كلية الطب البشري
Faculty of Medicine

Neuroscience I Module 2024

Physiology Lectures
(L1-L12)

Presented by:

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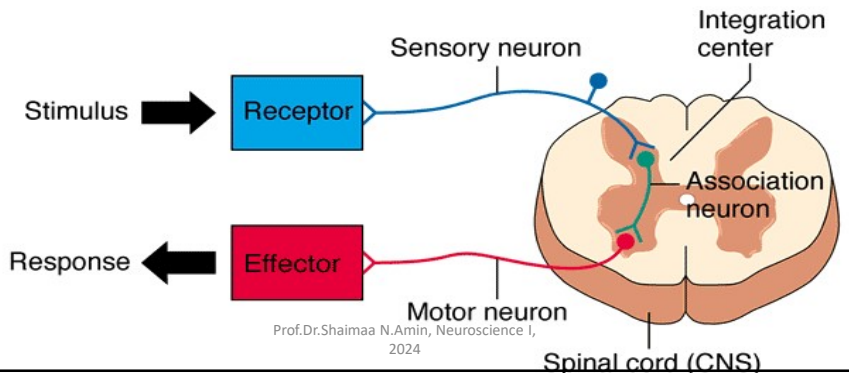
Professor of Medical Physiology

1

Sensations & Perceptions

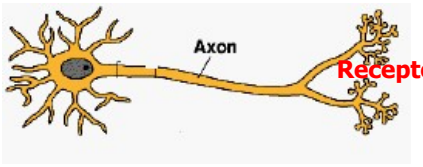
Sensation : is an awareness of sensory stimuli in brain.

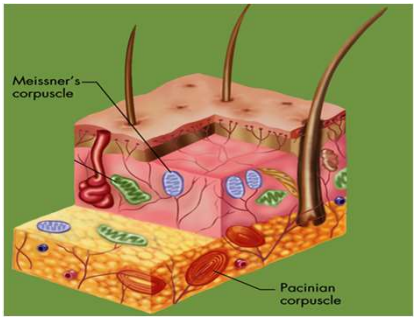


2

Sensory receptors

1. Specialized structures **or**
2. modified n. endings at the peripheral termination of afferent fibers.





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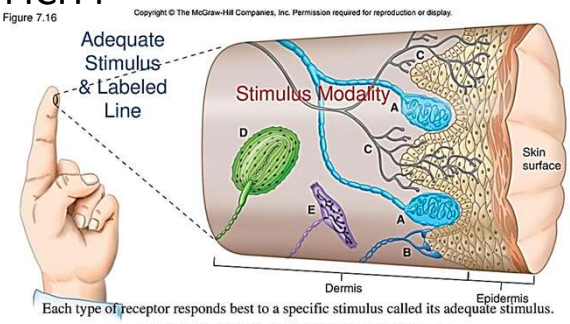
Classification (by stimulus type)

1. Mechanoreceptors
2. Thermoreceptors
3. Pain receptors
4. Electromagnetic receptors
5. Chemoreceptors

4

Properties Of The Sensory Receptors

(1) SPECIFICITY



Each type of receptor responds best to a specific stimulus called its adequate stimulus.

A. Meissner's corpuscle—rapidly adapting mechanoreceptor, touch and pressure
B. Merkle's corpuscle—slowly adapting mechanoreceptor, touch and pressure
C. Free nerve ending—slowly adapting, some are nociceptors, some are thermoreceptors, and some are mechanoreceptors
D. Pacinian corpuscles—rapidly adapting mechanoreceptor, vibration and deep pressure
E. Ruffini corpuscle—slowly adapting mechanoreceptor, skin stretch

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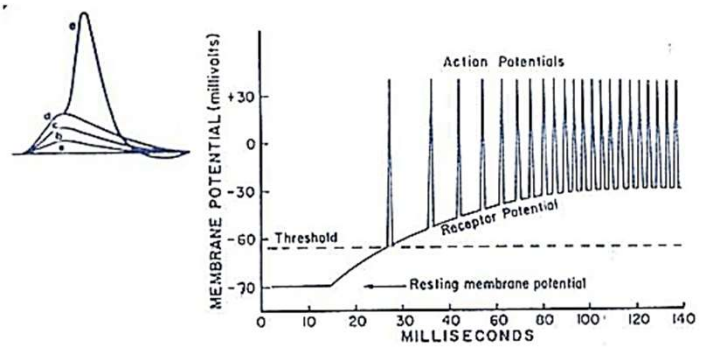
Properties Of The Sensory Receptors

(2) EXCITABILITY (THE RECEPTOR POTENTIAL)



Relation between the receptor potential (RP) & action potentials (APs). As the (RP) rises above threshold, the frequency of (APs) increases.

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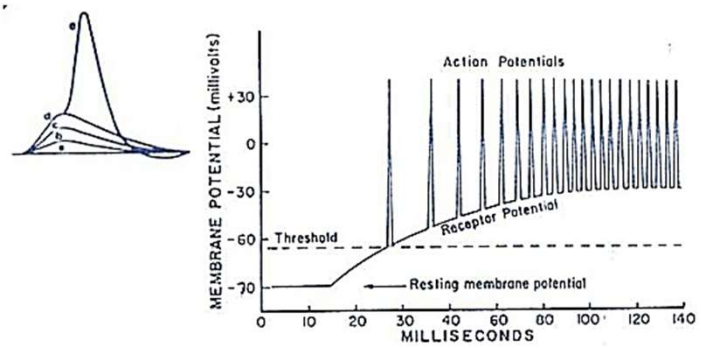
Properties Of The Sensory Receptors

(3) DISCHARGE OF IMPULSES



Relation between the receptor potential (RP) & action potentials (APs). As the (RP) rises above threshold, the frequency of (APs) increases.

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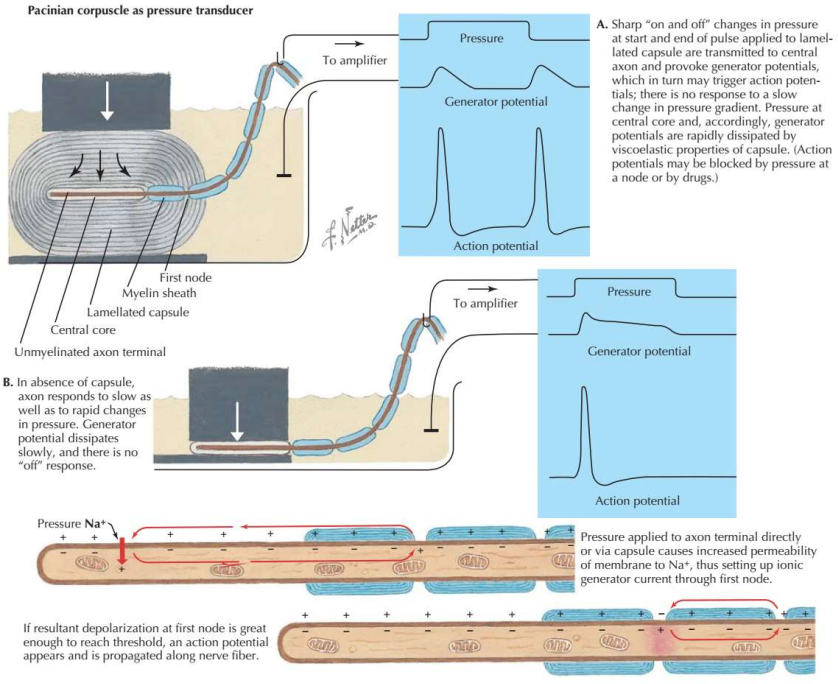

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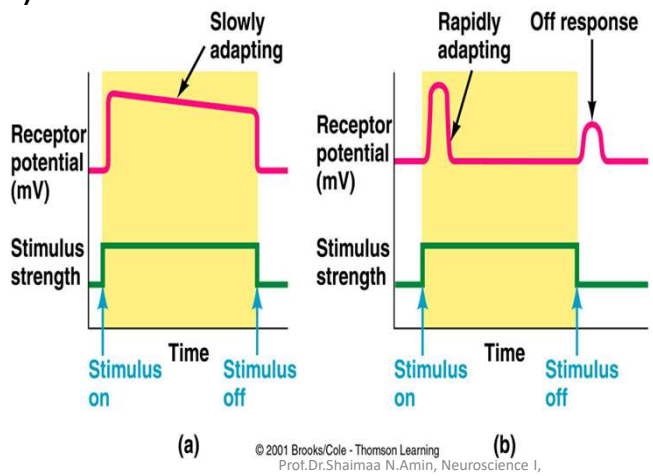
Properties Of The Sensory Receptors

(4) ADAPTATION



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Coding of sensory information

1-Modality (Muller's Law, Labeled line Principle)

2-Locality (Law of Projection)

3-Intensity (Recruitment of receptors, frequency on impulses).

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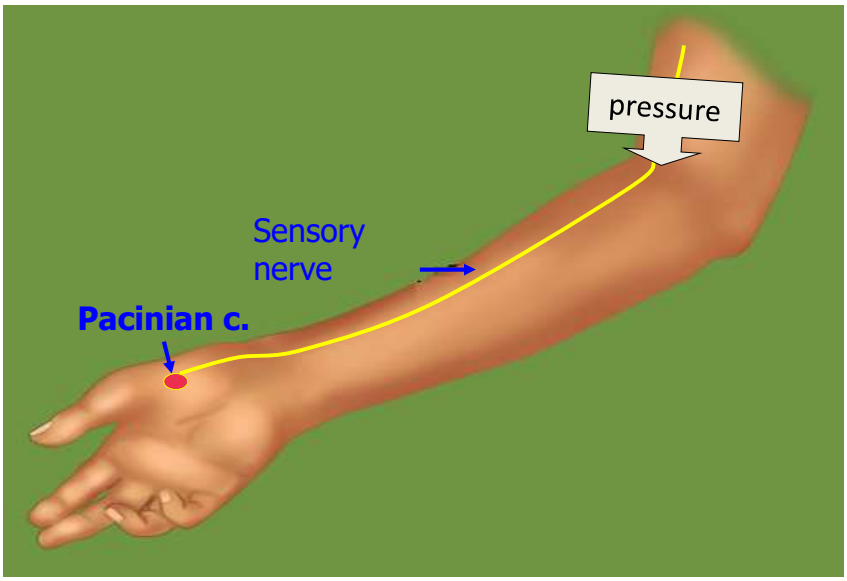
pressure
Sensory
nerve
Pacinian c.

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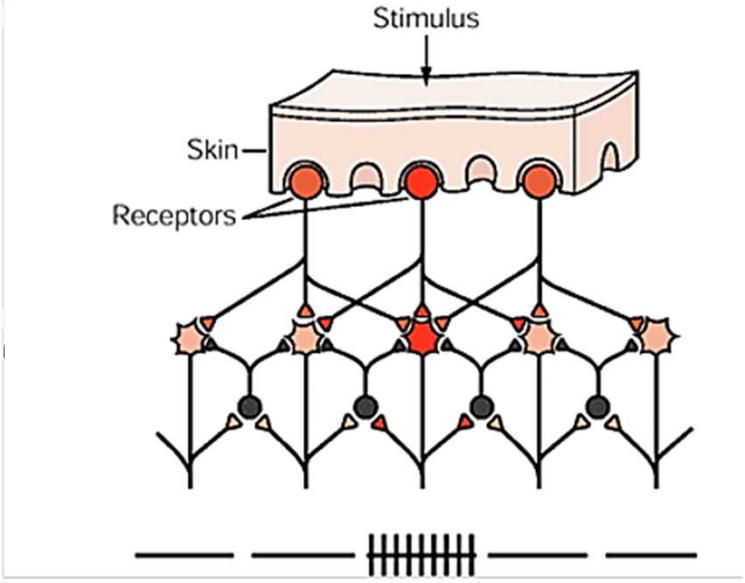
Stimulus
Skin
Receptors

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LAWS IN SENSORY PHYSIOLOGY

1. **Muller's Doctrine of specific nerve energies:** No matter where along the nerve pathway one stimulates, the type of sensation will depend on which part of the brain is finally going to be stimulated.
2. **Law of Projection:** No matter where along the nerve pathway one stimulates, the sensation will be felt at the site of the receptor Phantom limb sensation is best described by this law.
3. **Bell-Megendie Law:** This law states that the dorsal root is sensory and ventral root is motor.
4. **Labelled-line theory:** All the sensations from the different parts of the body travel along specified paths. For example, in the posterior column, fibers from the lower parts of the body are placed medially.
5. **Weber-Fechner's law:** In 1834, Weber demonstrated that the *sensitivity of a sensory system* to differences in intensity depends on the absolute *strength of the stimuli*. We easily perceive that 1 kg is different from 2 kg, but it is difficult to distinguish 50 kg from 51 kg. Yet both sets differ by 1 kg. This relationship is expressed in the equation now known as:
   - **Weber's law: $\Delta S = K. S$** 舰舰…[where ΔS is the minimal difference in strength between a reference stimulus S and a second stimulus that can be discriminated, and K is a constant.]
   - **Fechner** extended Weber's law to describe the relationship between the stimulus strength (S) and the intensity of the sensation (I) experienced by a subject: **$I = K \log (S/S_0)$**…… [where S_0 is the threshold amplitude of the stimulus and K is a constant.]
   - **Stevens' law** states that: **$I = K (S - S_0)^n$**

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Weber-Fechner principle:

This is a logarithmic function which states that: **the perceived sensation is proportional to log intensity of the stimulus.**

$$R = \log S \times K$$

R: perceived sensation .

S: stimulus intensity.

K :constant.

This means that 100 fold increase in stimulus intensity, will increase the perceived sensation by 2 times, 1000 fold increase will increase sensation by 3 times and so forth.

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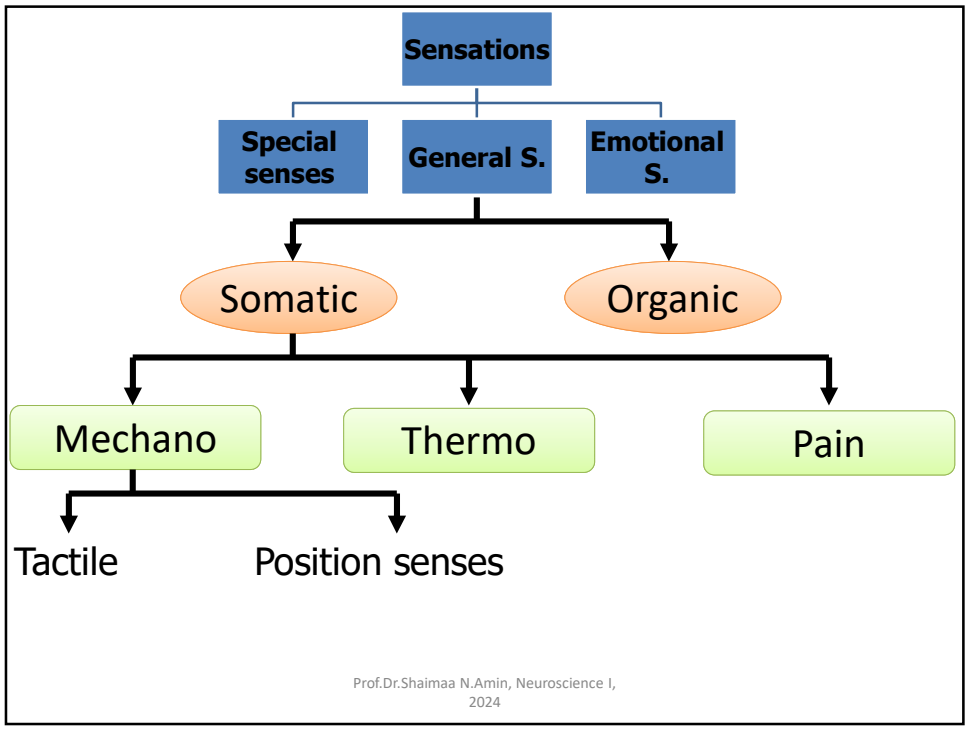
Sensations
Special senses
General S.
Emotional S.
Somatic
Organic
Mechano
Thermo
Pain
Tactile
Position senses
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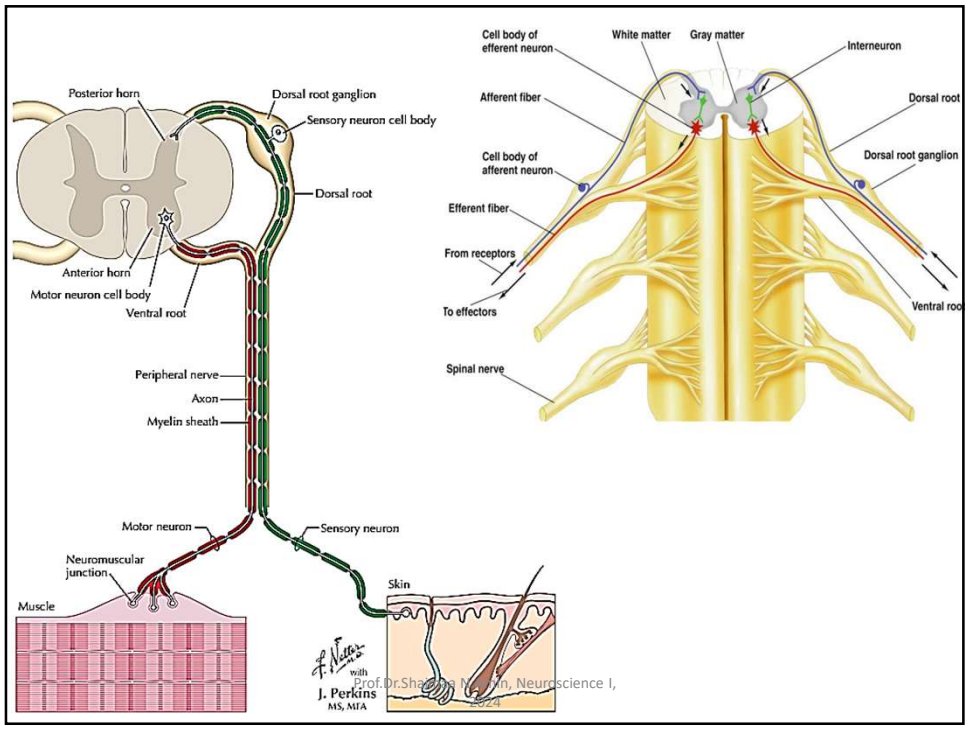
Posterior horn
Dorsal root ganglion
Sensory neuron cell body
Dorsal root
Anterior horn
Motor neuron cell body
Ventral root
Peripheral nerve
Axon
Myelin sheath
Motor neuron
Sensory neuron
Neuromuscular junction
Muscle
Skin
with
J. Perkins
MS, MFA
Cell body of efferent neuron
White matter
Gray matter
Interneuron
Afferent fiber
Dorsal root
Cell body of afferent neuron
Dorsal root ganglion
Efferent fiber
From receptors
To effectors
Ventral root
Spinal nerve
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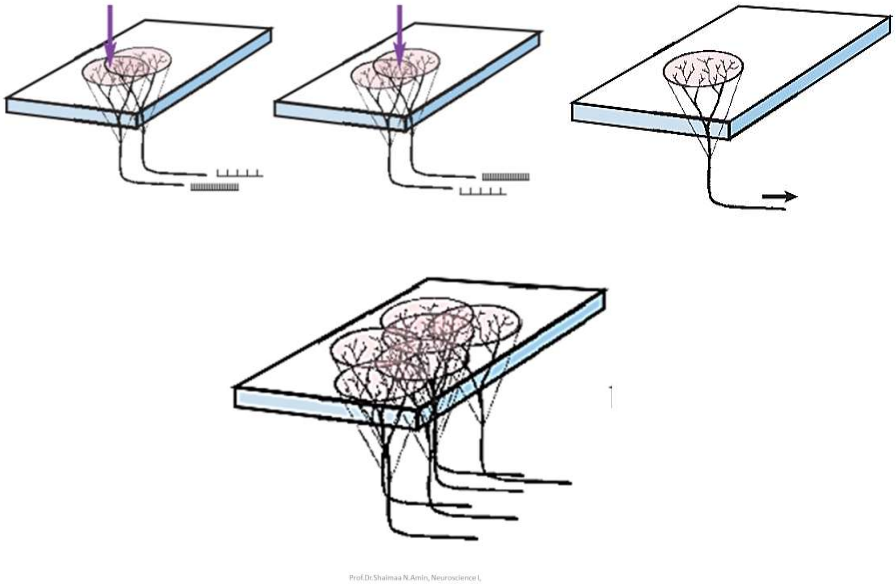

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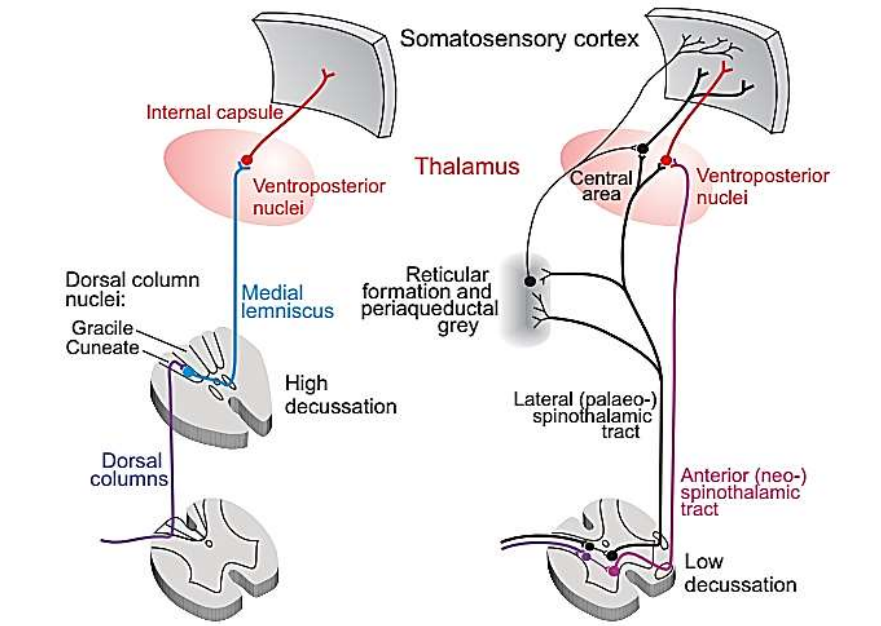
Somatosensory cortex
Internal capsule
Thalamus
Ventroposterior nuclei
Central area
Ventroposterior nuclei
Dorsal column nuclei:
Medial lemniscus
Reticular formation and periaqueductal grey
Gracile
Cuneate
High decussation
Lateral (palaeo-) spinothalamic tract
Dorsal columns
Anterior (neo-) spinothalamic tract
Low decussation
Lemniscal system
Anterolateral system

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Each pathway consists of three populations of neurons :—

First-order, Second-order and Third-order.

Unipolar, with its cell body in a sensory ganglion

Its axon
- decussates.
- ends in the thalamus.

Cell body in thalamus. Axon ends in cortex.

