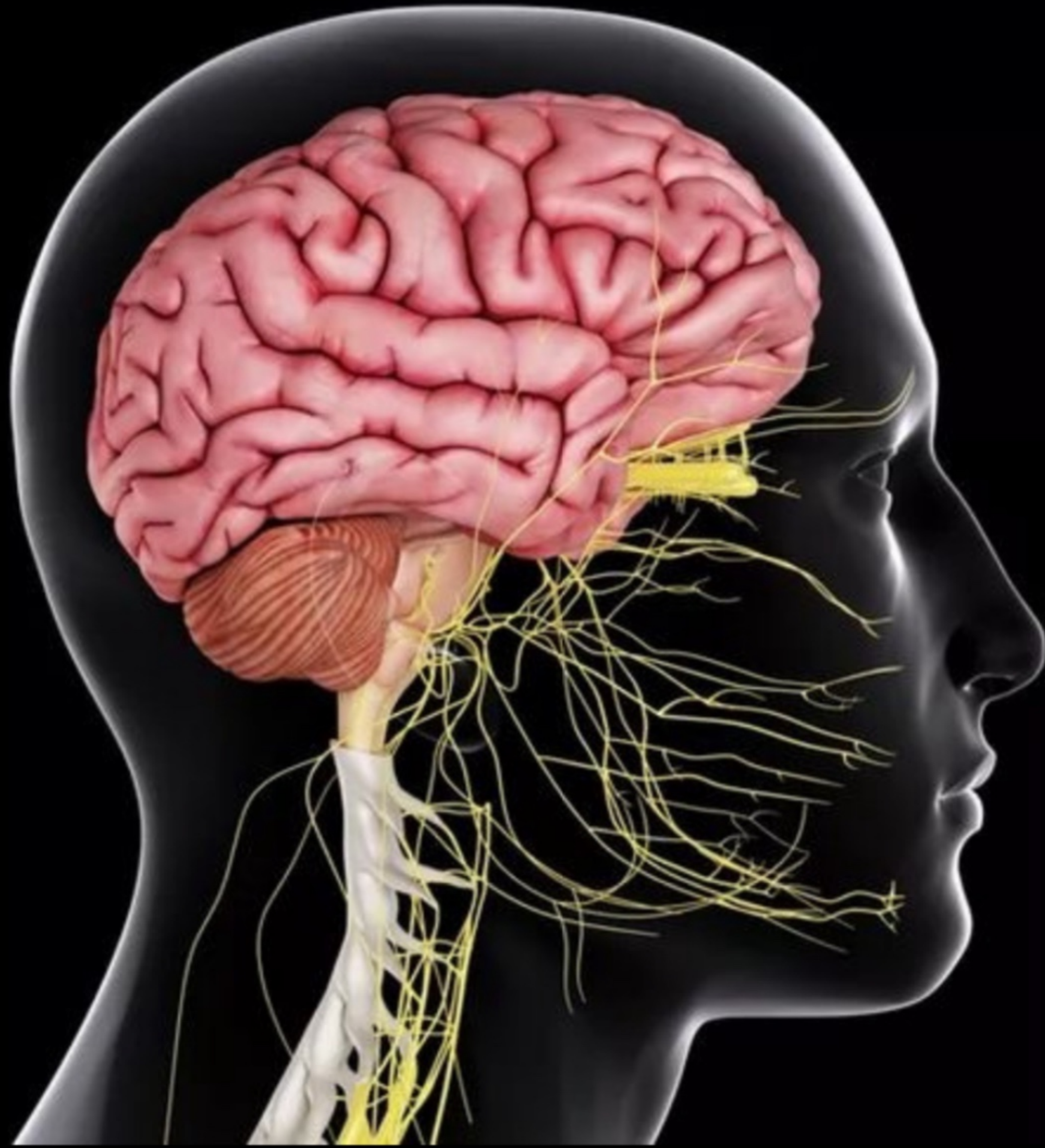




CENTRAL NERVOUS SYSTEM



SUBJECT : RF + UMN L / LMNL

LEC NO. : 6

DONE BY : Nehayer

THE RETICULAR FORMATION

This is a network of neurons located in the brain stem, extending upwards to the diencephalon (thalamus, hypothalamus, and subthalamus) and downwards to the upper part of the spinal cord.

Many nuclei and centers are present within its meshes (e.g., the respiratory and cardiac centers, the substantia nigra, and the red, vestibular, and raphe nuclei). It is divided into sensory and motor parts.

(A) THE SENSORY PART OF THE RETICULAR FORMATION

This consists of small neurons with multiple interconnections (which allows for convergence, divergence, and after discharge). It receives a rich sensory input (afferent fibers) from (1) All ascending lemnisci, (2) The visual, auditory, and olfactory nervous pathways, (3) The basal ganglia, (4) The cerebellum, (5) The cerebral cortex (Via corticofugal fibers) (6) The hypothalamus (7) The vestibular apparatus.

