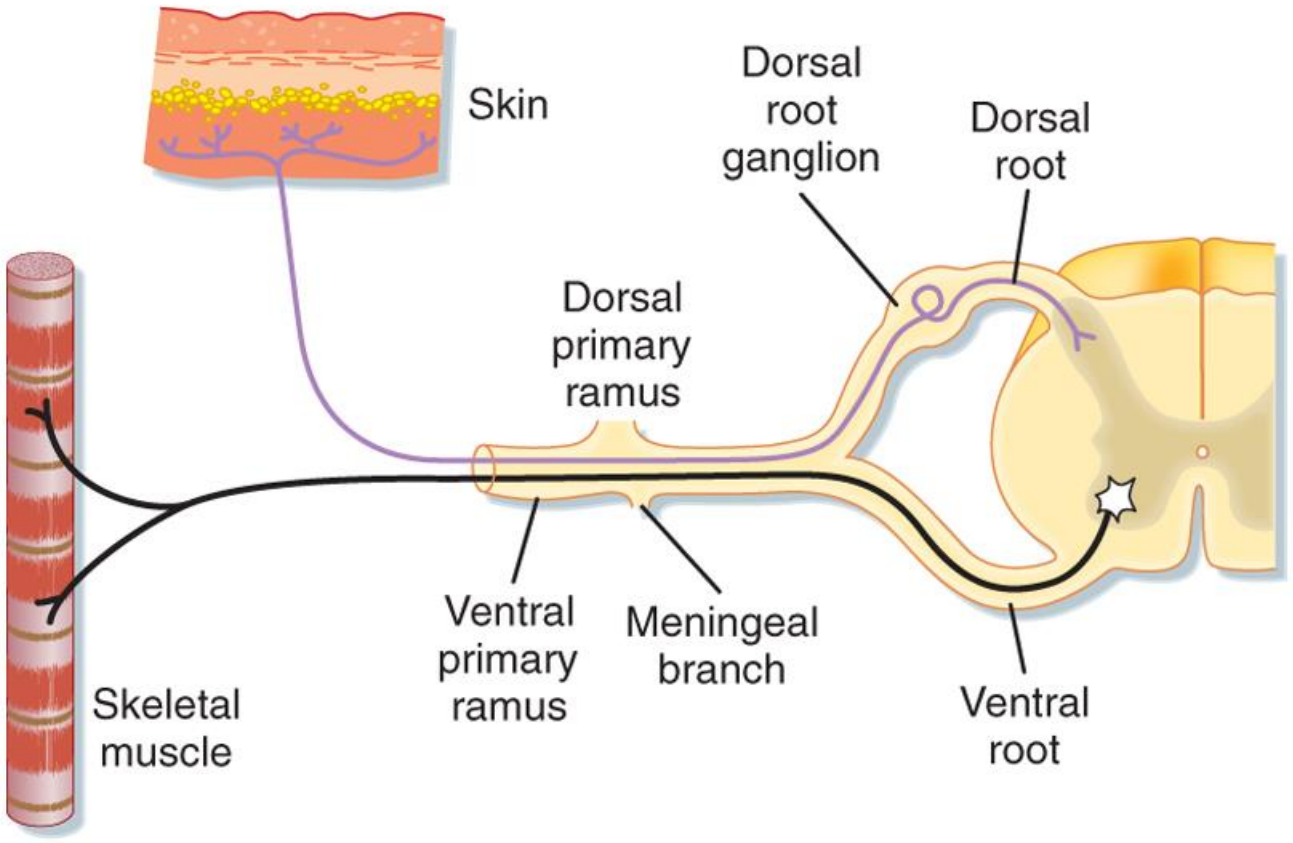
    Maintain posture, Balance

  - **Distal Muscles**

    Regulate fine skilled voluntary movements



PERIPHERAL NERVOUS SYSTEM


Skin
Dorsal root ganglion
Dorsal root
Dorsal primary ramus
Ventral primary ramus
Meningeal branch
Ventral root
Skeletal muscle

- **Anteror horn cells**

-**Medial group of Anterior horn cells**

innervate proximal muscles

-**Lateral group of Anterior horn cells**

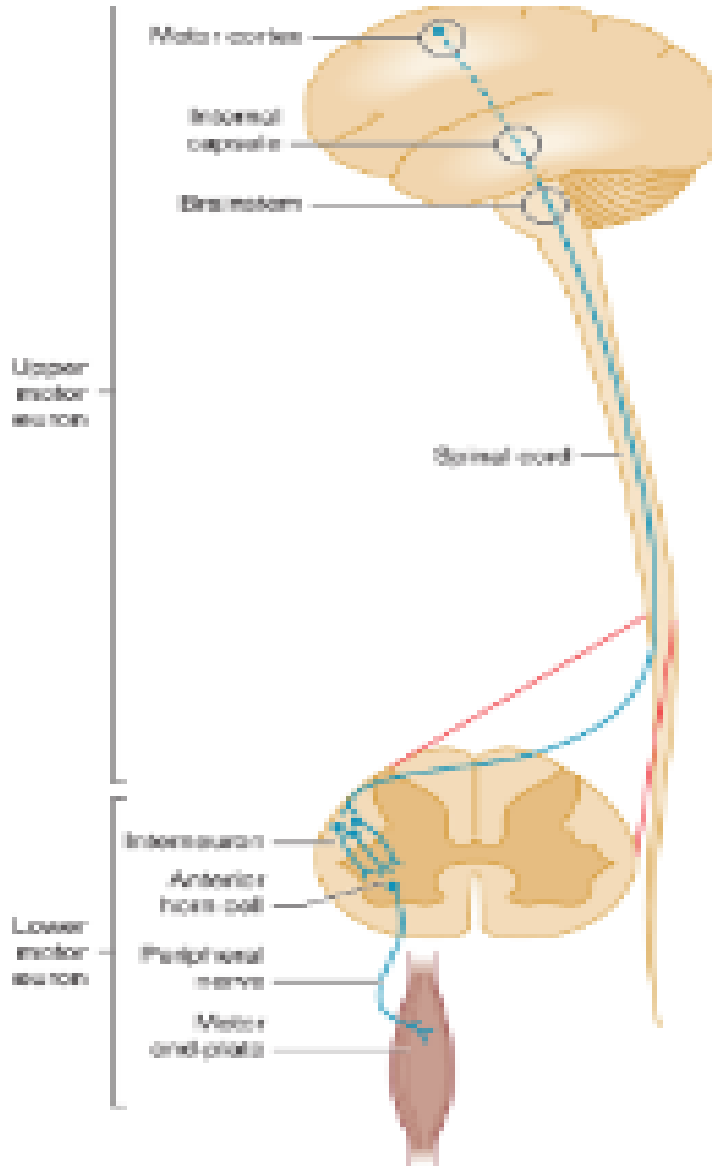
innervate distal muscles

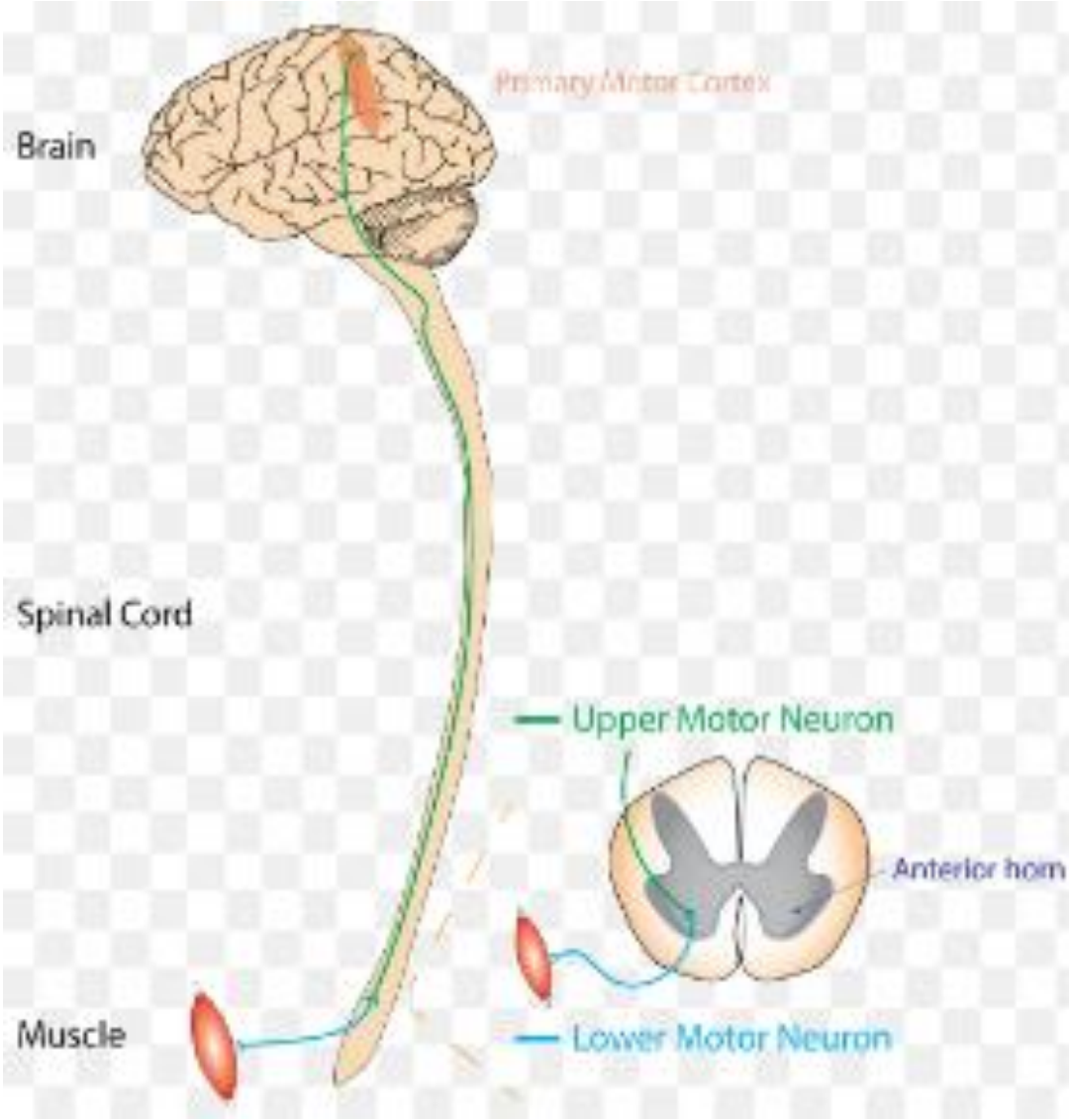
- **Upper Motor Neuron**

  Descending tracts

- **Lower Motor Neuron**

  Final common pathway


Upper motor neuron
Lower motor neuron


Primary Motor Cortex
Brain
Spinal Cord
Upper Motor Neuron
Anterior horn
Muscle
Lower Motor Neuron

Descending Tracts

- **Pyramidal tracts (corticospinal tract)**

  Regulate fine skilled voluntary motor activity

- **Extra Pyramidal tracts**

  Rubro spinal, Reticulo spinal,
  Vestibulo spinal, Tecto spinal,
  Olivo spinal.

  Regulate tone, posture and balance

- **Medial motor system**

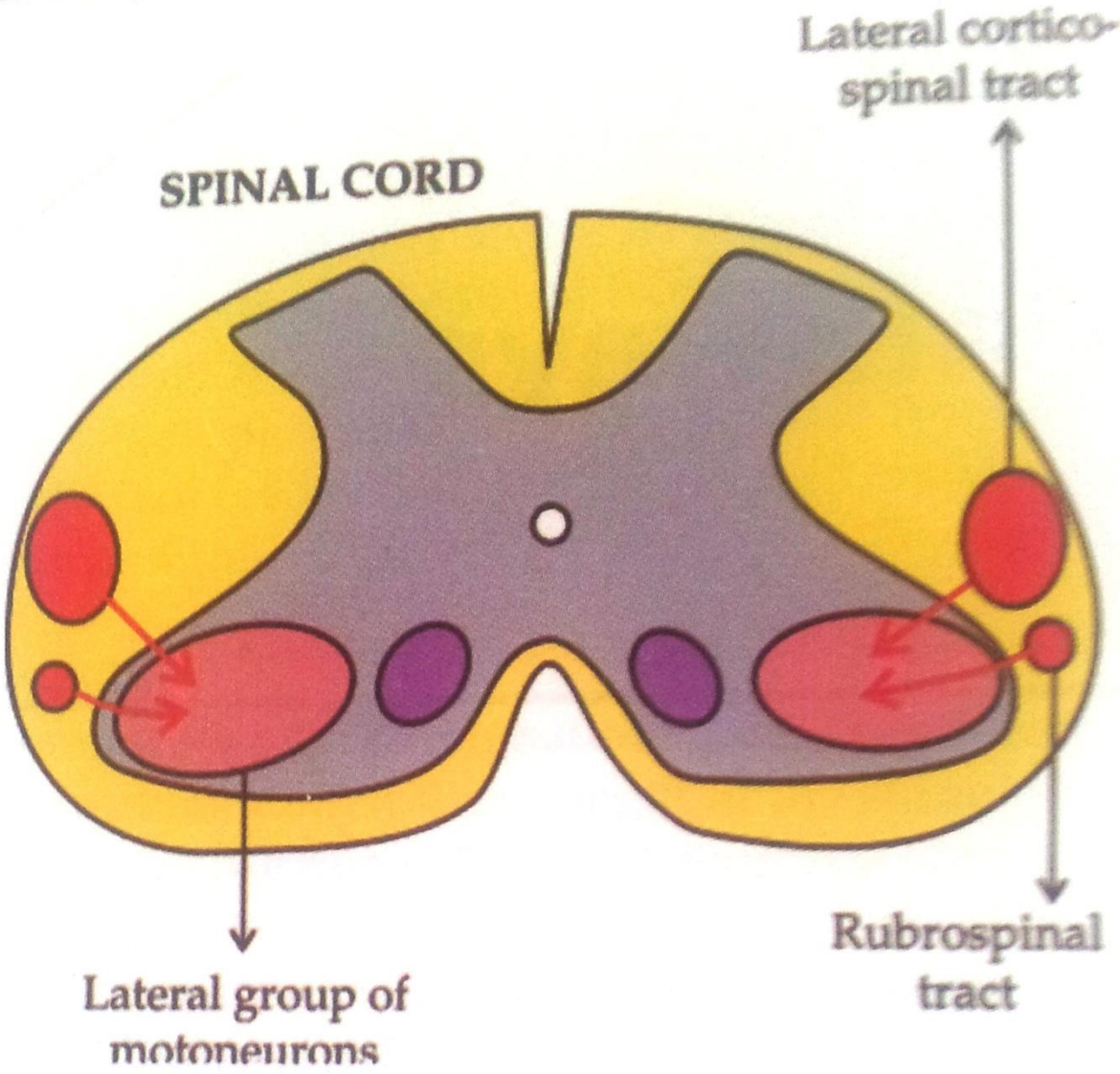
  Extrapyramidal tracts except Rubrospinal tract

  Anterior corticospinal tract

- **Lateral motor system**

  Lateral corticospinal tract

  Rubrospinal tract


Lateral cortico-
spinal tract
SPINAL CORD
Lateral group of
motoneurons
Rubrospinal
tract