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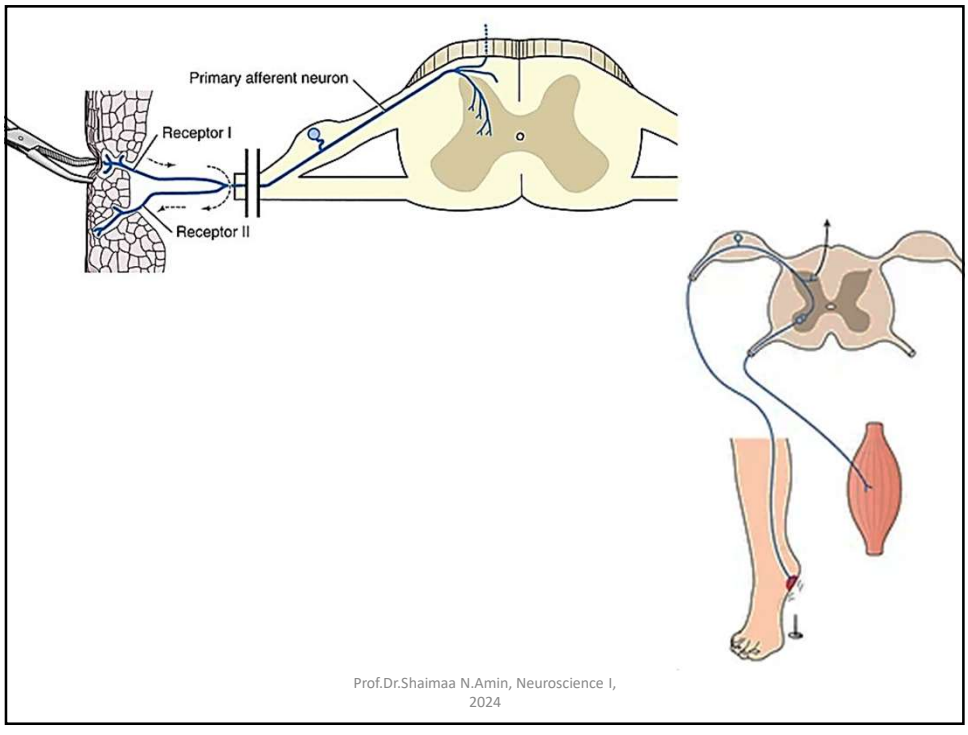

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Sensations carried by dorsal column

Sensation	Receptor	Afferent fiber
Fine touch	M & M	Aβ
Stereognosis	mixture	Aβ
Pressure	Pacinian & spray	Aβ
Vibration S.	Pacinian & Me.	Aβ
Position S.	Pacinian R & S	Aα

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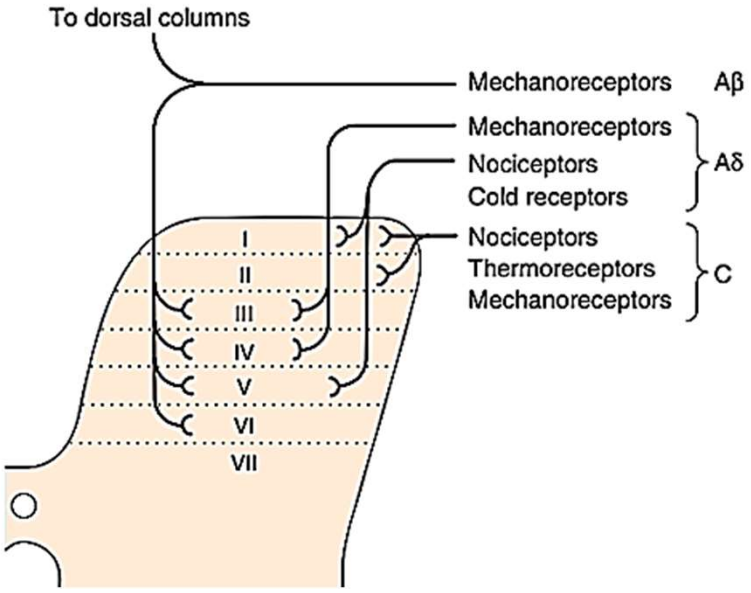

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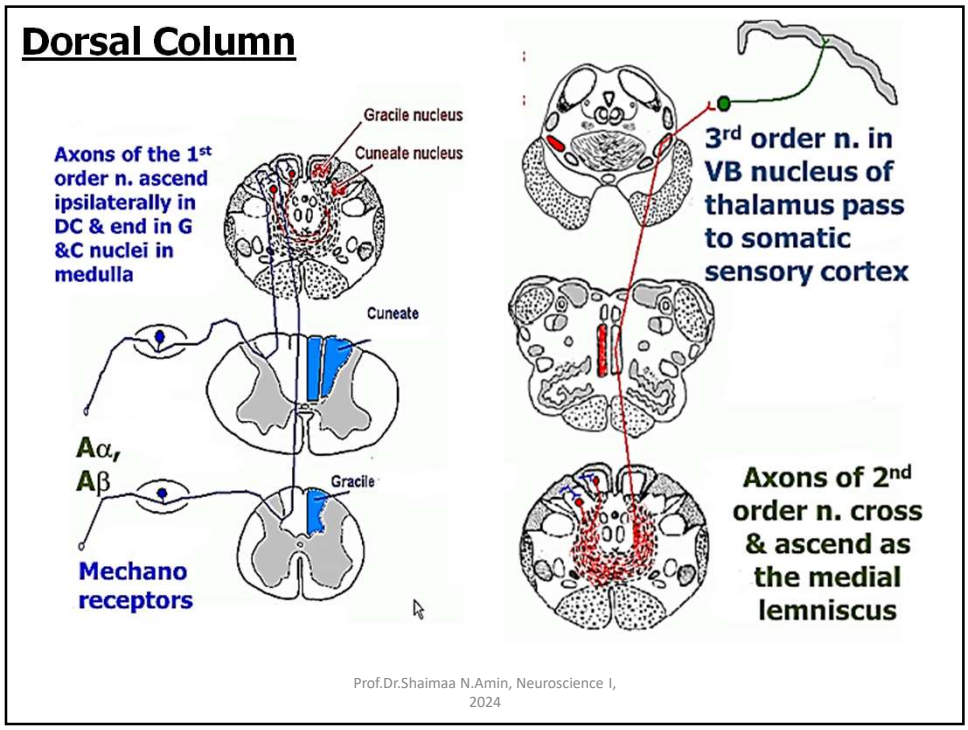
Dorsal Column
Axons of the 1st order n. ascend ipsilaterally in DC & end in G &C nuclei in medulla
Gracile nucleus
Cuneate nucleus
Cuneate
Gracile
Aα, Aβ
Mechano receptors
3rd order n. in VB nucleus of thalamus pass to somatic sensory cortex
Axons of 2nd order n. cross & ascend as the medial lemniscus
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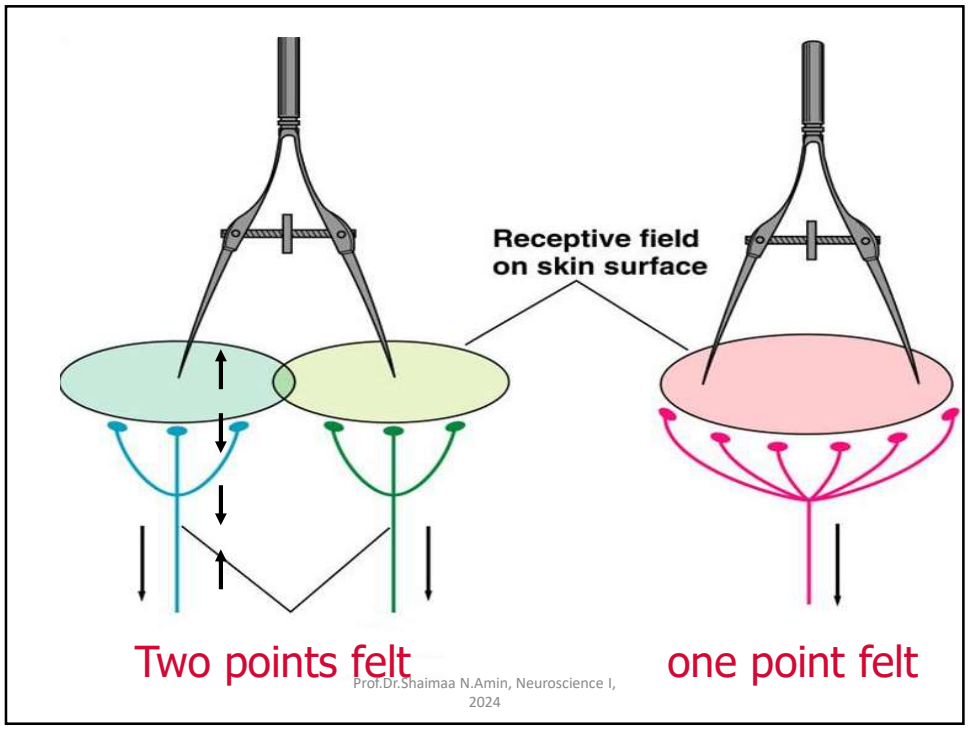
Receptive field on skin surface
Two points felt
one point felt
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Important

Two-point Discrimination:

- 1 mm on the fingertips of young adults; by the sixth or seventh decade of life it declines on average to ~ 2 mm.
  - **Receptor: *Merkel cell*>>*Meissner's*** corpuscle (as Merkel cell has the smallest receptive field).
- Braille Reading: For blind person, **Braille** dots (spaced ~ 3 mm apart) are perceived as separate dot because of **Merkel cell**. Meissner's corpuscle also contributes to detection of Braille patterns because they sense motion.

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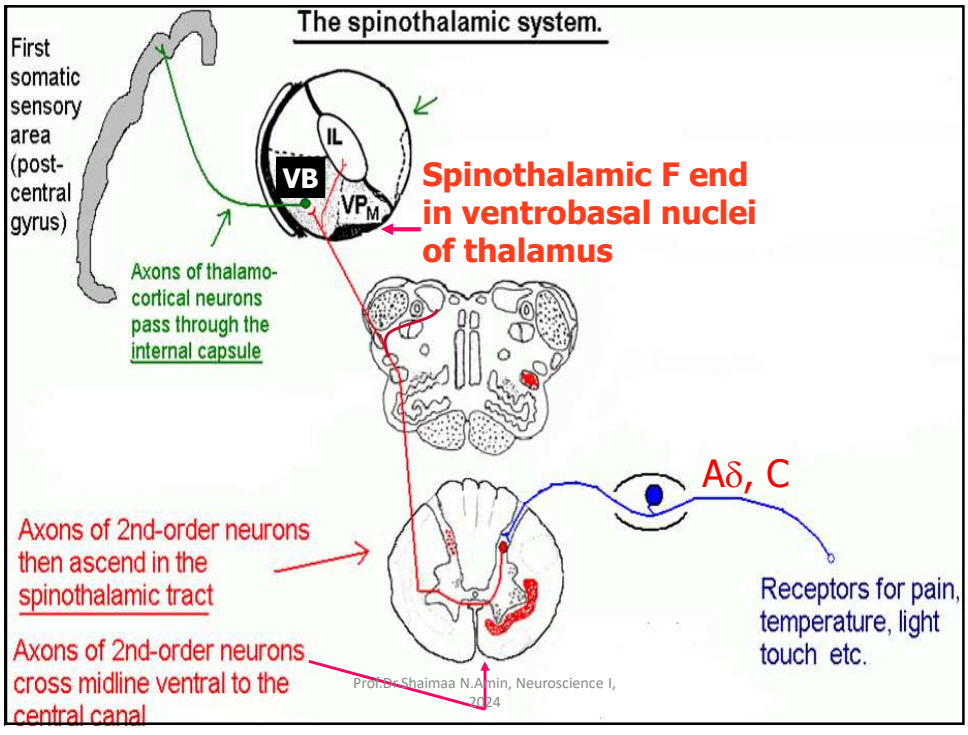

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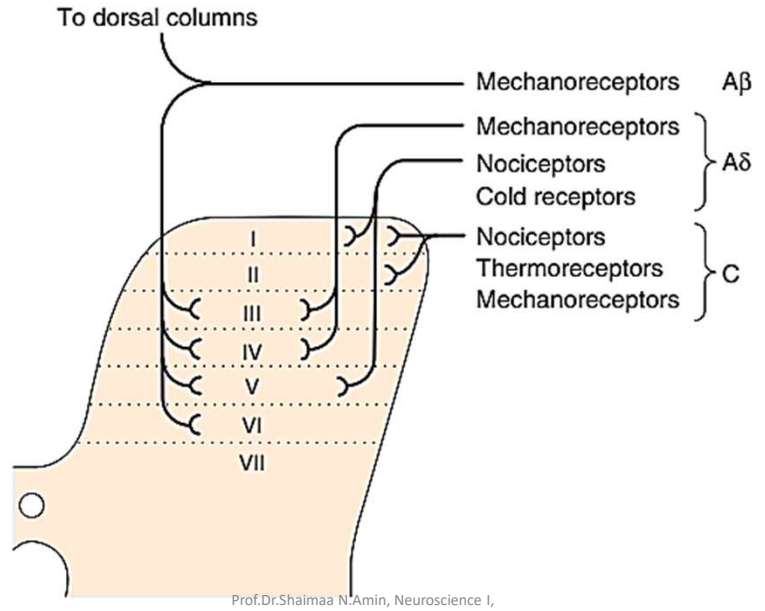

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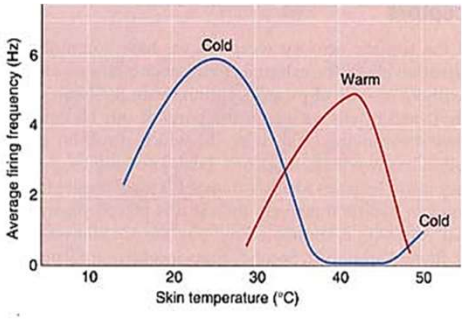
Thermoceptive Sensation

Characters of thermal receptors

- Have a small receptive field and widely separated.
- Cold R. are 10 times more numerous than warm R.
- Cold R. adapt more slowly than warm.



Thermoreceptors are stimulated chemically by changing the metabolic rate

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Pain

Nociceptors

- Free nerve endings, slowly (non) adapting to prolonged stimulation

- 4 types:
  - a. Mechanical pain receptors.
  - b. Thermal pain receptors.
  - C. Chemical pain receptors.
  - d. Polymodal pain receptors

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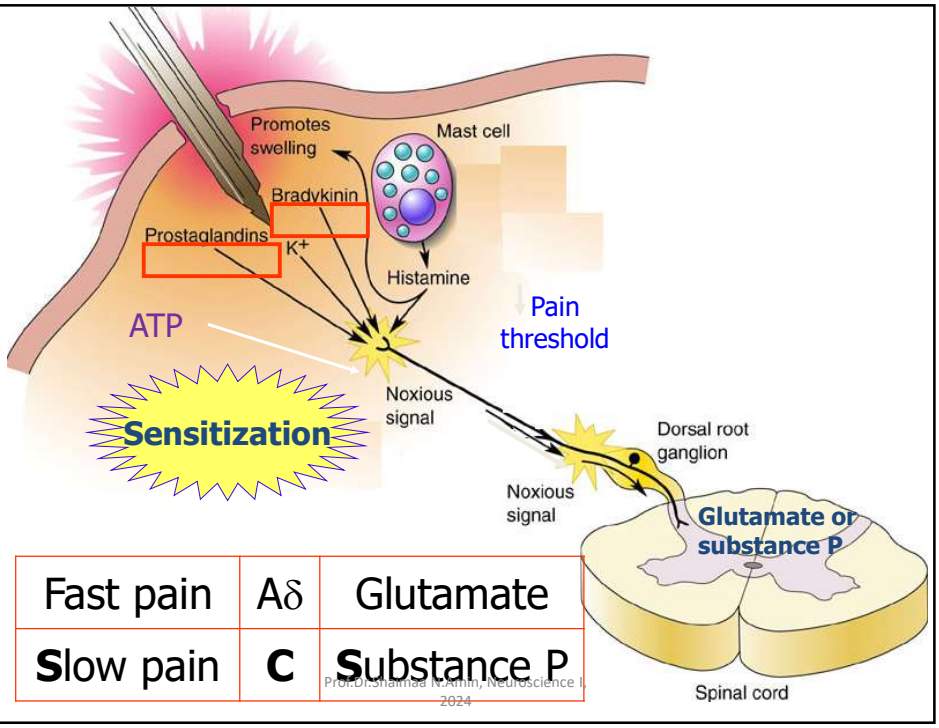

Fast pain	Aδ	Glutamate
Slow pain	**C**	**S**ubstance P

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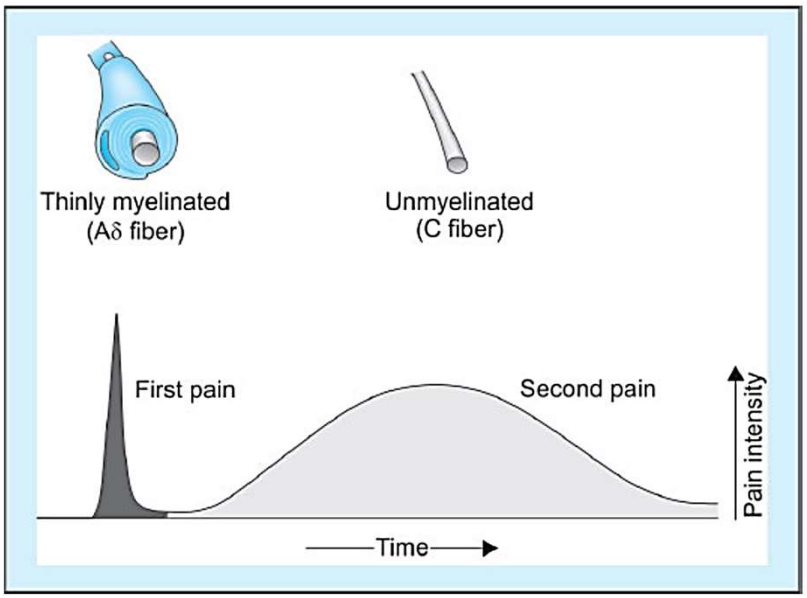

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Types of pain

Fast pain	Slow pain
Felt within 0.1 sec	Felt within 1 sec or more
Short duration	May be prolonged
Well localized	Poorly localized
Mechanical or thermal	All types of receptors
Usually in skin, rare in deep tissues	Skin, deep tissues & viscera

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Fast pain	Slow pain
Carried by Aδ, blocked by pressure	Carried by C, blocked by local anaesthetics
Aδ release Glutamate	C release Substance P
Transmitted by Neo-spinothalamic T	Transmitted by Paleospinothalamic T
Its fibers end in sensory cortex	End in RF ⟶Non-specific thalamic nuclei ⟶whole cortex

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Distribution of pain receptors

Widely distributed

Sup. Layers of skin
Periosteum
Arterial walls
Joint surface
Flax & tentorium of cranial cavity

Less distributed

Deep tissues & Viscera

Absent

Liver Parenchyma
Lung alveoli
Brain tissue

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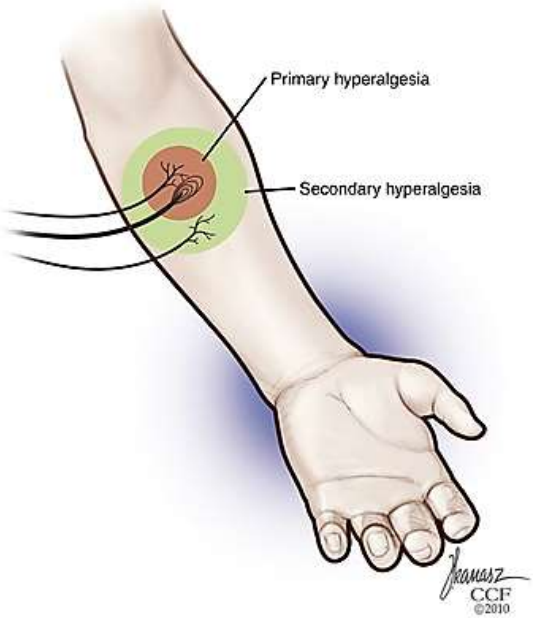

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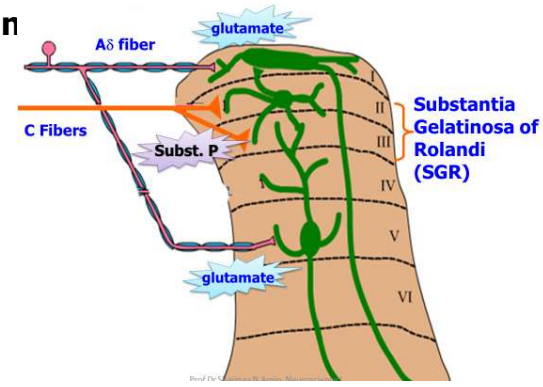
Roles of the cortex in pain perception are

1. **Localization of pain.**
2. **Discrimination of pain**
3. **Modulation of pain.**



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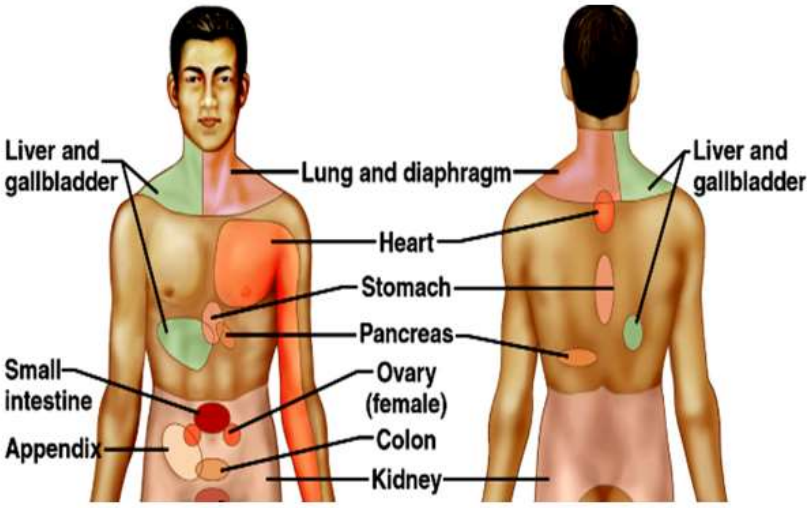
Referred pain



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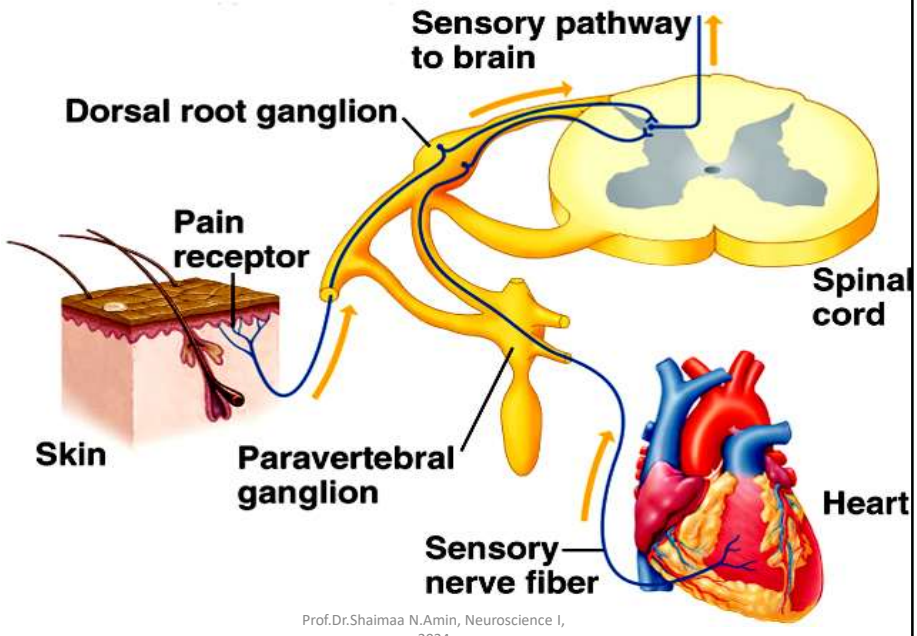
Convergence Projection theory



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Headache

Pain referred to the surface of the head from deep structures.

I. Headache of intracranial origin

- **The brain is insensitive to pain**
- **Pain receptors are found only in :**
  - Venous sinuses
  - Dural arteries
  - Dura
  - Tentorium

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I. Causes of intracranial H.