(B) THE MOTOR PART OF THE RETICULAR FORMATION

This consists of large neurons that receive signals from the sensory part, and their axons constitute the output (efferent) fibers from the reticular formation. It contains facilitatory and inhibitory parts:

(1) Facilitatory (excitatory) reticular formation: (last text)

This is located mainly in the pons. It has an inherent activity, and the axons of its neurons divide into 2 branches:

(a) An ascending branch that excites the cerebral cortex is called the Ascending Reticular Activating System or ARAS.

(b) A descending branch (= Ventral reticulospinal tract) exerts a facilitatory effect on the spinal gamma motor neurons.

(2) Inhibitory reticular formation: This is located mainly in the medulla oblongata. It has no inherent activity, and it descends as the lateral reticulospinal tract, which inhibits the spinal gamma motor neurons.

see p
next page

FUNCTIONS OF THE RETICULAR FORMATION

- (1) Control the level of consciousness through the ascending reticular activating system.
- (2) Regulation of the stretch reflex and muscle tone through the reticulospinal tracts.
- (3) Pain inhibition by the raphe Magnus nucleus.
- (4) Control of sleep by 2 specific centers in its meshes (see below)
- (5) Control visceral functions (e.g., cardiac activity) by controlling the spinal lateral horn cells.

ASCENDING RETICULAR ACTIVATING SYSTEM ARAS or RAS

This multineuronal polysynaptic system of nerve fibers originates at the facilitatory reticular formation. Its fibers extend upwards, then some project directly to the cerebral cortex, while the majority relay first at the nonspecific thalamic nuclei, from which other fibers arise and project diffusely to almost all parts of the cerebral cortex. The latter pathway is called the reticulo-thalamo-cortical pathway.

FUNCTIONS OF THE ARAS

The ARAS controls the electric activity of the cerebral cortex and is concerned with consciousness and production of the alert response, so reduction of its activity leads to sleep.

FACTORS THAT AFFECT THE ACTIVITY OF THE ARAS

(A) Factors that increase the ARAS activity

- (1) Sensory signals (especially pain).
- (2) Signals from the cerebral cortex (via the corticofugal fibers) that increase alertness and resist the desire to sleep (e.g., during emotions and voluntary movements).
- (3) Certain drugs are called analeptic, e.g., catecholamines, amphetamine, and caffeine.

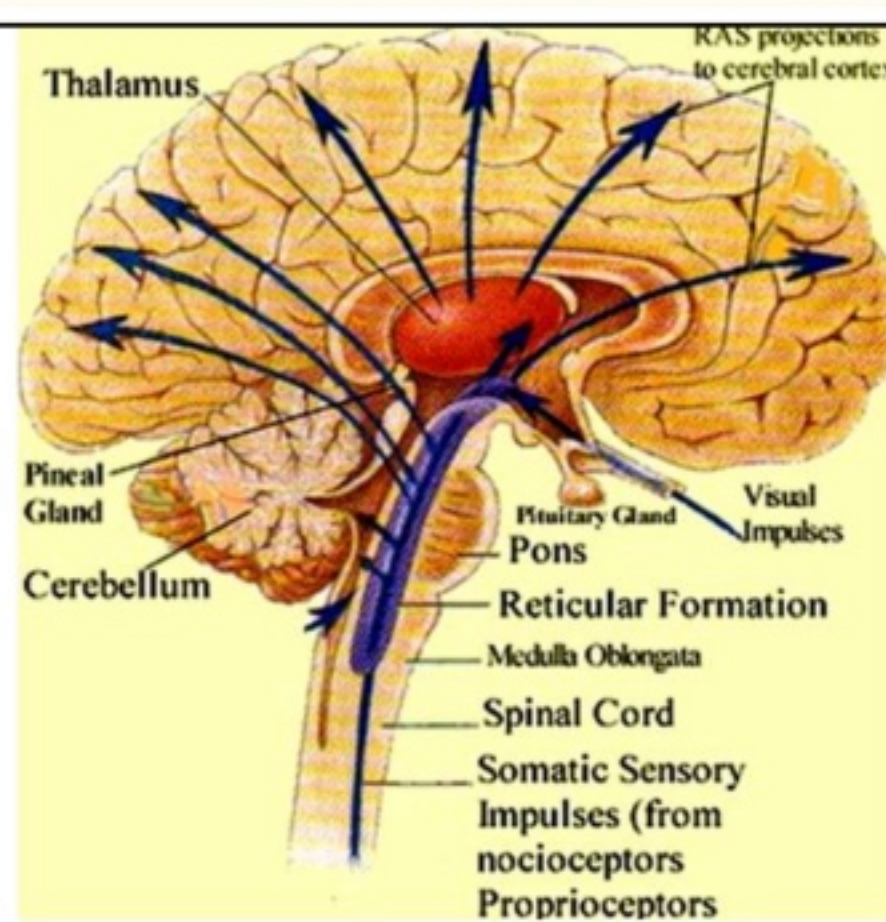
(B) Factors that decrease the ARAS activity