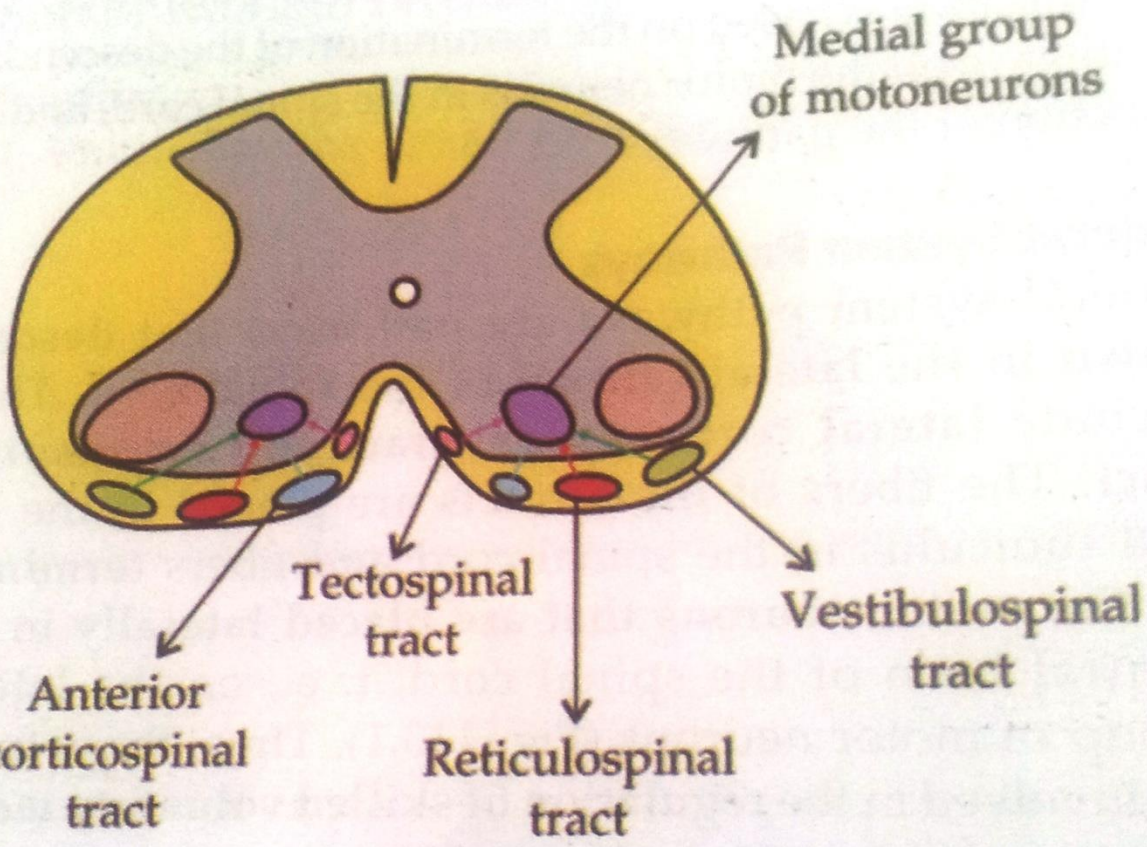
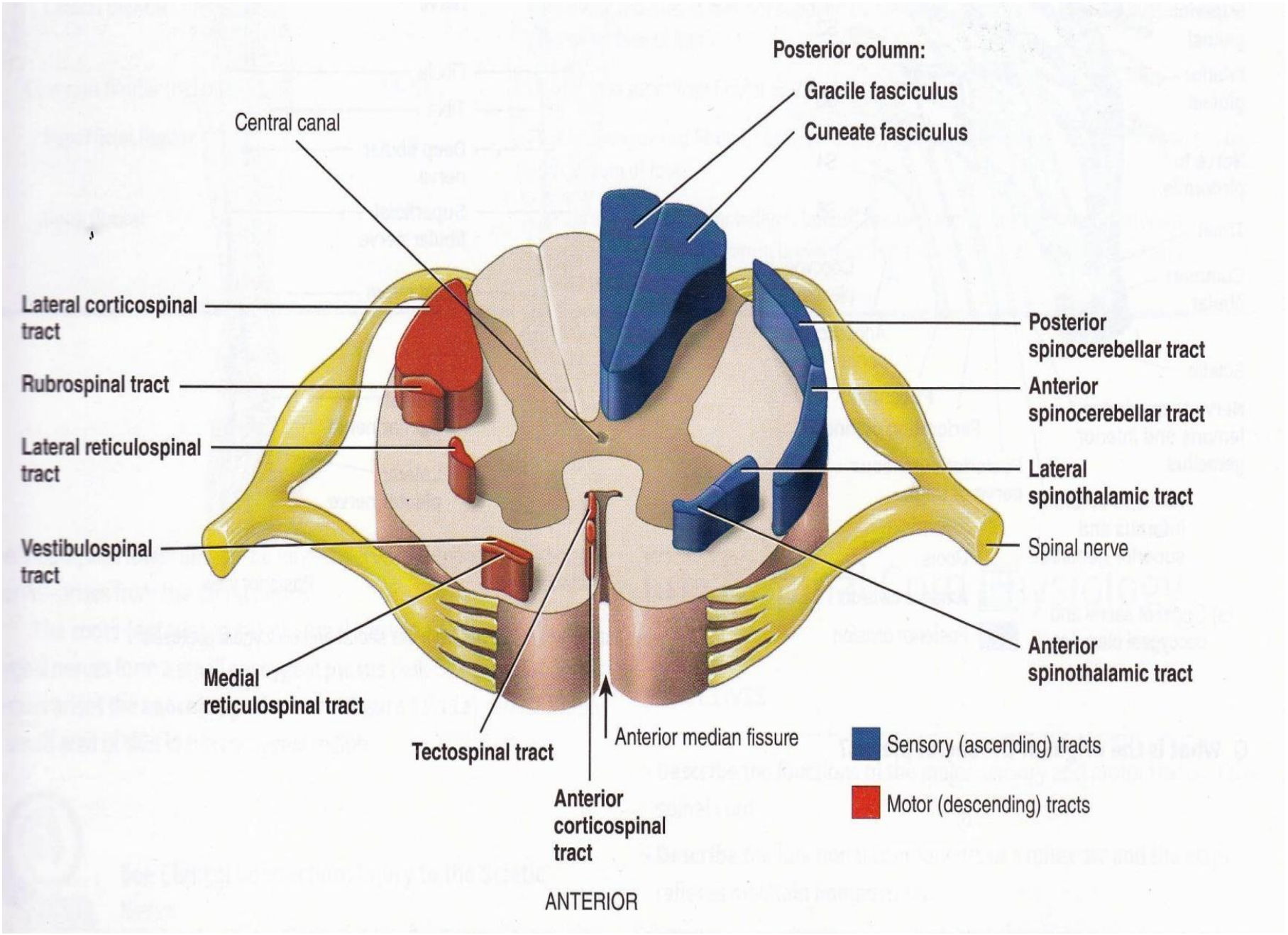
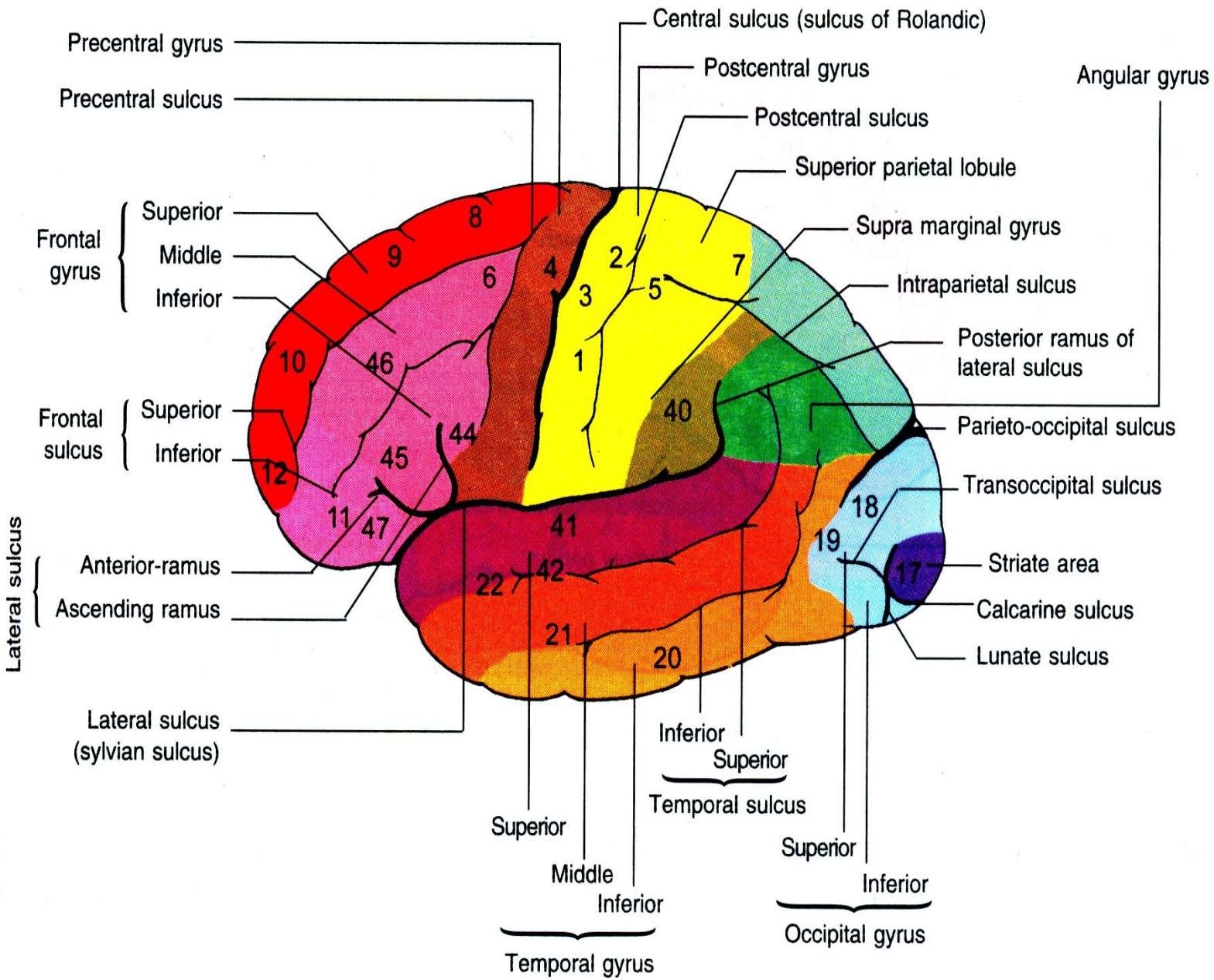


Figure 113.2 Placement of medial descending pathways in spinal cord.


Posterior column:
Gracile fasciculus
Cuneate fasciculus
Central canal
Lateral corticospinal tract
Rubrospinal tract
Lateral reticulospinal tract
Vestibulospinal tract
Posterior spinocerebellar tract
Anterior spinocerebellar tract
Lateral spinothalamic tract
Spinal nerve
Anterior spinothalamic tract
Medial reticulospinal tract
Tectospinal tract
Anterior median fissure
Anterior corticospinal tract
Sensory (ascending) tracts
Motor (descending) tracts
ANTERIOR


Precentral gyrus
Precentral sulcus
Central sulcus (sulcus of Rolandic)
Postcentral gyrus
Postcentral sulcus
Angular gyrus
Superior parietal lobule
Supra marginal gyrus
Intraparietal sulcus
Posterior ramus of lateral sulcus
Parieto-occipital sulcus
Transoccipital sulcus
Striate area
Calcarine sulcus
Lunate sulcus
Frontal gyrus
Superior
Middle
Inferior
Frontal sulcus
Superior
Inferior
Lateral sulcus
Anterior-ramus
Ascending ramus
Lateral sulcus (sylvian sulcus)
8
9
6
4
2
3
5
7
1
10
46
40
44
45
12
11
47
41
18
19
17
22
42
21
20
Superior
Middle
Inferior
Temporal gyrus
Inferior
Superior
Temporal sulcus
Superior
Inferior
Occipital gyrus

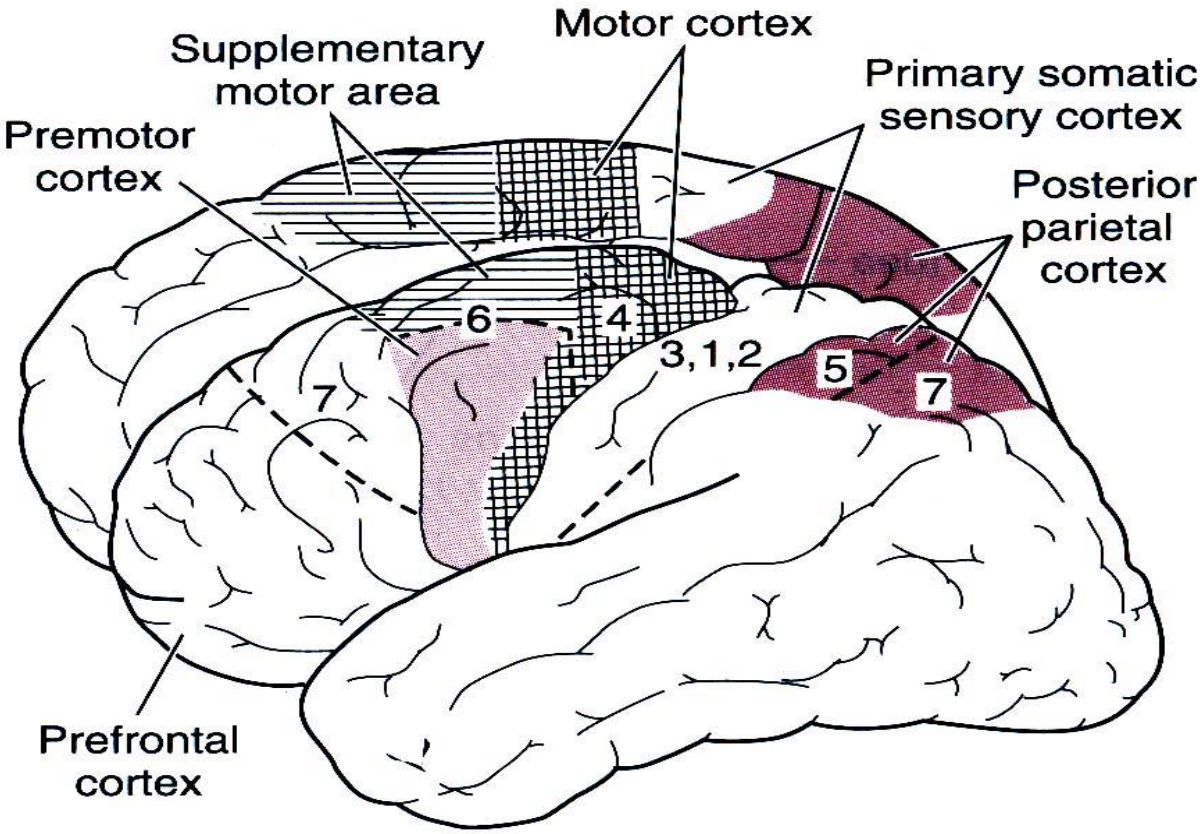


Figure 12–3. Medial (**above**) and lateral (**below**) views of the human cerebral cortex, showing the motor cortex (Brodmann's area 4) and other areas concerned with control of voluntary movement, along with the numbers assigned to the regions by Brodmann. (Reproduced, with permission, from Kandel ER, Schwartz JH, Jessell TM [editors]: *Principles of Neural Science,* 4th ed. McGraw-Hill, 2000.)

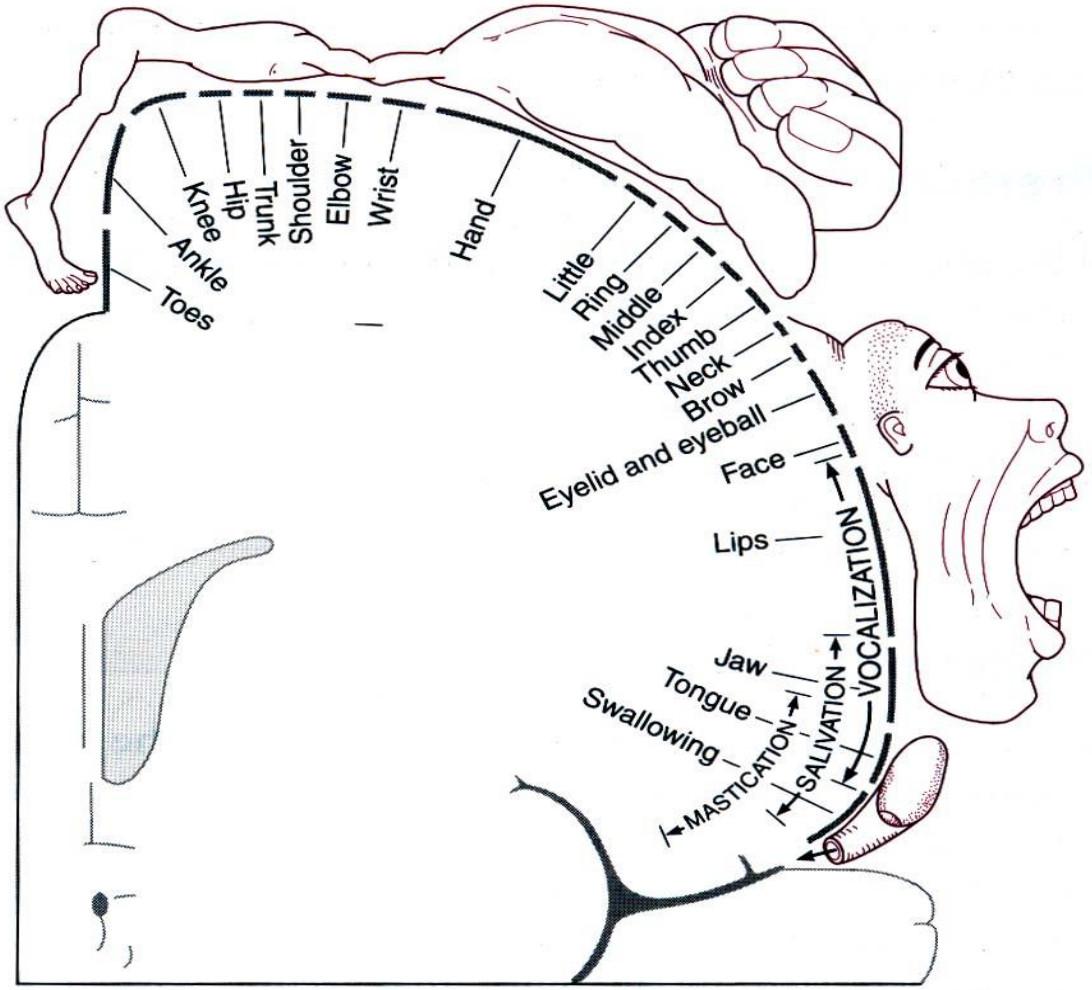


Figure 12–5. **Motor homunculus. The figure represents, on a coronal section of the precentral gyrus, the location of the cortical representation of the various parts. The size of the various parts is proportionate to the cortical area devoted to them. Compare with Figure 7–5.** (Reproduced, with permission, from Penfield W, Rasmussen G: *The Cerebral Cortex of Man.* Macmillan, 1950.)