Meningeal causes

1. Meningitis
2. Brain tumour
3. Alcohol
4. Trauma
5. Constipation

Non Meningeal intracranial causes

1. Hypertension
2. Drop of intracranial pressure
3. Migraine

II. Causes of extracranial H.

1. Muscular spasm
2. Inflammation of nasal sinuses
3. Errors of refraction
4. Otitis media
5. Toothache
6. Systemic disorders
7. Trigeminal neuralgia

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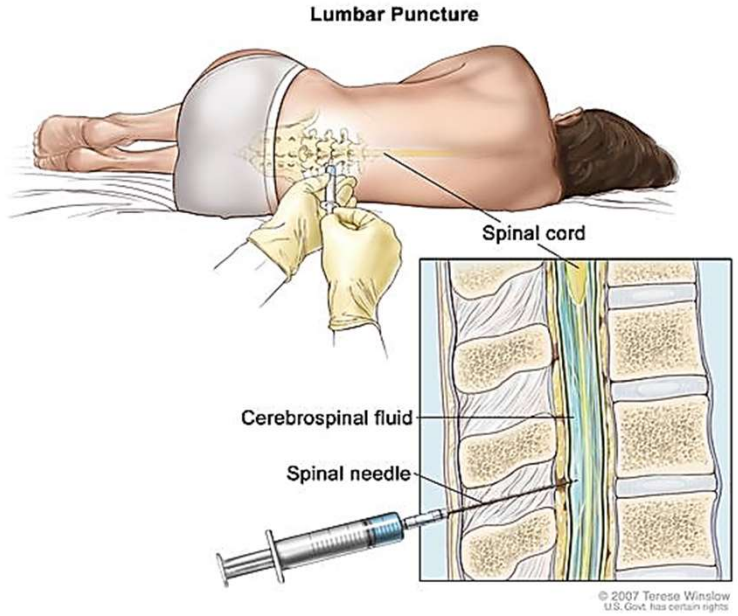

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Pain control

The gate theory

The dorsal horn cells of the spinal cord, in particular the cells of the SGR, act as a gate for the transmission of pain sensation to the brain

Opening of the gate	Closure of the gate
Impulses in C fibers	Analgesia system
	Opioid peptides
	Impulses in Aβ or Aδ

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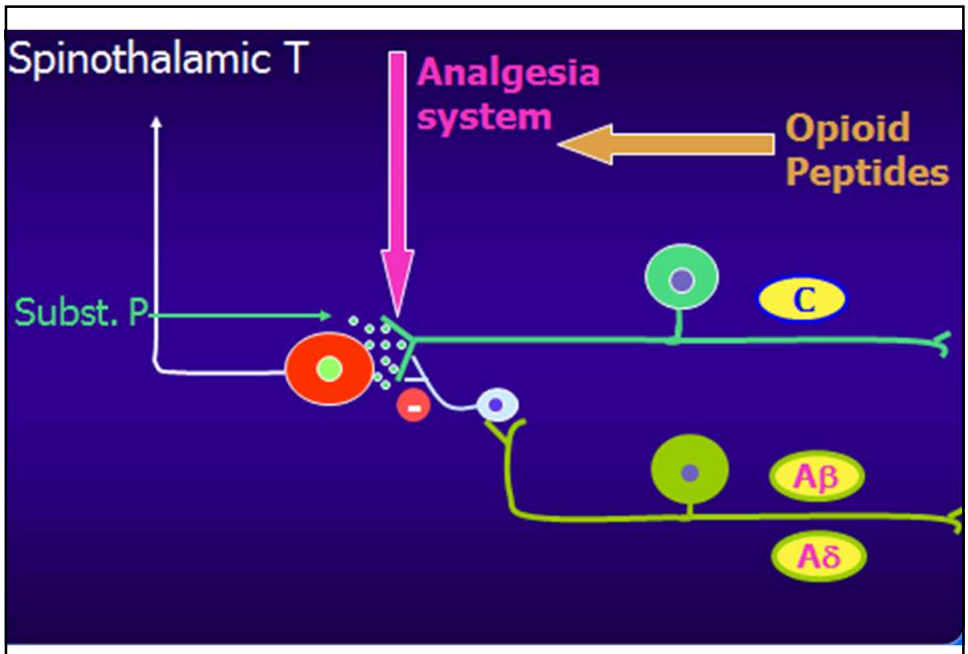
Spinothalamic T
Analgesia system
Opioid Peptides
Subst. P
C
Aβ
Aδ

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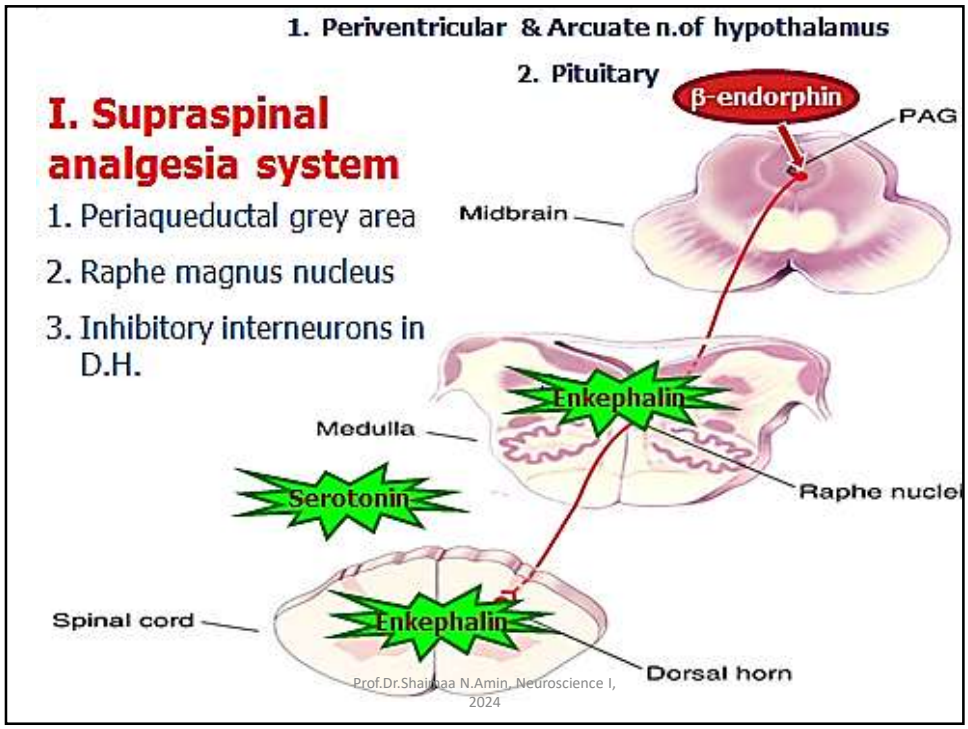
1. Periventricular & Arcuate n.of hypothalamus
2. Pituitary
β-endorphin
PAG
I. Supraspinal analgesia system
1. Periaqueductal grey area
2. Raphe magnus nucleus
3. Inhibitory interneurons in D.H.
Midbrain
Enkephalin
Medulla
Raphe nuclei
Serotonin
Spinal cord
Enkephalin
Dorsal horn

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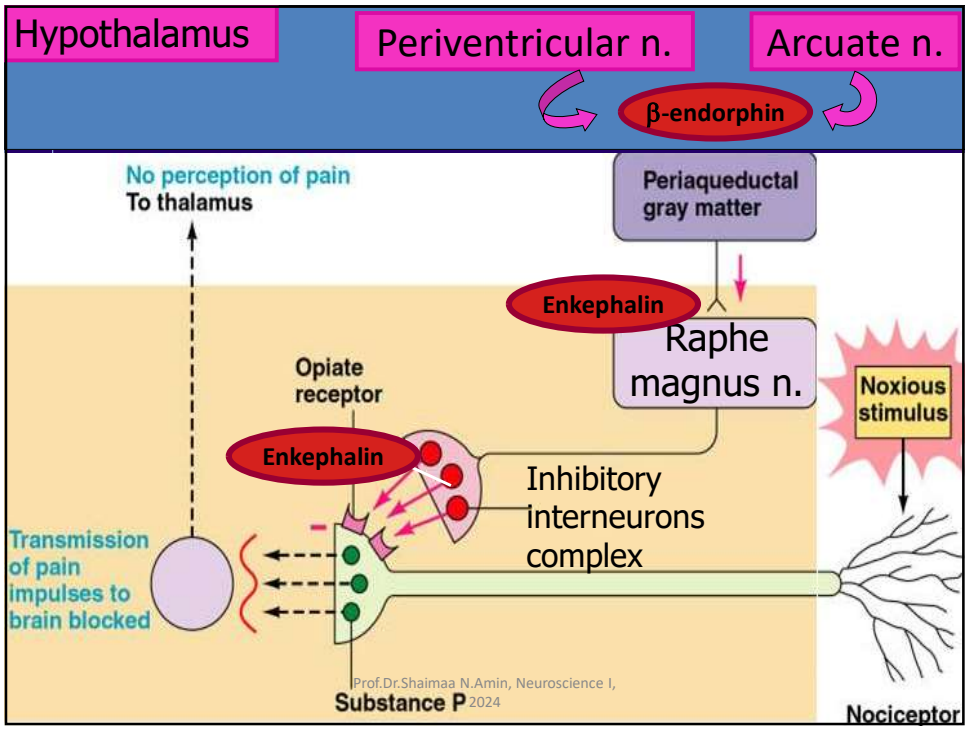

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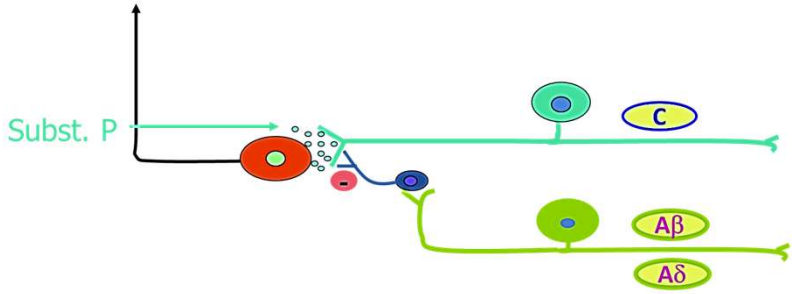
Spinal impulses

a. Collaterals from Aβ Fibers (rubbing of skin)

b. Collaterals from Aδ (counter irritation and acupuncture)



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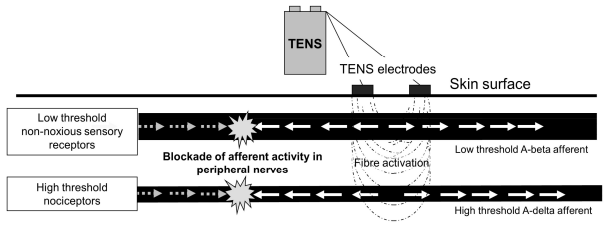
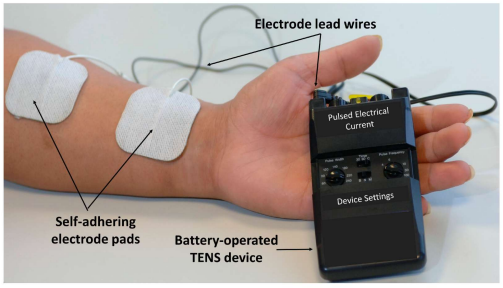
Transcutaneous Electrical Nerve Stimulation (TENS)





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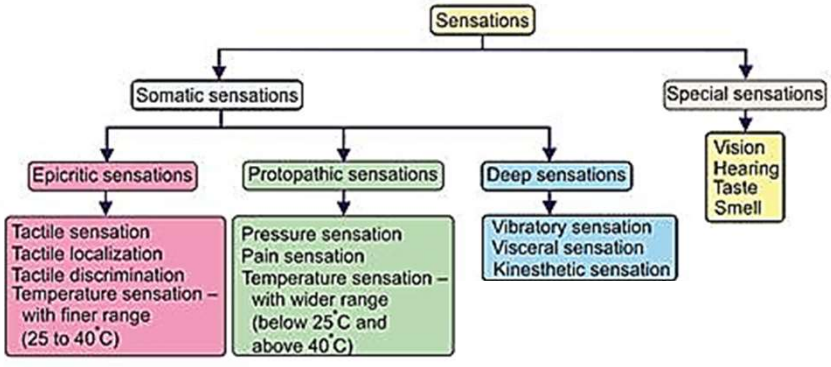

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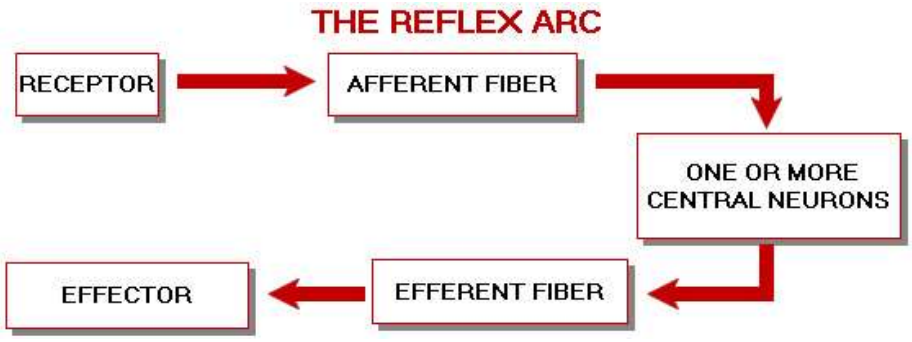
What is meant by a reflex?

The reflex arc

-The basic circuit that underlies a reflex.



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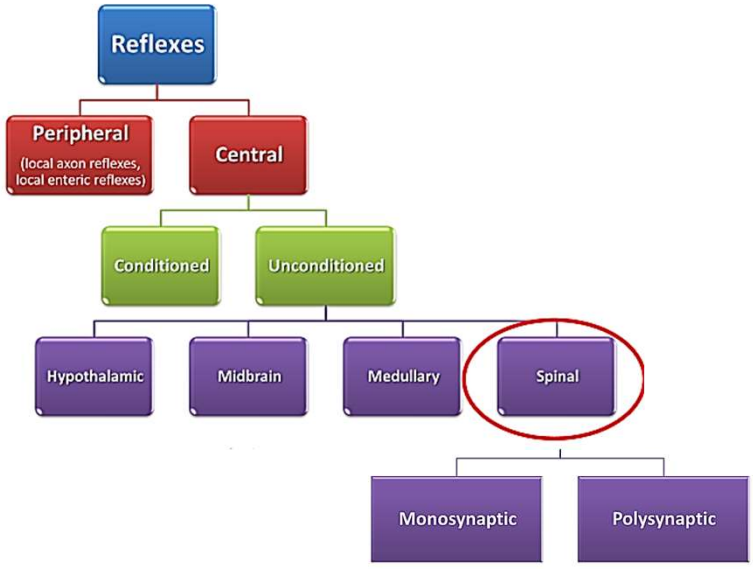

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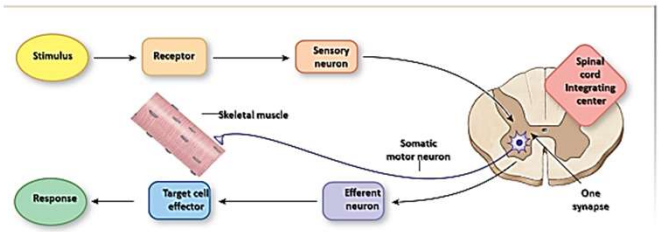
Monosynaptic Reflex



Polysynaptic Reflex

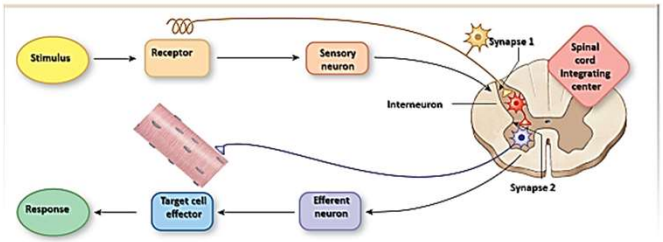


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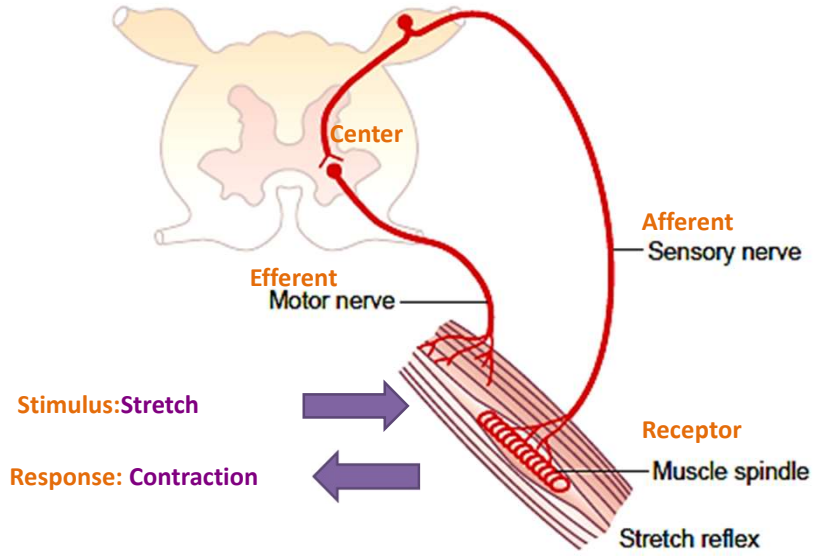
Table 36-1 Classification of Sensory Fibers from Muscle

Type	Receptor	Axon	Sensitive to
Ia	Primary spindle endings	12-20 μm myelinated	Muscle length and rate of change of length
Ib	Golgi tendon organs	12-20 μm myelinated	Muscle tension
II	Secondary spindle endings	6-12 μm myelinated	Muscle length (little rate sensitivity)
II	Nonspindle endings	6-12 μm myelinated	Deep pressure
III	Free nerve endings	2-6 μm myelinated	Pain, chemical stimuli, and temperature (important for physiological response to exercise)
IV	Free nerve endings	0.5-2 μm nonmyelinated	Pain, chemical stimuli, and temperature

52

Stretch Reflex arc



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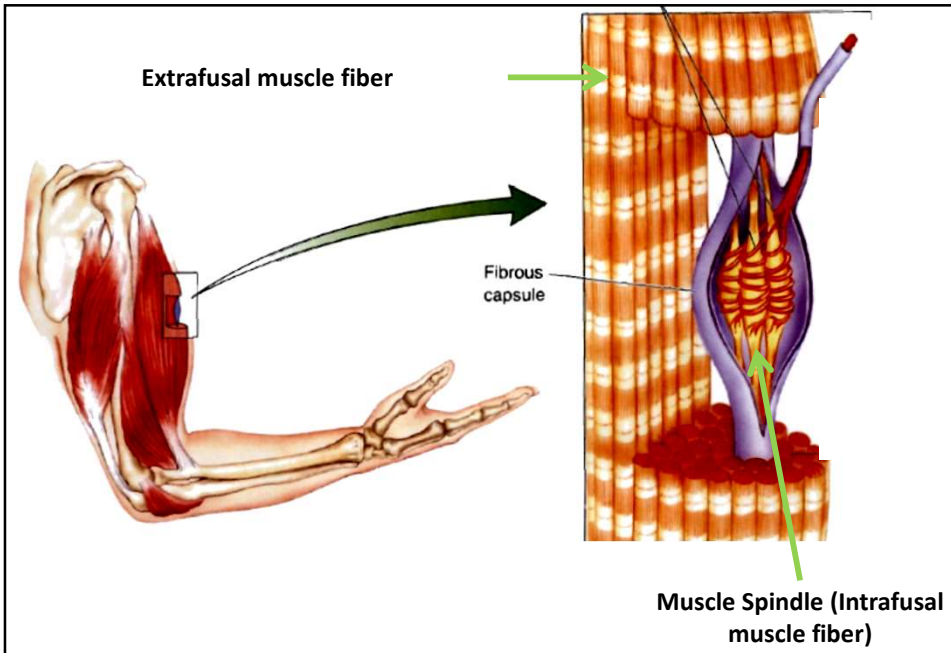

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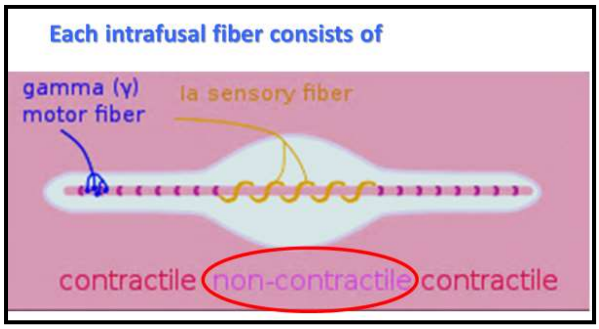
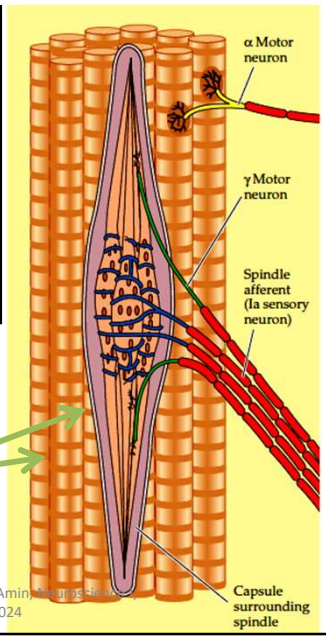
Functional anatomy of the muscle spindle

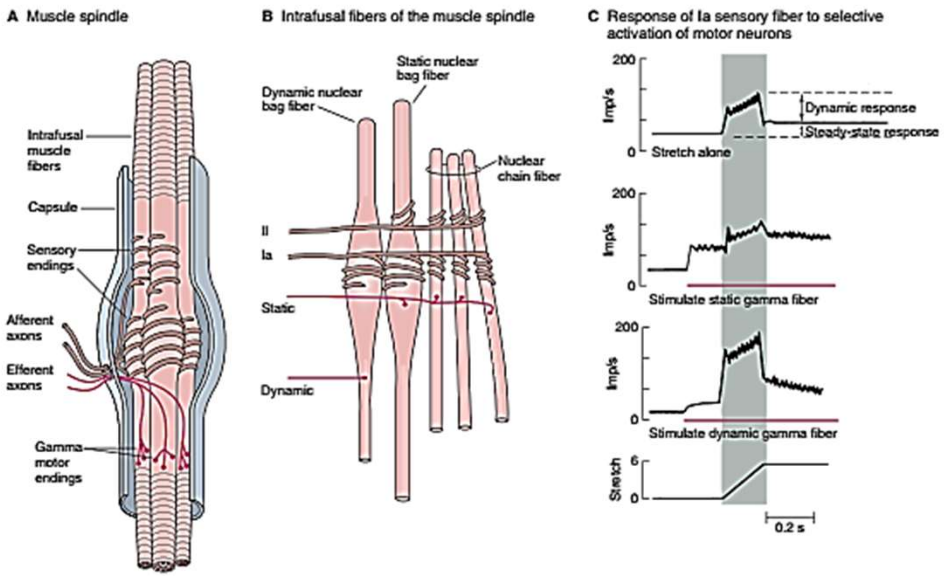




Parallel arrangement of intrafusal and extrafusal muscle fiber

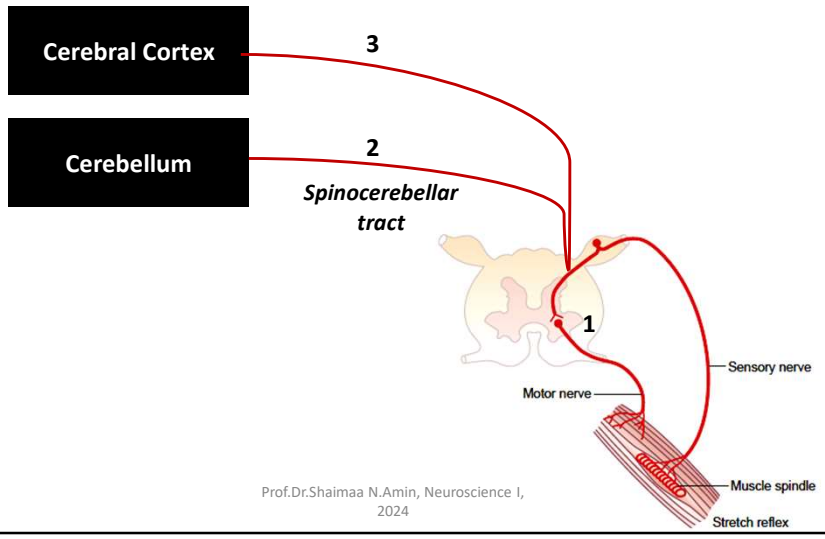
55

Types of intrafusal fibres



56

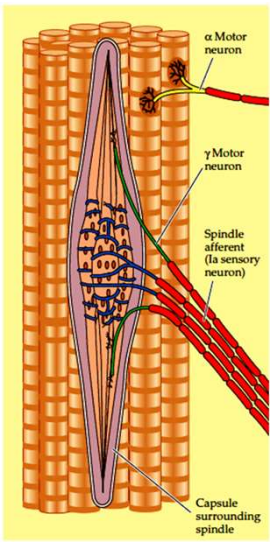
Central connections of the afferent fibres

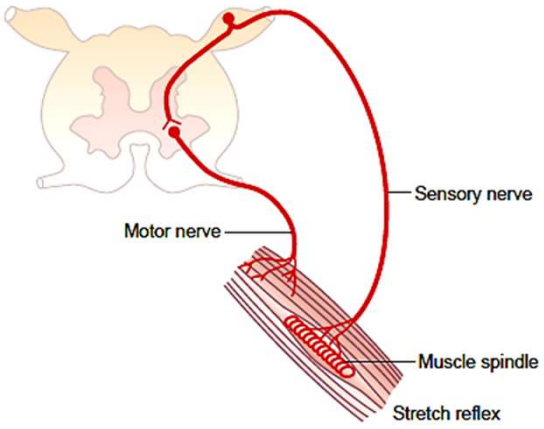


57

Physiological significance of the stretch reflex

1- Helps to maintain the muscle length.

