① CNS.

① **SYNAPSE**: is the junction b/w ② neurons
- ≠ anatomic continuation
- only physiological continuation b/w ② neurons

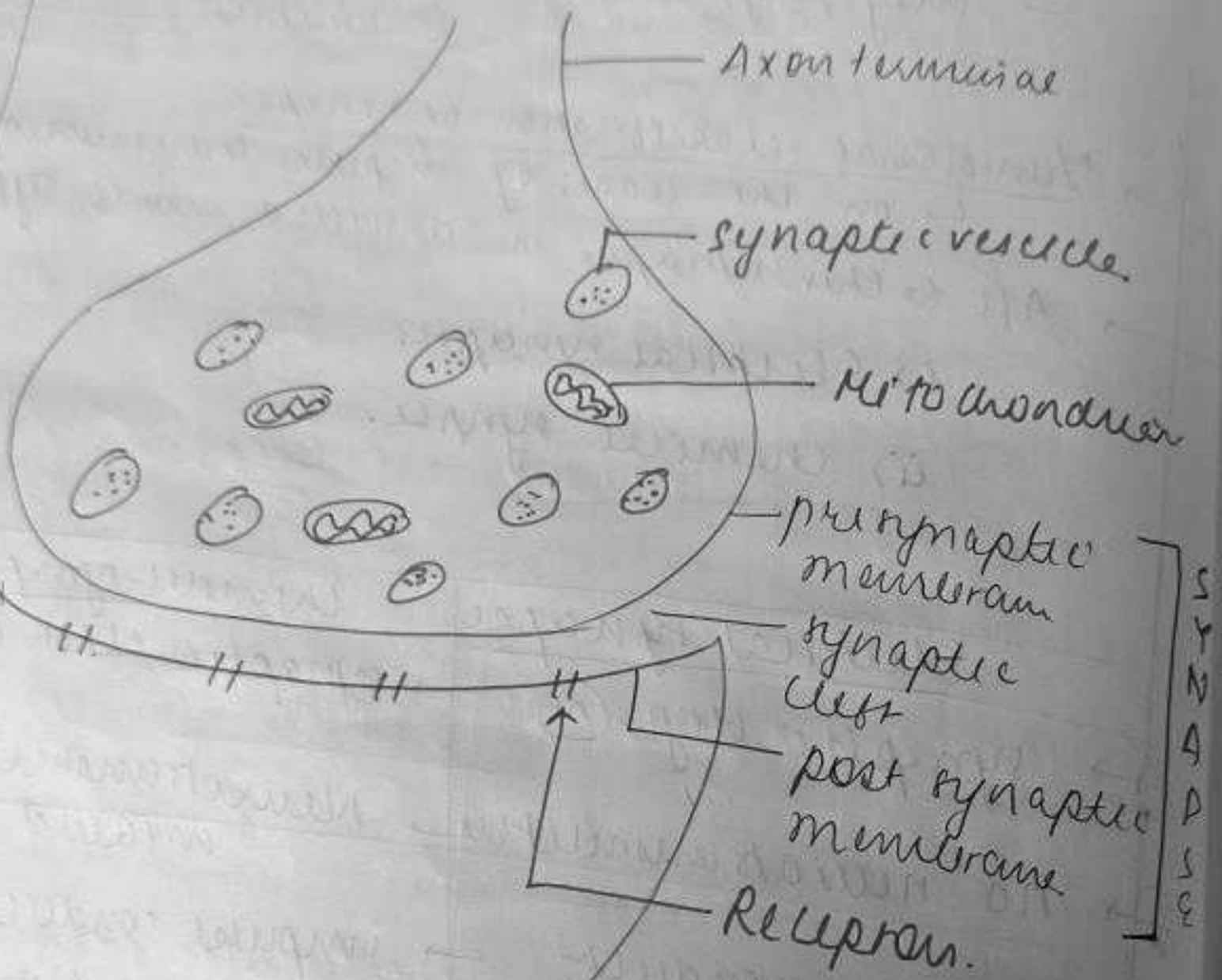
→ **Functional classification of synapse**:-
- on the basis of impulse transmission.

→ A/c to this synapse is divided into ② type:-

i) Electrical synapse. →
ii) Chemical synpse. →

Electrical synapse	Chemical synapse.
(i) → synaptic cleft (NIL)	→ synaptic cleft is present
(ii) → no neurotransmitters	→ Neurotransmitters involved.
(iii) → impulse conduction is faster	→ impulse conduction is slower.
(iv) → A.P. is transmitted as if it is in a single neuron.	→ AP is transmitted through neurotransmitter.
(v) → synaptic delay is less	→ synaptic delay is ↑.
(vi) → occurs in • cardiac m/s., • smooth m/s of • intestine, etc.	

Structure of chemical synapse :-



FUNCTIONS OF SYNAPSE:-

- Main function of synapse is to transmit impulses from 1 neuron → another
- some synapses ⊖ impulses
  impulses are ✗ transmitted to post synaptic neuron.

On the basis of function.

↓

SYNAPSES

EXCITATORY synapse	INHIBITORY synapse
↓	↓ ⊖
transmits the impulses (excitatory function)	transmission of impulses (inhibitory function)

→ Excitatory Neurotransmitters:-

(i) ACh.
(ii) Glutamate

→ Inhibitory neurotransmitters:-

(i) GABA
(ii) Glycine.
(iii) Serotonin
(iv) Dopamine.

→ Both excitatory & Inhibitory neurotransmitters:-

(i) Adrenaline
(ii) Noradrenaline.

→ EXCITATORY FUNCTION :-

Sequence of Event during Excitatory post synaptic Transmission / EPSP.

PRE-SYNAPTIC NEURON.

Arrival of A.P. in axon terminal
↓
opening of Ca^{++} channels in presynaptic membrane
↓
Influx of Ca^{++} from ECF into axon terminal
↓⊕
movement of synaptic vesicles towards presynaptic membrane
↓
fusion with presynaptic membrane & release (Ach) into synaptic cleft.
↓
Passage of Ach through synaptic cleft
↓
Ach. binds to receptors on post synaptic membrane
↓
POST-SYNAPTIC MEMBRANE.

Formation of Ach-receptor complex.
↓⊕
opening of (Na^+ channels) & influx of Na^+ from ECF.
↓
Development of EPSP
↓
opening of Na^+ channels in initial segment of axon
↓
Spread of A.P. through axon of post synaptic neuron.

INHIBITORY FUNCTION

sequence of events during post synaptic inhibition / IPSP:-

PRE SYNAPTIC MEMBRANE

Arrival of A.P. in axon terminal

↓

opening of Ca^{++} channels in pre synaptic membrane.

↓

influx of Ca^{++} from ECF → pre synaptic neuron.

↓ ⊕

movement of synaptic vesicles towards presynaptic membrane

↓

synaptic vesicles fuses with pre-synaptic membrane & release the neurotransmitter GABA.

↓

passage of GABA through synaptic cleft

↓

GABA binds with receptors on post synaptic membrane.

↓

formation of GABA receptor complex.

↓ ⊕

POST SYNAPTIC MEMBRANE

opening of K^+ channels

↓

efflux of K^+ into ECF

opening of Cl^- channels

↓

influx of Cl^- into post synaptic membrane

↓

↑ negativity inside post synaptic neuron.