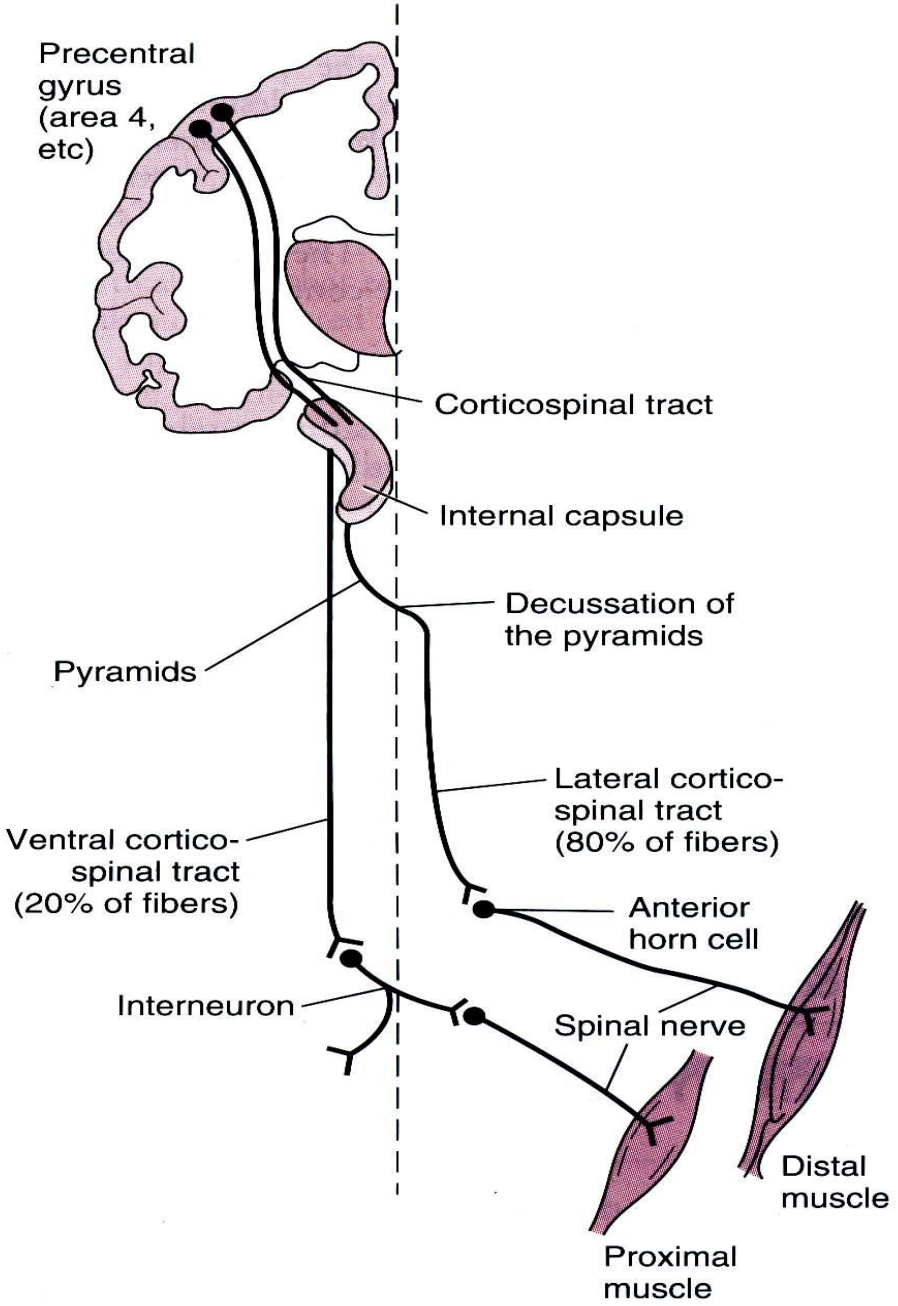
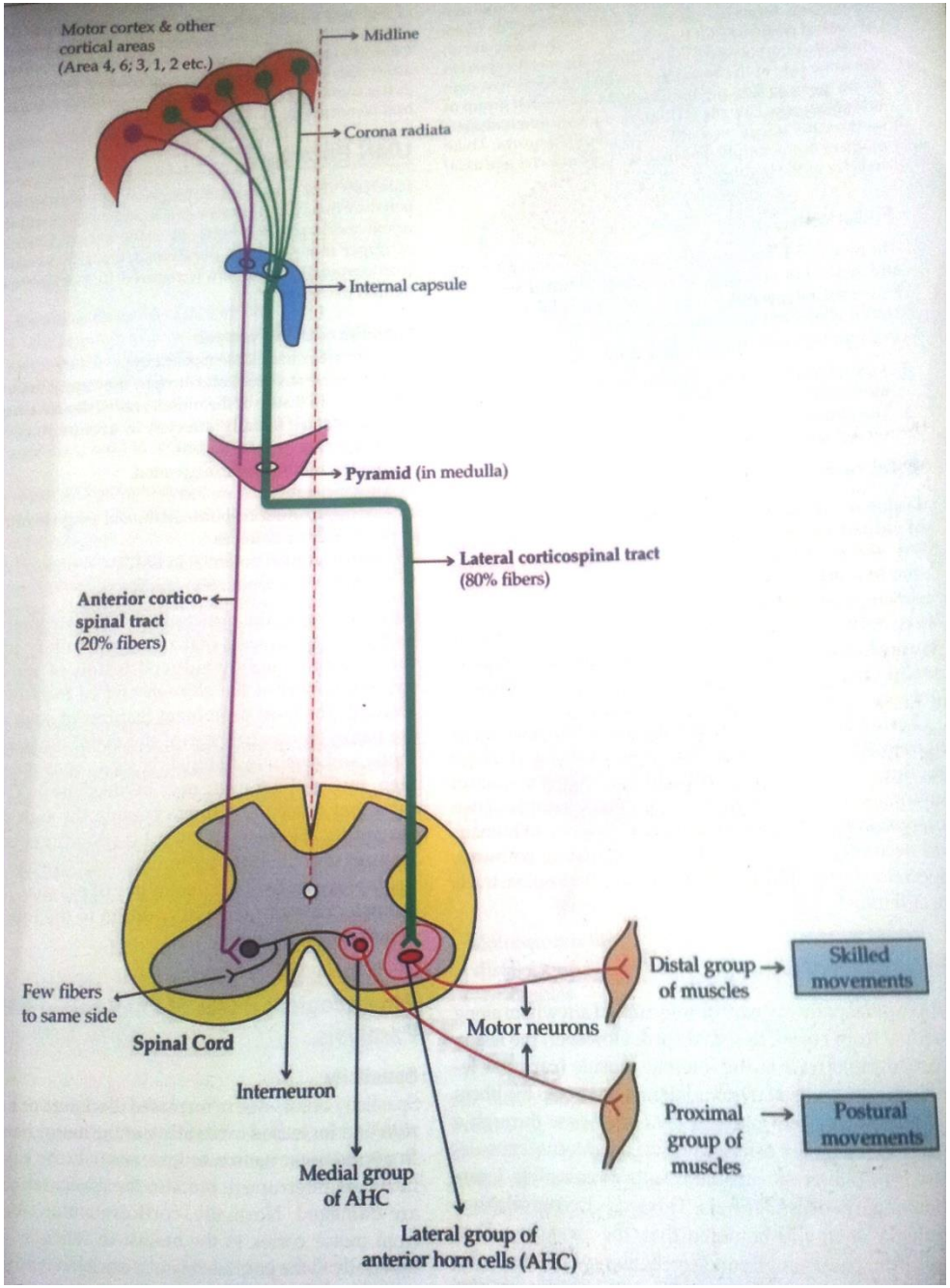
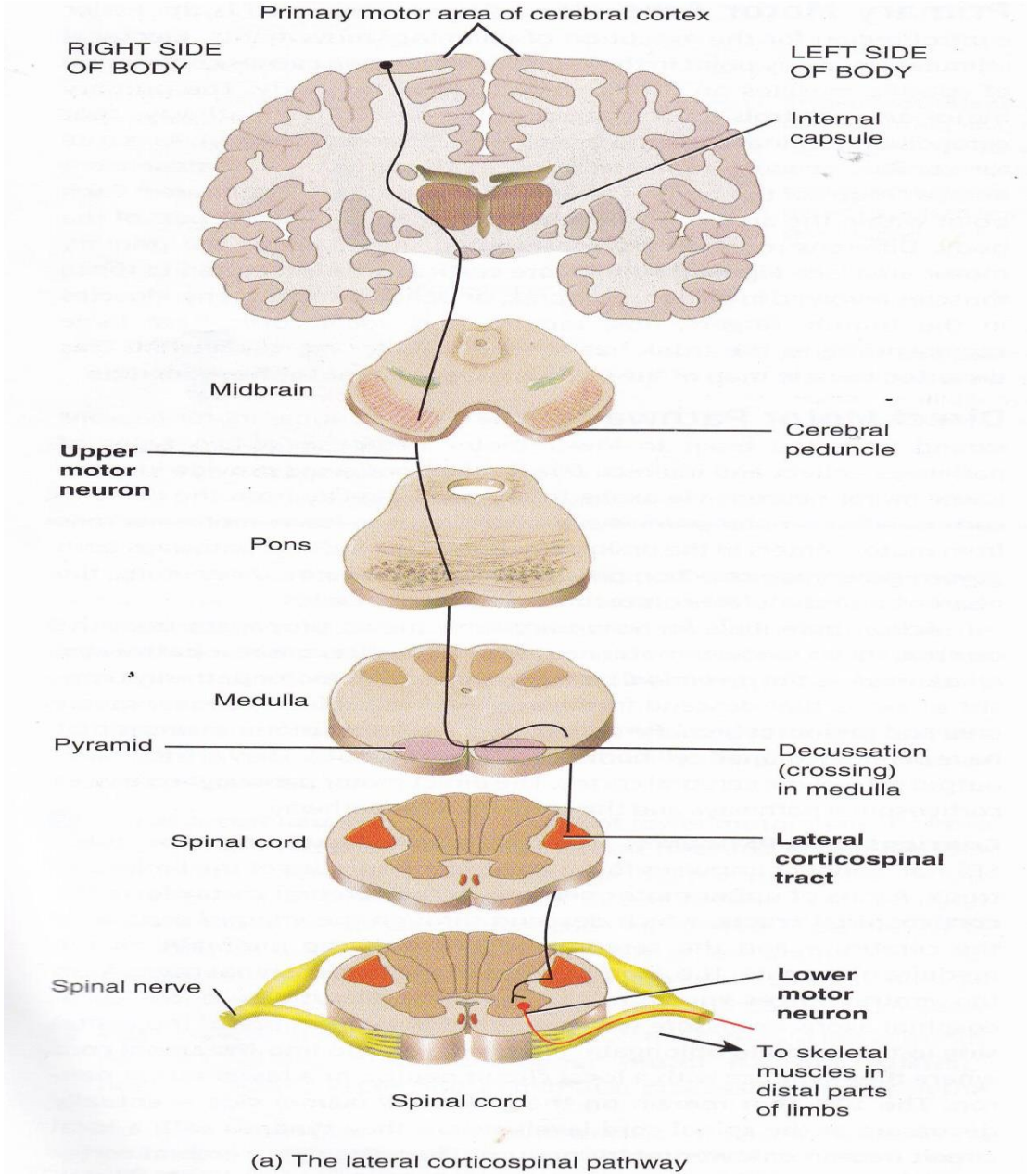


Figure 12–2. The corticospinal tracts.


Motor cortex & other cortical areas (Area 4, 6; 3, 1, 2 etc.)
Midline
Corona radiata
Internal capsule
Pyramid (in medulla)
Lateral corticospinal tract (80% fibers)
Anterior cortico-spinal tract (20% fibers)
Few fibers to same side
Spinal Cord
Interneuron
Medial group of AHC
Lateral group of anterior horn cells (AHC)
Motor neurons
Distal group of muscles
Skilled movements
Proximal group of muscles
Postural movements



(a) The lateral corticospinal pathway

Effects of lesion at different levels of CST

- Motor cortex - Discrete lesions
- Corona Radiata - Monoplegia
- Internal capsule - Contralateral hemiplegia
- Brainstem - Contralateral hemiplegia with cranial nerve involvement
- Below C_5 segment - Quadriplegia
- Thorasic or Lumbar segments – Paraplegia

REFLEXES

- Definition

Reflex is an involuntary, inborn response to a stimulus

1. Clinical classification

- **Superficial reflexes**

  Ex : Skin - Plantar reflex

  mucous membrane – conjunctival reflex

- **Deep reflexes (stretch reflexes)**

  Ex : Biceps jerk, Knee jerk

- **Visceral reflexes**

  Ex : Carotid sinus reflex, Micturition reflex

- **Pathological Reflexes –** Babinski Reflex

2. **Based on number of synapses**

- **Monosynaptic reflexes** Ex: Stretch Reflex
- **Polysynaptic Reflexes**

  Ex: Flexion Withdrawl reflex

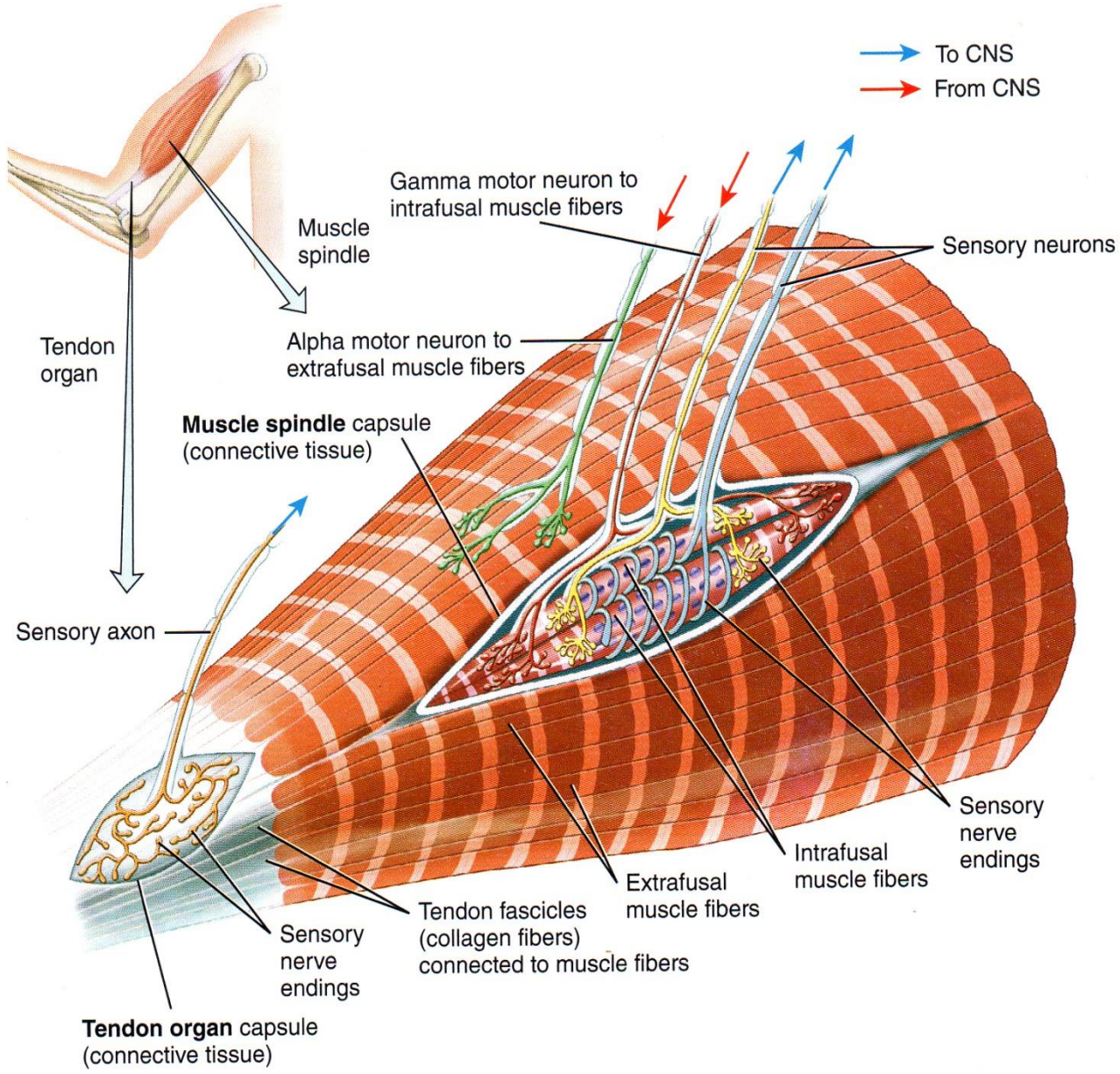
3. **Inborn or Aquired**

- **Inborn or Unconditioned**
- **Aquired or Conditioned**

4. **Physiological classification**

- **Flexor reflexes** Ex: Flexion withdrawl reflex
- **Extensor reflexes** Ex: Stretch reflex

5. **Anatomical -** Cortical, cerebellar, Midbrain, Cord


To CNS
From CNS
Gamma motor neuron to intrafusal muscle fibers
Muscle spindle
Sensory neurons
Tendon organ
Alpha motor neuron to extrafusal muscle fibers
Muscle spindle capsule (connective tissue)
Sensory axon
Sensory nerve endings
Intrafusal muscle fibers
Extrafusal muscle fibers
Tendon fascicles (collagen fibers) connected to muscle fibers
Sensory nerve endings
Tendon organ capsule (connective tissue)

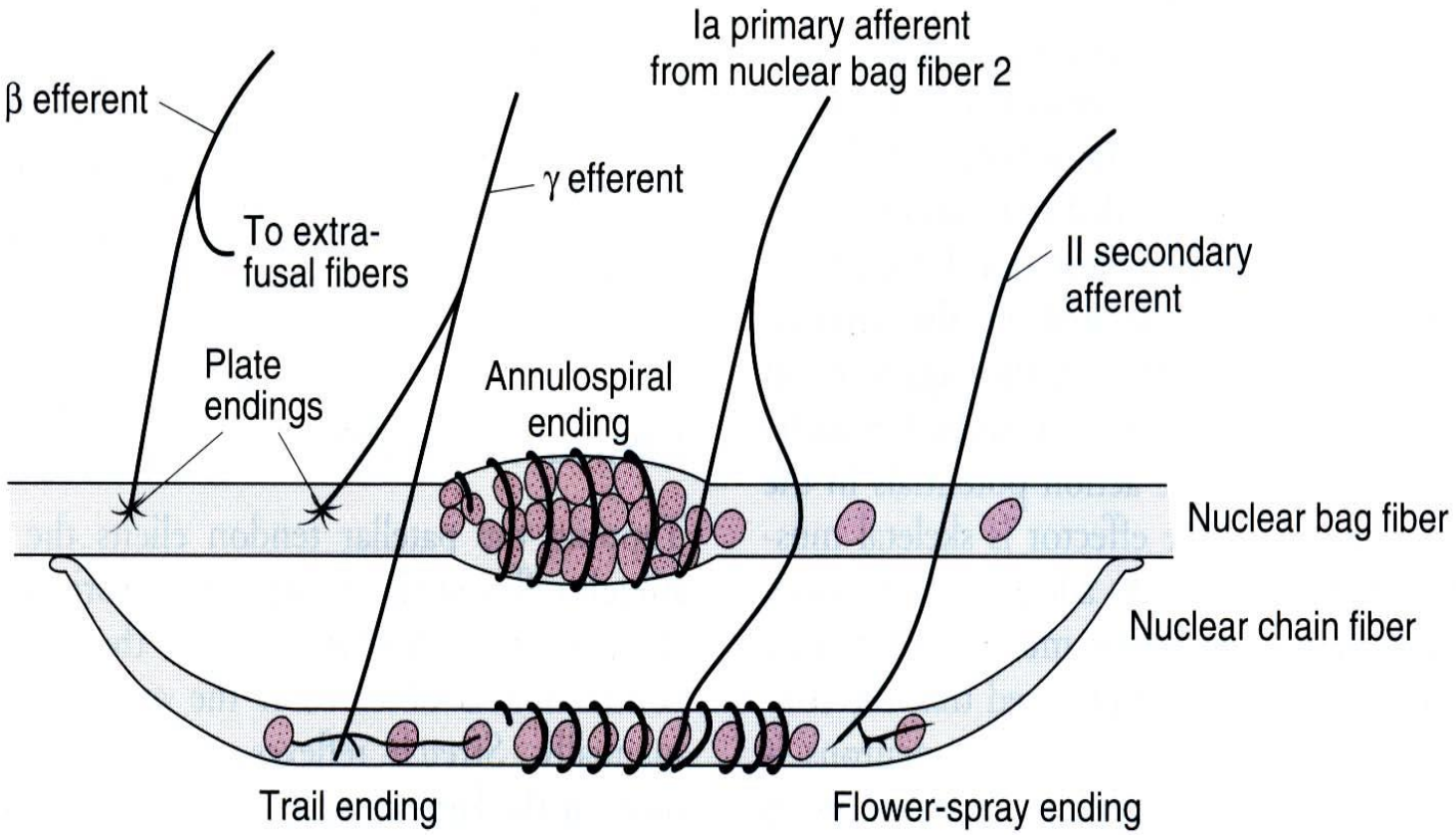


Figure 6–2. Diagrammatic representation of the main components of mammalian muscle spindle. Each spindle has a capsule and usually contains two nuclear bag fibers and four or more nuclear chain fibers.