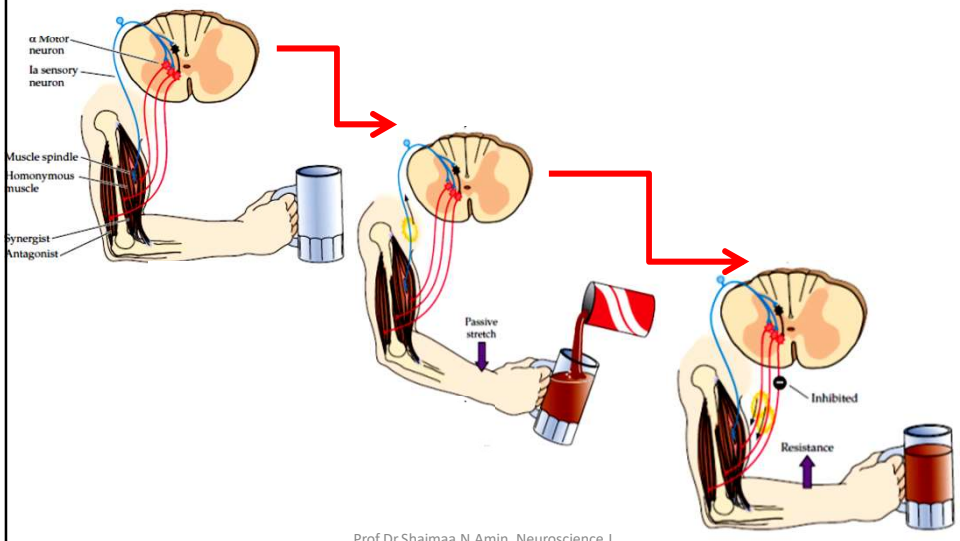




58

Physiological significance of the stretch reflex

1- Helps to maintain the muscle length.



59

Physiological significance of the stretch reflex

2-Muscle tone

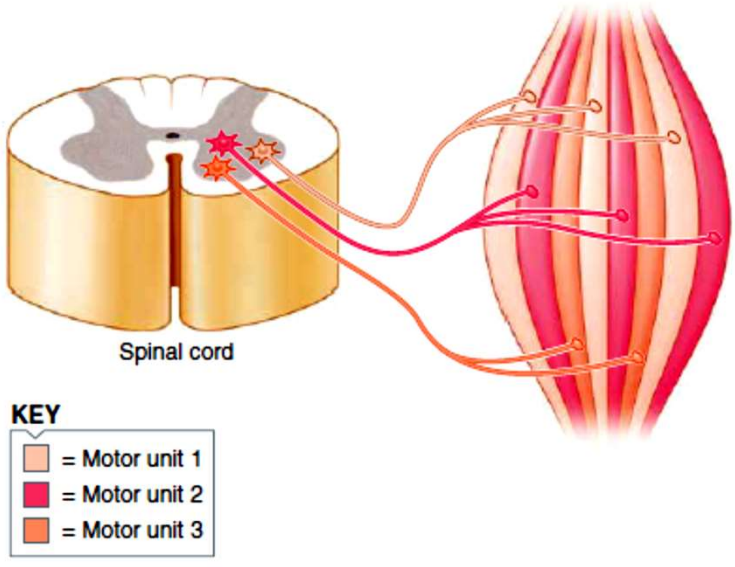
Def: Muscle tone is **continuous**, **alternating**, **reflex**, **subtetanic** contraction of muscle fibres.

–It may also defined as the resistance of the muscle to stretch.

–Base of muscle tone: Static stretch reflex

60

61



62

During rest the muscle spindle is continuously stretched

63

Continous impulses from the supraspinal centers to gamma motor neurons

↓

Contraction of the peripheral contracile part of intrafusal muscle fiber

↓

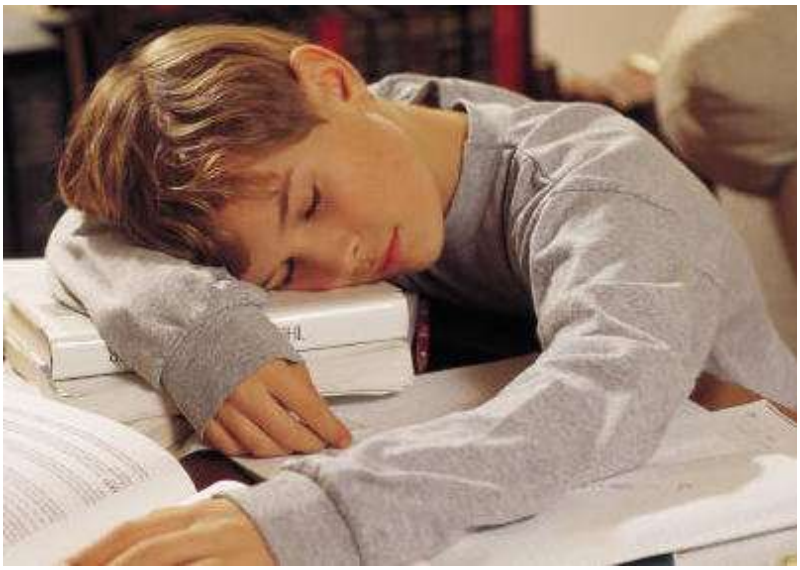
Stretch of the central receptor part of the spindle

↓

Stretch reflex

Gamma Spindle loop (Gamma led contraction)

64



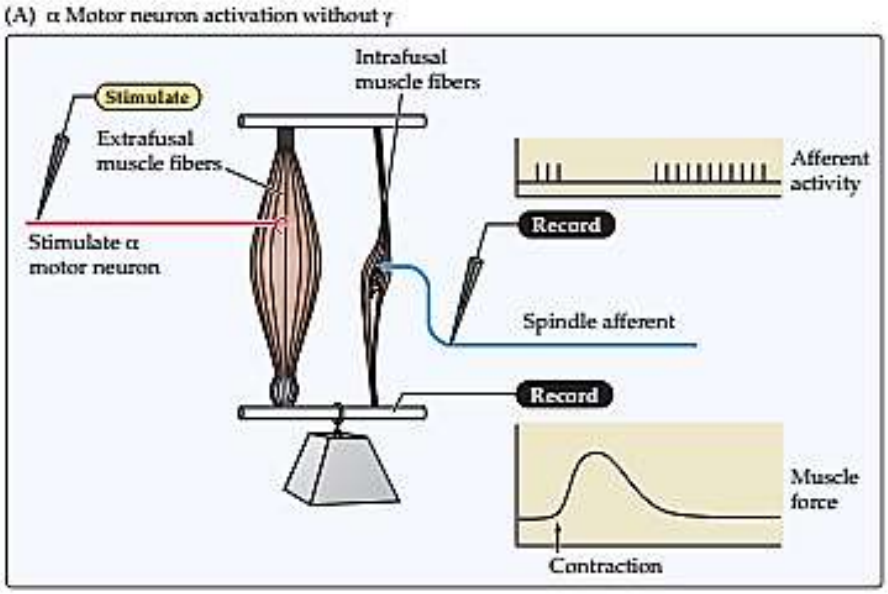
65

Muscle tone does not cause fatigue ?

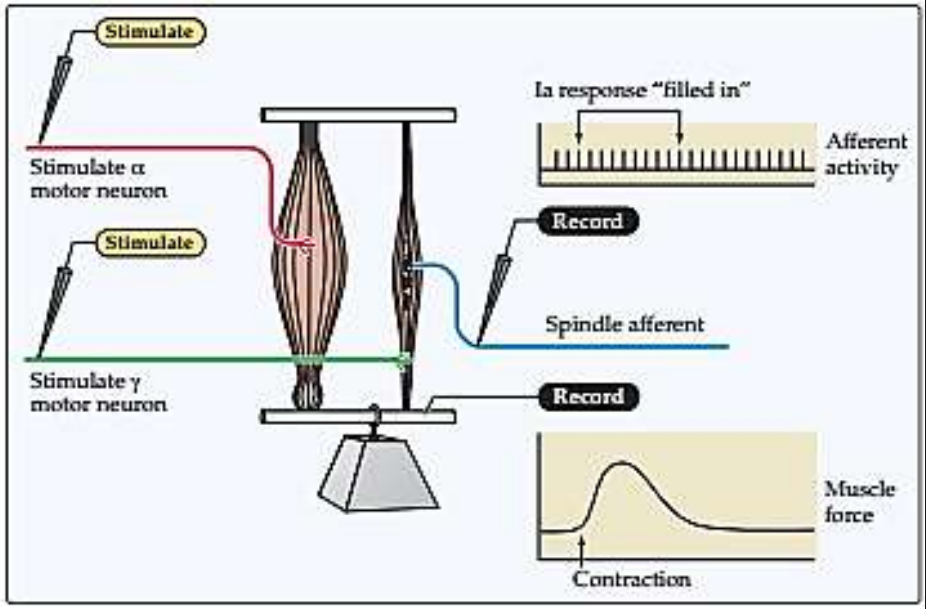
66

Testing Muscle Tone

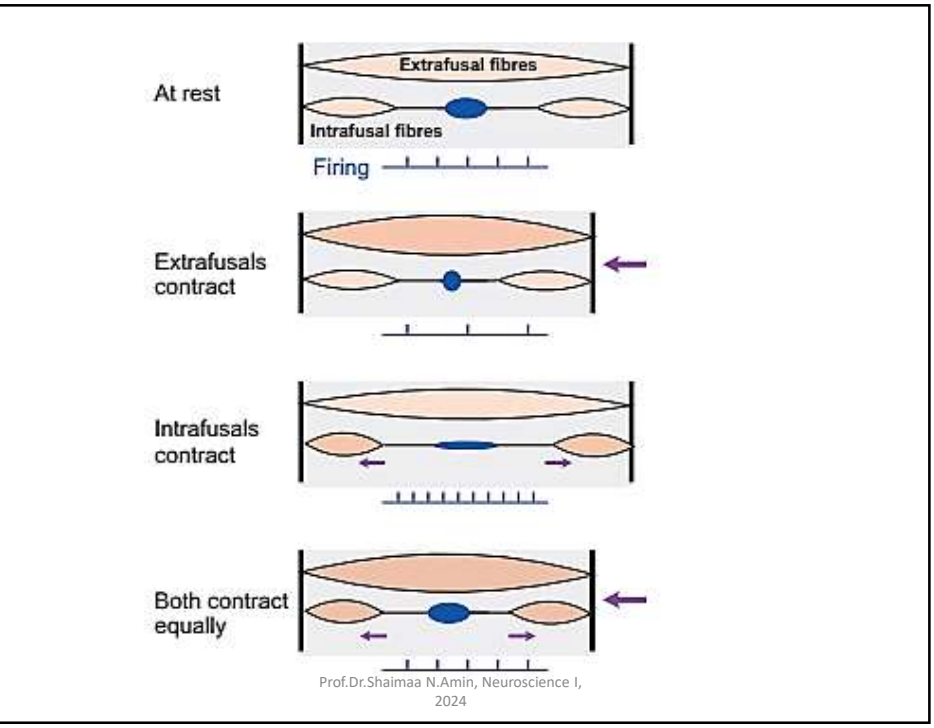
67



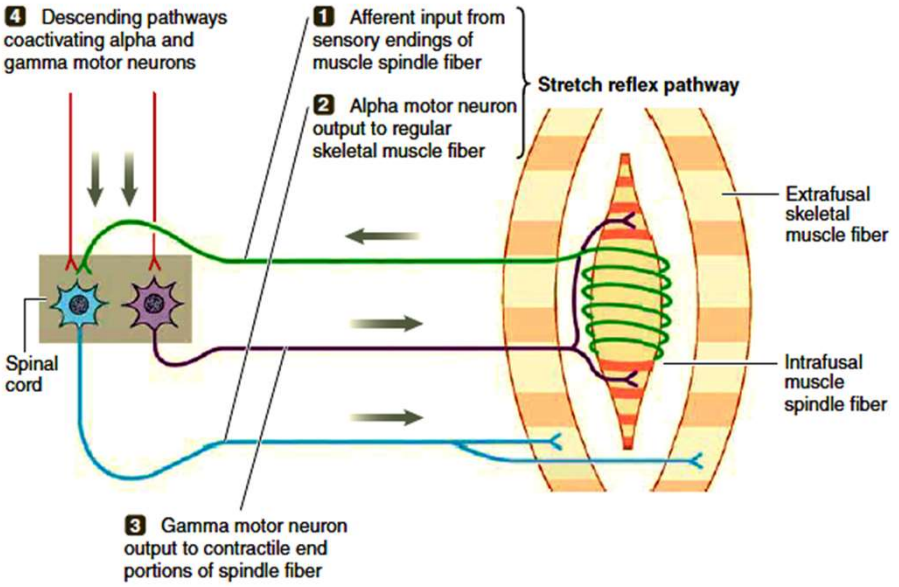
68


(B) α Motor neuron activation with γ
Stimulate
Stimulate α motor neuron
Stimulate
Stimulate γ motor neuron
Ia response "filled in"
Afferent activity
Record
Spindle afferent
Record
Muscle force
Contraction

69


At rest
Extrafusal fibres
Intrafusal fibres
Firing
Extrafusals contract
Intrafusals contract
Both contract equally

70

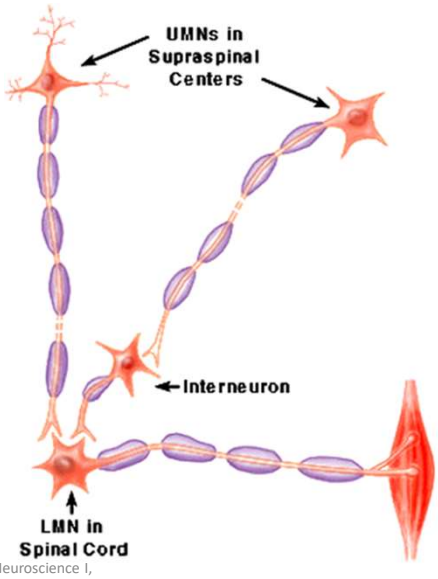


Pathways involved in monosynaptic stretch reflex and coactivation of alpha and gamma motor neurons

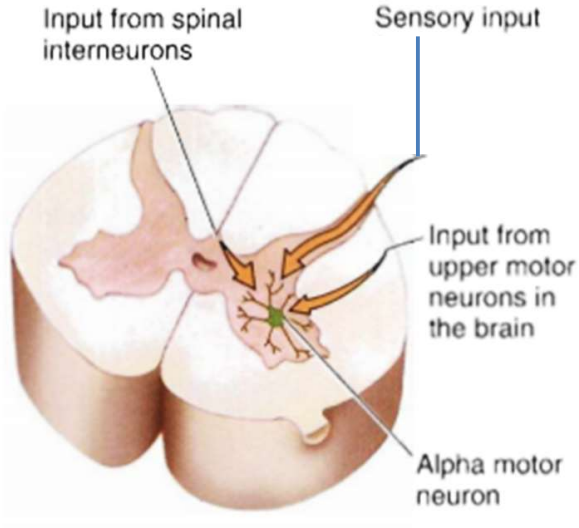
71

- Cells that carry signals from supraspinal centers to LMNs of the spinal cord are **the Upper Motor Neurons (UMNs).**

-**UMNs** carry **motor commands** to the LMNs, which **execute them** by causing muscle contraction.

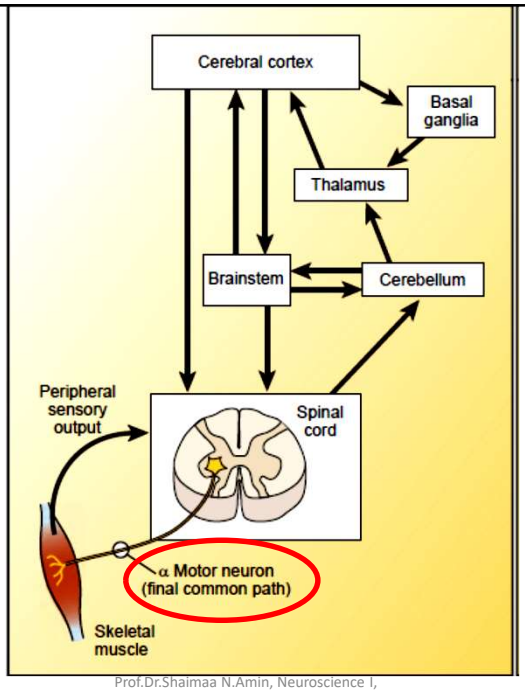


72

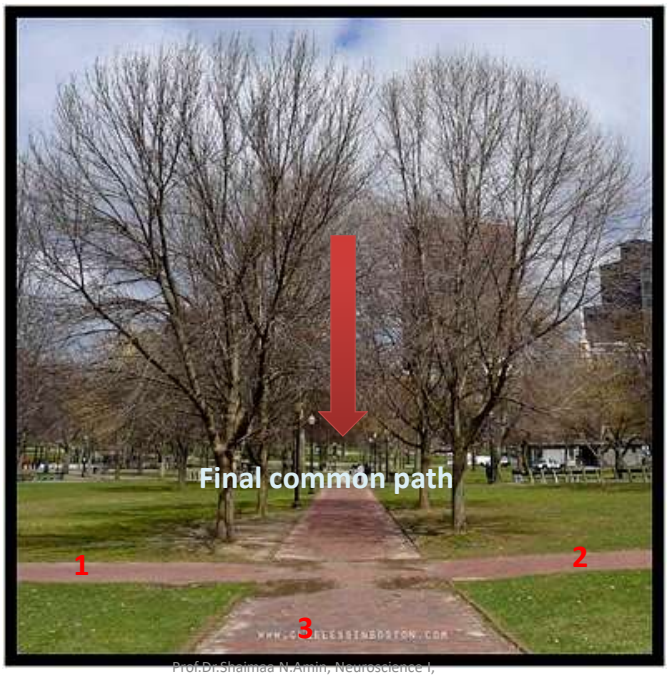


Alpha motor neuron and its three sources of input

73



74

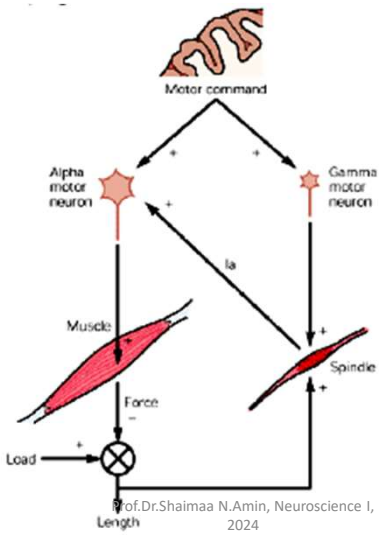


75

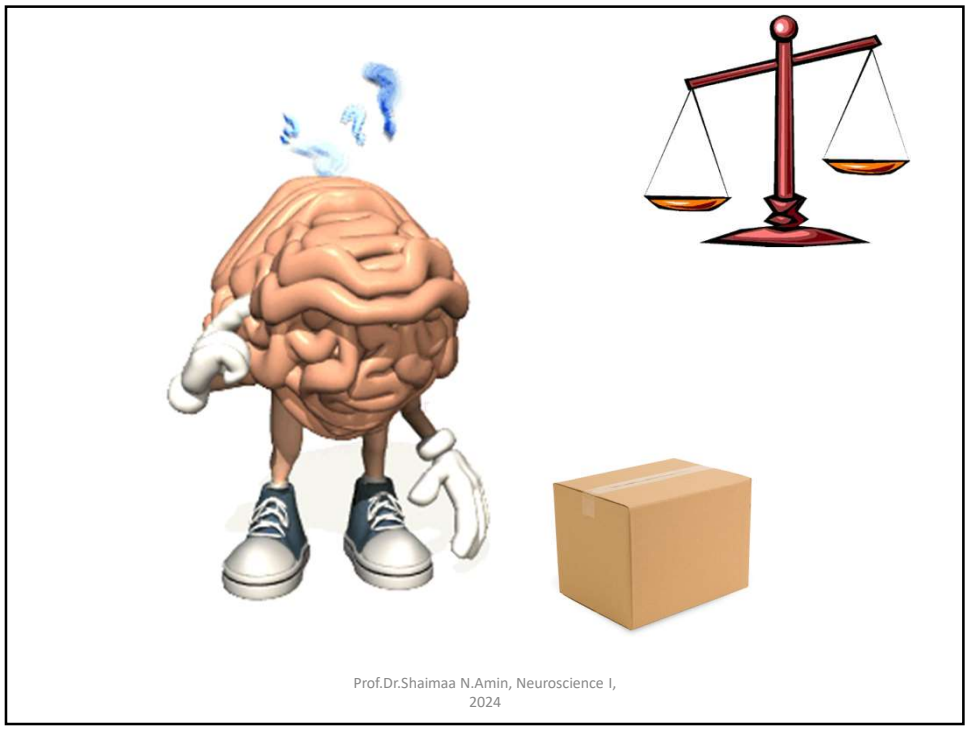
Physiological significance of the stretch reflex

2- Control of voluntary movements.

a-Servo-assistant function :



76



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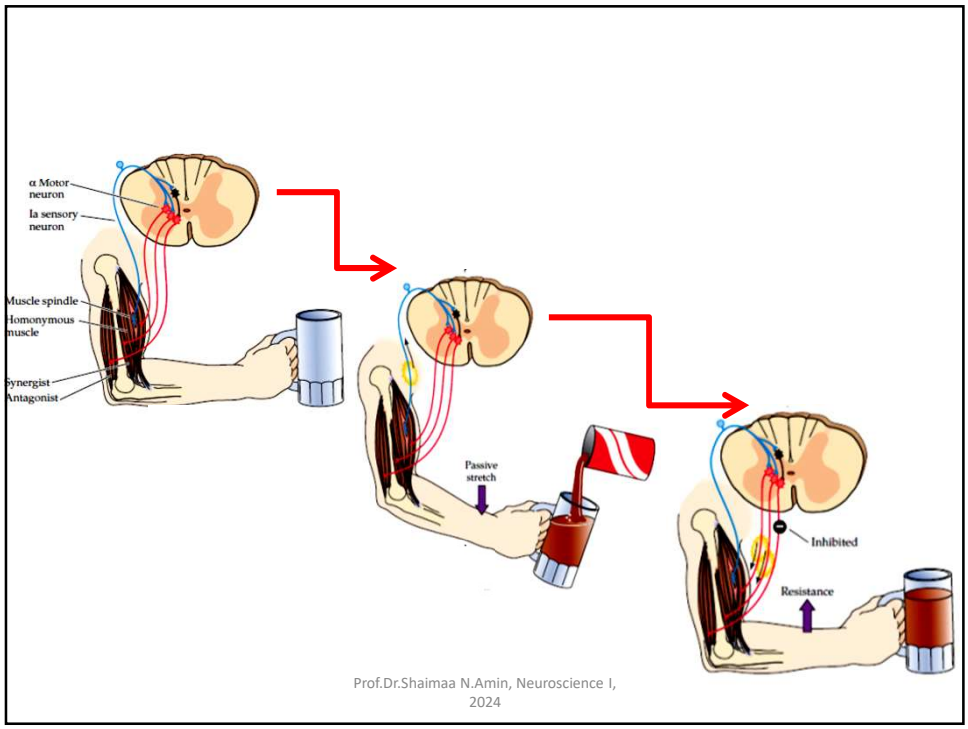
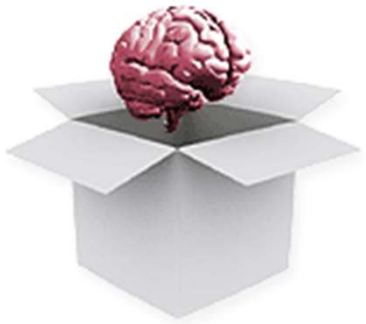

α Motor neuron
Ia sensory neuron
Muscle spindle
Homonymous muscle
Synergist
Antagonist
Passive stretch
Inhibited
Resistance