↓

Hyperpolarisation - IPSP

↓ ⊖

synaptic transmission

PROPERTIES OF SYNAPSE:-

(i) One way conduction - Bell-Magendie law:-
A/c to this law,
impulses are transmitted only in
(a) direction i.e.
from presynaptic neuron
↓
post synaptic neuron

(ii) Synaptic delay:- 0.3-0.5 msec
└ Of the causes for reaction time of reflex activity.
└ Short delay that occurs during the transmission of impulses through synapses.
— It is due to the time taken for:-
(a) Release of neurotransmitters.
(b) Passage of neurotransmitter through synaptic cleft & binding to receptors
(c) Action of neurotransmitter
↓
open ion channels on post synaptic membrane.

(iii) Fatigue:-
└ During continuous muscular activity, ~~for~~ synapses becomes fatigue along with ~~but~~ more area of frontal lobe of cerebral cortex.
→ Fatigue of synapse occurs due to depletion of neurotransmitter. (Ach)

REFLEX ACTIVITY

→ Defin^n:- response to peripheral nerve stimulation which occurs without our conciousness

→ It is a type of protective mechanism & protects the body from unfavourable damages.

→ Reflex Arc:-
└ anatomical nervous pathway for reflex action

— A simple reflex arc consists of 5 components

① Receptor - End organ - receives stimulus

② Afferent neuron (or sensory) : transmits impulses from R → C

③ Centre - S.C / brain → receives impulses via afferent neuron & generates appropriate motor impulses

④ Efferent neuron (or Motor) - transmits impulse from C → E

⑤ Effector - Ex - m/s - activity occurs in response to impulses.

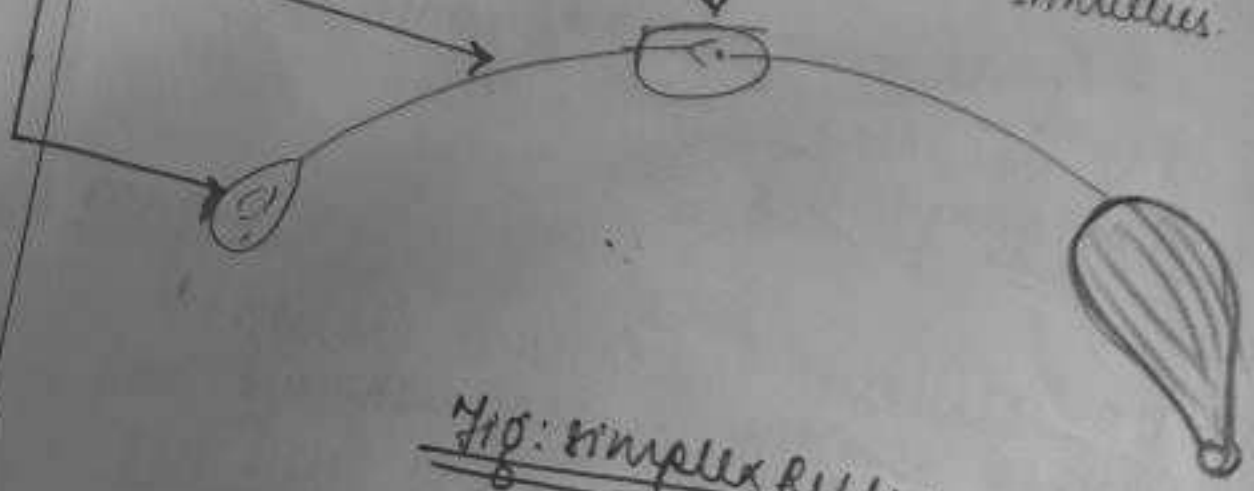


Fig: Simple Reflex Arc

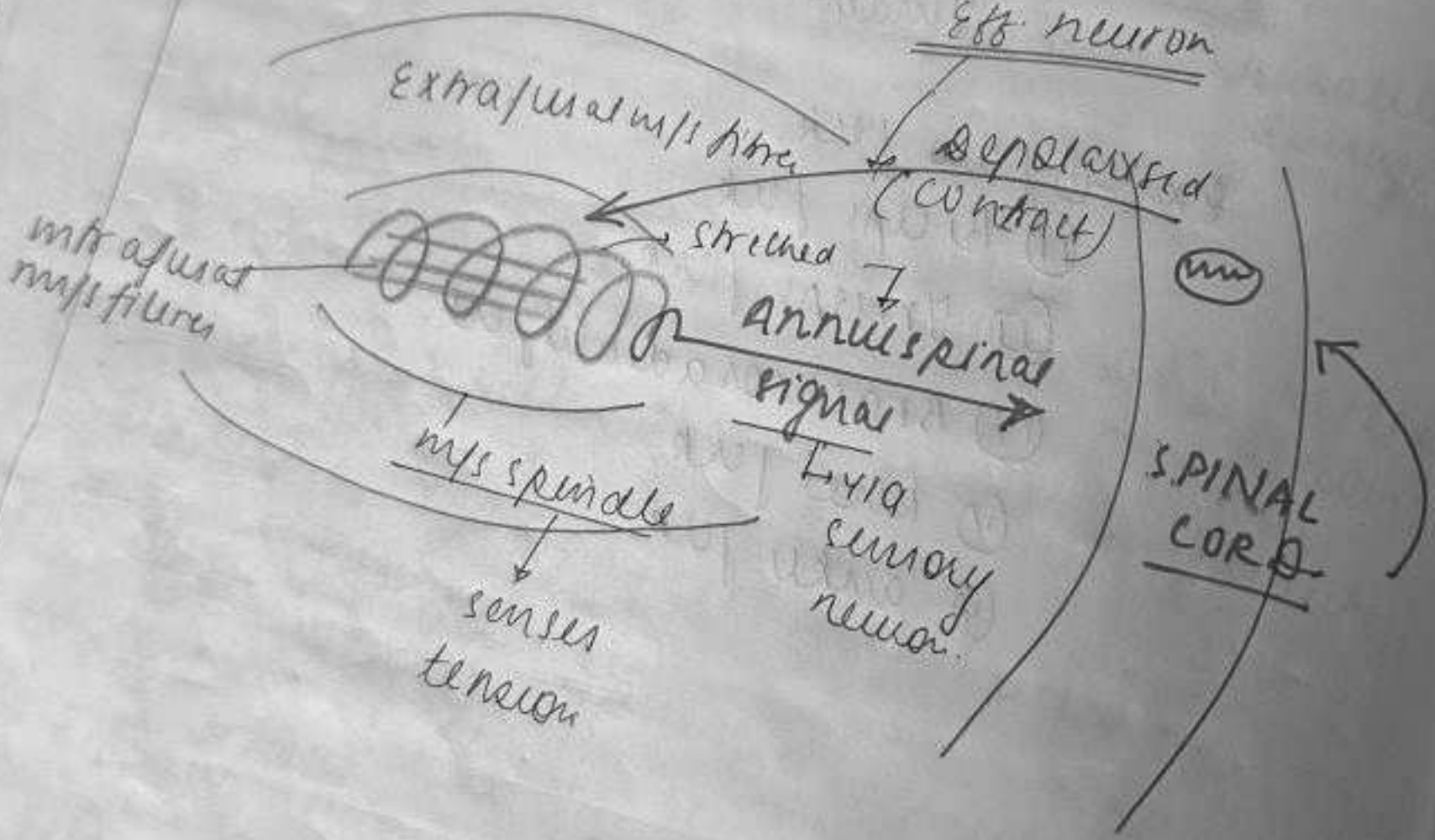
→ Reflex is classified upon clinical parameters:-

① superficial Reflex.