Intrafusal fibers

Nuclear Bag Fibers

Nuclear Chain Fibers

Sensory Innervation

- Ia for NBF & NCF– Central Annulospiral Endings

- II for NCF - Peripheral Flower Spray Endings

Motor supply

γ motor to NBF – peripheral Plate Endings

γ motor to NCF – Peripheral Trail Endings

Dynamic and Static Response

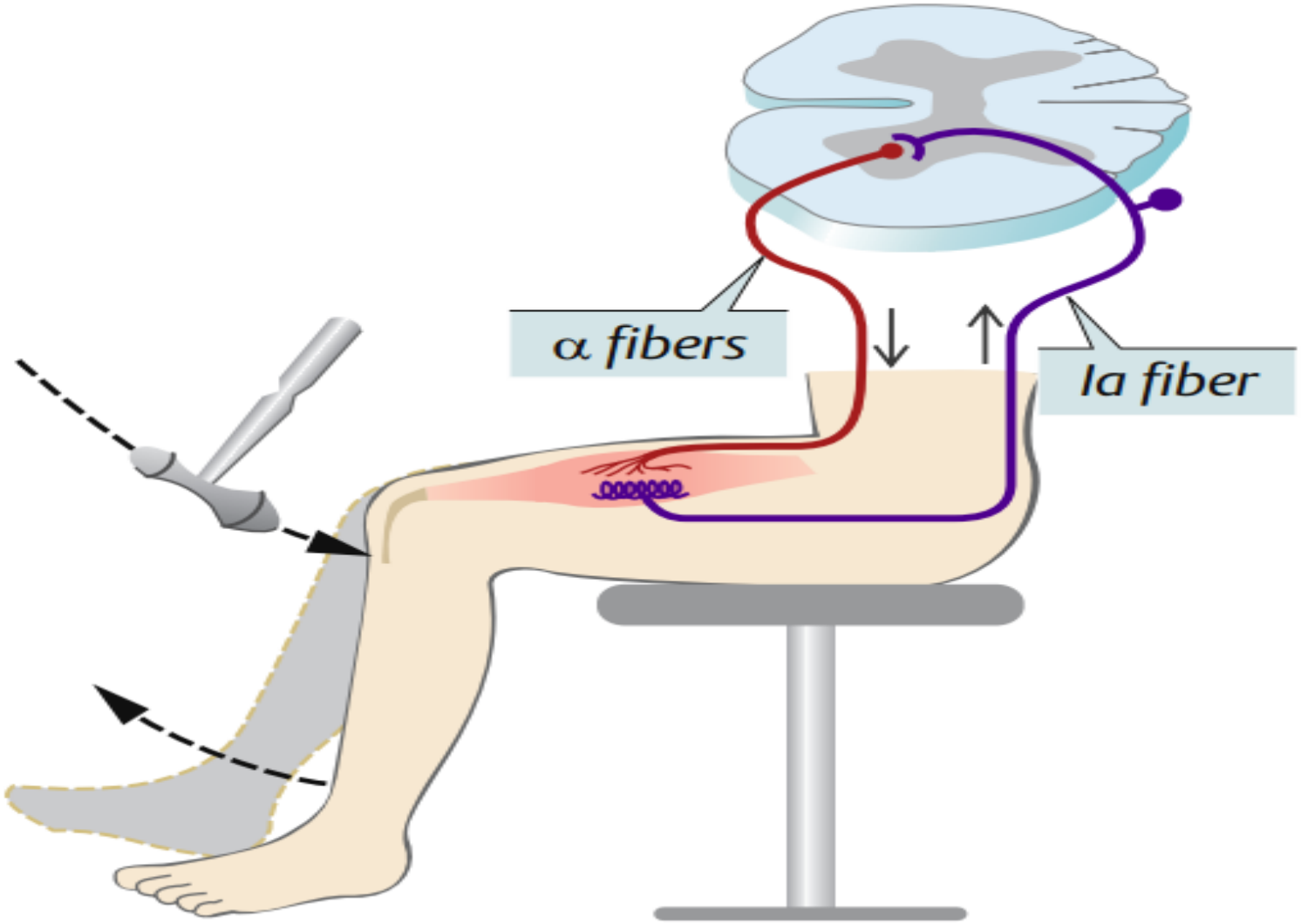
- **Stretch reflex**

Deep reflex, Tendon jerk, Monosynaptic reflex, Myotatic reflex

Stimulus : stretch of the muscle

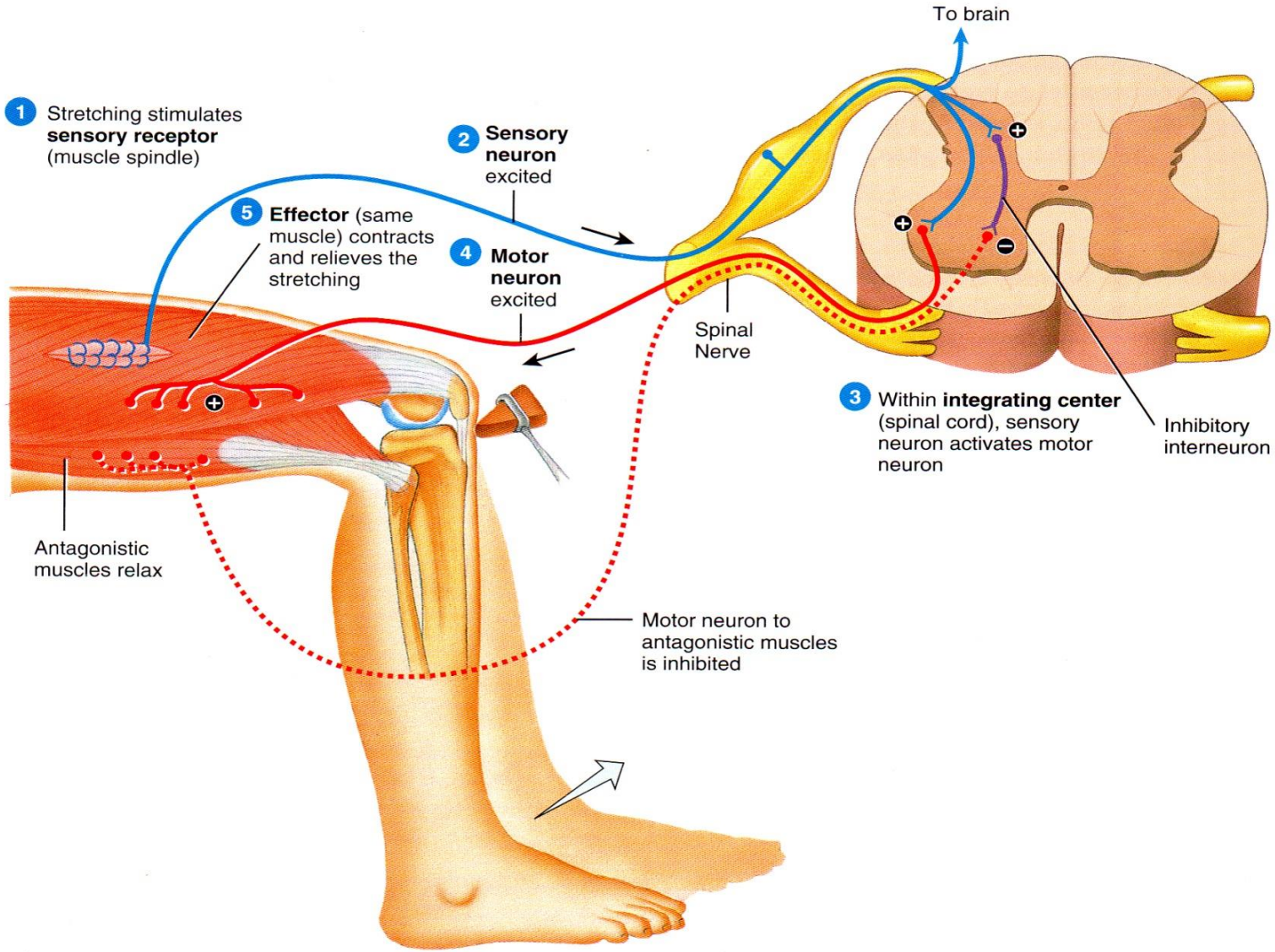
Response : Contraction of the muscle

C. Monosynaptic stretch reflex



Stimulus	**Stretch of the muscle**
	↓
Receptor	**Muscle spindle**
	↓
Afferenents	**Ia and II fibers**
	↓
Center	**Spinal cord**
	↓
Efferents	**α (Alpha) motor neurons**
	↓
Effector	**Skeletal muscle (extrafusal)**
	↓
Response	**Contraction of the muscle**

The stretch reflex causes contraction of a muscle that has been stretched.



Q What makes this an ipsilateral reflex?

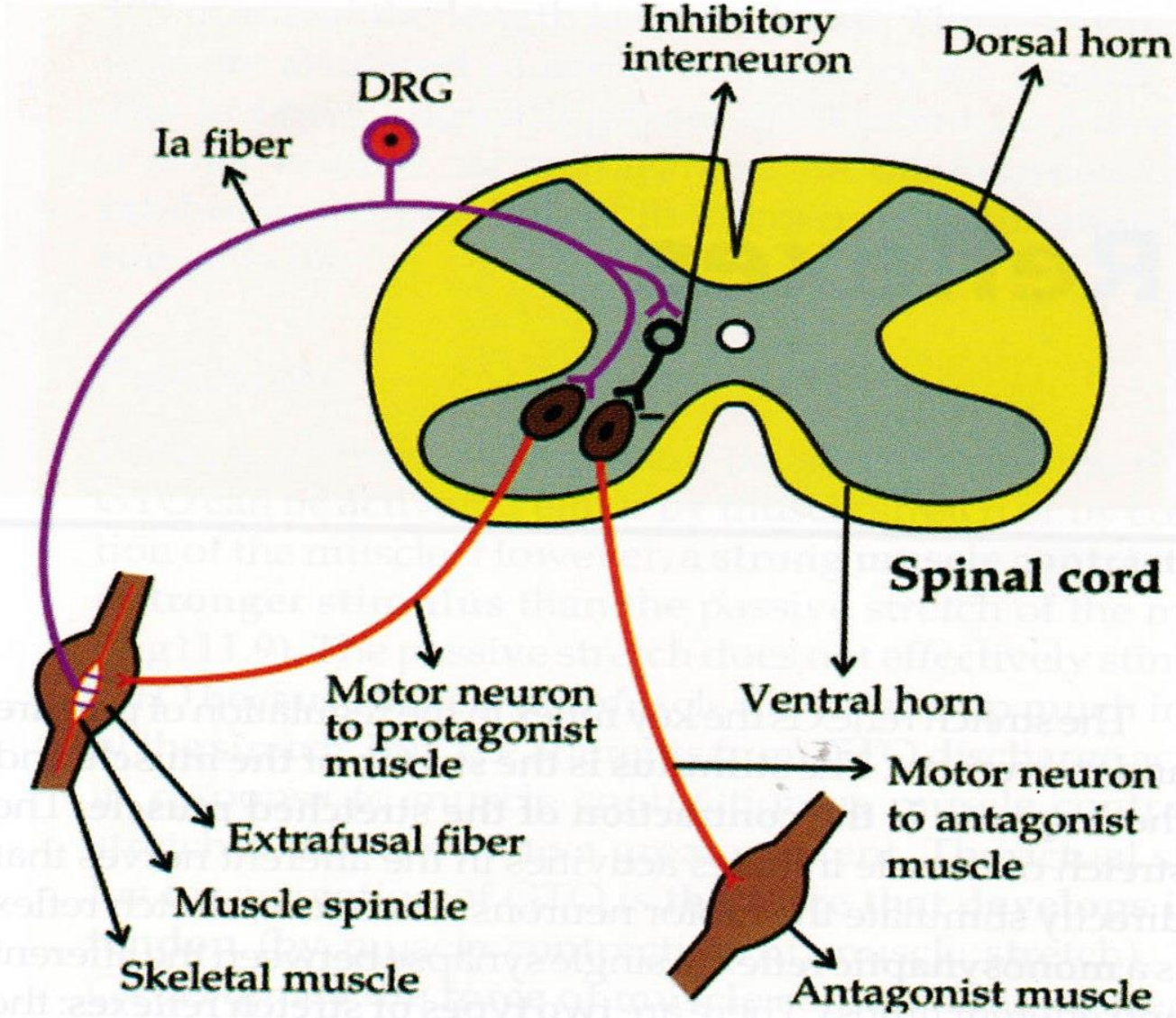


Figure 112.2 Schematic representation of a stretch reflex.

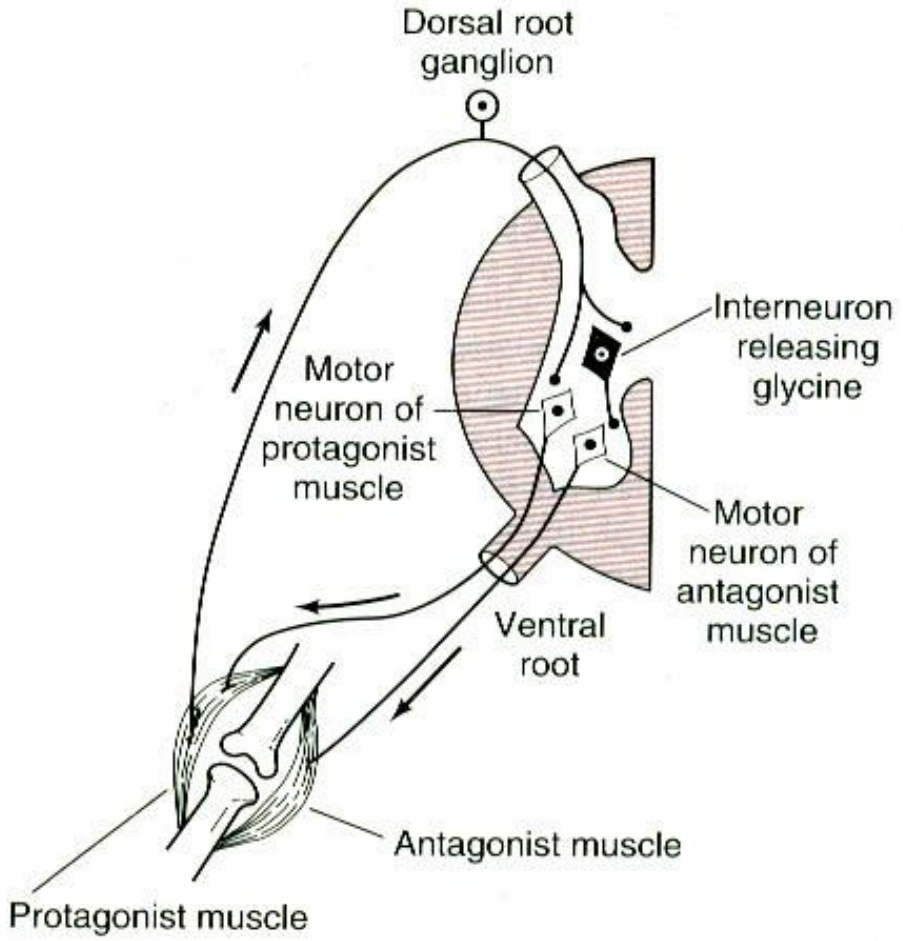


Figure 4–10. Diagram illustrating the anatomic connections responsible for inhibiting the antagonists to a muscle contracting in response to stretch. Activity is initiated in the spindle in the protagonist muscle. Impulses pass directly to the motor neurons supplying the same muscle and, via branches, to inhibitory interneurons that end on the motor neurons of the antagonist muscle.

- Direct muscle contraction through **α (Alpha)** motor neurons
- Indirect muscle contraction through **γ** motor neurons

α – γ co- activation

- γ motor neurons are controlled by Reticulo spinal tract
- UMNL – reflexes are exaggerated
- LMNL – reflexes are lost
- **Jendrassik maneuver**

- Muscle tone

  Sustained partial contraction of muscle at rest

  Tone depends on “γ” discharge

UMNL - Hypertonia - Spasticity or Rigidity

LMNL - Hypotonia - Flaccidity

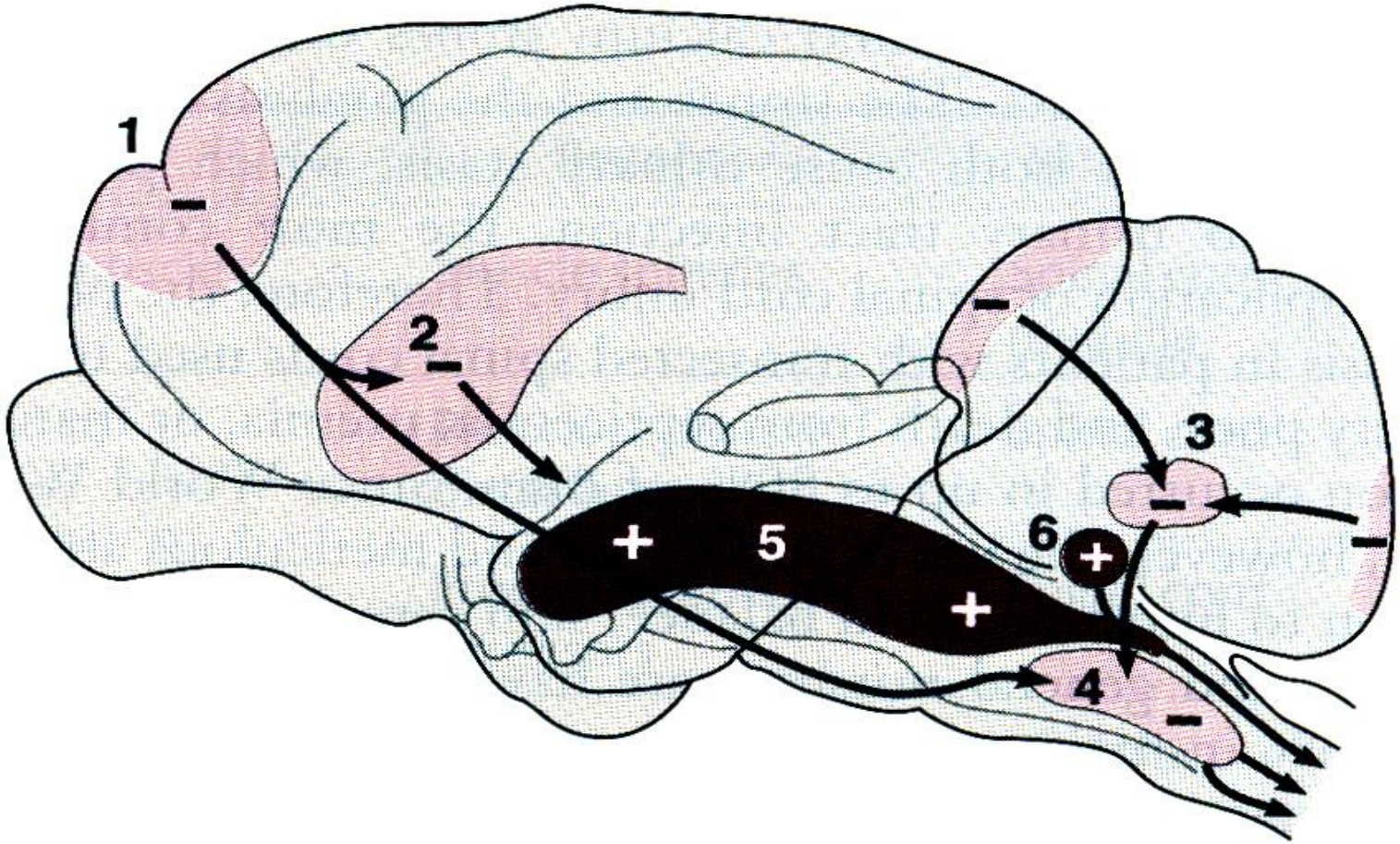


Figure 12–6. Areas in the cat brain where stimulation produces facilitation (plus [+] signs) or inhibition (minus [–] signs) of stretch reflexes. 1, motor cortex; 2, basal ganglia; 3, cerebellum; 4, reticular inhibitory area; 5, reticular facilitatory area; 6, vestibular nuclei.