78



Supraspinal control of stretch reflex

A-SUPRA SPINAL FACILITATORY AREAS

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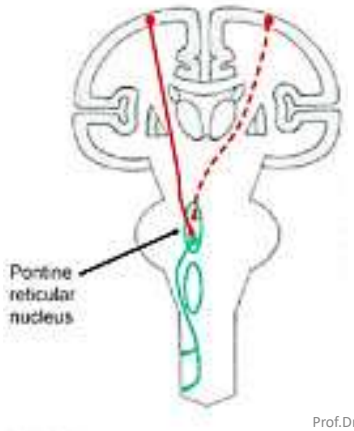
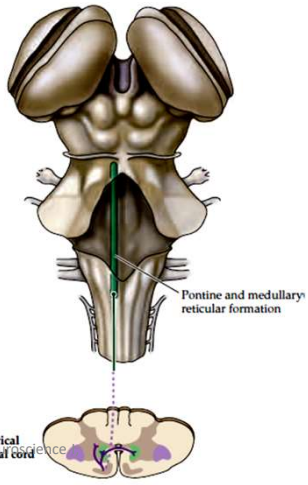
Supraspinal Facilitatory Areas

1-Facilitatory reticular formation

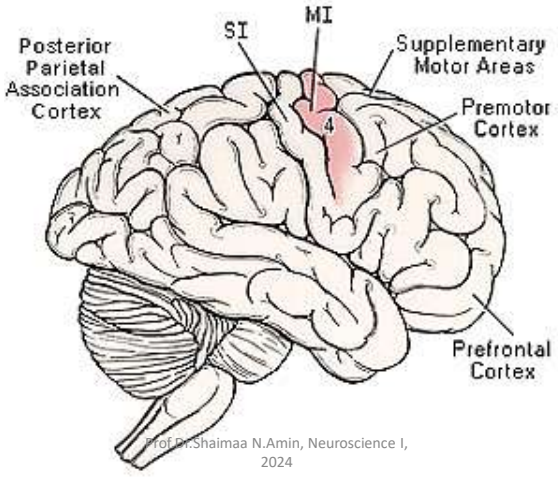




80

Supraspinal Facilitatory Areas

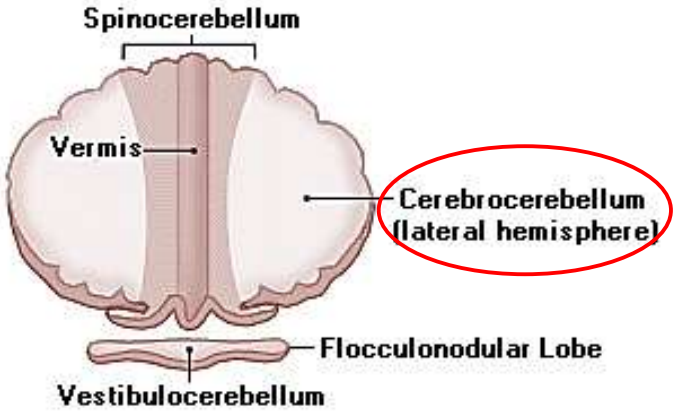
2-Primary motor area 4



81

Supraspinal Facilitatory Areas

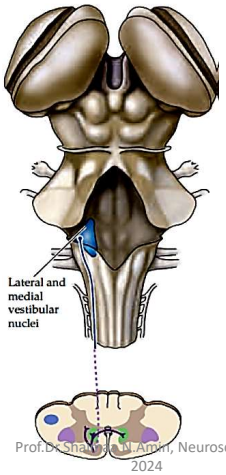
3-Neocerebellum



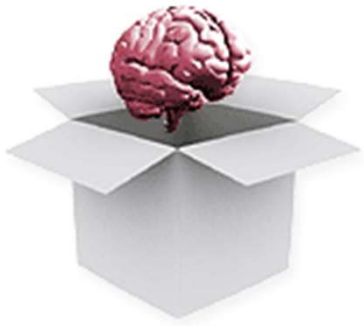
82

Supraspinal Facilitatory Areas

4-The lateral Vestibular nucleus



83



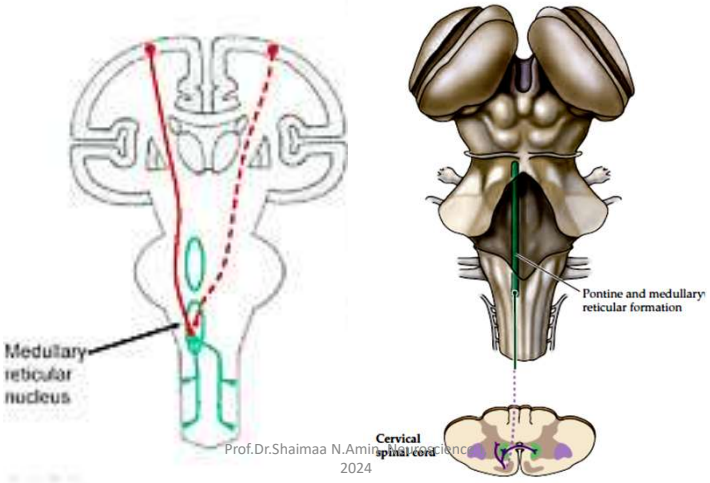
Supraspinal control of stretch reflex

A-SUPRA SPINAL INHIBITORY AREAS

84

Supraspinal Inhibitory Area

1-Inhibitory reticular formation



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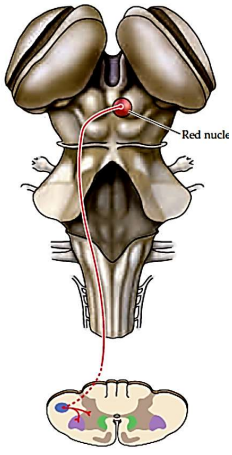
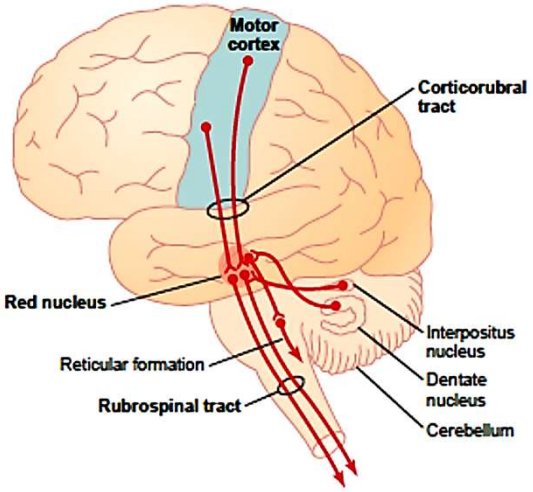
Supraspinal Inhibitory Area

2-Red Nucleus





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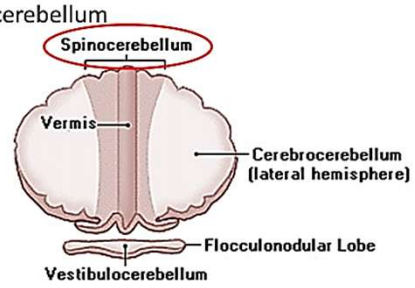
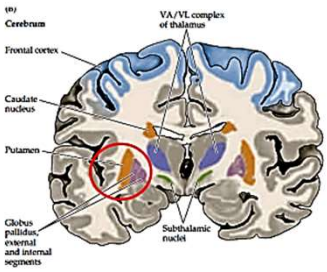
Supraspinal Inhibitory Area

3-Paleocerebellum

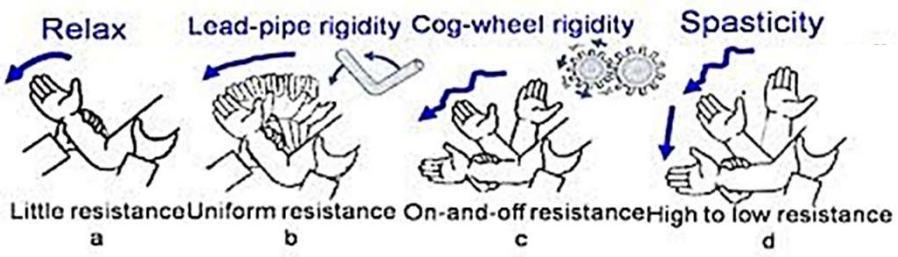


4-Basal ganglia (lentiform neucleus)



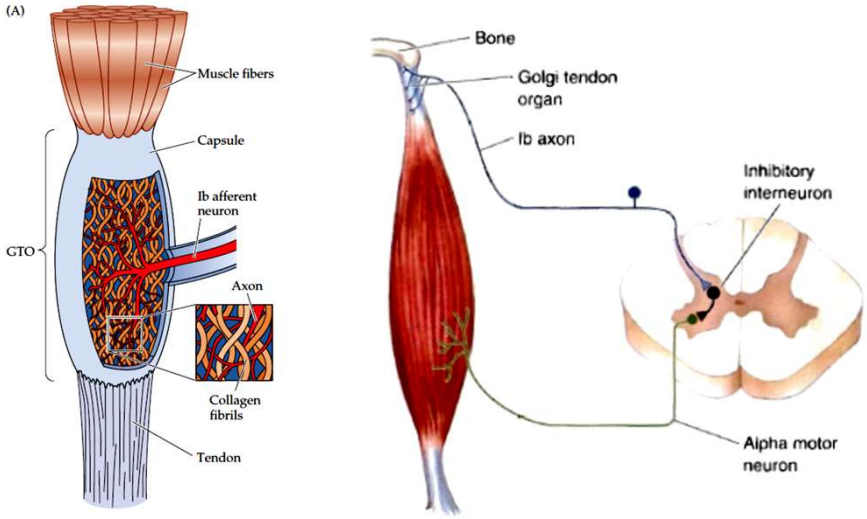
5-Cortical suppressor area (4S)

87



88

Inverse stretch Reflex
(Autogenic inhibition)



89

	STRETCH REFLEX	INVERSE STRETCH REFLEX
synapses	Monosynaptic	Disynaptic
Receptor	spindles	Golgi tendon organs
Stimulus	Muscle stretch (increased muscle length)	Excessive muscle stretch (increased muscle tension)
Effect	Muscle contraction	Muscle relaxation
Function	Maintenance of erect posture and smoothing movements	It prevents the tearing of tendons during strong muscle contraction

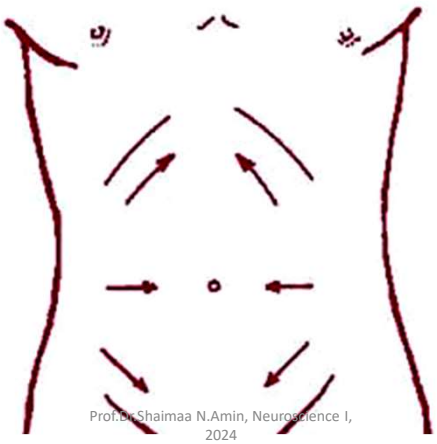
90

CLONUS
This is alternating rhythmic contractions and relaxations of a muscle in response to sudden maintained stretch.

91

Polysynaptic reflexes

- **Superficial abdominal reflexes**



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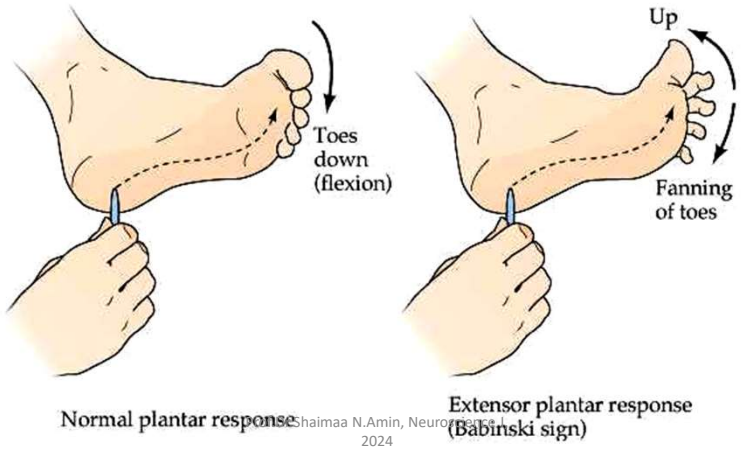
Polysynaptic reflexes

- Planter reflex



93

Polysynaptic reflexes

- Planter reflex

- Positive supporting reaction

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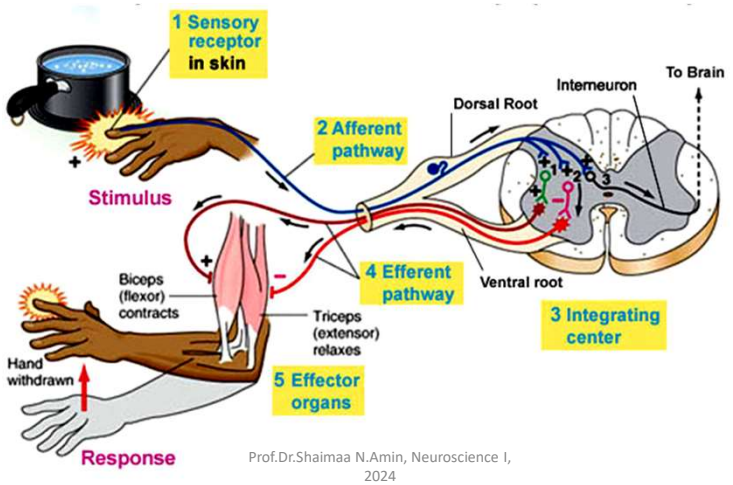
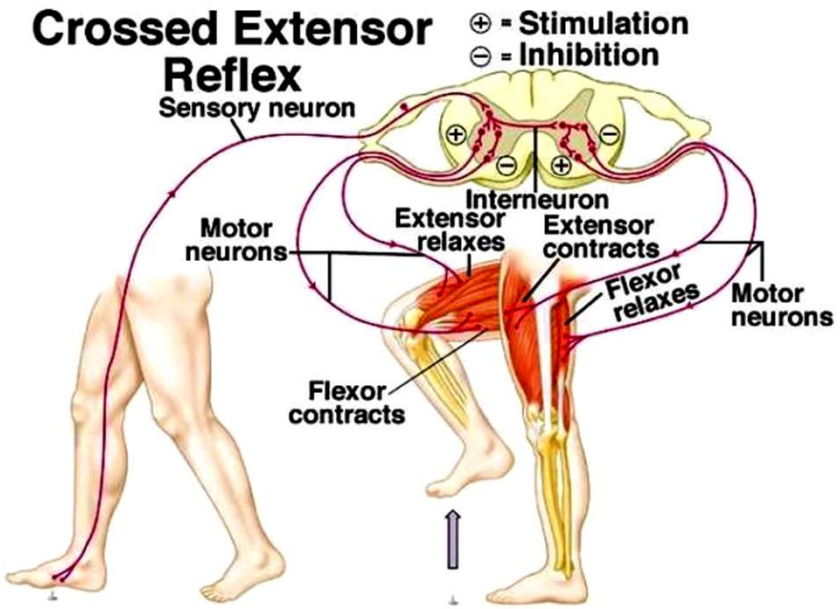
Polysynaptic reflexes

- **Withdrawal reflex**



95

Crossed Extensor Reflex



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Polysynaptic reflexes

- Scratch reflex

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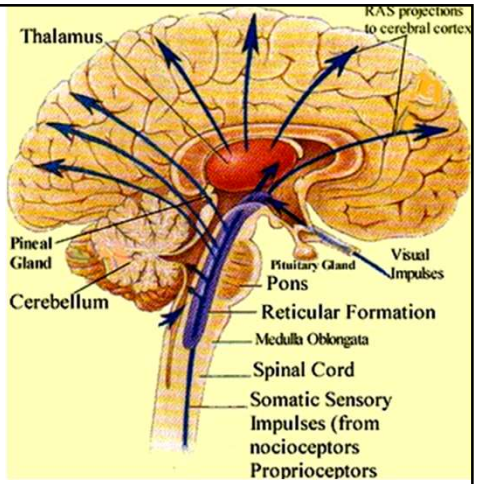
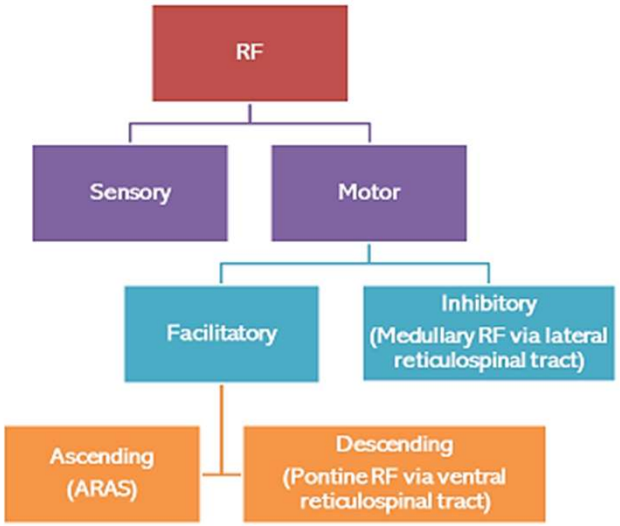
The Brain Stem

- The brain stem is a complex extension of the spinal cord, which performs sensory, motor and reflex functions.
- The reticular formation is a large structure occupying the core of the brain stem.



98



- **What are the factors that increase or decrease the activity of ARAS affecting the arousal response?**

99

- The pontine reticular system "facilitatory reticular formation":

- **Has spontaneous intrinsic activity which is enhanced by impulses from:**
  - **Motor area 4 of the cerebral cortex.**
  - **The vestibular nucleus.**
  - **The neocerebellum.**
  - **The classical sensory pathways.**

- The Medullary Reticular System"inhibitory reticular formation":

- **It does not discharge spontaneously, but driven by impulses from the:**
  - **Suppressor area of the motor cortex.**
  - **Basal ganglia.**
  - **Paleocerebellum.**

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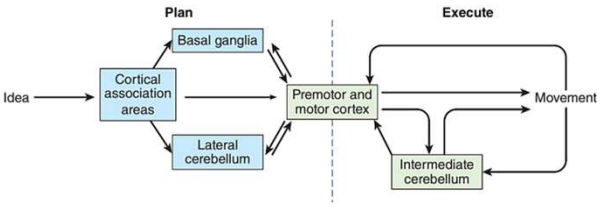
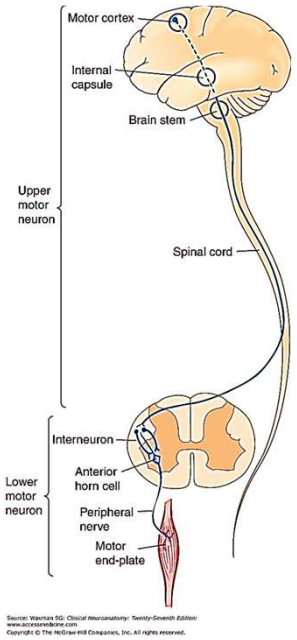
UMNL vs LMNL/Spinal cord transection





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UMNL

Cause:

Cerebrovasular accidents (strokes) **due to :**

1- hemorrhage or

2- thrombosis

in the posterior limb of the internal capsule.

- There is damage of both pyramidal and extrapyramidal fibres.

LMNL

Cause:

Is due to :

1- lesion of the lower motor neurons as in poliomyelitis.

2- damage of motor nerves e.g. diabetes mellitus, and alcoholism.

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UMNL	***LMNL***
1. Paralysis:	***1. Paralysis:***
Occurs on the opposite side of the body i.e. contra lateral hemiplegia. It is widespread affecting lower half of the face, upper limb and lower limb. Recovery is poor	Occurs in the muscles supplied by the affected segment only e.g. muscles of the limb only, on the same side of the lesion. Recovery may occur.

103

UMNL	***LMNL***
2. Hypertonia	***2. Hypotonia or atonia:***
The paralyzed muscles show increased tone of the spastic type. **(Clasp Knife Type).** Hypertonia is due to ???	• The paralyzed muscles show decrease or loss of tone, referred to as flaccid paralysis. • Hypotonia is due to interruption of the stretch reflex.

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GAMMA RIGIDITY AND ALPHA RIGIDITY

	Gamma rigidity	Alpha rigidity
Cause	Increased gamma discharge	Increased alpha discharge
Muscles affected	Antigravity muscles	All muscles
Resistance to movement	Uni-directional	Bi-directional
Type of rigidity	Clasp -knife	Lead- Pipe or cogwheel
Effect of velocity	Increases with velocity	Not velocity-dependent
Tendon jerks	Exaggerated and clonus may also be present	Not necessarily exaggerated
Common diseases	Upper motor neuron lesion	Parkinsonism

105

UMNL

3. Exaggerated Tendon Reflexes.-

- Deep reflexes are exaggerated on the affected side .g. exaggerated knee jerk and ankle jerk, and are due to the release of the stretch reflex from cerebral inhibition.
- **Clonus** is present (see before).

LMNL

3. Absent Deep Reflexes.

This occurs in the muscles supplied by the affected segments or motor nerves.

Have you done Jendrassik maneuver before reporting areflexia??

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Figure 1-33 Prof.Dr.Shaimaa N.Amin, Neuroscience I, 2024
The Jendrassik maneuver.

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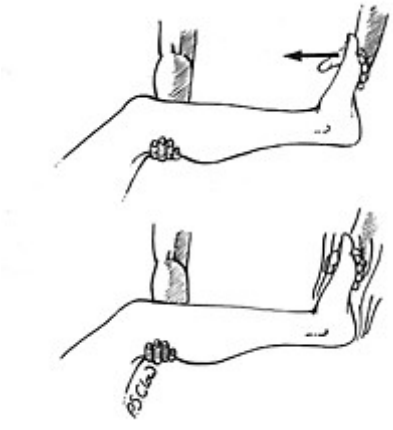
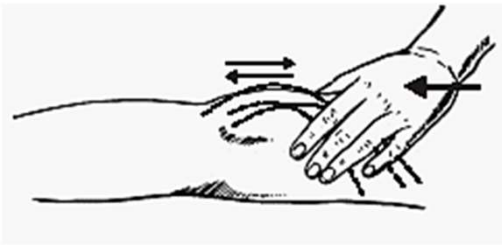


Clonus.



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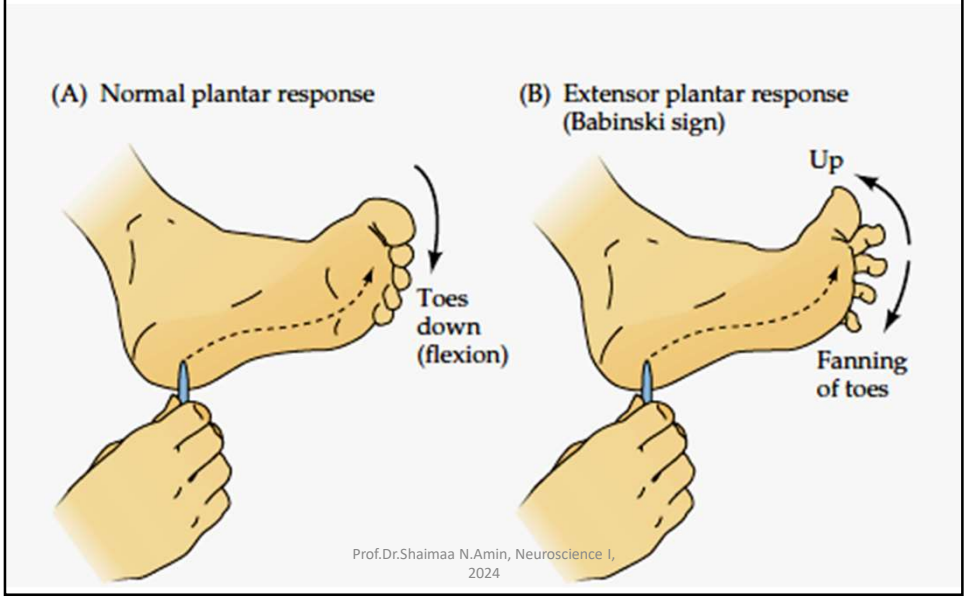
UMNL	***LMNL***
4. Loss Of Superficial Reflexes	***4. Loss Of Superficial Reflexes***
• Occurs on the affected side, due to loss of supra-spinal facilitation. • plantar reflex becomes extensor, known as a **positive Babinski's sign.**	**S**een in the affected segments only.