② Deep Reflex: Elicited from deeper str. like tendon or deep to skin. also called Tendon Reflex:-

Ex:- (i) Jaw jerk.
(ii) Biceps jerk.
(iii) Triceps jerk.
(iv) Brachioradialis jerk.
(v) knee jerk.
(vi) ankle jerk.

Stretch Reflex:-



PATHOLOGICAL REFLEX

+ BABINSKI SIGN :-
- abnormal plantar reflex.
- also called Babinski reflex / phenomenon.
- discovered by scientist Babinski.
- +ve in UMNL.

- Normal plantar reflex :-
  gentle scratch over outer edge of sole of foot
  ↓
  plantar flexion → down.
  &
  abduction of all toes

- In Babinski sign,
  ↓
  dorsiflexion ↑ of great toe
  &
  fanning of other toes.

- Babinski reflex +ve → Babinski +ve
- Babinski reflex -ve → Babinski -ve.

UMNL	LMNL.
→ all superficial reflex lost	→ all superficial & deep reflexes lost.
→ Babinski sign +ve.	
→ Deep reflex exaggerated.	

UMN & LMN.

Neuron of motor system.

Based on location of terminations

UMN

- neuron of higher centre of brain
- controls LMN.
- (3) types
  - (1) Motor neurons in cerebral cortex
  - (2) Motor neurons in basal ganglia & brainstem nuclei
  - (3) Neurons in cerebellum

LMN.

- common final pathway of motor system
- constitutes
  - ant. gray horn cells of SC
  - cranial N. nuclei situated in brain stem
- innervate the m/s directly

Clinical confirmation

LMN.
on fibre
ay of motor
tion
y horn cells of
V. nuclei
ted in lower
Stem
he m/s
difficulty

→ Effects of UMN Lesion & LMN Lesion:-

EFFECTS	UMN Lesion.	LMN Lesion.
(I) Mis tone	hypotonia	hypotonia
(II) Paralysis	spastic type of P.	flaccid type of paralysis.
(III) wastage of m/s occurs	✓	✓
(IV) superficial Reflex	lost.	lost.
(V) Plantar Reflex	Babinski sign	(NP)
(VI) Deep Reflex	exaggerated	lost.
(VII) Clonus	✓	X.
(VIII) Electrical activity	✓	X.
(IX) Mis affected	grp. of m/s	individual m/s
(X) Fasciculation twitching EMG	X	✓

Clinical confirmation

PATHWAYS FOR PAIN SENSATION

→ carried to brain by different parts of body by different pathways:-

i) Pathway from skin & deeper str.
ii) Pathway from face.
iii) Pathway from viscera
iv) Pathway from pelvic region.

① PATHWAY FROM SKIN & DEEPER STR.

① Receptor:- free nerve endings distributed throughout the body

↓

1st order neuron - cells in post horn of grey matter

- Substantia gelatinosa
- Marginal nucleus

2° order neuron

- substantia gelatinosa → receives slow pain fibres i.e. C-type
- marginal nucleus → receives fast pain fibres i.e. Aδ

2° order neuron

- fast pain fibres arise from marginal nucleus. — cross median
- slow pain fibres ↳ arise from substantia gelatinosa. ↳ cross median & ascend along paleospinothalamic tract

ascend in the form of lateral spinothalamic tract.

Post central gyrus of sensory cortex. Parietal lobe

Cortical cortex

Thalamus.

periaqueductal grey matter

Reticular formation

M.B

Analgesic pathway

Nu raphe magnus

M.O

pain transmission

(1) posterior nerve root Ganglion

Axon.

(1st spinothalamic tract)

pain pathway

spinal cord.

pain receptor

(2) post grey horn of s.c

FAST → Aδ Afferent → Marginal nucleus of

SLOW → C type Afferent → substantia gelatinosa of Rolando

- 3rd Order Neuron
  - are the neurons in:-
    - i) Nucleus of V.B.
    - ii) grey matter around aqueduct of Sylvius
    - iii) Thalamic nucleus
    - iv) Reticular formation

- Area for pain sensation:-
  - Post central gyrus of parietal cortex

- Neurotransmitters involved in Pain sensation.
  - Glutamate.
  - Sub. P.
    - Slow pain
      - C-type of afferent neuron
        - Secretes Substance P.
    - Fast pain.
      - Aδ type of afferent neuron.
        - Secretes Glutamate.

(N)

(iii) Pain sensation from Face:-
carried by CN-5

(iii) Pain sensation from Viscera:-

Thoracic & Abdominal viscera
↓
transmitted by sympathetic (thoracolumbar nerves).

Esophagus, Trachea & pharynx.
↓
carried by CN-10 & CN-9.
(Glossopharyngeal nerve)

(iv) Pain from Pelvic Region:-
↳ carried by sacral parasympathetic fibers.

ANALGESIA system

pain control system.

Body has its own analgesia system in brain which provides short term relief from pain

- **analgesic pathway:-**

analgesic pathway that intervenes with the transmission of pain is often considered as descending pathway.

① Fibres of analgesic pathway arise from frontal lobe of cerebral cortex.

↓

② terminate in grey matter surrounding 3rd ventricle & periaqueductal grey matter.

↓

③ Fibres from here descend down to brainstem & terminate on
- Nucleus raphe magnus
- Nu. reticularis

↓

④ Fibres from reticular nucleus descend through the lateral white column of SC & reach the synapse of neuron in afferent pain pathway in anterior horn of grey matter.

- Synapses of afferent pain pathway are L/w:-

① Aδ type of afferent fibres & neurons of marginal nucleus.

(ii) C type of afferent fibres & neurons of substantia gelatinosa.

(b) At synaptic level, the analgesic fibres release neurotransmitter - serotonin

⊖

pain transmission before being relayed into brain

GATE CONTROL THEORY

└ for pain to express pain suppression

→ A/c to this the pain stimuli transmitted by afferent fibres is blocked by gate mechanism