- (1) Reduction of signals from the sensory pathways or the cerebral cortex.
- (2) Stimulation of the sleep centres.
- (3) Extensive damage of the ARAS (e.g., by tumors). = coma
- (4) General anesthetic drugs: These drugs lead to unconsciousness by depressing the ARAS activity (inhibiting the synaptic transmission between its neurons).

check
pic next
slide

The Brain Stem

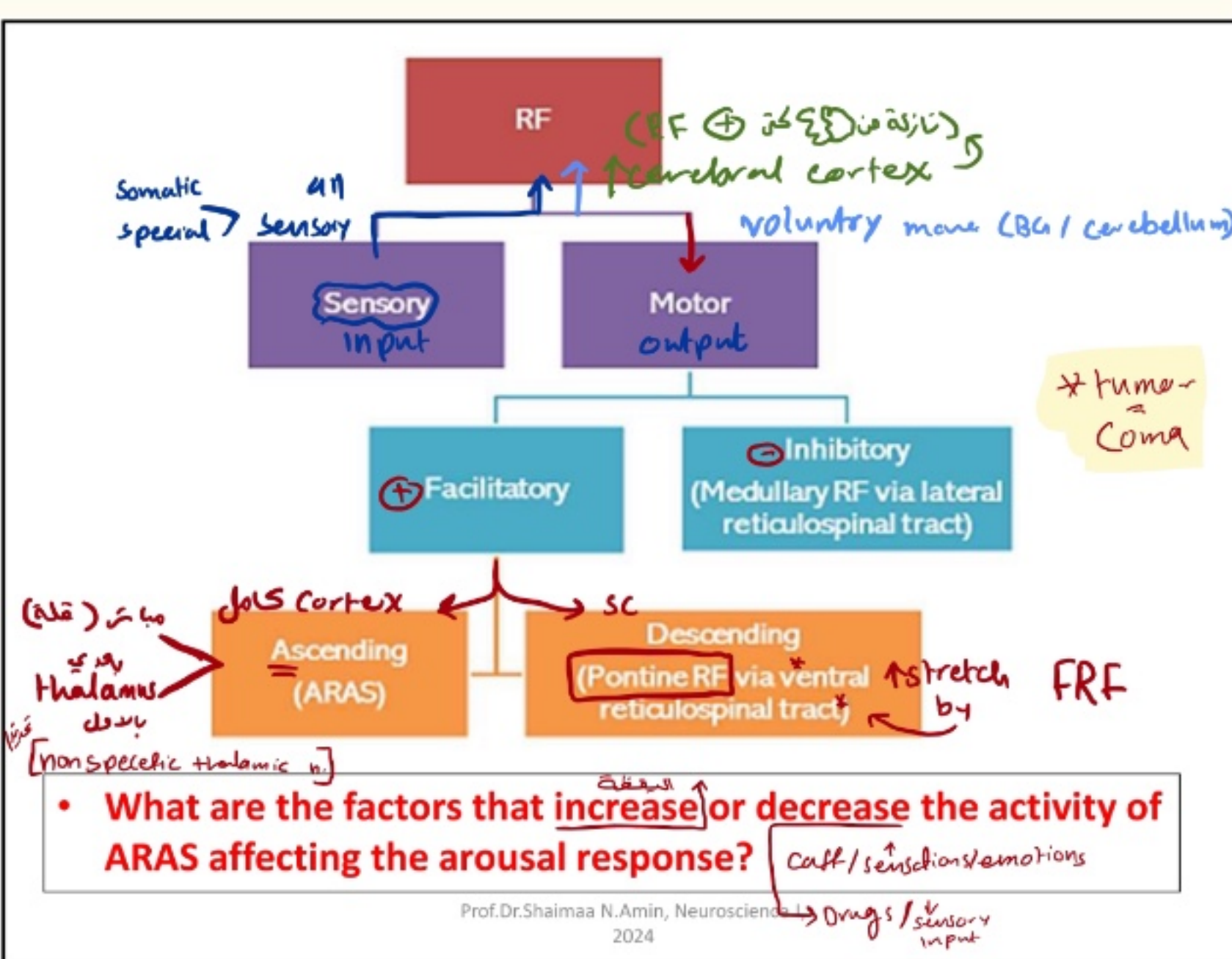
- The brain stem is a complex extension of the spinal cord, which performs sensory, motor and reflex functions.
- The reticular formation is a large structure occupying the core of the brain stem.



network of neurons in core of BS

CVS / on reflex / resp مراكز حسية

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

Call / sensations / stimulations / Drugs / sensory inputs

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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:

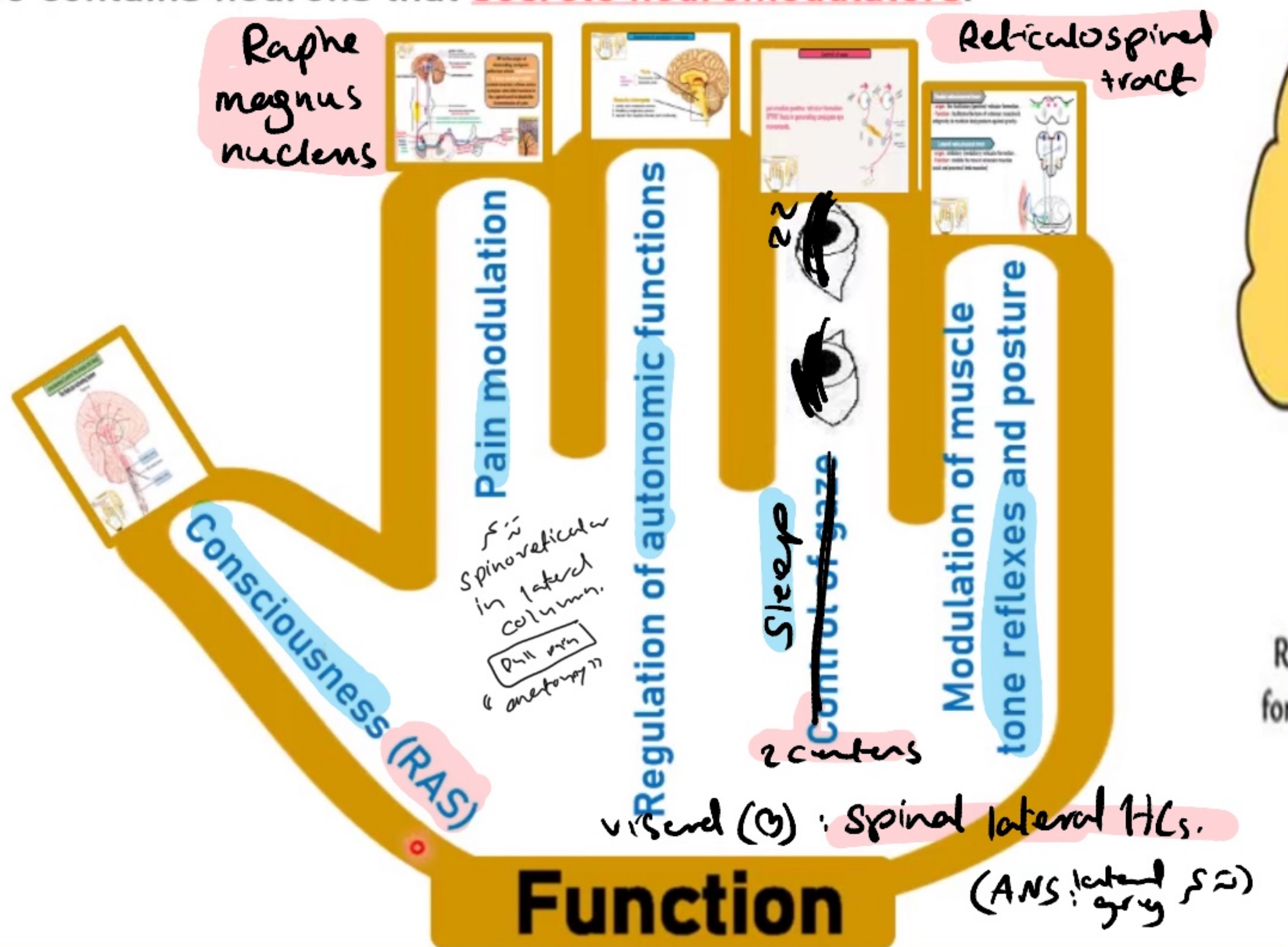
- Suppressor area of the motor cortex.
- Basal ganglia.
- Paleocerebellum.

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RETICULAR FORMATION

Definition :

- anatomic area in the brainstem made up of various neurons and fibers.
- It also contains neurons that secrete neuromodulators.



Function

+

Impulses from sensory pathways and motor cortex.

Sympathomimetic drugs.

Stress, Caffeine, Thyroid H.

-

Impulses from medullary and pontine sleep centers.