INVERSE STRETCH REFLEX

- Stimulus : strong stretch
- Receptor : Golgi tendon organ
- Response : Relaxation of the muscle

AUTOGENIC INHIBITION

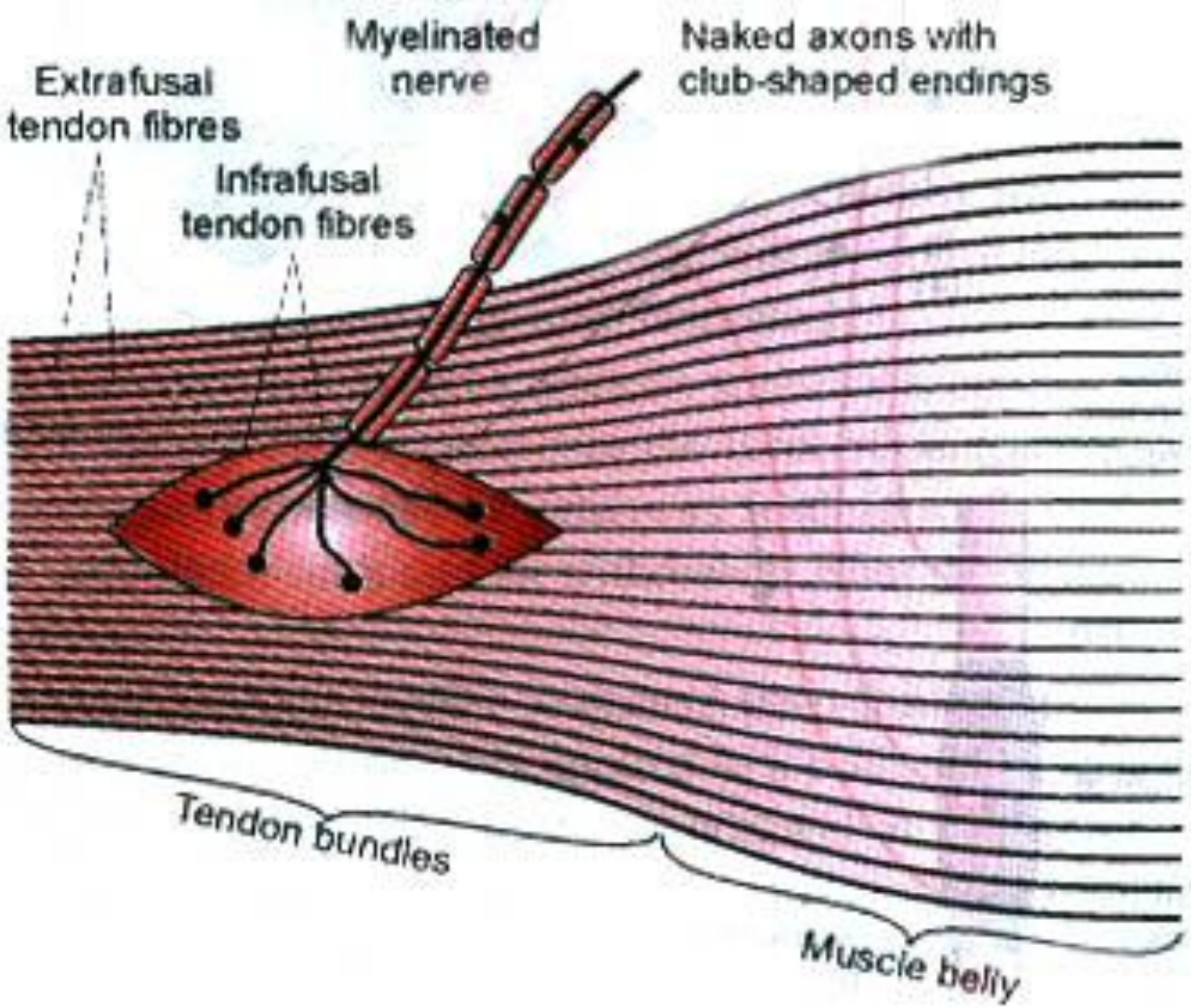
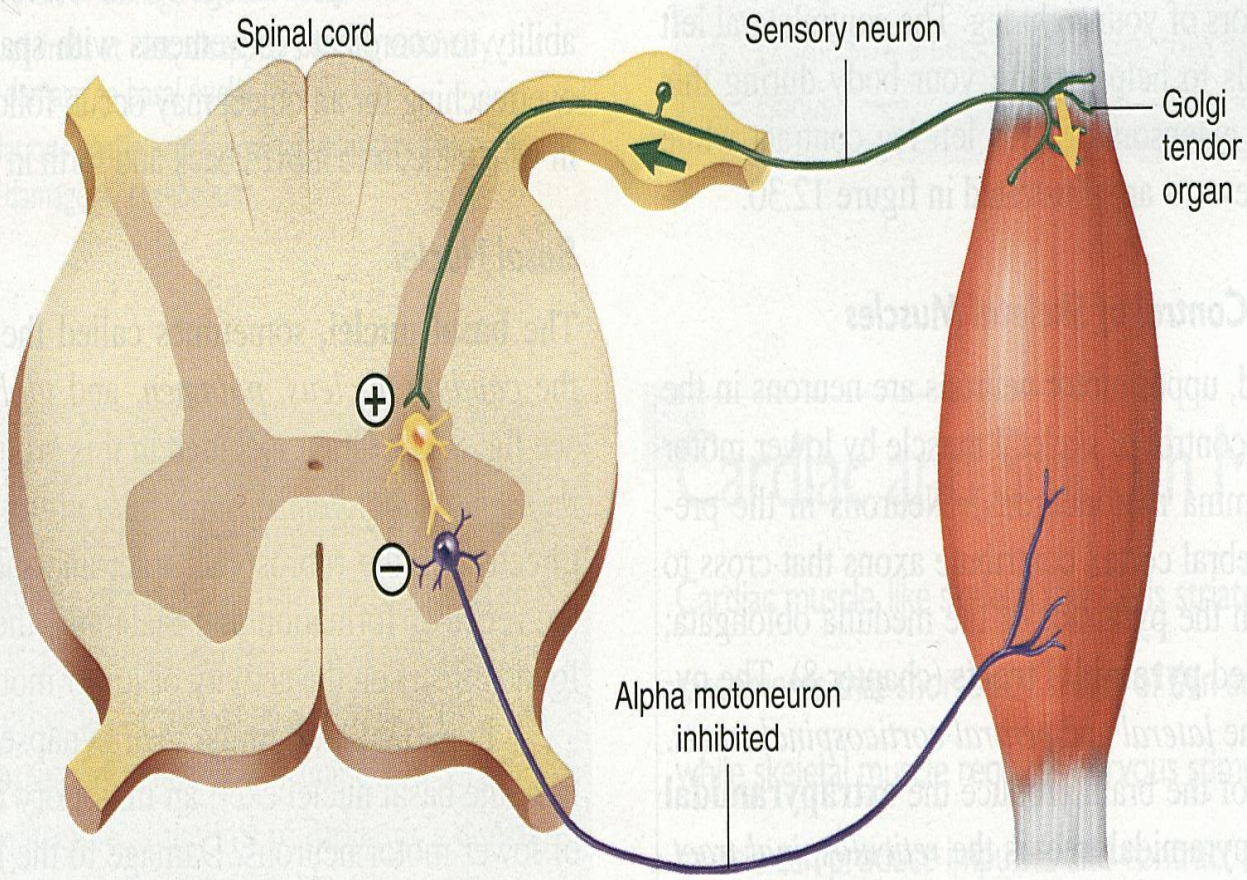


Fig. 10.9-14. *Structure of Golgi tendon organ.*



ure 12.28 **The action of the Golgi tendon organ.** An increase in muscle tension stimulates the activity of sensory nerve endings in the Golgi organ. This sensory input stimulates an interneuron, which in turn inhibits the activity of a motor neuron innervating that muscle. This is therefore a tic reflex.

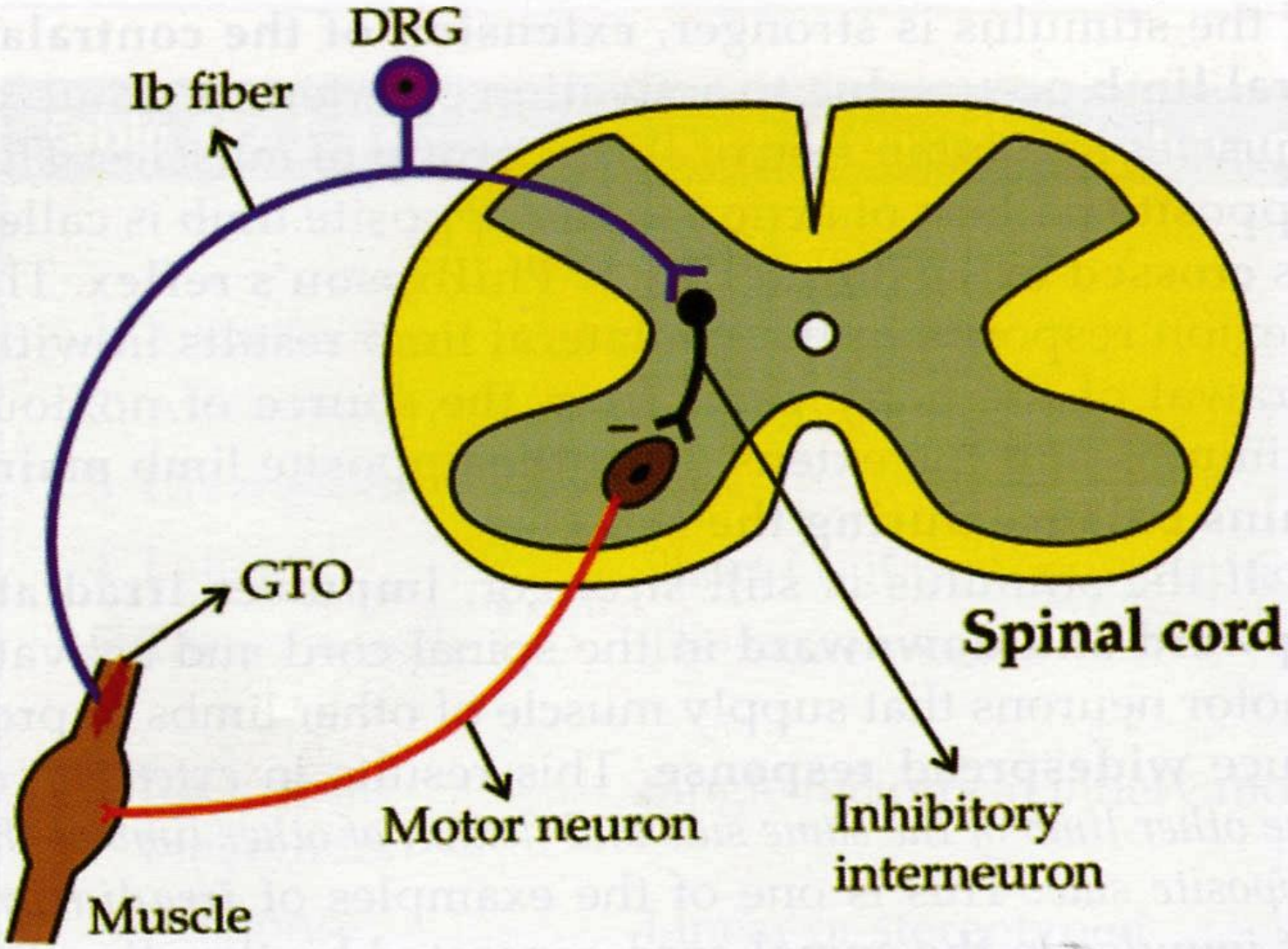
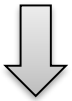
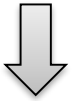


Figure 112.3 Schematic representation of an inverse stretch reflex.

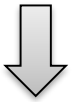
STIMULUS **[VERY STRONG STRECH**].



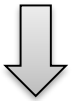
RECEPTOR **[GOLGI TENDON ORGAN].**



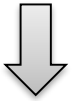
AFFERENT **[Ib FIBERS].**



COORDINATING CENTER **[SPINAL CORD].**

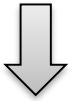


INHIBITORY INTERNEURON

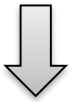


GLYCINE

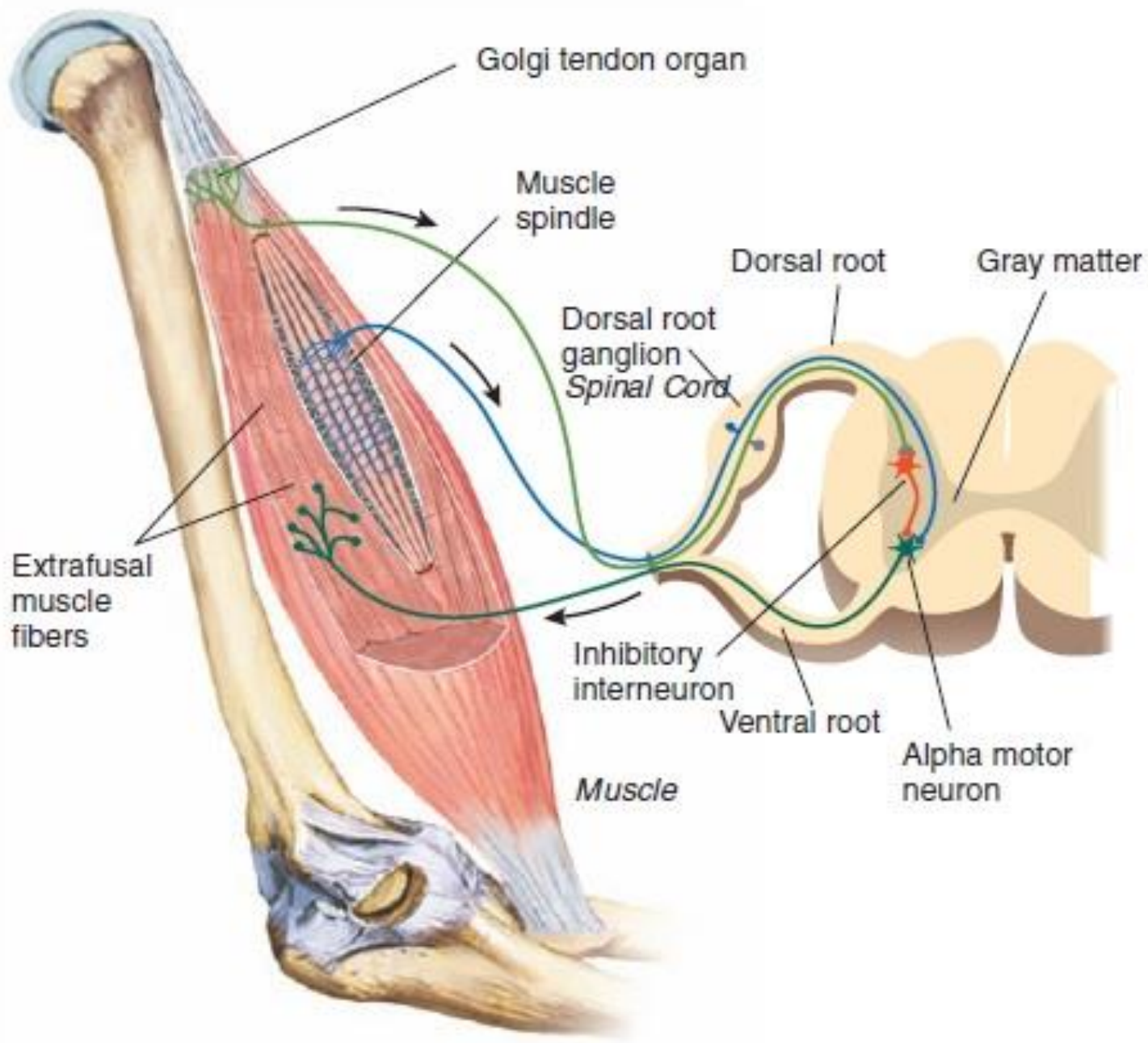
EFFERENT **[α MOTOR NEURON].**

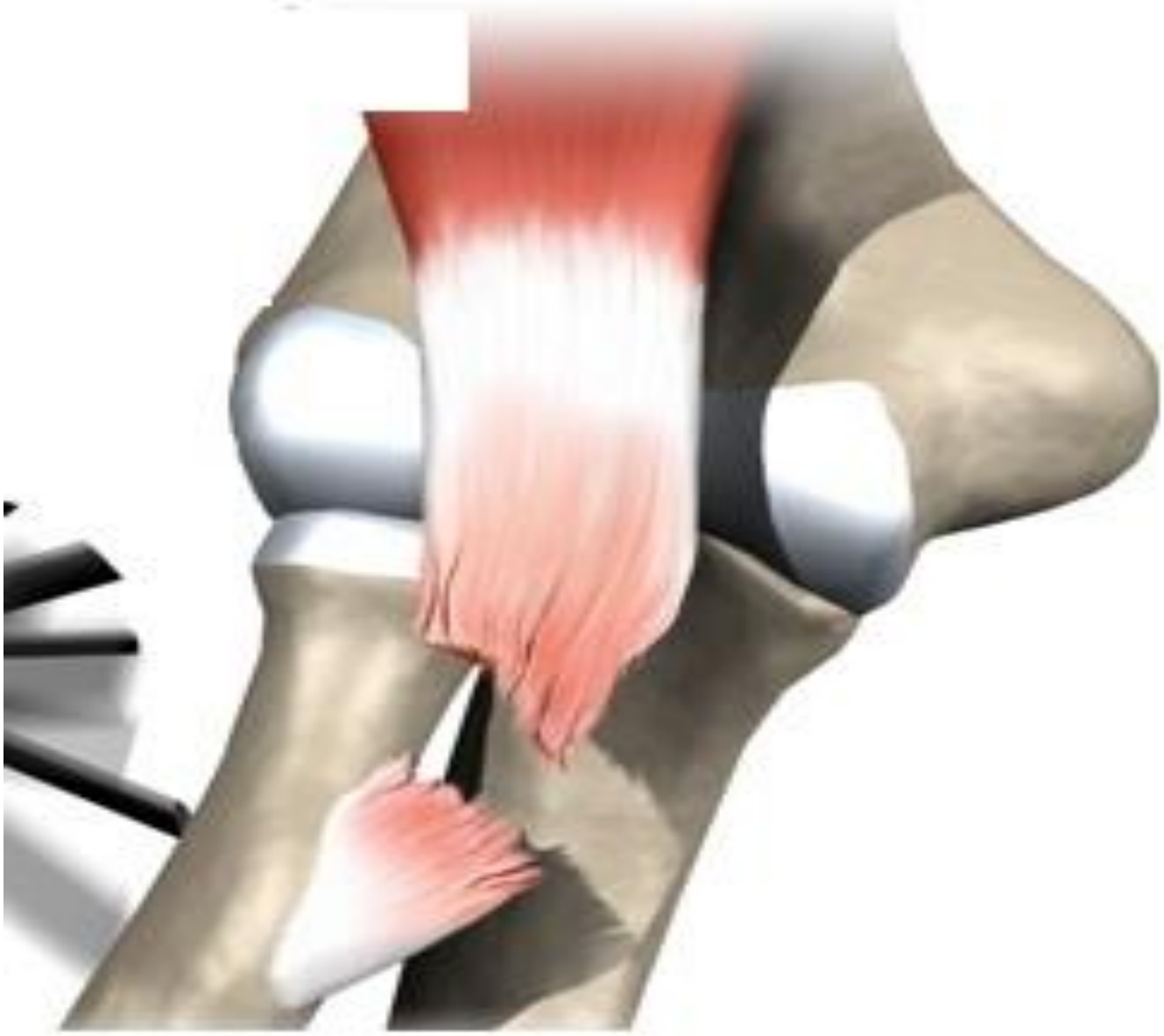


EFFECTOR ORGAN **[AGONIST SKELETAL MUSCLE].**



EFFECT **[RELAXATION BY INHIBITION].**


Golgi tendon organ
Muscle spindle
Dorsal root
Gray matter
Dorsal root ganglion
Spinal Cord
Extrafusal muscle fibers
Inhibitory interneuron
Ventral root
Alpha motor neuron
Muscle