109

The Planter reflex



110

UMNL	***LMNL***
5. delayed Muscle-Wasting :	***5. early Marked Muscle-Wastin) :***
This is because : paralyzed muscles are still innervated and can contract reflexly.	The affected muscles show marked atrophy due to the inability of the muscles to contract neither reflexly nor voluntarily.

111

Response to electric stimulation:

Normally, stimulation of skeletal muscles by Faradic (alternating) currents produces tetanus while their stimulation by Galvanic (direct) currents produces contraction only at the make closing) and break (= opening) of the circuit. whether the cathodal or the anodal electrode was used for stimulation. Therefore. in the latter condition, there are 4 contraction states (1) CCC (= cathodal closing contraction) (2) COC (= cathodal opening contraction) (3) ACC anodal closing contraction) (4) AOC anodal opening contraction). and the strength of contraction was found to be as follows:

CCC > ACC > AOC > COC

112

In UMNL, the paralyzed muscles respond normally to both currents and their chronaxies are also normal. On the other hand, the response in the case of LMNL is altered and is called the reaction of degeneration.

113

REACTION OF DEGENERATION (RD)

This is an abnormal response of skeletal muscles to electric stimuli that occurs in LMNL only. It is characterized by:

(l) The chronaxie of the paralyzed muscles is prolonged.

(2) There is no response to Faradic currents.

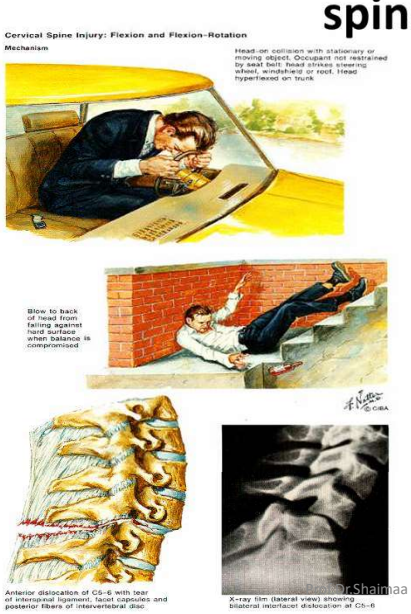
(3) There is a sluggish (= weak) response to Galvanic currents.

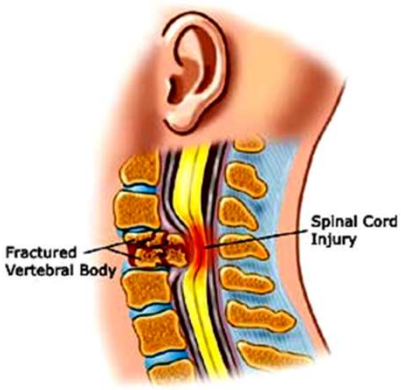
(4) The ACC becomes stronger than the CCC (due to unknown causes).

The RD is one of the manifestations of muscle denervation. The reaction of denervation is the diagnostic feature of LMNL.

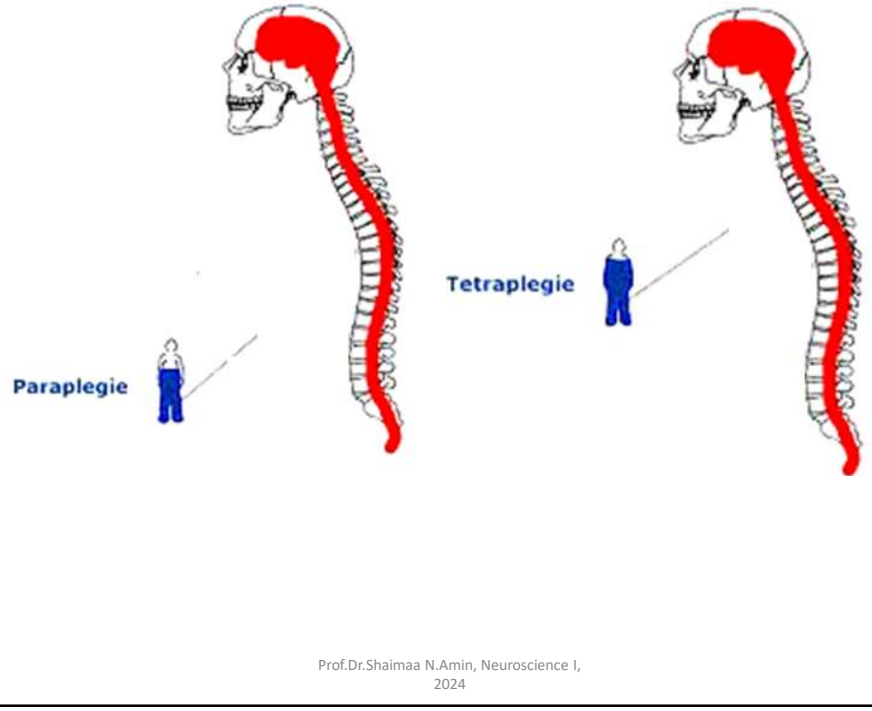
114

Complete transection of the spinal cord





115



116

Stages after spinal cord lesion

A. Stage of spinal shock

B- Stage of recovery of reflexes

C- Failure of reflex activity (rare) .

117

A -Stage of spinal shock

Duration of spinal shock

- Varies in duration depending on the degree of encephalization .
- Usually occupies a period of 2-6 weeks in human. (human>dog>cat>rat>frog).

Characterized by the following features, all of which occur below the level of the lesion:

1- Loss of all reflexes (superficial, deep and visceral).

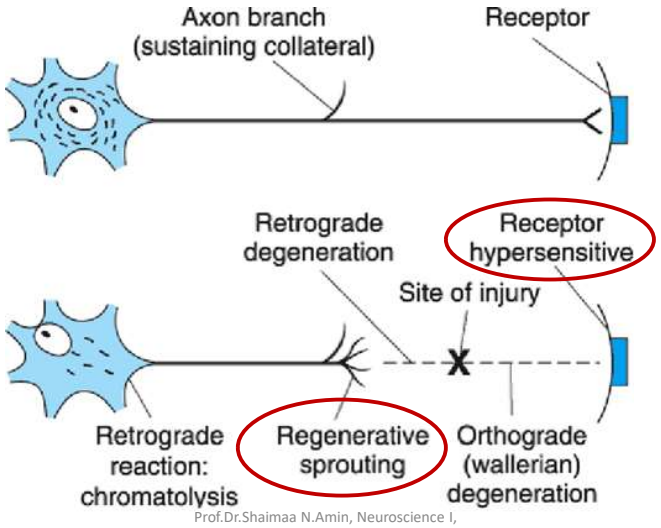
2- Permanent Loss of all sensations .

3- Permanent Loss of all voluntary movements.

Micturation reflex: Retention with overflow

118

B -Stage of recovery of reflex activity

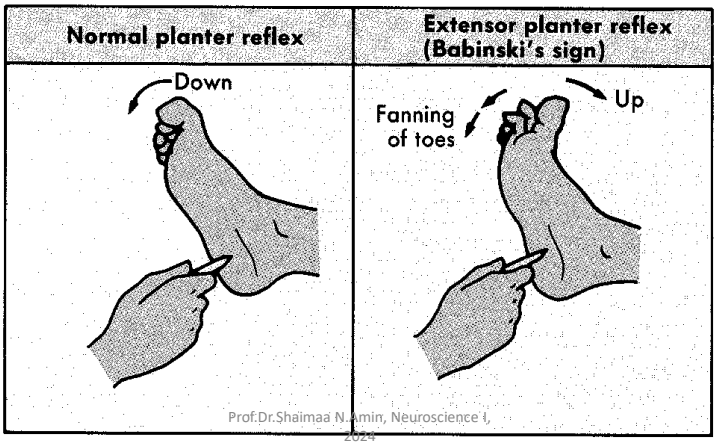


119

2- *Return of spinal reflexes* :

a- Flexor reflexes return earlier than extensor ones.

Planter reflex ? +ve Babniski sign ???



120

3- Return of visceral reflexes

automatic evacuation, **but** voluntary control over micturation and sensation of bladder fullness are **permanently lost**.

4- Mass reflex

A minor painful stimulus to the skin of the lower limbs will cause:

1- Flexion withdrawal of the limb.

2- Evacuation of the bladder and rectum.

3- Sweating of the skin.

4- Rise of blood pressure.

intentional mass reflex → MICTURATION & DEFECATION

121

Advanced stage OF Recovery

1- The tone in extensor muscles gradually returns and becomes greater than in flexors "paraplegia in extension".

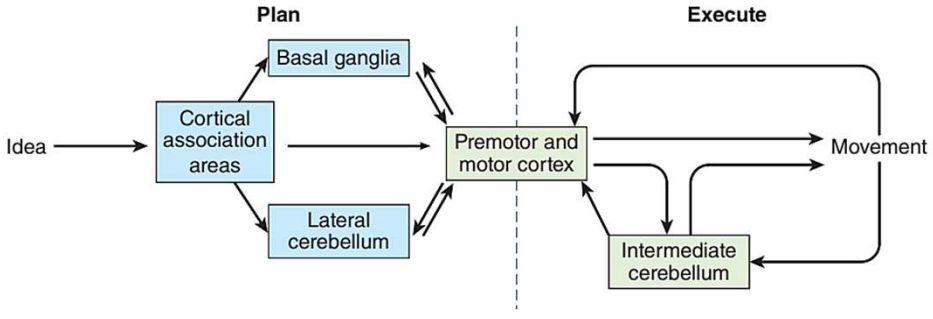
2- Positive supporting reflex becomes well developed and the patient can stand without support. (But he will never walk).

122

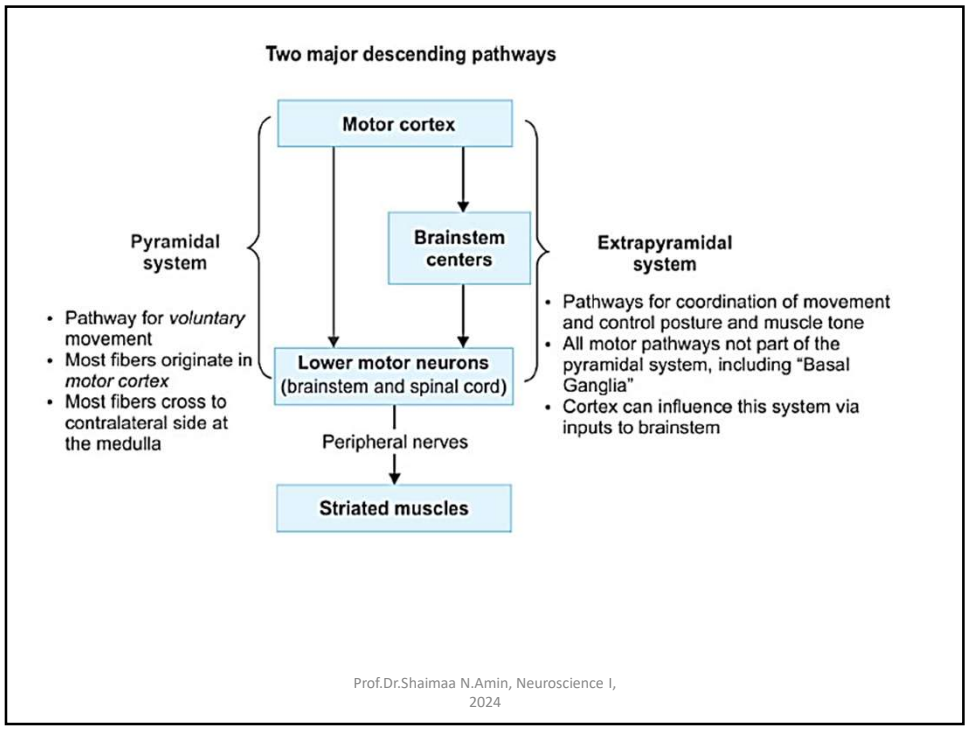
STAGE OF FAILURE OF SPINAL REFLEX ACTIVITY

- This is a terminal stage that usually results from bad management during the recovery stage. It is often associated with general toxemia due to infection of the bed sores or the urinary tract (and the latter frequently terminates by uremia).

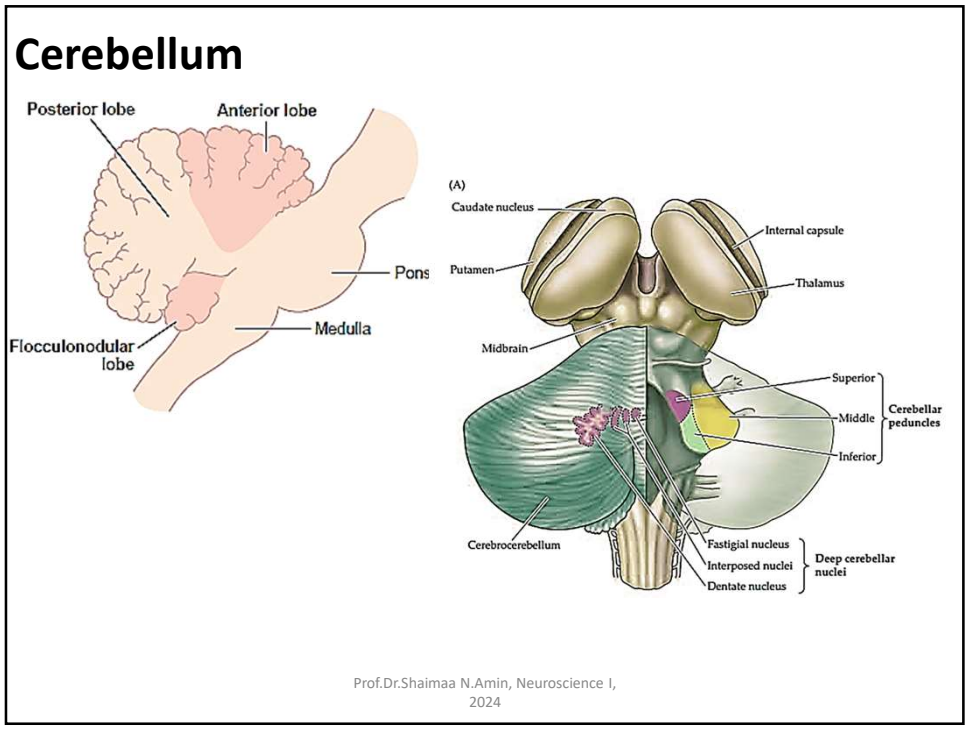
123



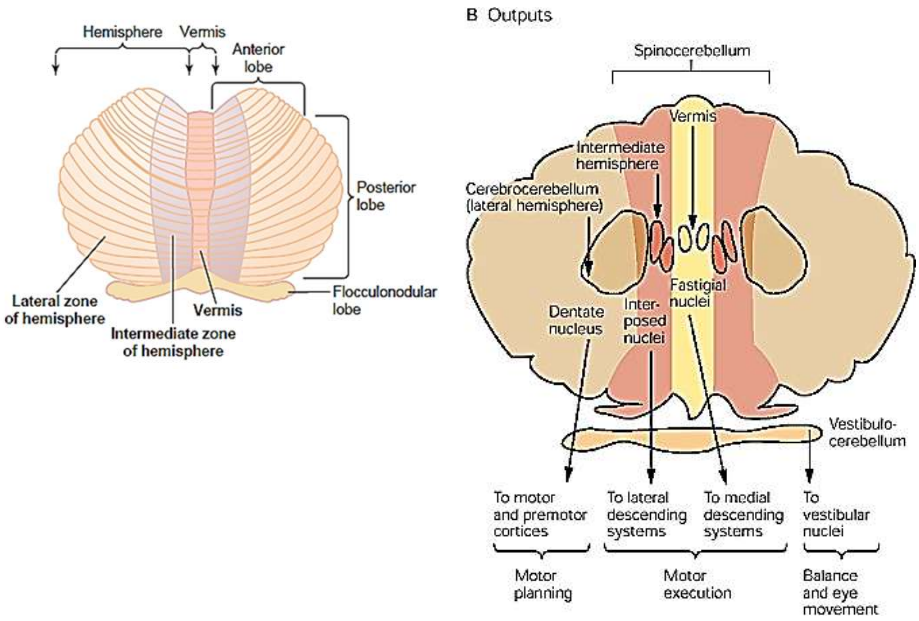
124


Two major descending pathways
Motor cortex
Brainstem centers
Lower motor neurons (brainstem and spinal cord)
Peripheral nerves
Striated muscles
Pyramidal system
• Pathway for *voluntary* movement
• Most fibers originate in *motor cortex*
• Most fibers cross to contralateral side at the medulla
Extrapyramidal system
• Pathways for coordination of movement and control posture and muscle tone
• All motor pathways not part of the pyramidal system, including "Basal Ganglia"
• Cortex can influence this system via inputs to brainstem

125

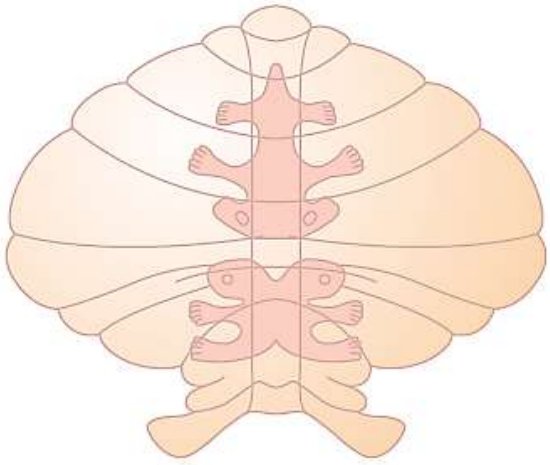

Cerebellum
Posterior lobe
Anterior lobe
Pons
Medulla
Flocculonodular lobe
(A)
Caudate nucleus
Putamen
Internal capsule
Thalamus
Midbrain
Superior
Middle
Inferior
Cerebellar peduncles
Cerebrocerebellum
Fastigial nucleus
Interposed nuclei
Dentate nucleus
Deep cerebellar nuclei

126



127

Topographic representation of the body in the cerebellum



128

Cerebellar Connections

- **Afferent fibres the pass to the cerebellum are:**

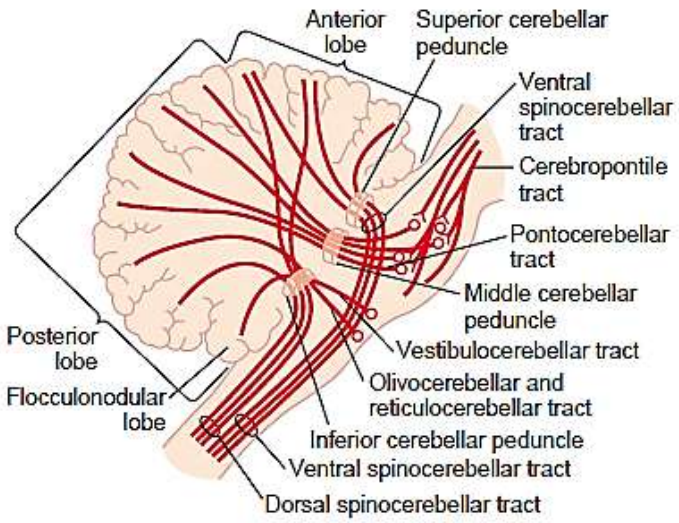
Peduncle	Afferent
1-Superior cerebellar peduncle	Ventral spino-cerebellar tract
2-Middle cerebellar peduncle	Cortico-ponto-cerebellar tract
3-Inferior cerebellar peduncle	A-Dorsal spinocerebellar tract B-Vestibulo-cerebellar tract C-Olivo-cerebellar tract

- **Efferent fibres from the cerebellum are:**

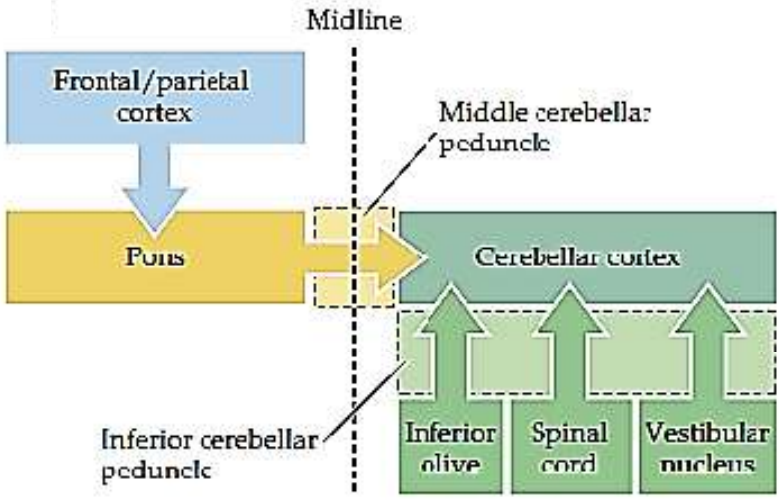
Peduncle	Afferent
1-Superior cerebellar peduncle	A-Dentato-thalamo-cortical tract B-Dentato-rubro-spinal tract.
3-Inferior cerebellar peduncle	A-fibers to reticular formation of the pons. B-Fibers to reticular formation of the medulla.