Anesthetic drugs

Injury of RAS in midbrain or thalamus

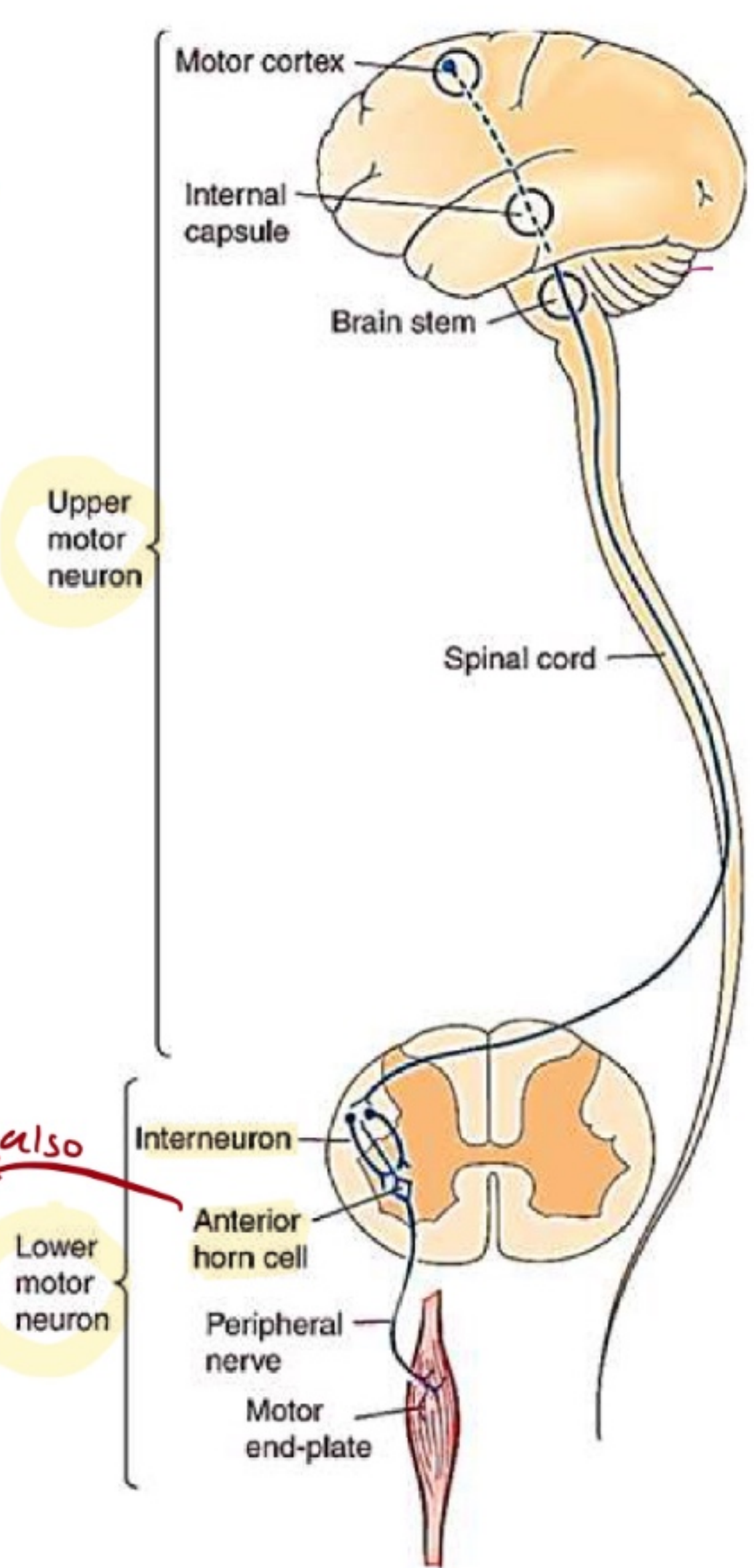
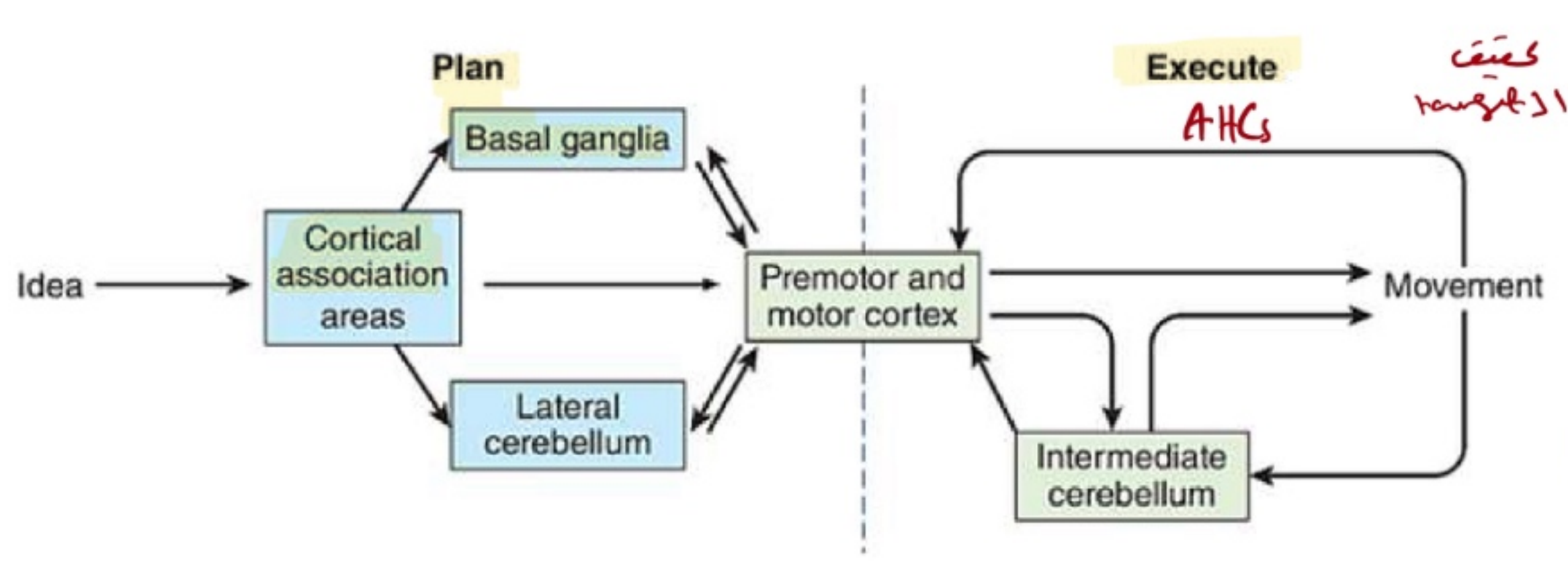
| | Gamma rigidity | Alpha rigidity |
|------------------------|--|-----------------------------|
| Cause | Increased gamma discharge | Increased alpha discharge |
| Muscles affected | Antigravity muscles | All muscles |
| Resistance to movement | Uni-directional | Bi-directional |
| Type of rigidity | 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 |