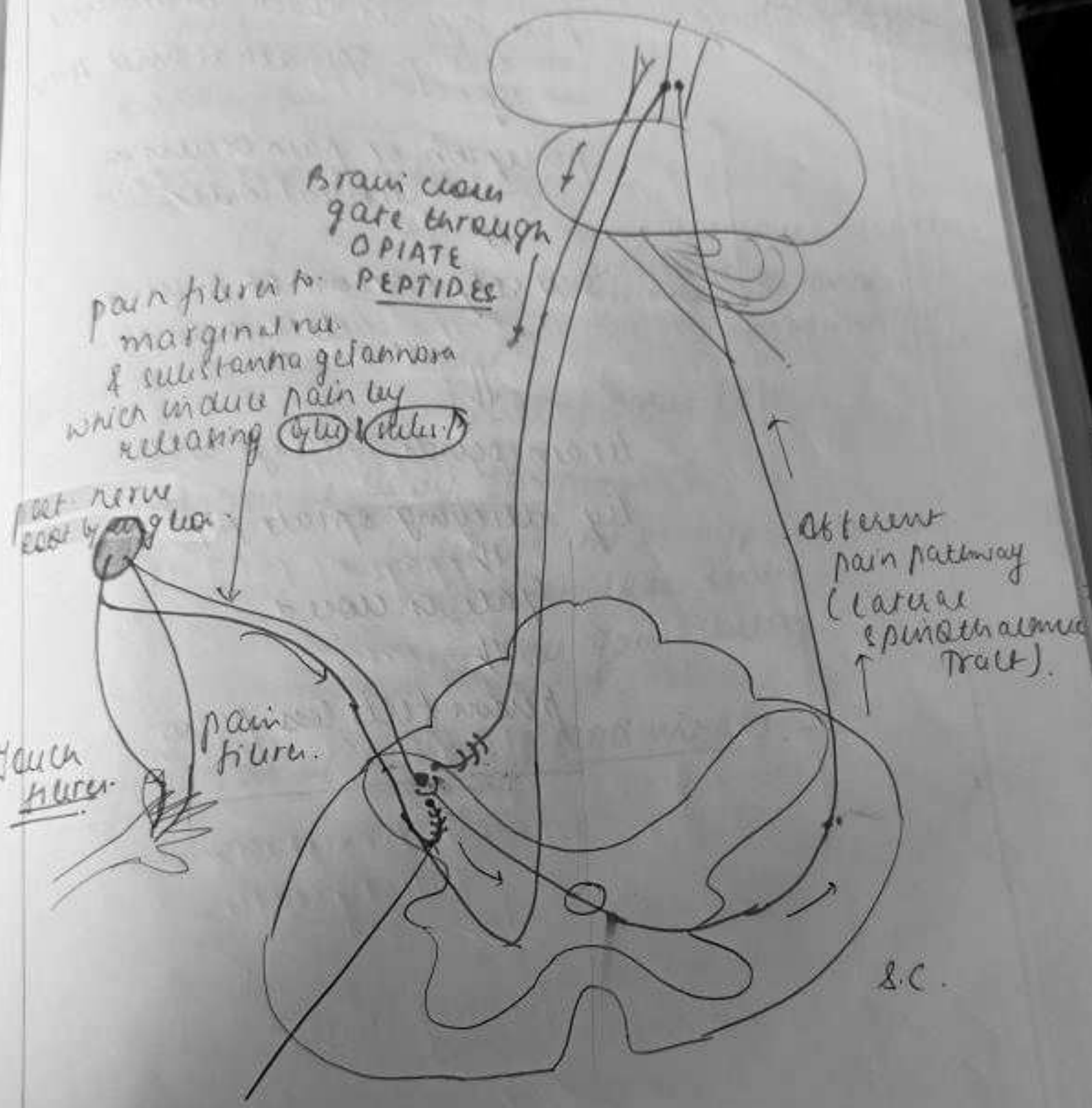
→ Gate open → pain felt
→ Gate closed → pain suppressed

MECHANISM OF GATE CONTROL AT S.C. LEVEL.

pain stimulus applied.
⇓⊕
touch receptors along with pain receptors
↓
reach S.C through post. nerve root ganglion
↓
send collaterals to neurons of pain pathway i.e. cell of marginal nu. & substantia gelatinosa.
↓⊖
release of glutamate & substance P from afferent pain fibres.
↓
closes the gate.
pain transmission is blocked

Role of Brain in gate control mechanism

gate



Collateral from
post. column fibres.
⊖
~~pain~~
Release of Glu & mes. P.
∴ block pain transmission.

Fig: GATE CONTROL SYSTEM

⇒ Role of brain in Gate Control Mechanism. —

gain in S.C. & ward.

⇓

pain signals reach thalamus through spinothalamic tract

⇓

perception of pain occurs in cerebral cortex.

⇓

severity & extent of pain is determined

⇓

brain sends message to S.C. by receiving opiate pipeline

⇓

Gate is closed.

⇓

person feels less pain

(i) Re

(ii) co

(iii) Con

(iv) Reg

(v) Regu

FUNCTIONS OF hypothalamus:-

(i) Release & control of hormones of post. pituitary -

PVN ——— ADH / vasopressin
&
supra-optic nuc. ——— oxytocin

(ii) control of ant. pituitary:-

by releasing releasing hormones (ex: GnRH
&
inhibiting hormones.
ex- somatostatin

(iii) Control of adrenal cortex:-

by releasing ACTH CRH

(iv) Regulation of body temp:-

(a) centres present in hypothalamus:-
(i) heat loss centre.
(ii) heat gain centre.

(v) Regulation of hunger & food intake:-

MECHANISM OF REGULATION OF FOOD INTAKE

→ GLUCOSTATIC MECHANISM:-

→ Food intake.

↓

↑ Blood glucose level.

↓ (+)

Satiety centre.
(Ant. of Hypothalamus)

↓ (−)

Feeding center.

↓

stoppage of food intake.

↓

after ~ 3 hrs.

↓

blood glucose level ↓

↓

no more stimulation of satiety centre.

↓ (+)

Feeding center.

↓

↑ Appetite

(VII) Regulation of water balance:-

1. Thirst mechanism.
2. ADH Mechanism

THIRST MECHANISM:-

ECF volume ↓

↓

Osmolality of ECF ↑

↓ ⊕

Osmoreceptors in hypothalamus. ← OLT SFO

↓ ⊕

Thirst centre.

↓

Feeling of thirst

↓

H_2O intake.

↓

↑ ECF vol.

↓ osmolality of ECF.

(VII) Role in Behavioural & Emotional changes:-

[7]
2017

→ Hypothalamus has ③ centres for behavioural & emotional changes.

① Reward centre
② Punishment centre.

① Reward centre:-
⊕ → situated in medial forebrain bundle & ventromedial nucleus of hypothalamus
pleases/satisfies the animal.

② Punishment centre:- ⊕ → situated in posterior & lateral nucleus of hypothalamus
pain, fear, escape reaction
& other elements of punishment.

→ Role of Reward & Punishment centre:-

Importance of reward & punishment centre lies in behavioural pattern of an individual.

If person feels rewarded/ satisfied.	If person feels punished.
↓	↓
he/she continues to do so.	he/she stops doing it.

→ ∴ These ② centres play imp. role in the development of behavioural pattern of an individual.

(VIII) Regulation of sexual function:-

↳ releasing GnRH.