129

I. Afferent fibres to the cerebellum

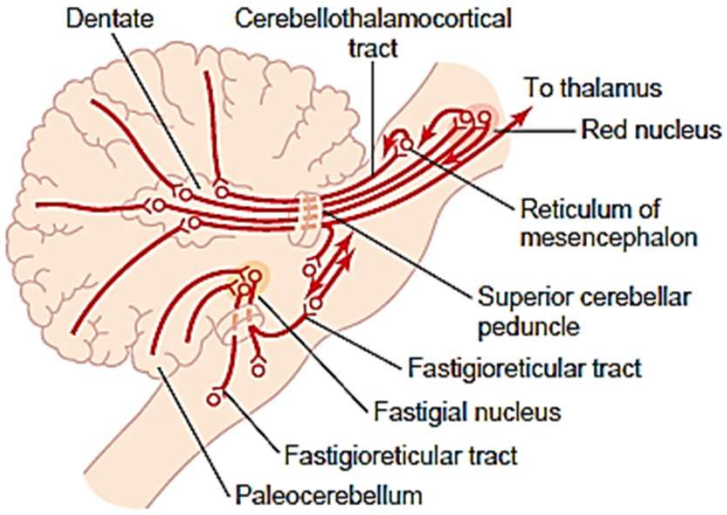


130

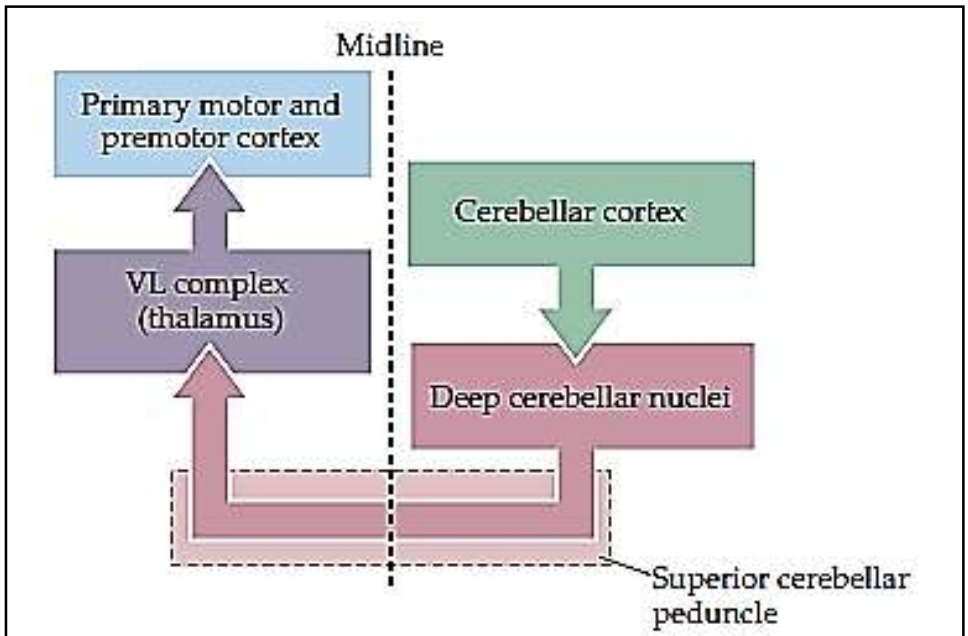


131

II. Efferent fibres from the cerebellum

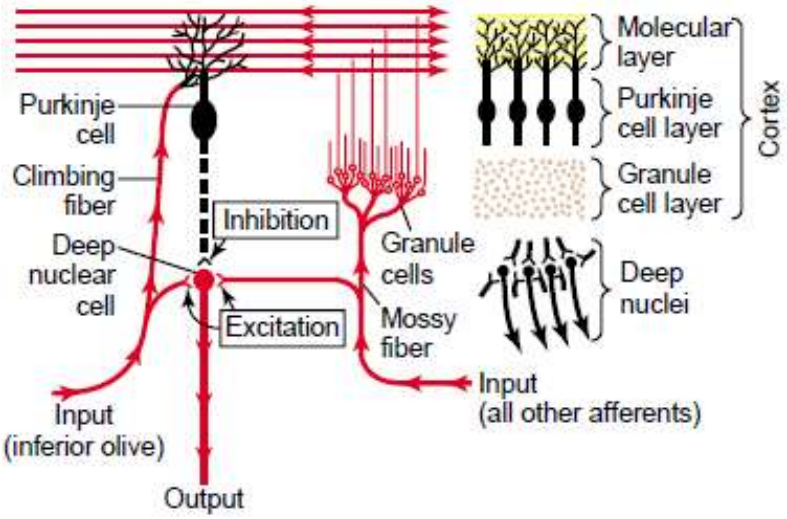


132

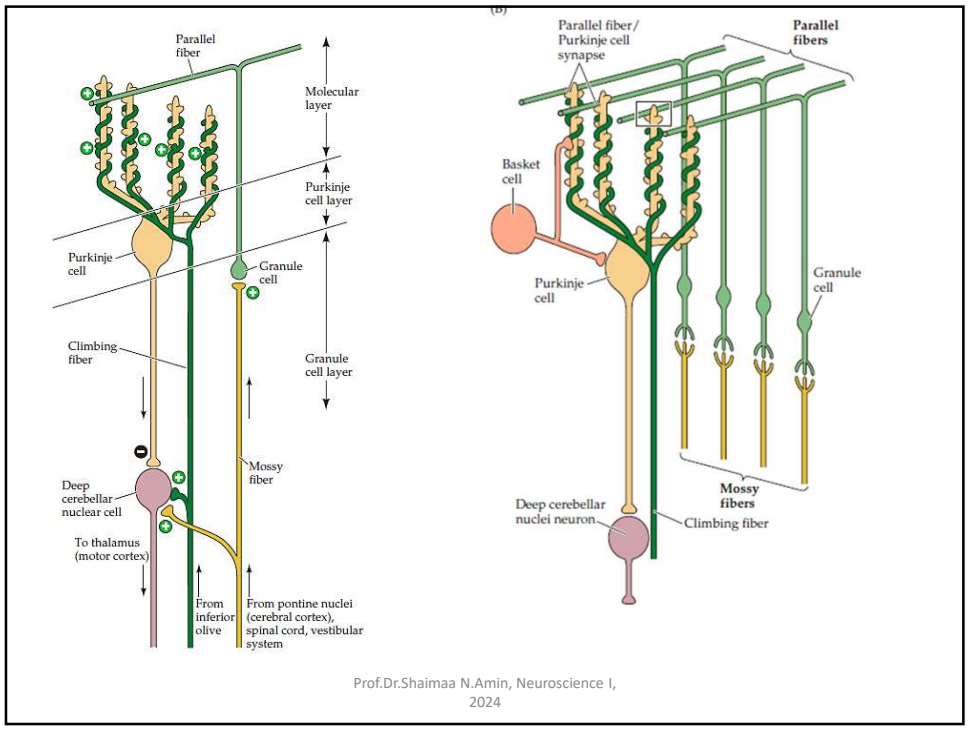


133

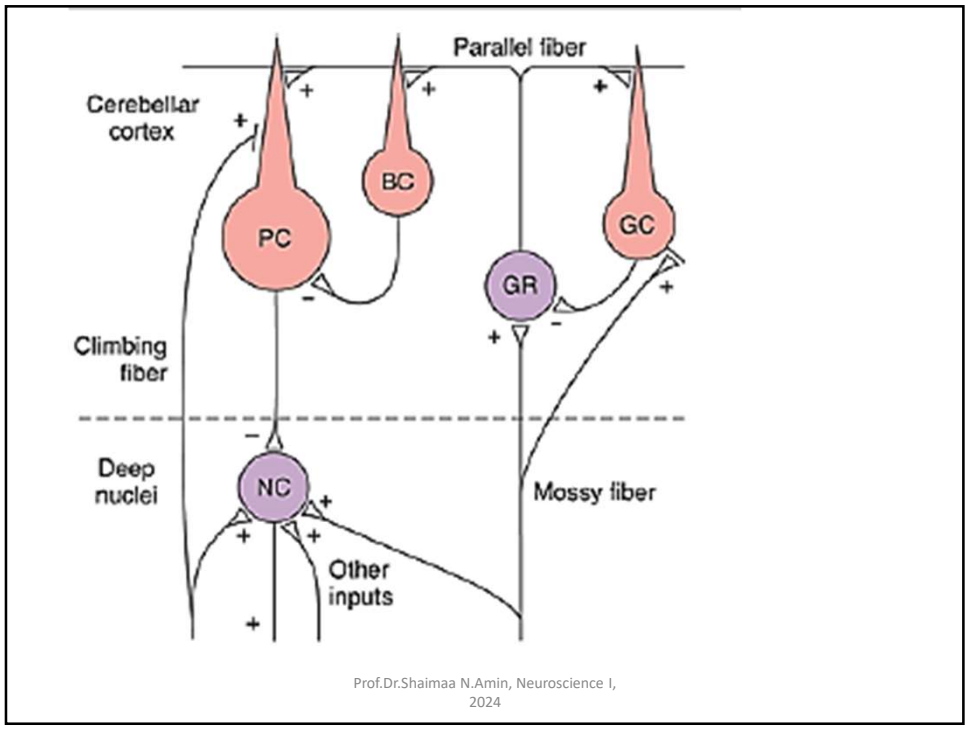
Histological Structure And Connections Of The Cerebellum



134


Parallel fiber
Molecular layer
Purkinje cell layer
Purkinje cell
Granule cell
Climbing fiber
Granule cell layer
Mossy fiber
Deep cerebellar nuclear cell
To thalamus (motor cortex)
From inferior olive
From pontine nuclei (cerebral cortex), spinal cord, vestibular system
Parallel fiber / Purkinje cell synapse
Parallel fibers
Basket cell
Purkinje cell
Granule cell
Mossy fibers
Deep cerebellar nuclei neuron
Climbing fiber

135


Parallel fiber
Cerebellar cortex
BC
PC
GC
GR
Climbing fiber
Deep nuclei
NC
Mossy fiber
Other inputs

136

Functions of the cerebellum

- A. Functions of the cerebellum in voluntary movements:

1-Servo-comparator function.

2-The braking effect.

3-Planning and timing function.

- B. Other functions:

1-Function in equilibrium.

2-Function in muscle tone.

137

The Neocerebellar syndrome

- Due to damage of the deep cerebellar nuclei as well as the cerebellar cortex.
- Manifestations occur on the *same side* of the lesion.

A.Ataxia:

B-Other manifestations

1- Disturbance of Posture and Gait
2- Dysarthria
3- Dysmetria
4-Intention tremors
5- Rebound phenomena
6- Adiadochokinesia
7- Decomposition of movements
8-Nystagmus
9-Hypotonia

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1- Disturbance of Posture and Gait :

a- head is tilted to the side of the lesion.

b- unsteady drunken gait (zigzag line) .

C- patient tends to fall towards the side of the lesion .

2- Dysarthria :

Slurred or Scanning Speech

= Staccato speech

3- Dysmetria :

----------→ hypermetria

X

----------→ hypometria

139

4-Intention tremors:

Mechanism:

Dysmetria initiates gross correction action → correction overshoot to the other side → So fingers oscillates back and forth

Appears only during movement

Absent : Rest and sleep

5- Rebound phenomena :

Inability to stop the movement at the proper time = inability to put on the brake .

6- Adiadochokinesia

Inability to do rapid succesive movements

e.g. repeated supination and pronation

140

141

7- Decomposition of movements :

Inability to do a complex movement that involves simultaneous motion at more than one joint

Test : Heel – knee test

8-Nystagmus :

This is tremor of the eye ball, which occurs when the patient attempts to fix his gaze on an object to the side of his head (*Horizontal nystagmus*).

It is due to absence of damping function.

9-Hypotonia:

Marked hypotonia on the side of the lesion due to loss of the facilitatory effect of the cerebellum on the stretch reflex.

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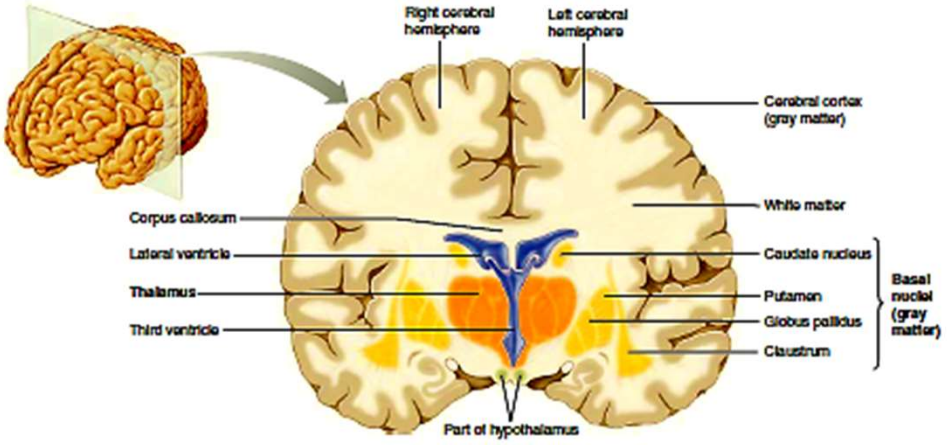
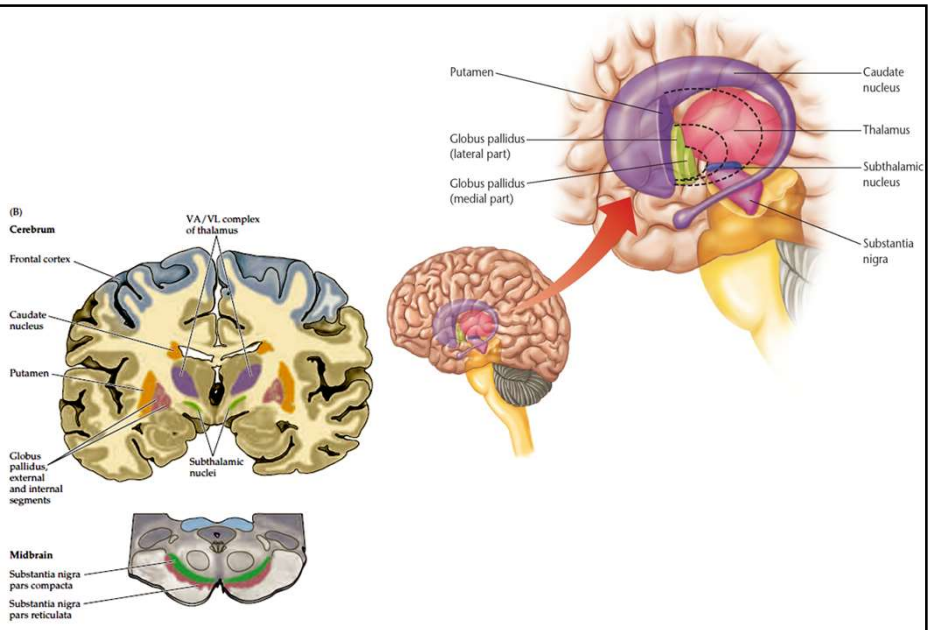
Basal Ganglia

Anatomical considerations



144



145

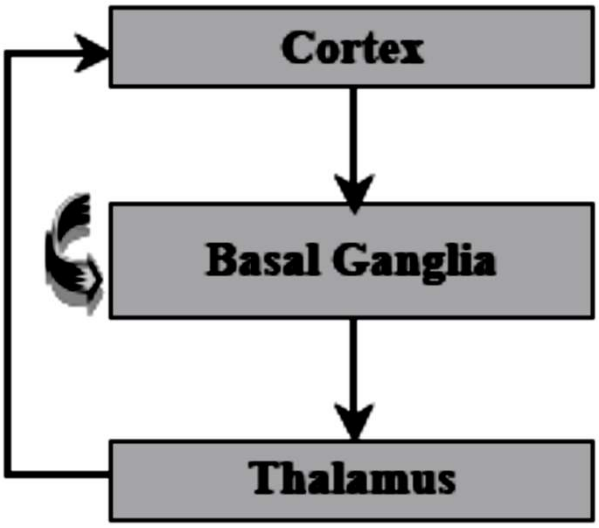
Connection of the Basal Ganglia

A-Cortical connections

B-Interconnections of the basal ganglia

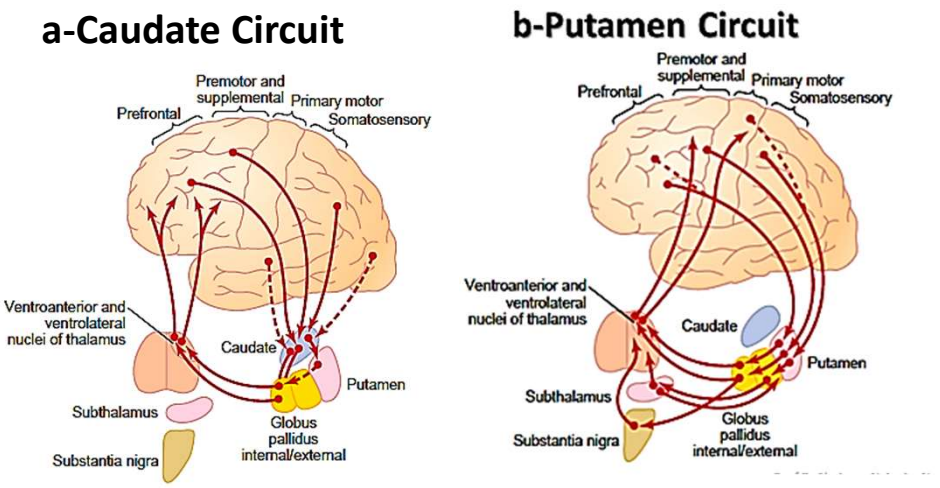
C-Brain stem Connections

146

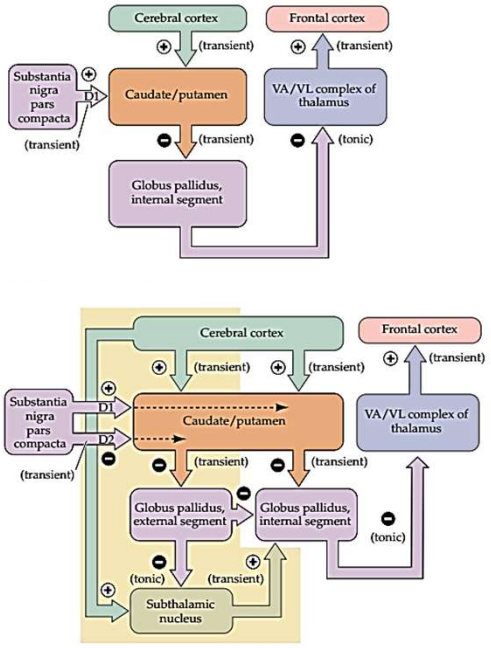


147

Cortical connections

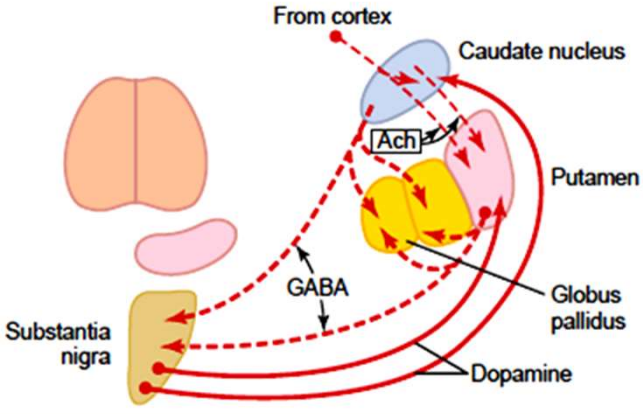


148



149

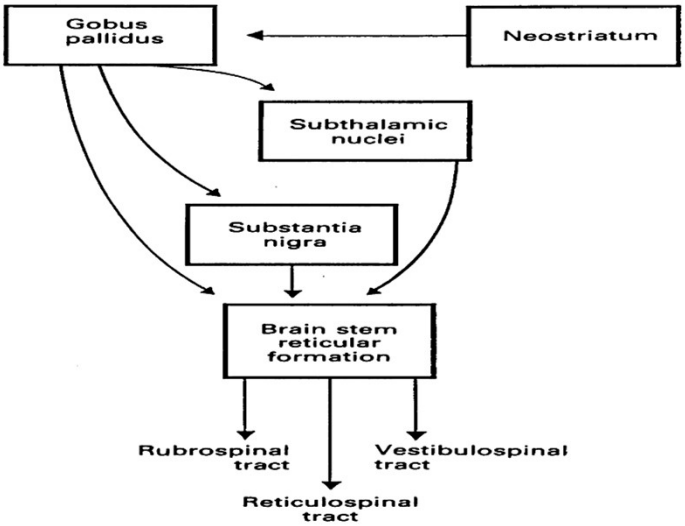
Interconnections of the basal ganglia



1- C .Cortex→ Glutamate → Neostriatum
2- Neostriatum → GABA → Substantia nigra
3- Substantia nigra → dopamine → Neostriatum

150

Brain stem connections



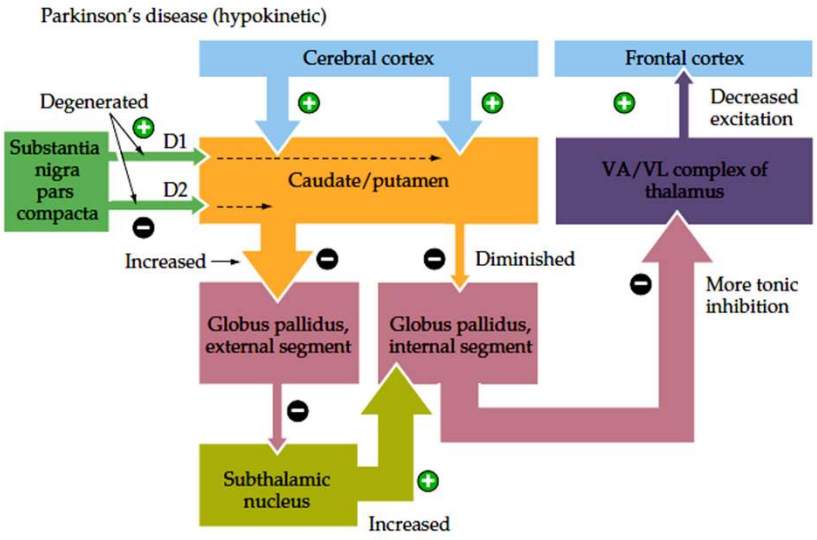
151

Functions of the Basal Ganglia

(A) Control of the muscle tone

(B) Control of voluntary movements

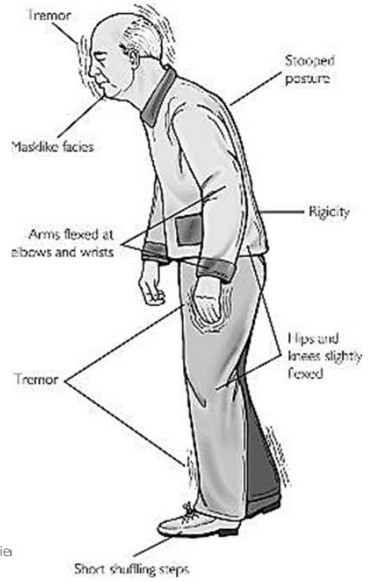
152



153

Characters of Parkinson's disease

- Rigidity



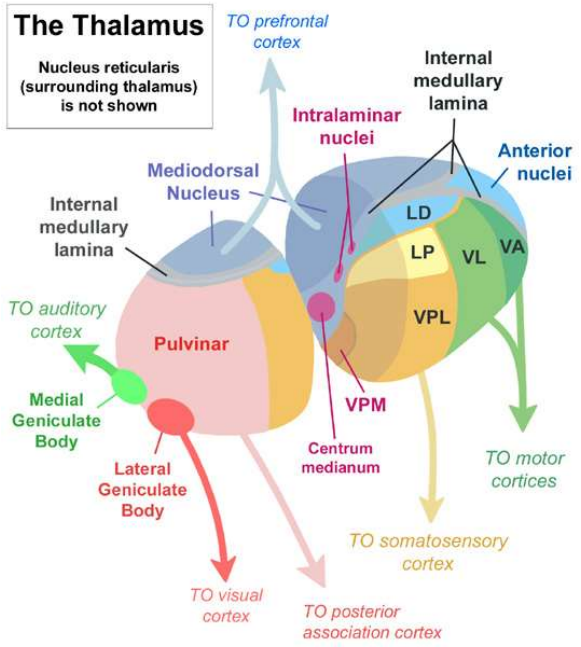
154

Motor CAUSES OF TREMORS

1. Static tremor: This occurs in Parkinsonism. It is present during rest and is associated with hypertonia.
2. Kinetic tremor: This occurs in neocerebellar disease. It is especially present at the end of movements and is associated with hypotonia.

	SENSORY ATAXIA	MOTOR ATAXIA
Most common cause	Tabes dorsalis	Neocerebellar disease
Gait	high steppage (stamping gait)	Staggering (drunken gait)
Romberg's sign	Positive	Negative
Effect of vision	Corrected by vision	Not affected by vision
Deep sensations	impaired or lost	Normal
Tremors	Absent	Kinetic tremors present
Nystagmus	Absent	Present
Speech	Normal	Scanning or staccato

155

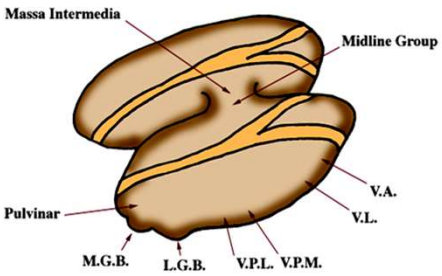


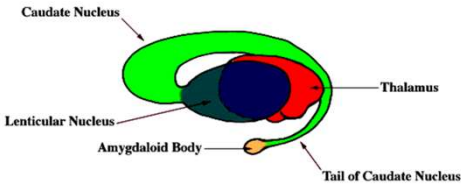
156

Thalamic Nuclei

1-Non-specific thalamic nuclei (midline, intralaminar)

2-Specific thalamic nuclei (Posteroventral, MGB, LGB, anterior thalamic nuclei)





157

Functions of the thalamus

1- It is a relay station for all types of sensation

2- The thalamus also relays signals, concerned with motor function, from the basal ganglia and cerebellum.

3- It relays autonomic signals and those related to emotional reactions.

4- It also has a role in coding, storing and recalling memories.

5- Impulses from non-specific thalamic nuclei are concerned with excitability of the cortex.

158

The Thalamic Syndrome

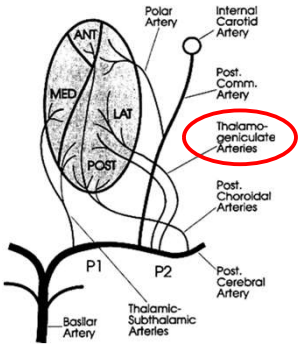
The Patient presents with:

1- Loss all sensation on the opposite side of the body.

2- Ataxia.

3- 2ry hyperalgesia.

4- Emotional disturbance.