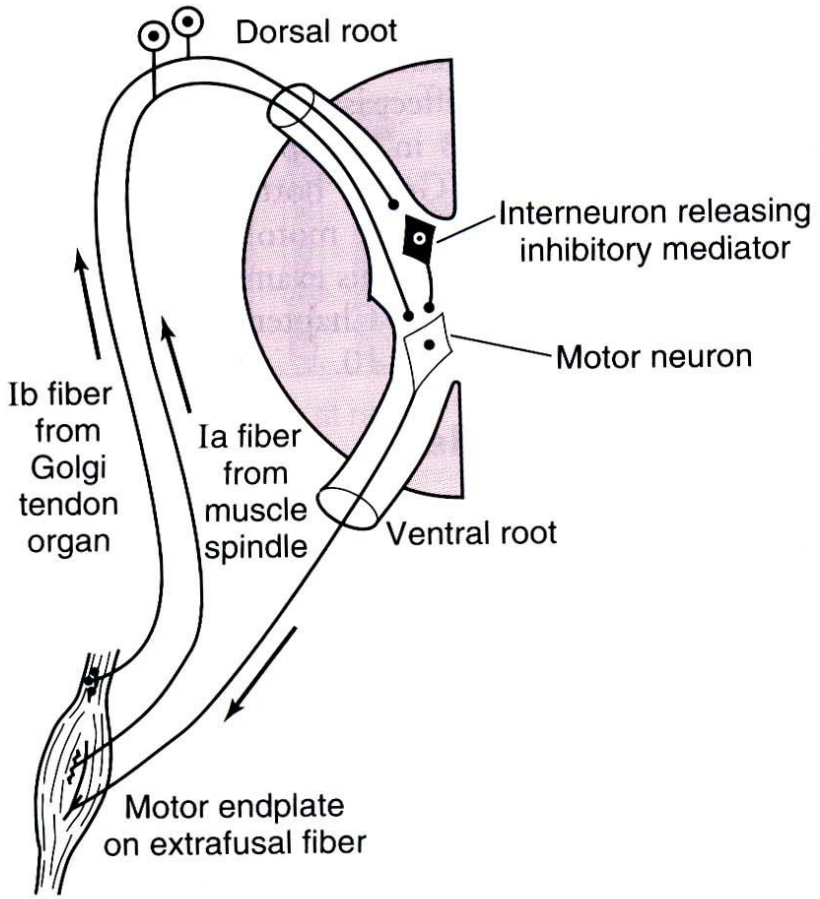


Figure 6–6. Diagram illustrating the pathways responsible for the stretch reflex and the inverse stretch reflex. Stretch stimulates the spindle, and impulses pass up the Ia fiber to excite the motor neuron. It also stimulates the Golgi tendon organ, and impulses passing up the Ib fiber activate the interneuron to release the inhibitory mediator glycine. With strong stretch, the resulting hyperpolarization of the motor neuron is so great that it stops discharging.

- **Clasp Knife Effect (Lengthening Reaction)**

- **Clonus (Patellar, Ankle)**

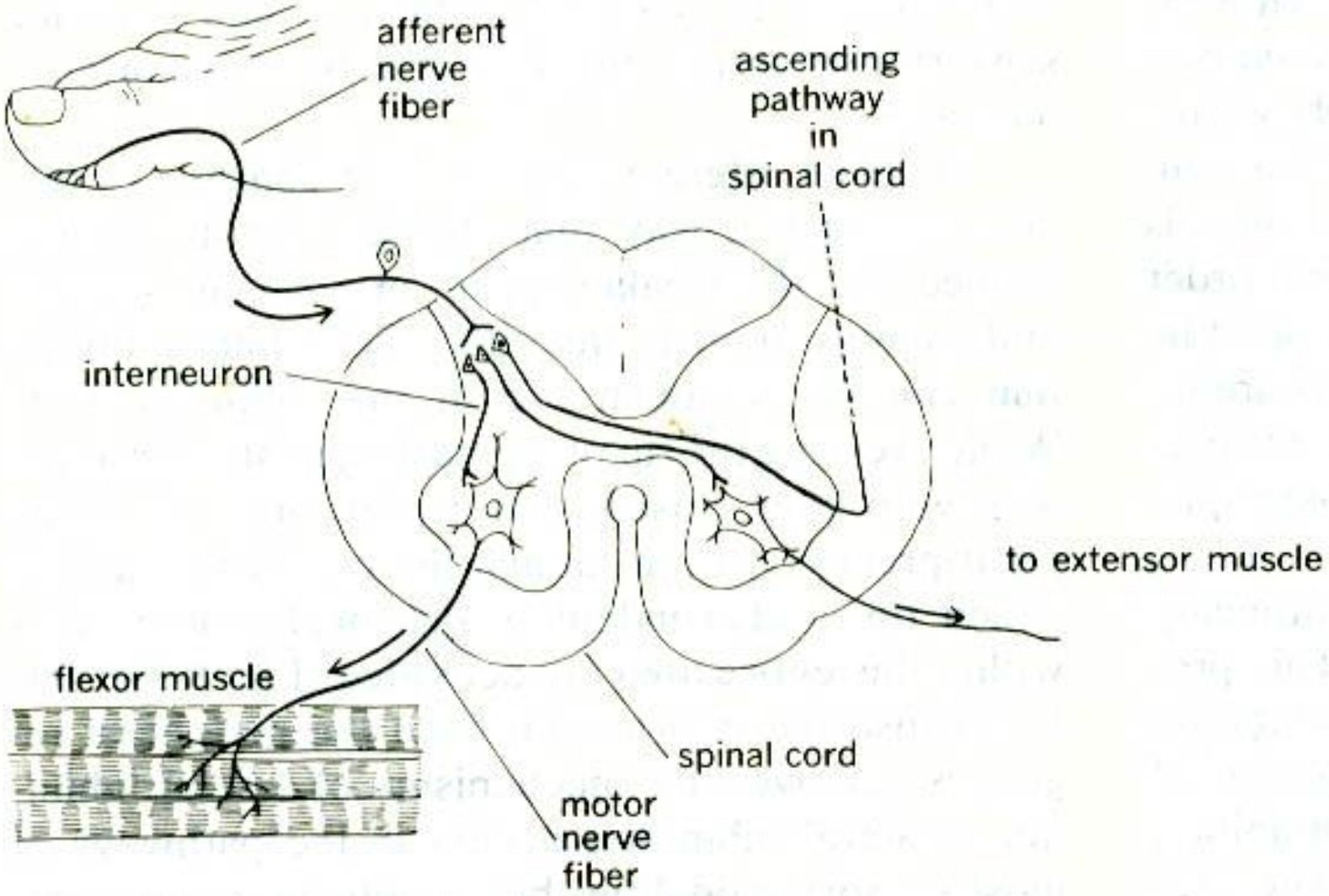
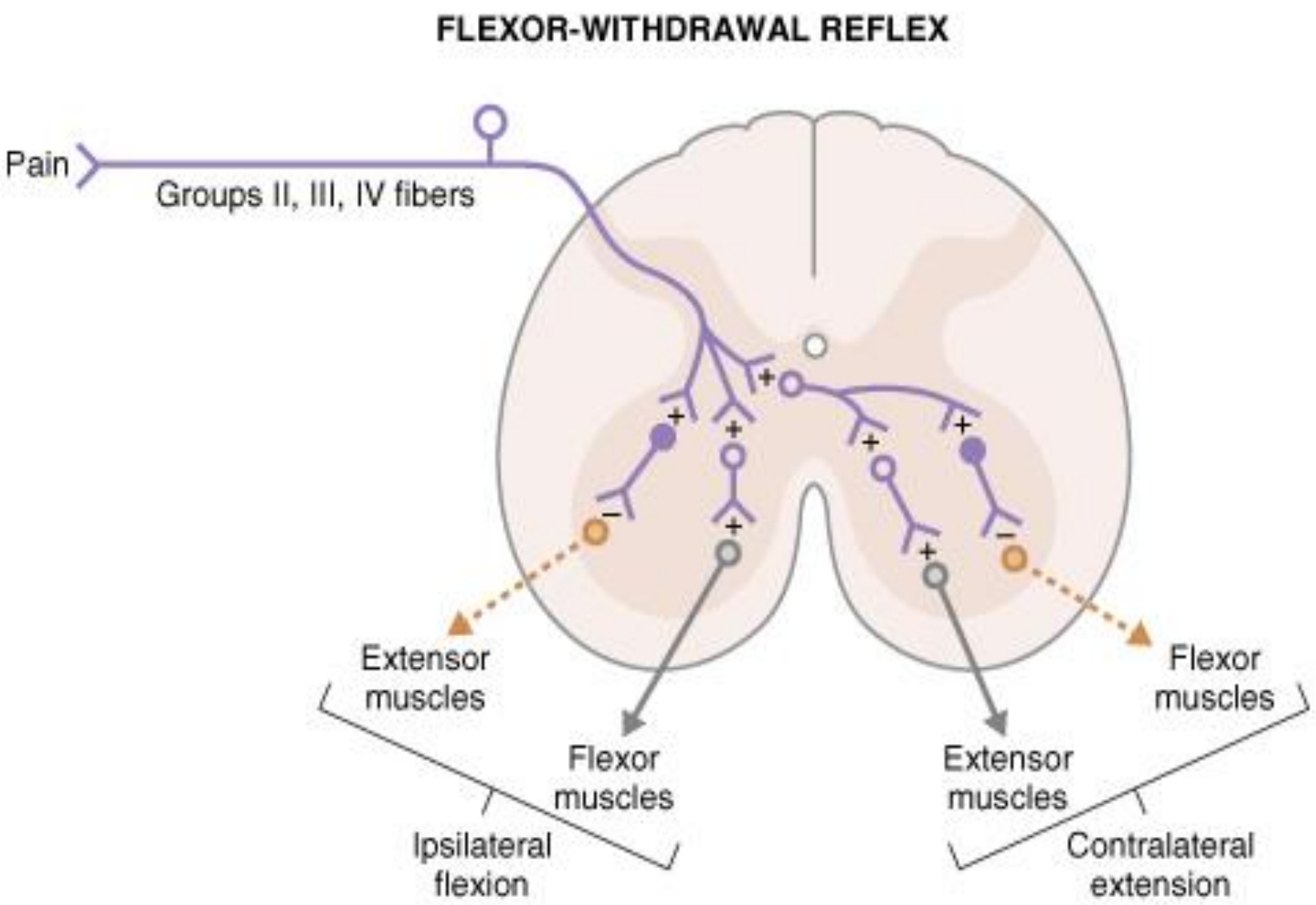


FIGURE 8-60. Flexion, or withdrawal, reflex pathway. Arrows indicate direction of action potential propagation.


FLEXOR-WITHDRAWAL REFLEX
Pain
Groups II, III, IV fibers
Extensor muscles
Flexor muscles
Ipsilateral flexion
Flexor muscles
Extensor muscles
Contralateral extension

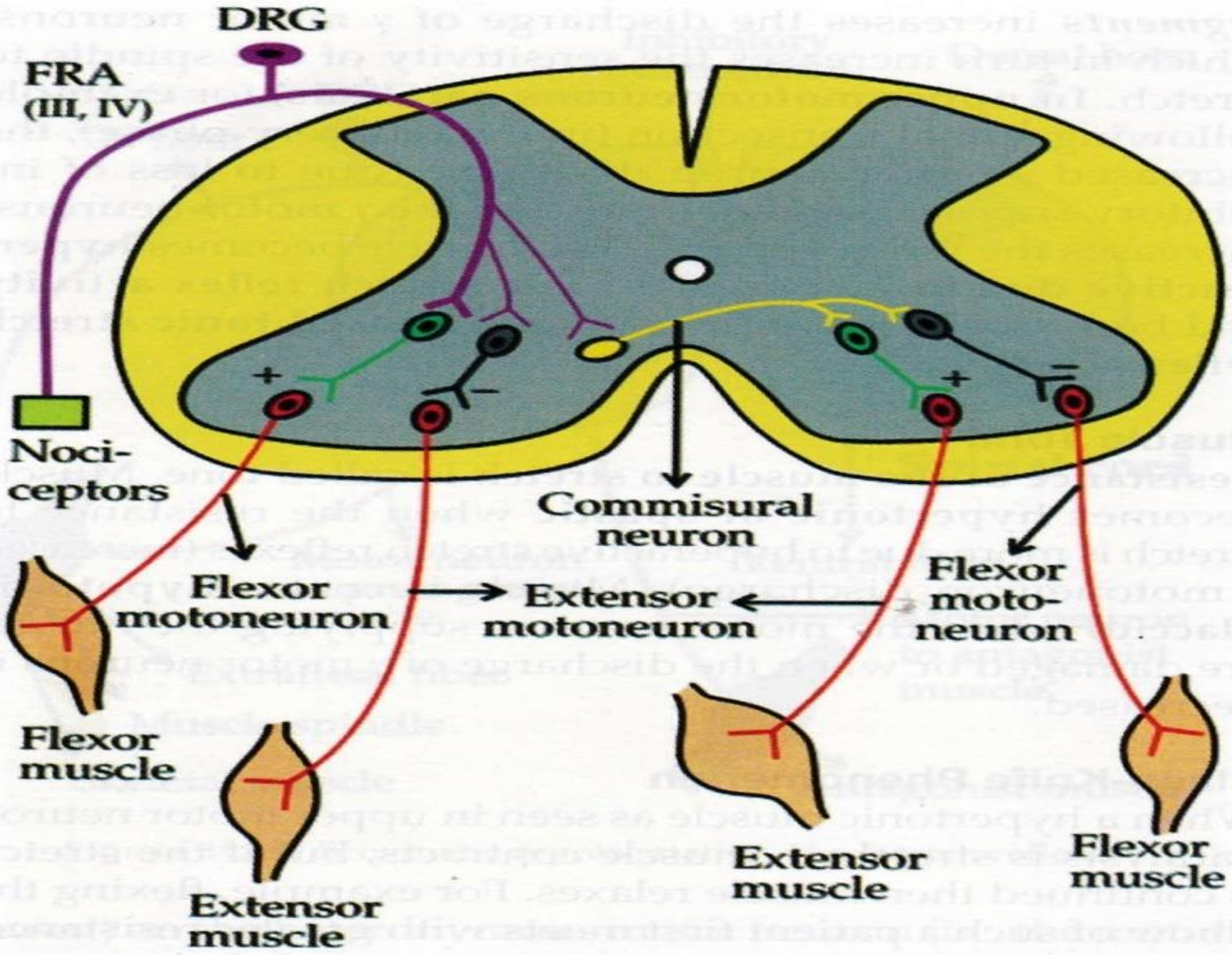


Figure 112.4 Schematic representation of a withdrawal reflex. FRA: flexion reflex afferents.