جود من استجابة القابلية

"revise previous lectures"

سج ناجي

UMNL vs LMNL/Spinal cord transection



remember anatomy

lower = nuclei

XII ⇒ LMNL

medial ↓ medullary syndrome

motor nuclei lesion LMN يعتبر lesion

UMNL
Cause:
 Cerebrovascular accidents (strokes) **due to**:
 1- hemorrhage or
 2- thrombosis
 in the posterior limb of the internal capsule. (مستطابق)
 • There is damage of both pyramidal and extrapyramidal fibres

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

2. Hypertonia
 The paralyzed muscles show increased tone of the **spastic type. (Clasp Knife Type).** (subton release) (extended stretch = inverse stretch)
 Hypertonia is due to ???

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).

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.**

UMNL
5. delayed Muscle-Wasting :
 This is because : paralyzed muscles are still innervated and can contract **reflexly.** (السيبي)
 "Both" atrophy ↑ Disuse لانها ما بتتحرك

LMNI
Cause: اي لته بآثر AHCs او axon طابع العضلات
Is due to :
 1- lesion of the lower motor neurons as in poliomyelitis. (الموتون)
 2- damage of motor nerves e.g. diabetes mellitus, and alcoholism

1. Paralysis:
 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.

2. Hypotonia or atonia:
 • The paralyzed muscles show decrease or loss of tone, referred to as flaccid paralysis.
 • Hypotonia is due to interruption of the stretch reflex. (absent)
 لانه ال reflex arc تآثر بآثر ال AHC

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?
 لانه حكيما قبل فكي من التوتر ما يلهو reflex بيدي ال جندراسيك وبعين هيك ان يمسو

4. Loss Of Superficial Reflexes
Seen in the affected segments only.
 ex. plantar reflex is **si** **si** ← متحركة خيس = reflex تآثر

LMNL
5. early Marked Muscle-Wasting) :
 The affected muscles show marked atrophy due to the inability of the muscles to contract neither **reflexly nor** voluntarily. (ما بتتحرك)
 (atrophy قلنا صحت كل ال muscle ما راسا) paralysis