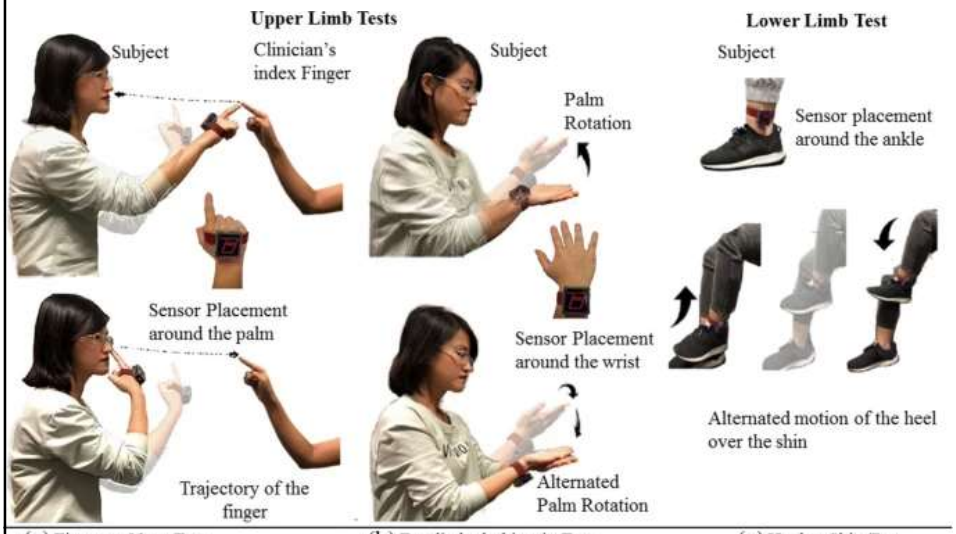


159

ATAXIA

• This means incoordination of voluntary movements without paralysis, and it is either sensory or motor (sometimes mixed in the thalamic syndrome).

160



161

Hypothalamus

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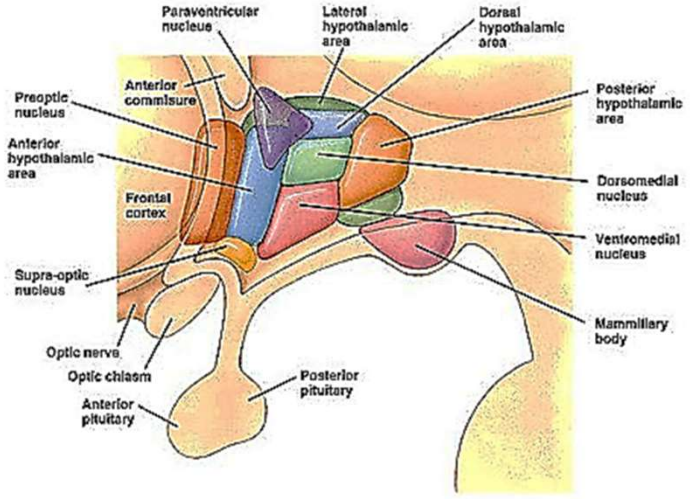
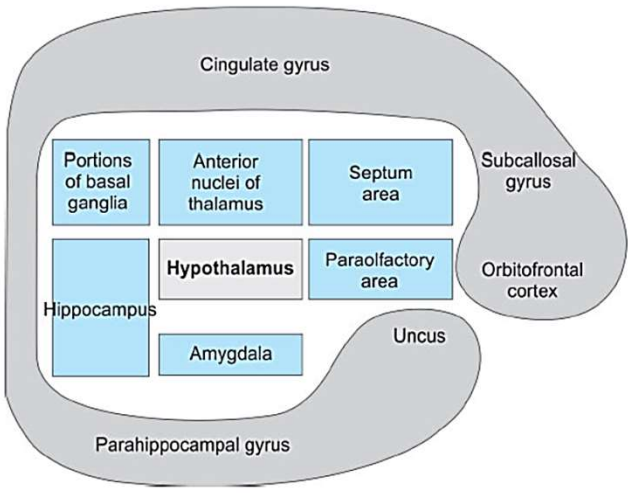
► Nuclei of the Hypothalamus



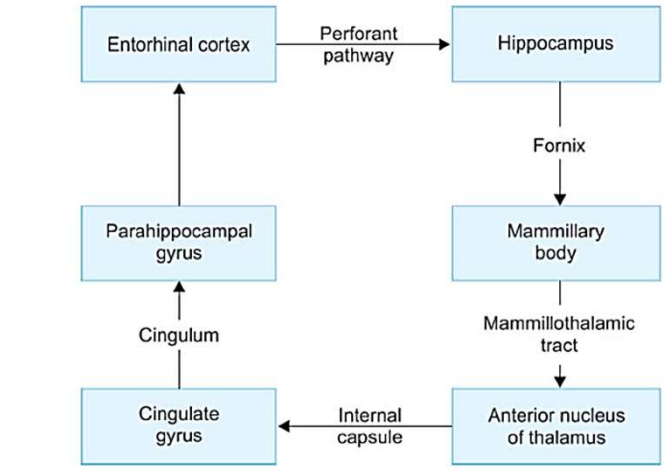
163

The Limbic System



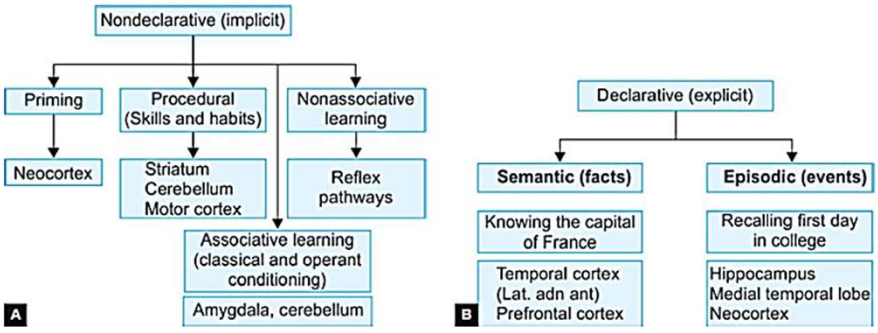
The limbic system, showing the central position of the hypothalamus.

164



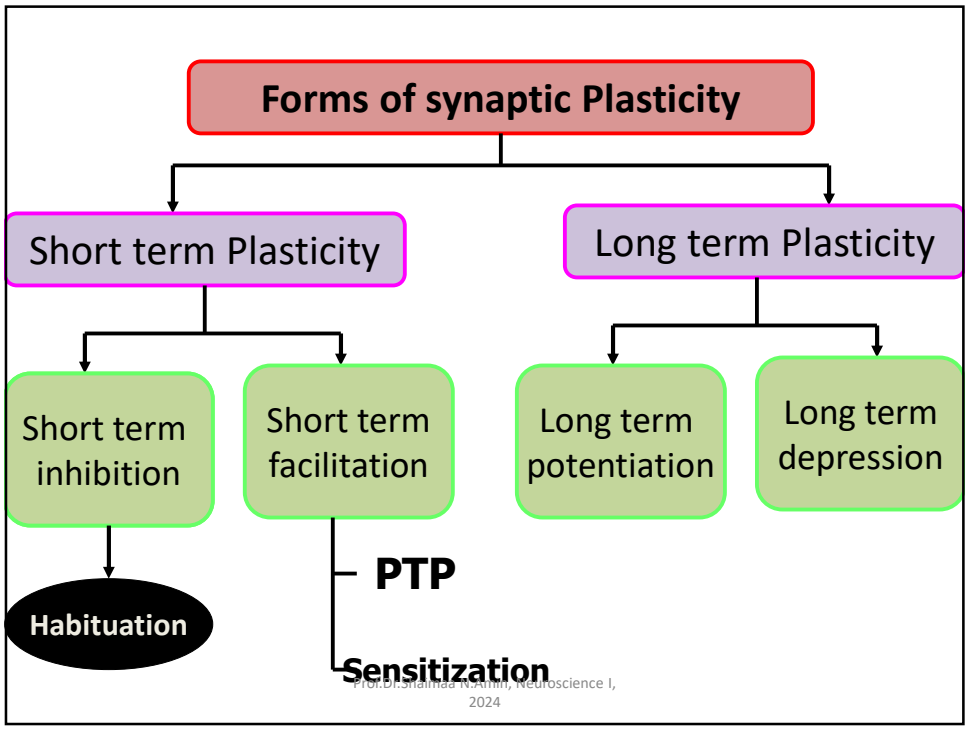
Component of papez circuit.

165



Parts of brain areas involved in processing and formation of implicit and explicit memory

166


Forms of synaptic Plasticity
Short term Plasticity
Long term Plasticity
Short term inhibition
Short term facilitation
Long term potentiation
Long term depression
Habituation
PTP
Sensitization
Prof.Dr.Shaimaa N.Amin, Neuroscience I, 2024

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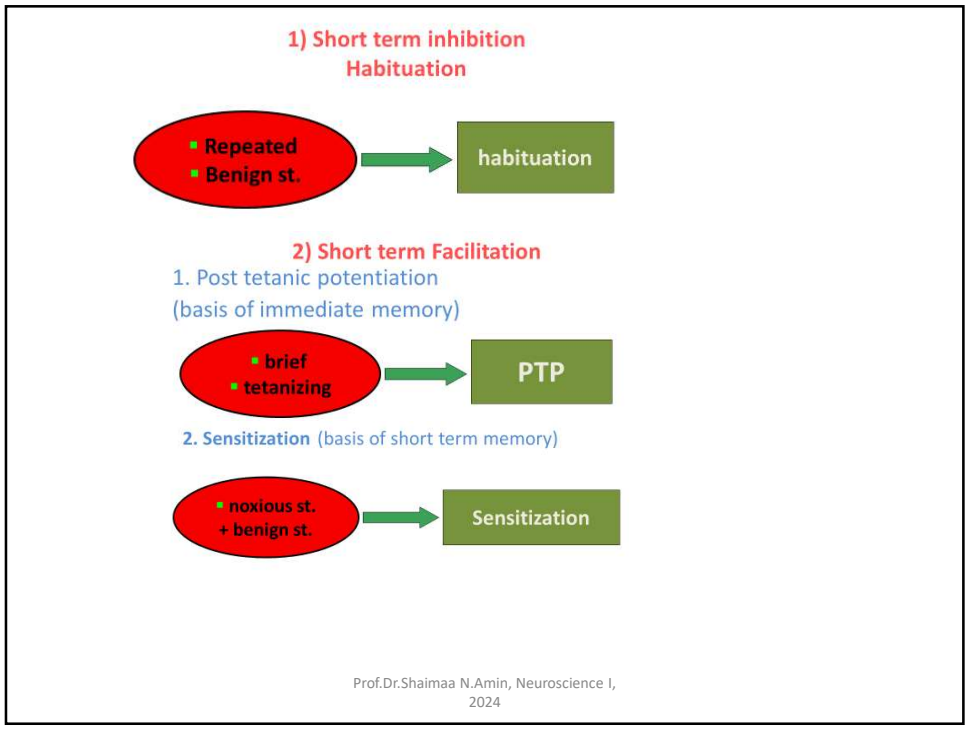
1) Short term inhibition
Habituation
Repeated
Benign st.
habituation
2) Short term Facilitation
1. Post tetanic potentiation
(basis of immediate memory)
brief
tetanizing
PTP
2. Sensitization (basis of short term memory)
noxious st.
+ benign st.
Sensitization
Prof.Dr.Shaimaa N.Amin, Neuroscience I, 2024

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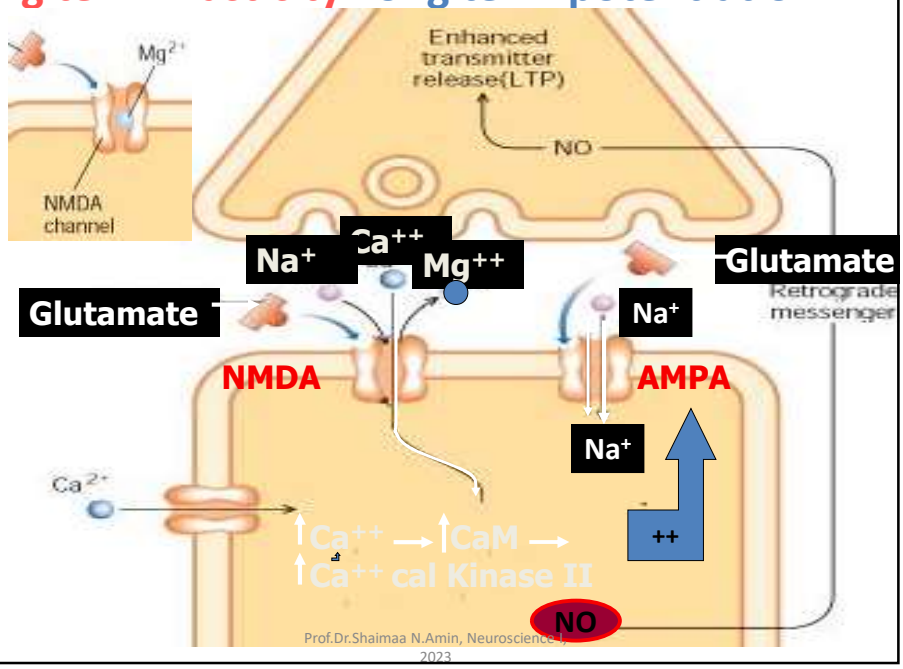
Long term Plasticity:Long term potentiation



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2. Long term Depression (LTD):

Presynaptic neuron stimulation ⟶ prolonged depression of postsynaptic neuron.

Ionic basis:

Occurs if presynaptic neuron stimulation ⟶ depolarization of postsynaptic neuron less than 20mv (threshold for NMDA receptors to open).

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Type of S. plasticity	Stimuli	Ionic basis
Habituation	▪ Repeated ▪ Benign	**Inactivation of presy. Ca^{++} ch.**
PTP (immediate m.)	▪ Brief ▪ tetanizing	**Presy. Ca^{++} accumulation**
Sensitization (STM)	▪ Noxious + benign	**Presynaptic facilitation**
LTP	▪ as PTP (brief & repeated)	**Activation of NMDA**
LTD	Slower st. of presyn. n.	**Closure of NMDA**

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Electrical activity of the Brain & Arousal response and sleep

- **Two types of brain potentials can be recorded:**

I- Evoked potentials : The electrical events that occur in the cortex after stimulation of a sense organ can be monitored with a recording electrode.

Types of Evoked potentials :

1- Somatosensory stimuli → SSEP

2- Auditory stimuli → AEP

3- Visual stimuli → VEP

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2- Spontaneous potential(Electroencephalogram)

the recording of the variations in brain potential.

The EEG can be recorded with scalp electrodes through the unopened skull or with electrodes on or in the brain. The term electrocorticogram is used for the recording obtained with electrodes on the pial surface of the cortex.

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(II) The Electro-encephalogram (EEG)

-The EEG is the record of the spontaneous electric activity of the brain.

-It can be recorded by applying electrodes on the scalp of patient.

-The electric activity appears on a multi-channel recorder as waves of variable intensity (0-200 mV) and frequency (1-50 Hz).

Conditions Required :

1- Recording of EEG should be done in a calm room at a comfortable temperature

2- The subject should be in complete physical and mental rest.

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Alpha

-8-13 Hz & 50 µv
-Regular & rhythmic
-Adult
-Parito-occipital region.
-Physical and mental rest
-Awake but with eyes closed

Beta

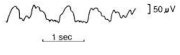
-18-30Hz & 20 µv
-Irregular & non-rhythmic
-Adult
-Frontal region
-Intense activation of the CNS i.e. during thinking and tension

Theta

4-7 Hz & 100 µv
Regular and large
Parietal & Temporal
In Children
& in adult during light
Sleep and may be in adults during emotional disappointment

Delta

-1-3 Hz & 100 µv
-Regular& high voltage
-Infants & in adults during deep sleep
coma, anasthtesia



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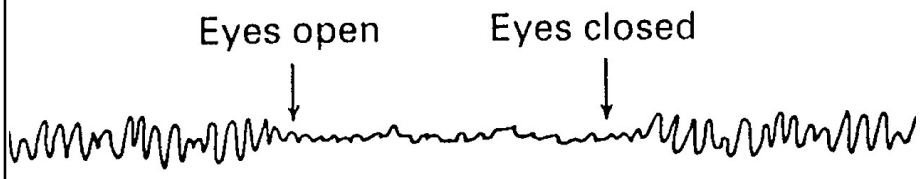
Desynchronization:alpha block : Alert or Arousal response :



While the eyes are closed, synchronous discharge of many cerebral neurons produces alpha waves, but when the eyes are opened, faster low voltage irregular beta waves are recorded. This is known as "alpha block". It has also been called "de-synchronization" because it represents breaking up of the synchronized neural activity necessary to produce regular waves.

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Clinical Uses of the EEG

1-Localizing brain tumors

2. Diagnosis of epilepsy

3.Diagnosis of sleep disorders.

4.Confirmation of brain death (flat EEG).

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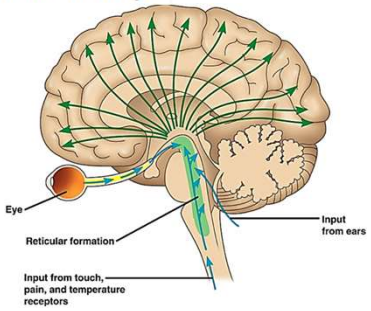
Wakefulness And Sleep Cycle

sleep and **wakefulness**

= state-dependent behavior

Reflected by changes in cortical electrical activity:
EEG changes

What are the factors that increase or decrease RAS activity?



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In mammals there are two sleep states:

- REM: rapid eye movement;
- NREM (non-REM)

Defined by:
- EEG
+ EOG, electroculography,
+ EMG, electromyography
(= polysomnography)

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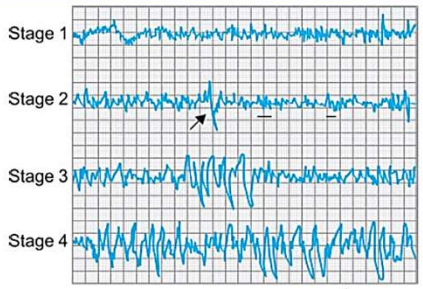
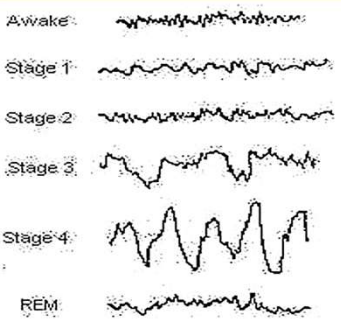
- REM: rapid eye movement;
- NREM (non-REM)

Recent stage	Old stage	% of total sleep duration	Features
N1	Stage 1	2–5%	Low amplitude, high frequency EEG activity. Alpha wave <50%
N2	Stage 2	45–55%	Sleep spindles (alpha-like 10–14/sec, 50 µv amplitude), K complexes
N3	Stage 3	10–15%	Low frequency, increased amplitude
	Stage 4		Maximum slowing (least frequency), large waves (rhythmic slow waves, synchronized)



EEG in NREM sleep. K-complex has been shown by an arrow.



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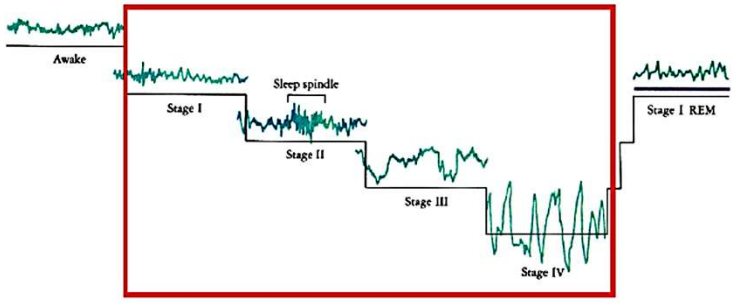
- REM: rapid eye movement;
- NREM (non-REM)



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Mechanisms of sleep

(1) Passive mechanism i.e. as a result of its *fatigue* (after a period of wakefulness) or by *decreasing its activity* through elimination of its exciting stimuli e.g. the visual, auditory, painful and other stimuli.

(2) Active mechanism : This is ***more accepted*** as a mechanism of sleep.

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One theory regarding the basis for transitions from sleep to wakefulness involves alternating reciprocal activity of different groups of RAS neurons.

In this model, wakefulness and REM sleep are at opposite extremes. When the activity of norepinephrine- and serotonin-containing neurons is dominant, there is a reduced level of activity in acetylcholine-containing neurons in the pontine reticular formation.

This pattern of activity contributes to the appearance of the awake state. The reverse of this pattern leads to REM sleep.

When there is a more even balance in the activity of the aminergic and cholinergic neurons, NREM sleep occurs. In addition, an increased release of GABA and reduced release of histamine increase the likelihood of NREM sleep

The orexin released from hypothalamic neurons may regulate the changes in activity in these brainstem neurons.

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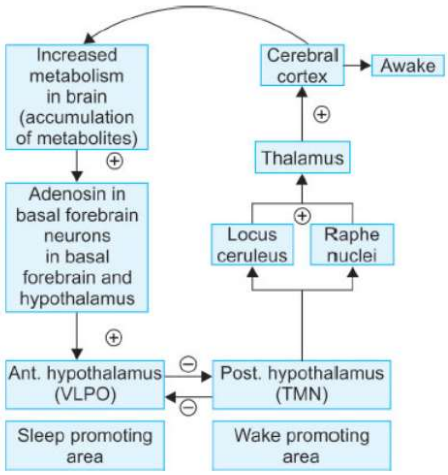

Fig. 14.3: Metabolite homeostatic theory of sleep. (VLPO: Ventrolateral preoptic nucleus; TMN: Tuberomammillary nucleus).

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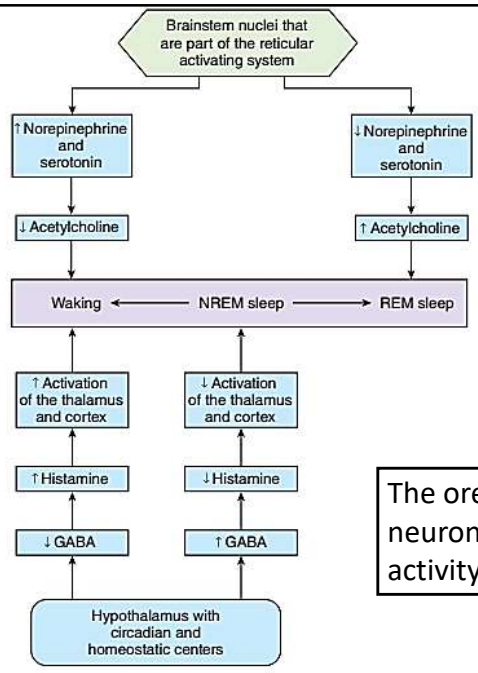

The orexin released from hypothalamic neurons may regulate the changes in activity in these brainstem neurons.

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Sleep disorders

1- Insomnia:

It is insufficient sleep that occurs in adults due to :

1- Psychological factors e.g. anxiety

2- Intake of analeptics e.g. coffee.

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2- Somnambulism:

"*sleep walking*"

-More common in male children.

-The person walks with eyes opened, and avoid obstacle and when awakened can not remember what he did.

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3- Narcolepsy:

Irresistible sleep during daytime activities which starts with sudden onset of REM sleep.

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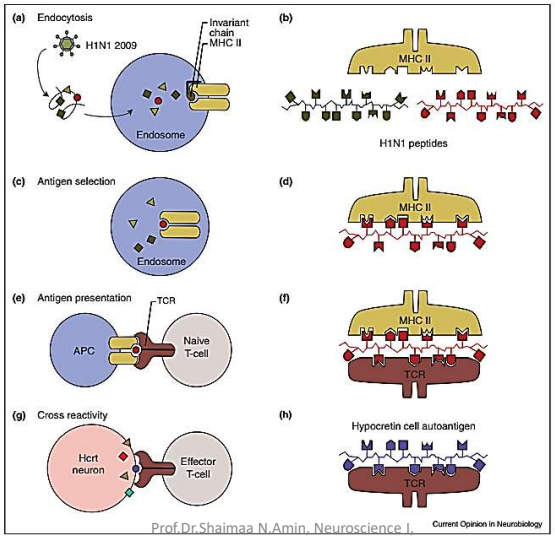
Autoimmune Basis of Narcolepsy



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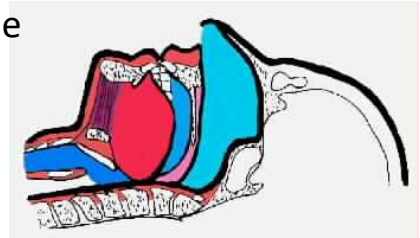
4- Sleep apnea:

-Caused by obstruction of the airways during sleep.

-Effort to overcome the obstruction awakens the person from sleep.



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5- REM behavior disorder:

- Hypotonia fails to occur.
- The patients with this condition act out their dreams, they even jump out of bed to do battle with imagined aggressors.

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3-Neuroscience, Purves et al. 6th edition
4-Review of Physiology, Soumen Manna, 5th edition

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