↓ ⊕

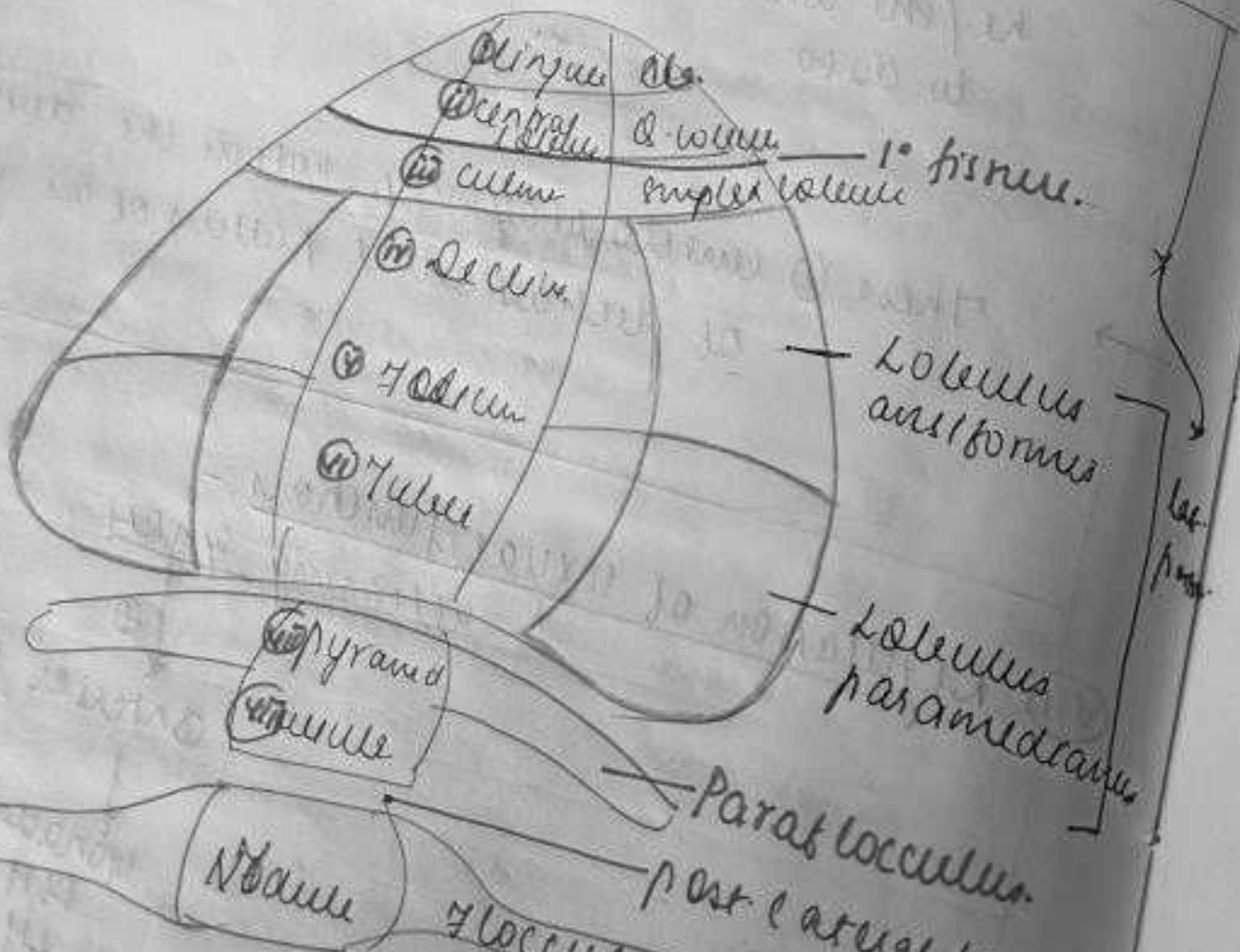
anterior pituitary

↓

Gonadotropins
FSH
LH

Physiological / Functional Division of Cerebellum

① Vestibulocerebellum / Archicerebellum
② Spinocerebellum = Vermis (nodule) + Int. lobe
③ Cerebrocerebellum.



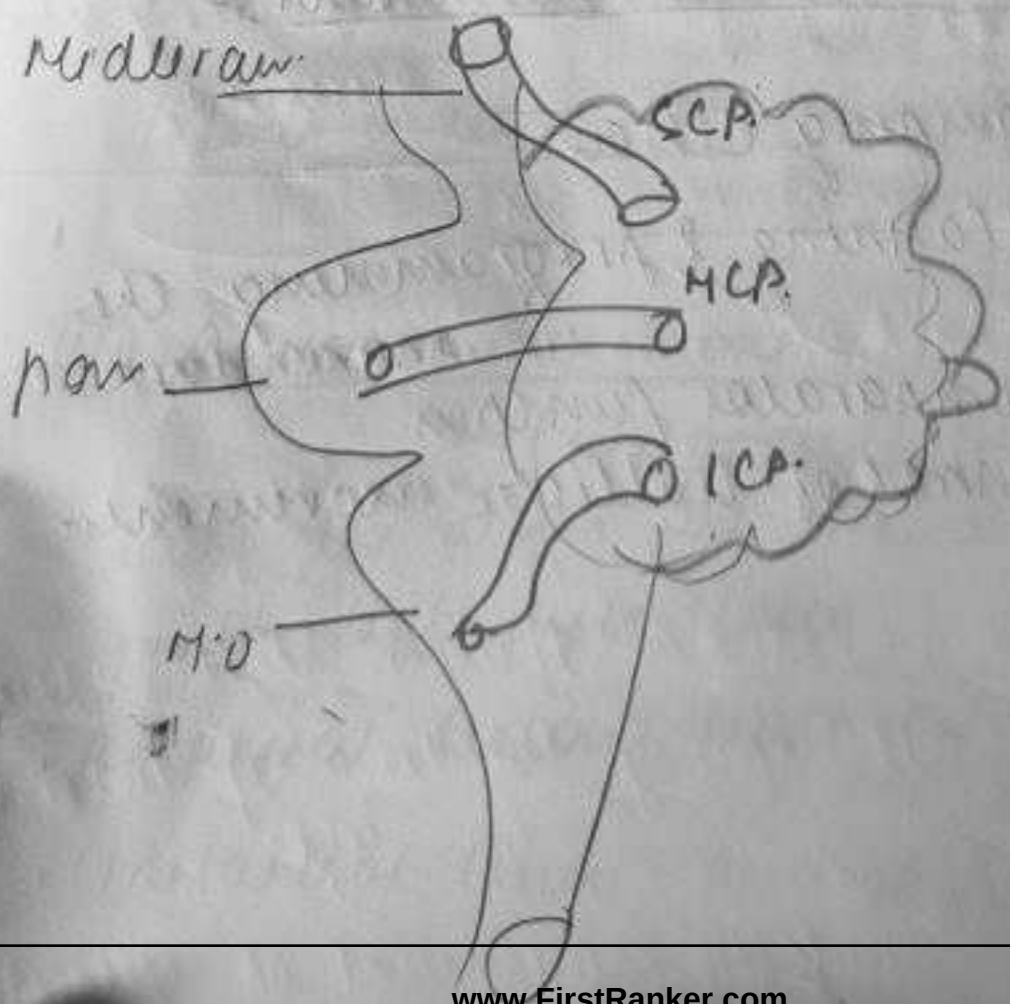
Connections of cerebellum:-

(3) pairs of cerebral peduncles:-
- (i) S.C.P
- (ii) M.C.P
- (ii) I.C.P

→ Superior cerebellar peduncle:-
- connects S.C → M.B
- mostly afferent fibre.

→ Middle cerebellar peduncles:-
- S.C → pons
- purely afferent fibres.

→ Inferior cerebellar peduncles:-
- S.C → M.O
- mostly afferent fibres.



FUNCTIONS OF CEREBELLUM.

① Regulation of tone, posture & equilibrium:-

VESTIBULO-CEREBELLUM/ ARCHICEREBELLUM.

(i) By receiving impulses from vestibular apparatus.

SPINOCEREBELLUM.

(ii) By receiving impulses from
- proprioceptors in m/s, tendons & joints.
- ~~pain~~ tactile receptors.
- visual receptors.
- auditory receptors.

(ii) Regulation of Co-ordinated movements:-

CORTICOCEREBELLUM/ NEOCEREBELLUM

(i) Damping action
(ii) Planning & programming the movement
(iii) Comparator function
(iv) control of ballistic movements.