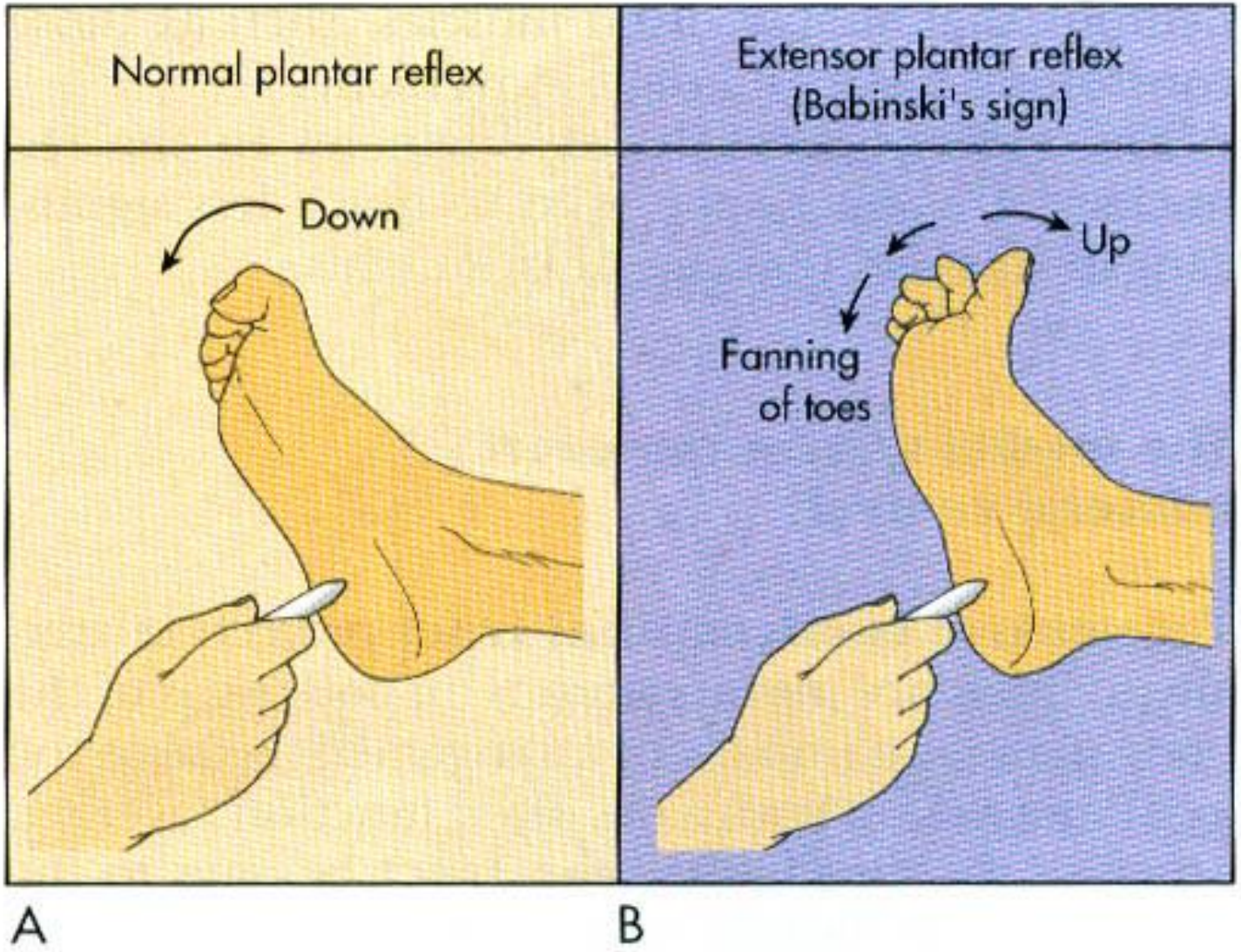


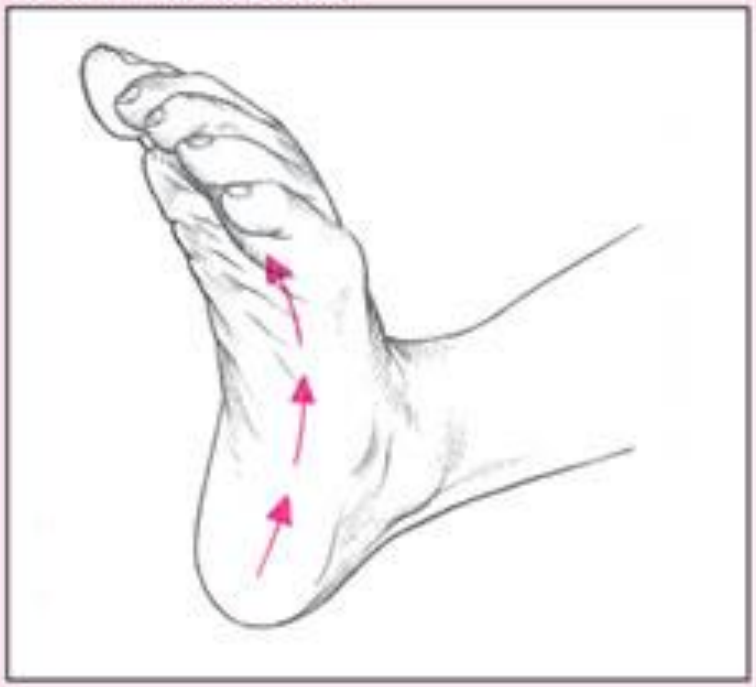
FIGURE 9–16. Babinski's sign. A, The normal response to stroking the plantar surface of the foot. **B,** Babinski's sign (extensor plantar reflex) in a person with interruption of the corticospinal tract.

EXAMINATION TIP

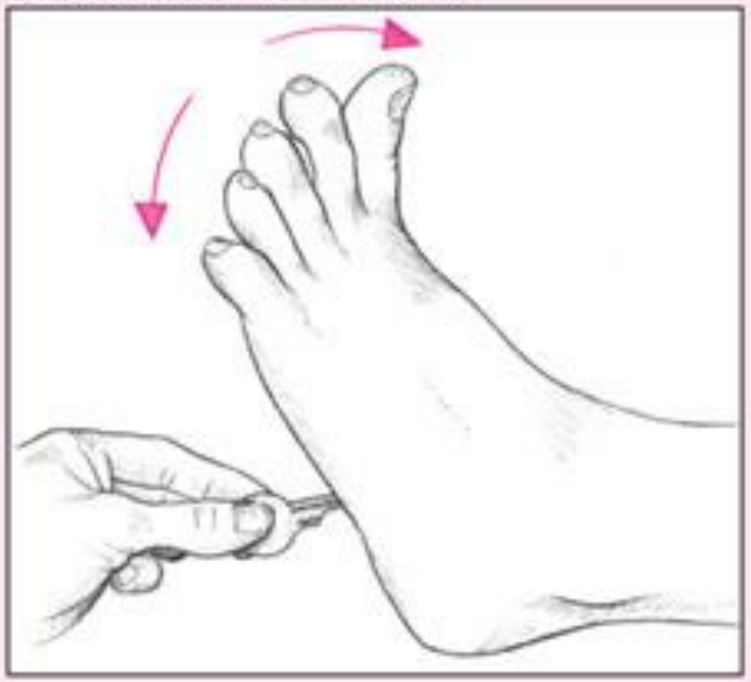
How to elicit Babinski's reflex

To elicit Babinski's reflex, stroke the lateral aspect of the sole of the patient's foot with your thumbnail or another moderately sharp object. Normally, this elicits flexion of all toes (a negative Babinski's reflex), as shown below in the left illustration. With a positive Babinski's reflex, the great toe dorsiflexes and the other toes fan out, as shown in the right illustration.

NORMAL TOE FLEXION



POSITIVE BABINSKI'S REFLEX



Properties of reflexes

- Adequate stimulus
- Central delay
- Convergence, Divergence, Summation, Occlusion, Sub liminal fringe
- **Prepotent**
- **After Discharge**
- **Irradiation**
- **Local sign**

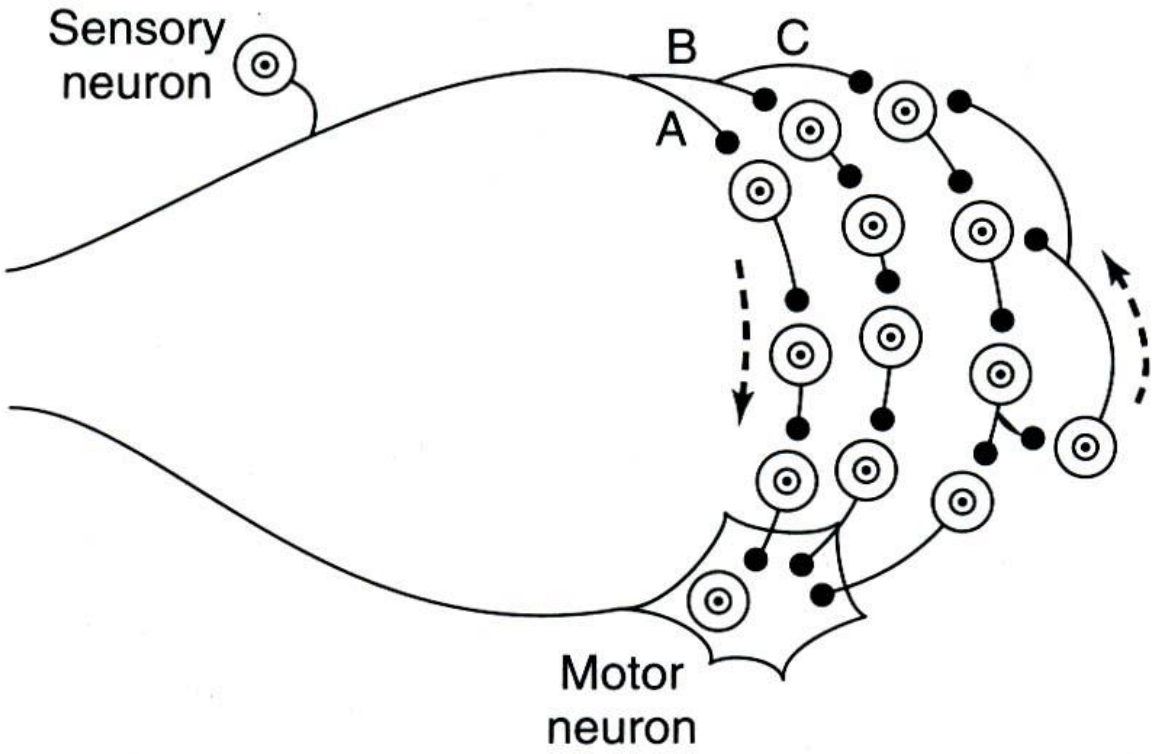
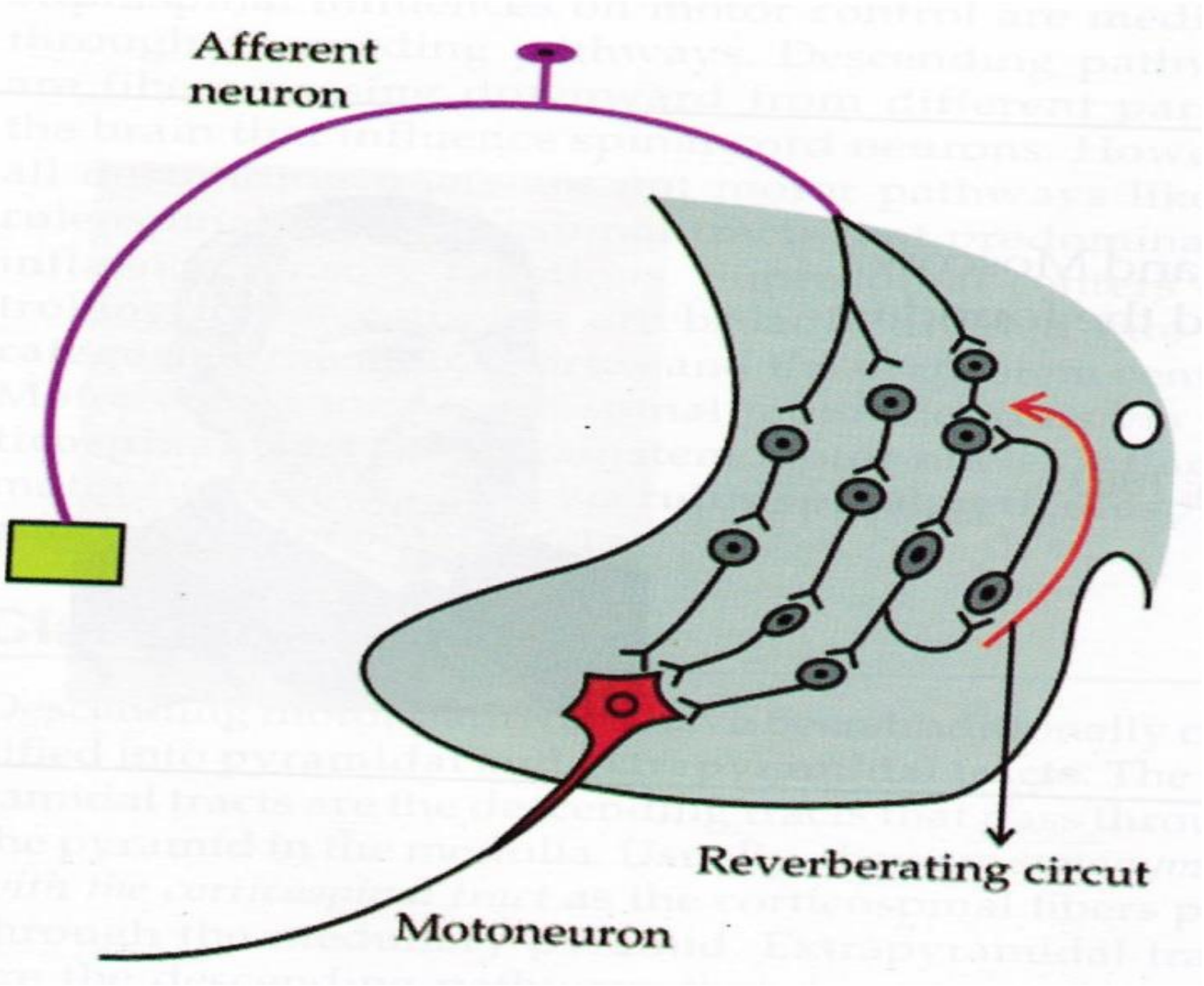


Figure 6–8. Diagram of polysynaptic connections between afferent and efferent neurons in the spinal cord. The dorsal root fiber activates pathway A with three interneurons, pathway B with four interneurons, and pathway C with four interneurons. Note that one of the interneurons in pathway C connects to a neuron that doubles back to other interneurons, forming reverberating circuits.


Afferent
neuron
Reverberating circut
Motoneuron

Sl. No.	Parameter	UMN Lesion (Spastic paralysis)	LMN Lesion (Flaccid paralysis)
1.	Definition	Lesion of C.S.T. from origin in C.C. Upto its termination in anterior horn cell of spinal cord	Lesion of anterior horn cell. or motor nerve (Peripheral Nerve) to skeletal muscle
2.	Common site of lesion	At the level of internal capsule (Stroke)	Anterior horn cell –Polio Peripheral nerve – Trauma
3.	Bulk of muscle	No change in size or bulk	Atrophy
4.	Power of muscle	Decreased (Paresis)	Lost (Plegia)
5.	Tone	Increased (Spasticity) (Hypertonia)	Decreased (Flaccidity) (Hypotonia)
6.	Superficial reflexes	Lost	Lost

Sl. No.	Parameter	UMN Lesion (Spastic paralysis)	LMN Lesion (Flaccid paralysis)
7.	Plantar reflex	Babinski's sign (Plantar extensor)	Babinski sign (Plantar flexor)
8.	Deep reflexes	Exaggerated	Lost
9.	Clasp knife reflex	Present	Absent
10.	Patellar and Ankle clonus	Present	Absent
11.	Muscles affected	Group of muscles	Individual muscles
12.	Fibrillations	Absent	Present
13.	Fasciculations	Absent	Present
14.	Nerve conduction studies	Normal	Abnormal

Complete Transection of Spinal cord

- **Common causes**
  - Gunshot injuries
  - Occlusion of blood vessels
- Stages
  1. Stage of **Spinal Shock**
  2. Stage of **Reflex Activity**
  3. Stage of **Reflex Failure**

1. Stage of Spinal Shock

Loss of all functions and activity below the level of transection immediately after injury.

Cause :

Cessation of tonic discharge of supraspinal pathways

- Duration of spinal shock depends on evolution of animal **(Encephalization). (3-4 weeks)**

- **Diaschisis**

- **Motor effects**
  - Tone is lost
  - Power is lost
  - Flaccid paralysis (Quadriplegia, Paraplegia)
  - Reflexes are lost – Areflexia
- **Sensory effects**
  - All sensations are lost - Anaesthesia
- **Vasomotor effects**
  - Damage to sympathetic neurons- Fall in BP
- **Visceral effects**
  - Urinary bladder and Rectum are paralysed
    Micturition reflex, Defecation reflexes lost
- Sexual function lost , Decubitus ulcers develop

2. **Stage of reflex activity (Recovery)**

- **Autonomic reflexes recover first**
  - Bladder - Automatic bladder
  - Defecation reflex - Automatic
  - Blood Pressure is restored
  - Bed sores heal up rapidly

- **Skeletal Muscle tone recovers (3 – 4 weeks)**
  - Tone in Flexors returns first

Paraplegia in flexion

- **Reflex activity recovers**

  - Flexor reflexes returns first (Babinski sign)

  - Extensor reflexes returns after 1-5 weeks
    Knee jerk, Ankle jerk

  - Mass reflex can be elicited
    Spasm of flexors
    Evacuation of Bladder and Rectum
    Sweating

3. Stage of reflex failure

- Reflexes become difficult to elicit

- Threshold for stimulus increases

- Mass reflex is abolished

- Muscles become flaccid and undergo wasting

Incomplete Transection of Spinal Cord

- Stage of Spinal Shock
- Stage of Reflex Activity
- Stage of Reflex Failure

- **Stage of Reflex Activity**

  Tone In the Extensors returns first

  Paraplegia in Extension

  - Phillipson Reflex
  - Extensor Thrust Reflex
  - Crossed Extensor Reflex

Hemisection of the Spinal Cord

Lesion involving one lateral half of Spinal Cord

Typical sensory and motor changes that develop after recovery from spinal shock constitute the

BROWN-SEQUARD SYNDROME

- **Changes at the level of lesion**
- **Changes below the level of lesion**
- **Changes above the level of lesion**