cause

فروقات

check pa lec
 كه فذكو
 اكو فذكو
 ال سائنه
 و بالاسائوني

paralysis

tone

tendon Reflex

sup. Reflex

muscle waste

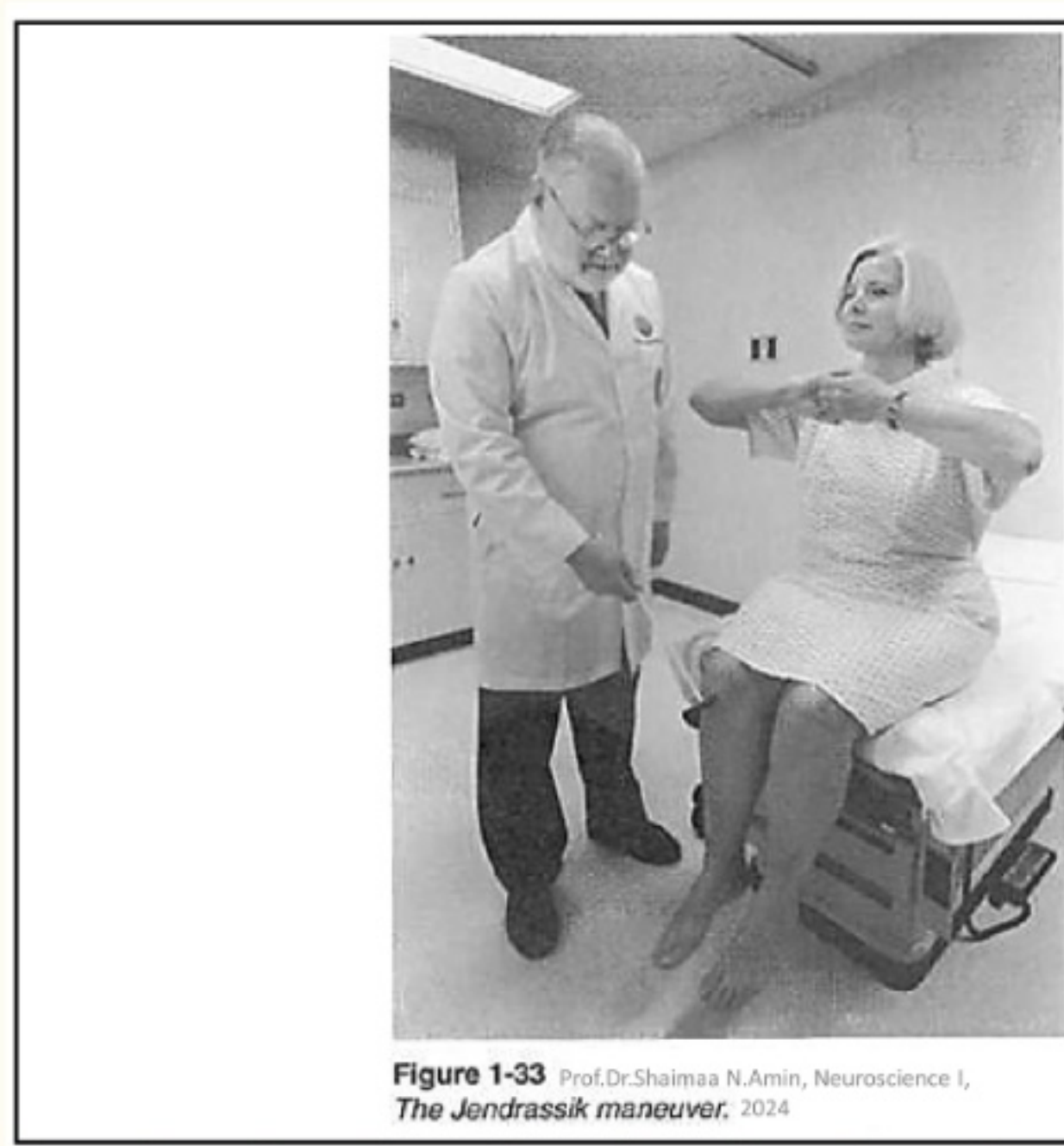
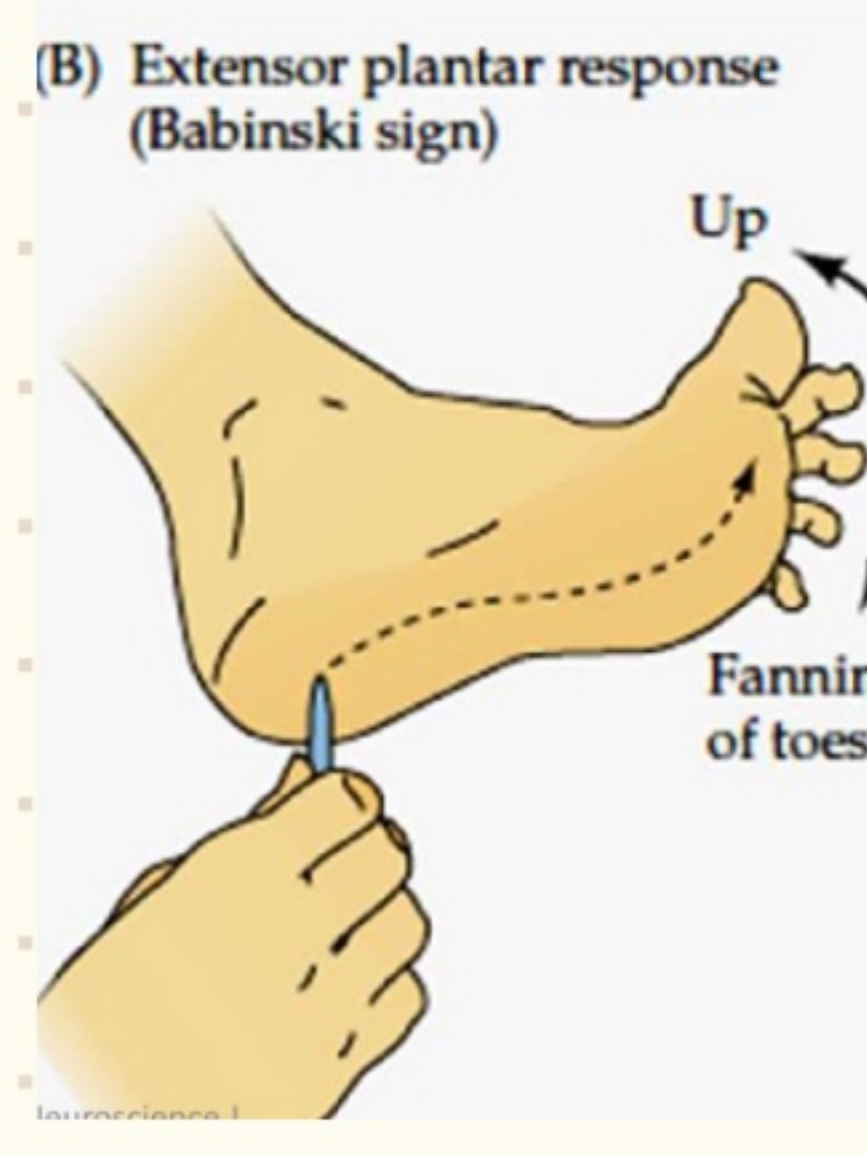