Cerebellar Lesions:- tumour, injury / vessels R-OT consumption

(i) disturbance in tone & posture
(ii) disturbance in equilibrium.
(iii) disturbance in movement.

(i) Disturbance in tone & posture:-

(1) Atonia or Hypertonia:-
- Atonia: loss of tone of m/c.
- Hypertonia: ↑ tone of m/s

→ Occurs due to loss of facilitatory impulses to γ-motor neurons in S.C.

(ii) Attitude:-

↳ Attitude of body changes in unilateral lesion of cerebellum.

- Trunk is bent with concavity towards affected side.

(iii) Deviation movement:-

↳ Lateral deviation of arms when both the arms are stretched in front of body with both eyes closed.

- Bilateral lesion - both arms deviate.
- Unilateral lesion - arm of affected side deviates

④ Effect on deep reflexes:-

↳ pendular movement occurs while eliciting a tendon jerk.

Ex: knee jerk.

(ii) Disturbances in Equilibrium:-

① While standing:-

- Legs spread to provide broad base
- Body sways side to side.
- oscillations of head.

② While moving - Gait:-

- Gait means manner of walking.
- staggering gait, drunken like gait is observed

(iii) Disturbances in movements:-

i) ATAXIA: lack of co-ordination of movements.

ii) ASTHENIA: weakness, slowness of mvt.

iii) INTENTION TREMOR:- Tremor that occurs while attempting to do any voluntary act.

iv) NYSTAGMUS:- To & fro movement of eyeball

2016

BASAL GANGLIA

- mass of grey matter, present in subcortical region of cerebral hemisphere.
- forms the part of extrapyramidal system, which is concerned with motor activities.

→ **Components of Basal ganglia :-**

(i) Corpus striatum.
(ii) ~~Lentiform nucleus~~ Substantia nigra.
(iii) Subthalamic nucleus of Luys.

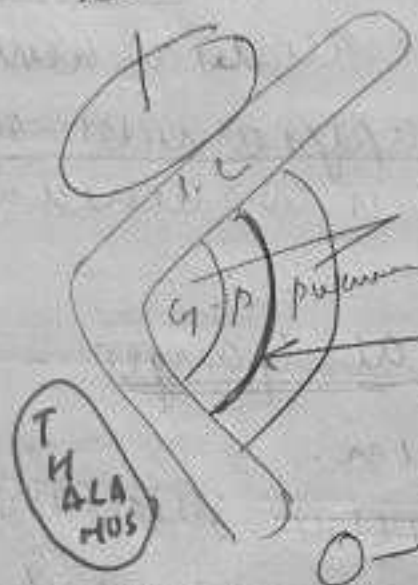
→ **Corpus striatum :-**

- mass of grey matter closely related to thalamus.
- divided into (2) parts by internal capsule
  (i) Caudate nucleus.
  (ii) Lentiform nucleus.

- Caudate nucleus ← head, tail — present medial to I.C.

- Lentiform nucleus ← Globus pallidus, putamen.
  — lat. of I.C

Head of caudate nu.



(I) Lenticular nucleus
- External medullary Lamina.
- tail of caudate nucleus.

(II) Substantia nigra:-
- part of M.B.
- separates the tegmentum & crus cerebri of M.B.
- contains ppty of (Fe)

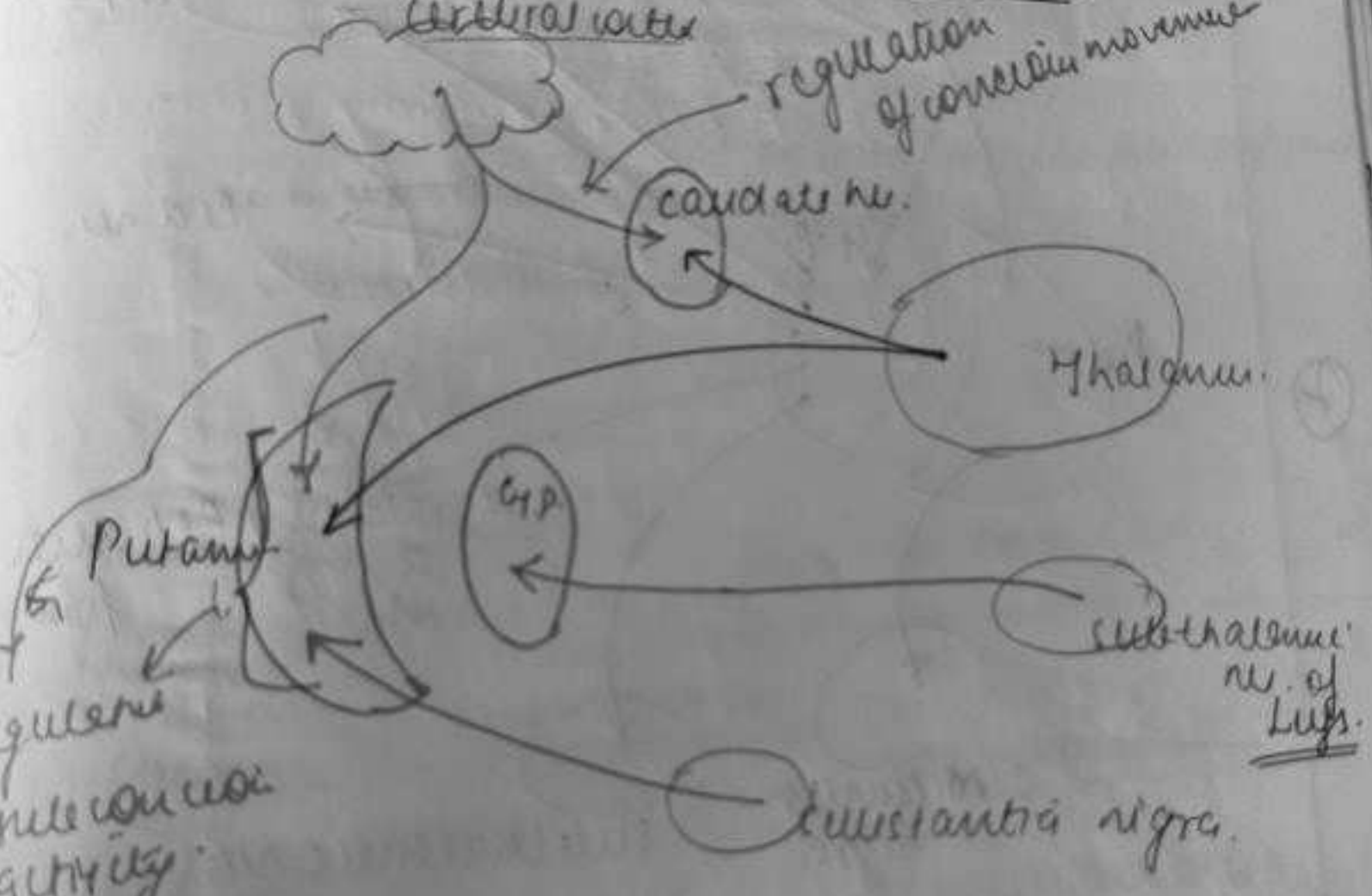
(III) Subthalamic Nucleus of Luys:-
- lateral to (Red nucleus) ★
- dorsal to substantia nigra.