Brown-Sequard syndrome

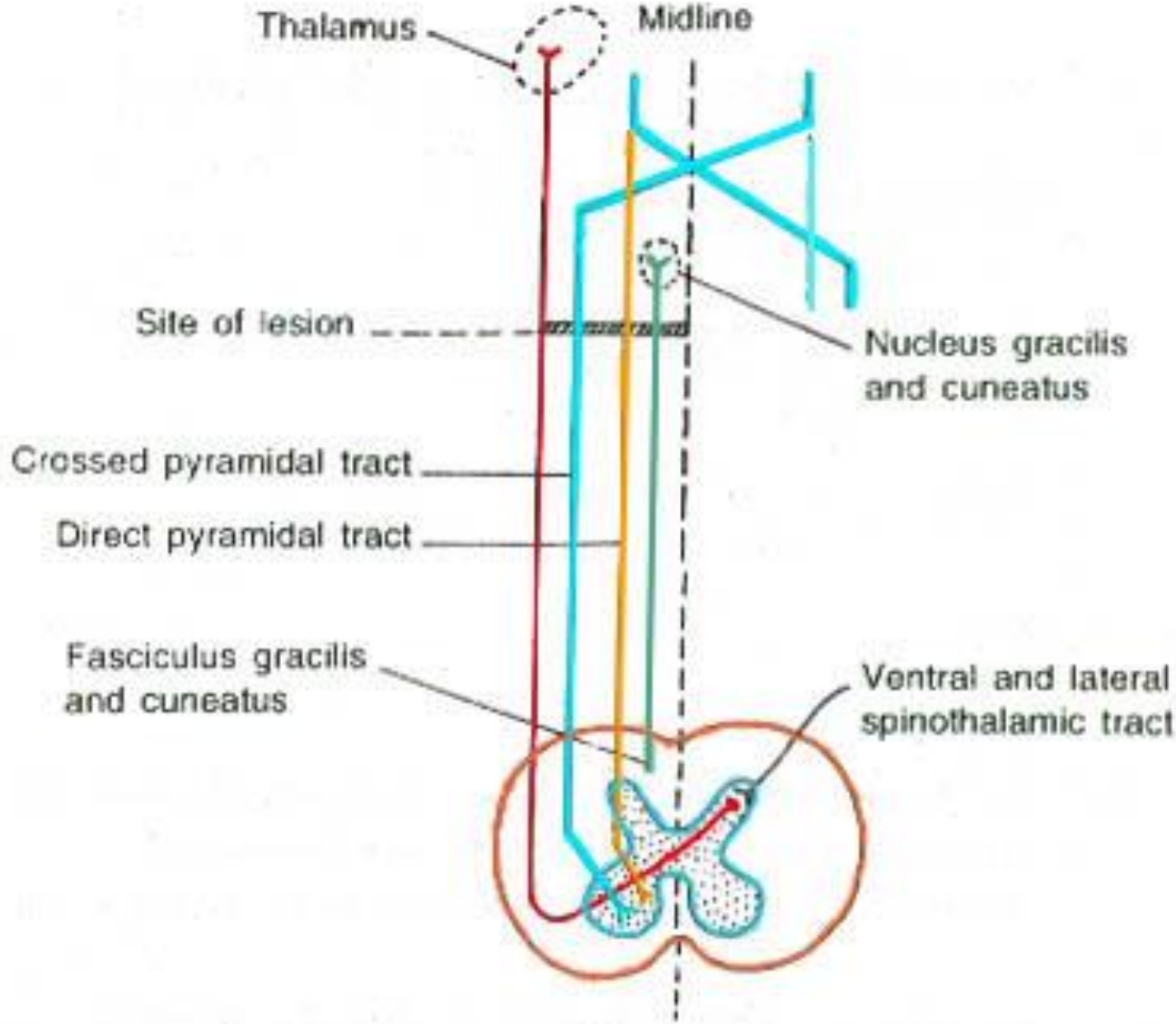


Fig. 11.8.1 Hemisection of the spinal cord

- **Changes at the level of lesion**

  On the same side

  - **Sensory changes :**

    All sensations are lost (complete anaesthesia)

  - **Motor changes :**

    LMN type of paralysis

  On the opposite side

  - **Sensory changes** :

    Crude touch, Pain and Temperature are lost

    Intact Dorsal Column Sensations

  - **Motor changes:** No motor changes (mild LMNL)

- **Changes below the level of lesion**

On the same side

- **Sensory changes**:

Dorsal column sensations are lost

Crude touch, Pain and Temperature are intact

- **Motor changes** :

UMN Type of paralysis

On the opposite side

- **Sensory changes** :

Crude touch, Pain and Temperature are lost

Dorsal column sensations are intact

- **Motor changes** : No motor changes (UMNL)

- **Changes above the level of lesion**

On the same side

- **Sensory changes** :

A band of Hyperaesthesia

- **Motor changes** :

Twitching of the muscle

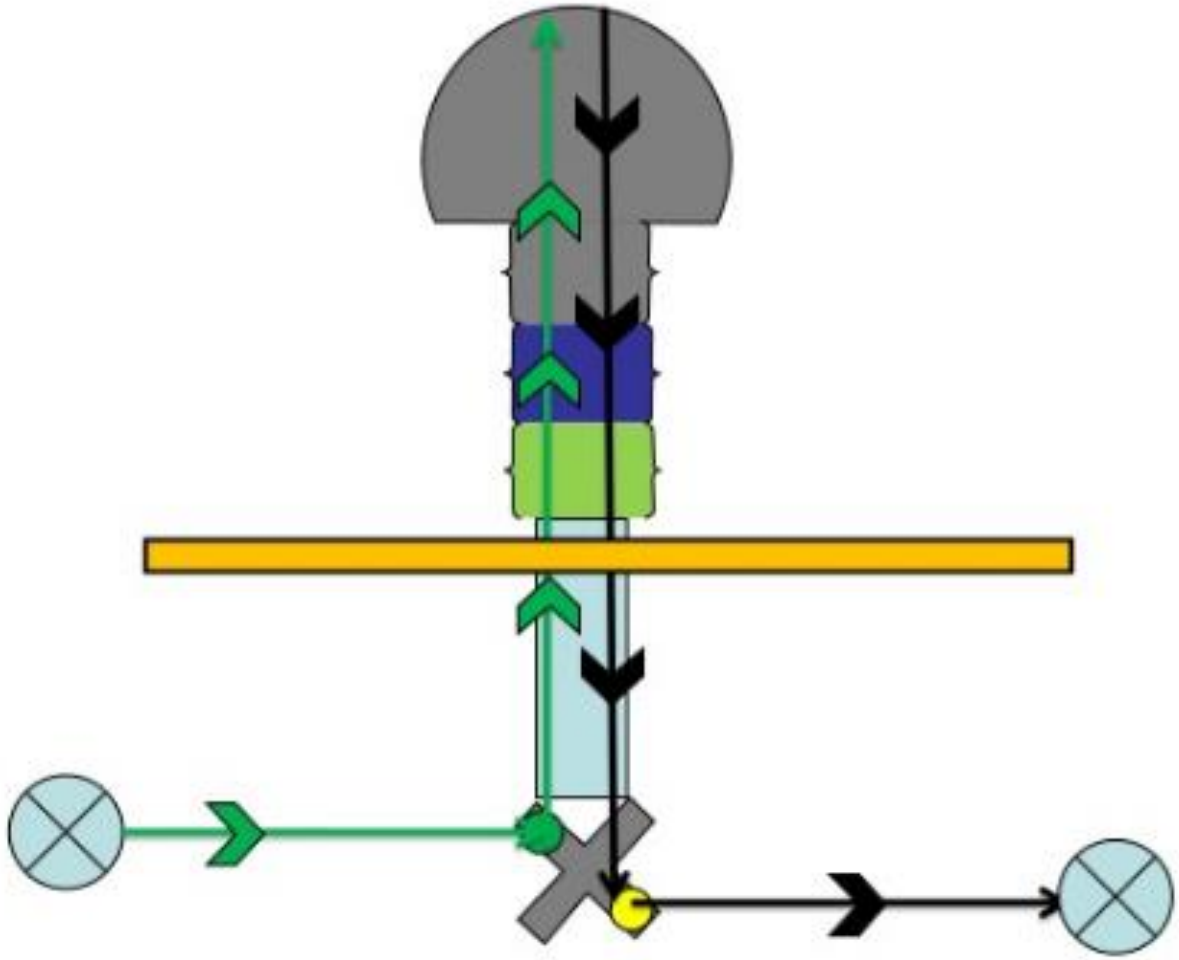
On the opposite side

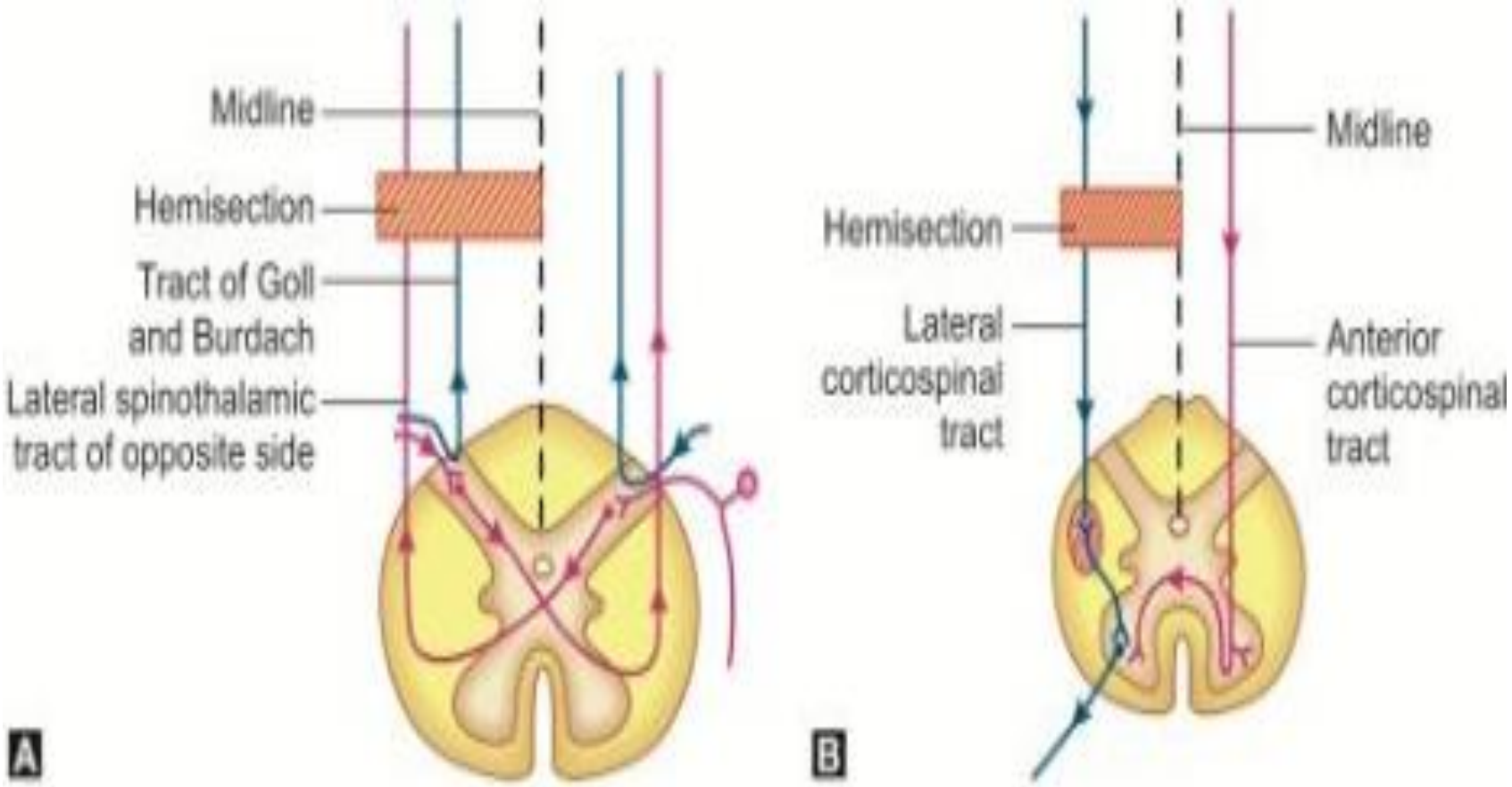
- **Sensory changes** :

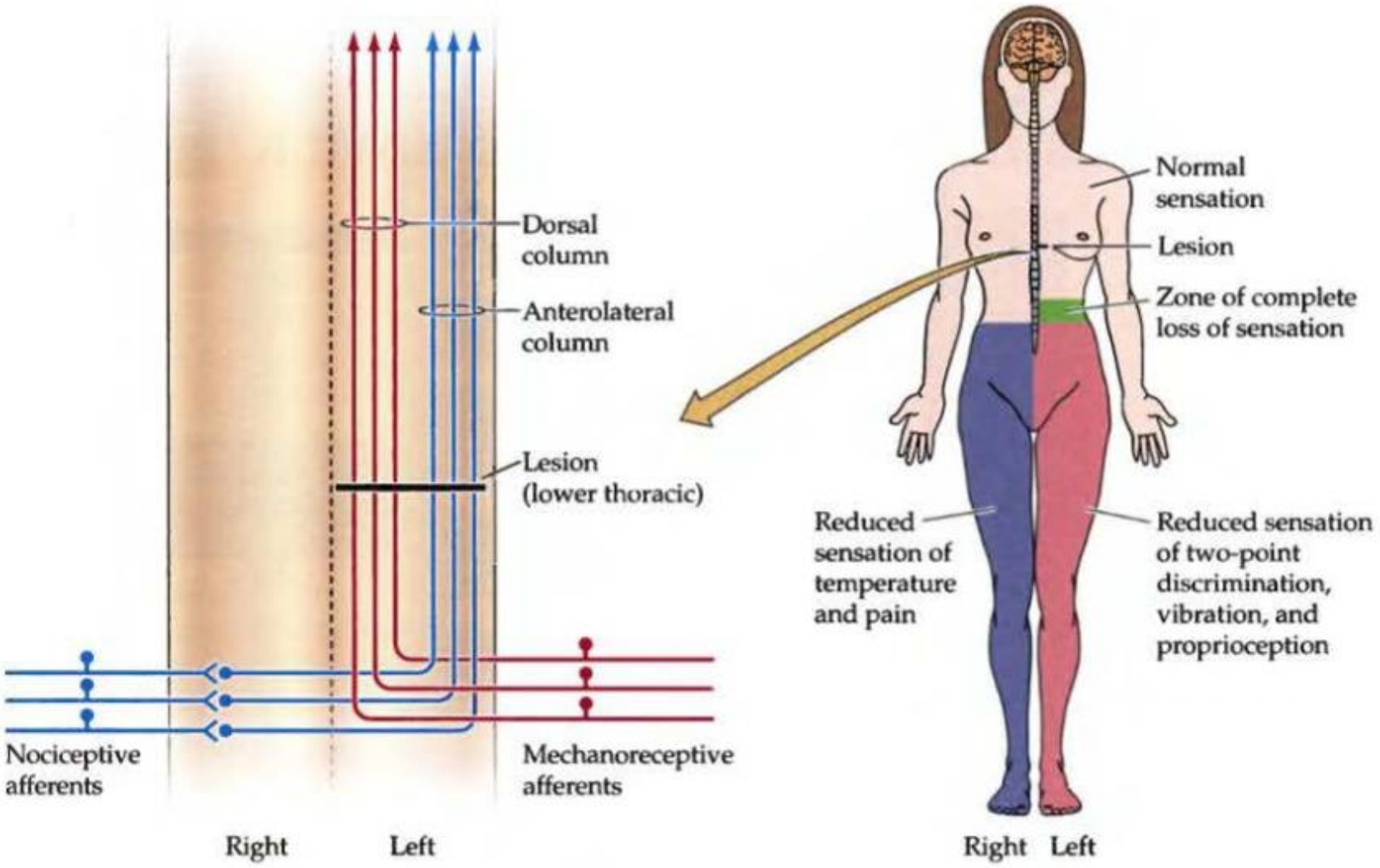
No sensory changes (Referred Hyperaesthesia)

- **Motor changes** :

No motor changes




Midline
Hemisection
Tract of Goll and Burdach
Lateral spinothalamic tract of opposite side
A
Midline
Hemisection
Lateral corticospinal tract
Anterior corticospinal tract
B



Brown-Séquard syndrome

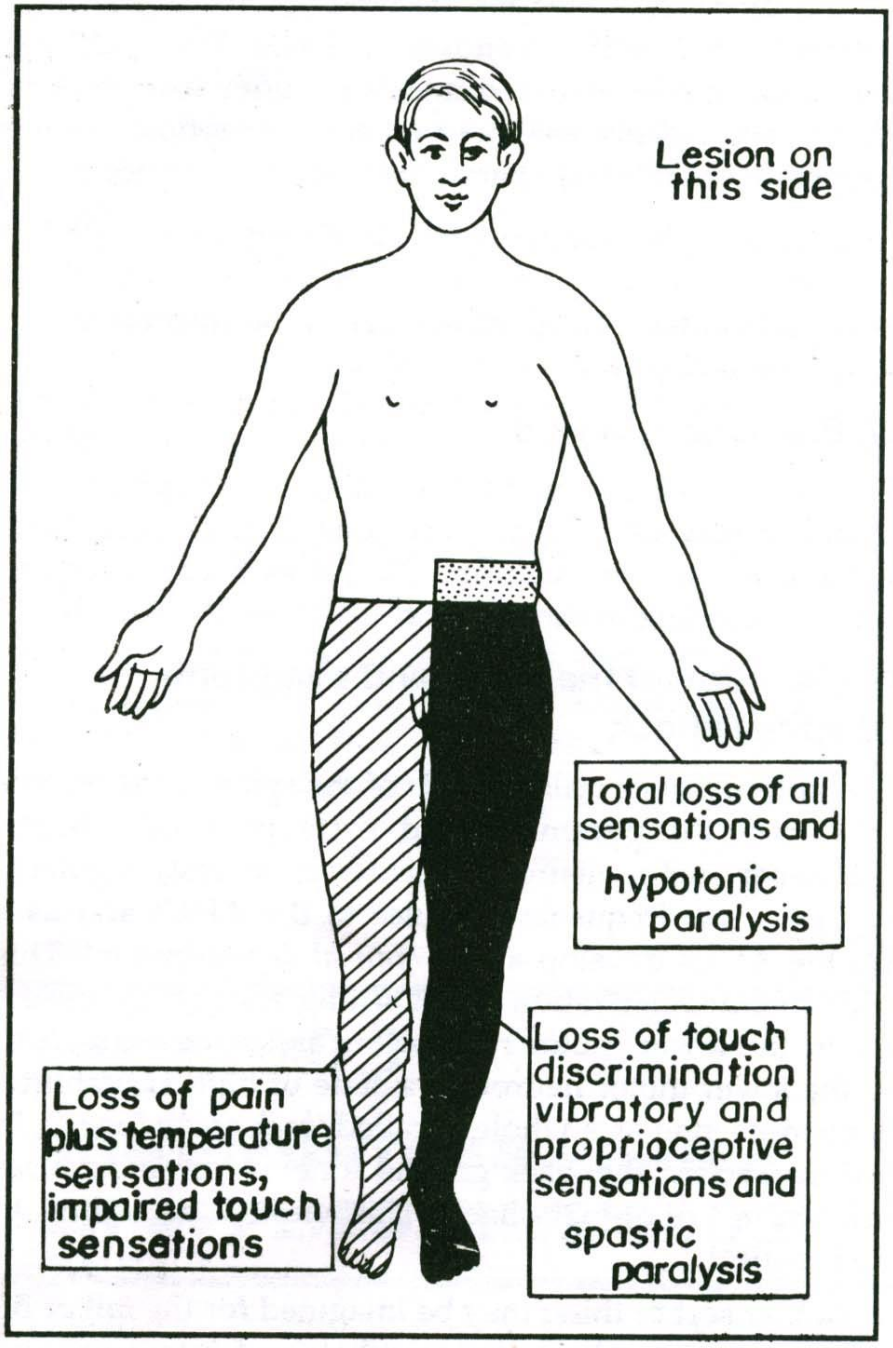


Fig. 10D.8.1 : Description in the text

- Sub acute Combined Degeneration of SC
- Tabes Dorsalis
- Syringomyelia