Figure 1-33 Prof. Dr. Shaimaa N. Amin, Neuroscience I, The Jendrassik maneuver, 2024



Prof. Dr. Shaimaa N. Amin, Neuroscience I, 2024



(B) Extensor plantar response (Babinski sign)

« والملقى الصعبكم بحلولة القلب »

UPPER and LOWER MOTOR NEURON LESIONS

UMNL results from damage of the cortical motor areas or anywhere along the course of their descending tracts, commonly in the internal capsule due to cerebral hemorrhage or thrombosis. On the other hand, a **LMNL** results from either damage of the spinal (or cranial) motor neurons by disease (commonly poliomyelitis) or injury of the motor nerves by trauma or disease (e.g. polyneuropathy).

DIFFERENCES BETWEEN UMNL and LMNL

Although both lesions result in paralysis of skeletal muscles, yet each has characteristic manifestations.

(1) **Extent of paralysis** (widespread in UMNL and localized in LMNL).

(2) **Site of paralysis:** This is always at the same side in LMNL, but it may be on either side in the case of UMNL e.g. a hemisection of the spinal cord at the cervical region leads to ipsilateral hemiplegia, while a lesion in the internal capsule leads to contralateral hemiplegia.

(3) **Recovery:** UMNL does not recover because the upper motor neurons cannot regenerate due to the absence of neurolemma. Conversely, LMNL can recover if it is due to injury of the motor nerves (because these nerves can regenerate due to the presence of neurolemma), but they cannot recover if the motor nerve cells themselves are damaged (e.g. in poliomyelitis).

(4) **Muscle tone:** In LMNL, there is hypotonia or atonia (i.e. muscle flaccidity) due to interruption of the efferent limb of the stretch reflex.

On the other hand, in UMNL there is hypertonia i.e. muscle spasticity mainly in the antigravity muscles. It is a type of gamma rigidity that shows the lengthening (clasp-knife) reaction. It occurs as a release phenomenon with reversed supraspinal balance on the gamma motor neurons from inhibition to excitation.

(5) **Tendon jerks:** These are lost in LMNL- and exaggerated in UMNL due to the same causes of hypertonia. Also, clonus often appears in UMNL and is the most diagnostic feature of this lesion.

(6) **Superficial reflexes:**

(a) The abdominal and cremasteric reflexes are lost in both lesions due to loss of pyramidal facilitation in UMNL and interruption of their efferent limbs in LMNL.

(b) The plantar reflex is lost in LMNL, but is modified in UMNLs and the response becomes dorsiflexion of the big toe and fanning of the other toes (probably due to interruption of the pyramidal and extrapyramidal fibers respectively). Such response is known as the positive Babinski's sign or the plantar extensor reflex. However, this sign may be present in some normal individuals (see below).

(7) **Muscle status:** Muscle wasting (atrophy) occurs rapidly and markedly in LMNL due to degeneration of the motor nerves that supply the muscles. On the other hand, the paralyzed muscles in UMNL are atrophied only after relatively long periods due to disuse atrophy).

wish you all the best ♡