2010

Connection of Basal ganglia :-

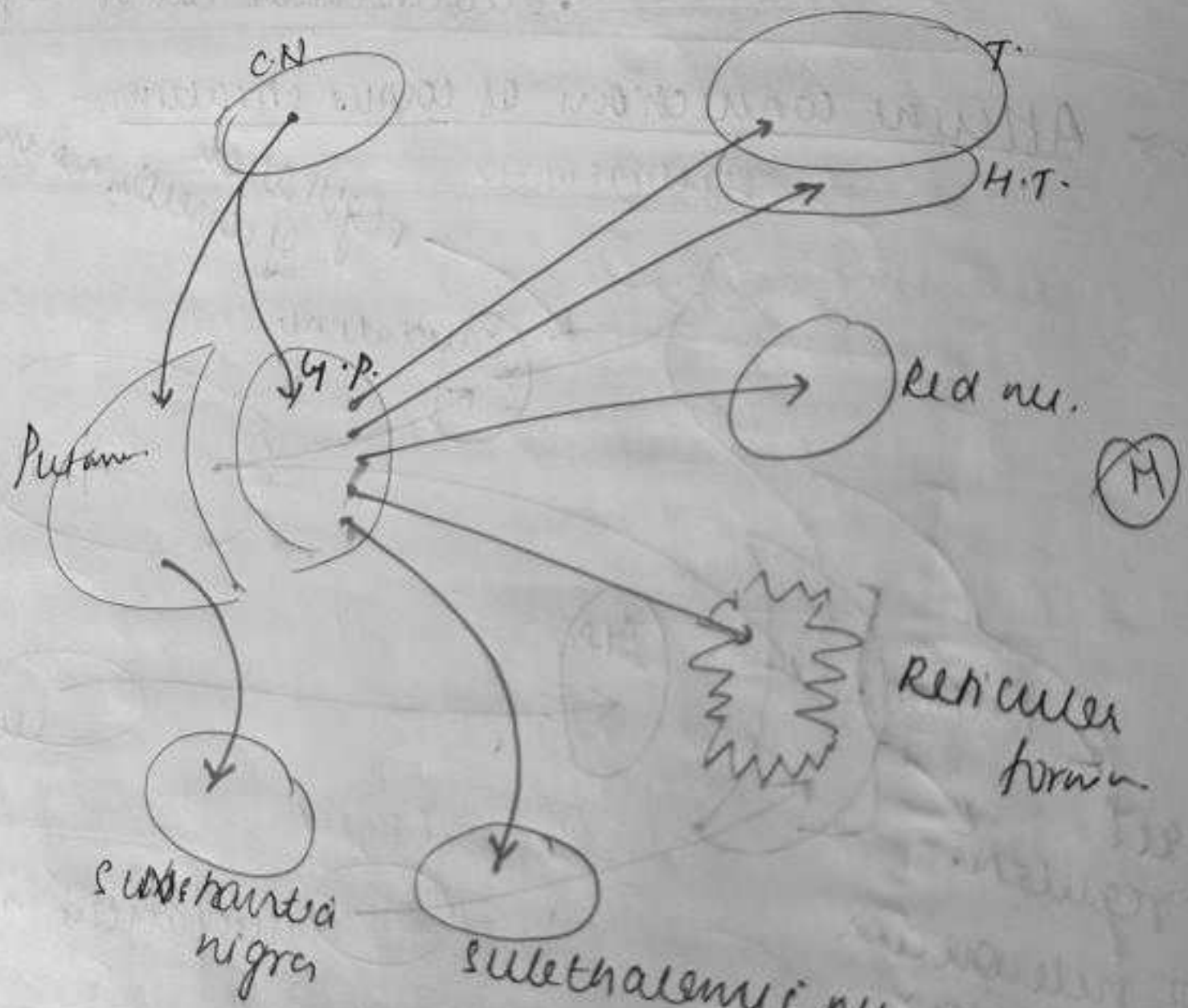
Afferent & Efferent connections of:
- Corpus striatum,
- Substantia nigra,
- Subthalamic nu. of Luys.

Afferent connection of corpus striatum :-



- Afferents come to putamen

Efferent & Intrinsic connections of corpus striatum.



(P) (H)

2016

FUNCTIONS OF BASAL GANGLIA.

- form part of extrapyramidal system.
- concerned with integration & regulation of motor activities

① Control of m/c Tone:-

↳ Basal ganglia control m/c tone
r. motor neuron - responsible for the development of tone m/c.

Basal ganglia
↓ ⊖
r-motor neuron
↓
[↓ m/c tone]

Lesion of Basal ganglia → m/c tone ↓

② Control of motor activity:-

① Regulation of voluntary movements:-
↓
controlled by basal ganglia which is in close association with cerebral cortex.

- Basal ganglia control motor activities through neuronal circuits b/w basal ganglia & other parts of brain.

- Neuronal circuits arises from ③ area of cerebral cortex:- ① 1° motor area.
② Premotor area.
③ supplementary motor area.

(ii) Regulation of conscious movements:-

→ Fibres u/g cerebral cortex & caudate nucleus responsible for regulation of conscious movements.

(iii) Regulation of unconscious movements:-

→ cortical fibres reaching putamen of LN. are directly concerned with the regulation of unconscious movements

↓

which trained takes place during trained motor activities.

Ex:- writing learned alphabets.

③ Control of Reflex Muscular activity:-

↓

particularly visual & labyrinthine reflexes.

↓

imp for maintaining posture

- Basal ganglia - responsible for integration & coordination of impulses from these reflex activities.

⑤ Control of automatic associated Movements:-

↓

movements which take place in the body along with some other motor activities.

Ex: ① swing of arms while walking

② appropriate facial expressions while talking.

- Basal ganglia is responsible for the control of automatic associated movements.

2016

PARKINSON'S DISEASE:-

↳ slowly progressive degenerative disease of nervous system, associated with destruction of brain cells which produce dopamine.

→ Causes of Parkinson's disease:-

↳ lack of dopamine,

⇑

damage to basal ganglia.

↓

mostly due to destruction of substantia nigra & nigrostriatal pathway

↓

contains dopaminergic fibres.

→ Damage to basal ganglia occurs because of the following causes:-

(i) viral infection of brain like encephalitis.
(ii) Injury to basal ganglia
(iii) cerebral arteriosclerosis.
(iv) Destruction/removal of dopamine in basal ganglia. It occurs mostly due to long term treatment with anti-hypertensive drugs.
(v) Parkinsonism due to drugs is called drug induced parkansonism.
(vi) Unknown causes:- Idiopathic parkinsonism.

Signs & symptoms of parkinson's disease:-

↳ develops very slowly.

① **Tremor:-** Tremor occurs during rest & static tremor.

↓

disappears when doing any work.

② **Slowness of movement:-**

movement starts slowing - Bradykinesia

↓

Gradually, patient becomes unable to initiate any voluntary activity.

(Akinesia)

③ **Poverty of movement:**

↳ Loss of all associated movements.

④ **Rigidity:-** stiffness of muscles.

↑

x-motor neurons.

Basal ganglia ⊖ ⊕

⑤ **Gait:-** ↳ mean manner of walking.

· Patient loses normal gait in parkinson's disease

↳ **Festinant gait**

(VI) speech problems:-

(VI) Emotional changes:- person suffering from parkinson's disease are often upset emotionally

(VII) Dementia:-
└ memory loss.
In later stages, patient develop dementia.

Huntington's chorea

Treatments of Parkinson's disease:-

→ Parkinson's disease occurs due to lack of Dopamine.
↑
damage to dopaminergic fibres.

→ It is treated by Dopamine injection

Dopamine ≠ cross BBB.

∴ another substance - Levodopa (L-Dopa)

is injected

crosses BBB.

converted to dopamine in brain

- since L-Dopa ~~is converted~~
└ LIVER, Dopamine

some side effects occur

→ along with L-Dopa, another substance called carbidopa.

carbidopa → administered.

L-Dopa —LIVER→ Dopamine

≠ cross BBB.

∴ L-dopa moves to brain tissue & is converted into Dopamine.

→ HUNTINGTON CHOREA:-

↓

inherited progressive neural disorder.

due to degeneration of neurons secreting GABA

in [corpus striatum
substantia nigra.

— occurs in middle age mostly.

— characteristics
(i) chorea.
(ii) hypotonia
(iii) dementia
(iv) bilateral wasting of m/s occurs.

LIMBIC SYSTEM:-

Learner called Rhinencephalon.

COMPONENTS of Limbic system:-

(i) Archicortical structure
- (i) Hippocampus
- (ii) Dentate gyrus

(ii) Paleocortical structure
- (i) Piriform cortex.
- (ii) Olfactory tubercle
- (iii) Olfactory tubercle

(iii) Juxtacortical str.
- (i) cingulate gyrus of limbic cortex.
- (ii) Orbitoinsulotemporal cortex.

(iv) subcortical str.
- (i) Amygdaloid complex.
- (ii) Thalamic nuclei
- (iii) hypothalamic nuclei
- (iv) caudate nucleus.
- (v) Reticular formation of midbrain.
- (vi) septal nuclei.

Efferent connection of Basal ganglia

STRETCH REFLEX.

↓

Reflex contraction of m/s when it is stretched.

- also called Myotactic reflex
- monosynaptic Reflex
- quickest of all reflex.

→ M/s spindles the receptor organ for stretch Reflex.

↓ ⊕

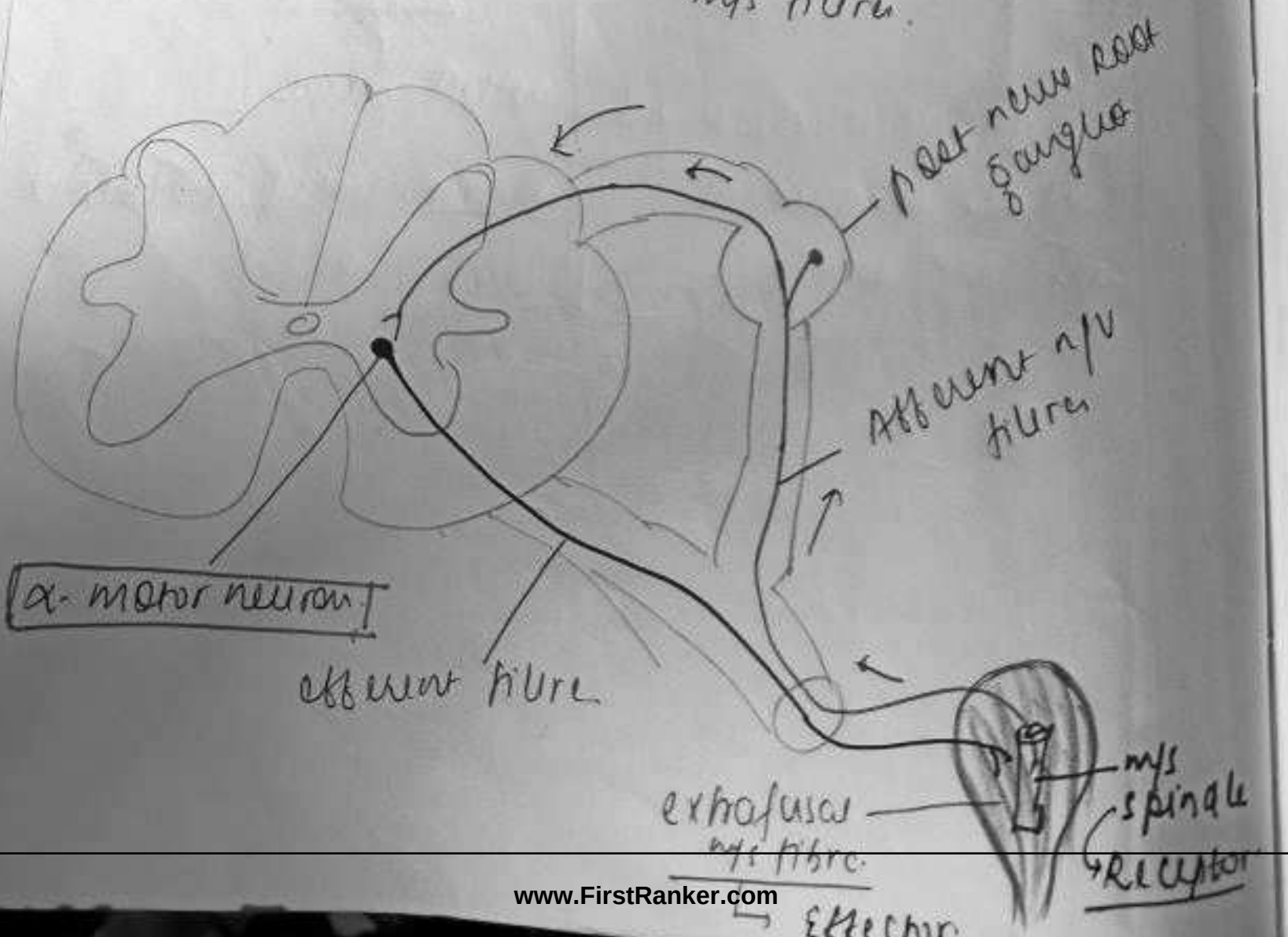
muscle spindle.

↓

stretch reflex.

- Intrafusal m/s fibres are present ll to extrafusal m/s fibres & are attached to tendon of m/s.

stretching of m/s fibres
↓
stretching of m/s spindle.
↓ ②
m/s spindle.
↓
discharges sensory impulses
↓
transmitted by 1° & 2° afferent nerve fibres
↓
to
↓
α motor neurons in S.C
↓
send motor impulses via efferent nerve fibres to
↓
contraction of extrafusal m/s fibres.



RESPONSE OF M/S SPINDLE TO STRETCH.

② types.

Dynamic Response

→ response depends on rate of change in length of m/s ($\frac{dl}{dt}$)

- 1° sensory n/v fibres discharge rapidly.

→ discharge becomes nil during constant stretching of m/s ($\frac{dl}{dt} = 0$)

Static Response:-

→ response depends on change in length as well as rate of change in l.

→ impulses are discharged rapidly throughout the period of m/s stretch by 1° sensory fibres of the nuclear chain fibres.

Ex: postural Reflexes.

Static Reflex:- postural reflexes that maintain posture at rest

Basic phenomena of posture

- Basic phenomena for maintainance of posture are :- ① M/s tone
  ② Stretch Reflex. ← purely Reflex process - spinal segmental Reflex. (static Reflex)

Defn:- **Muscle Tone** :- also called (Tonus)

└ state of passive partial contraction of m/s with certain vigour & tension

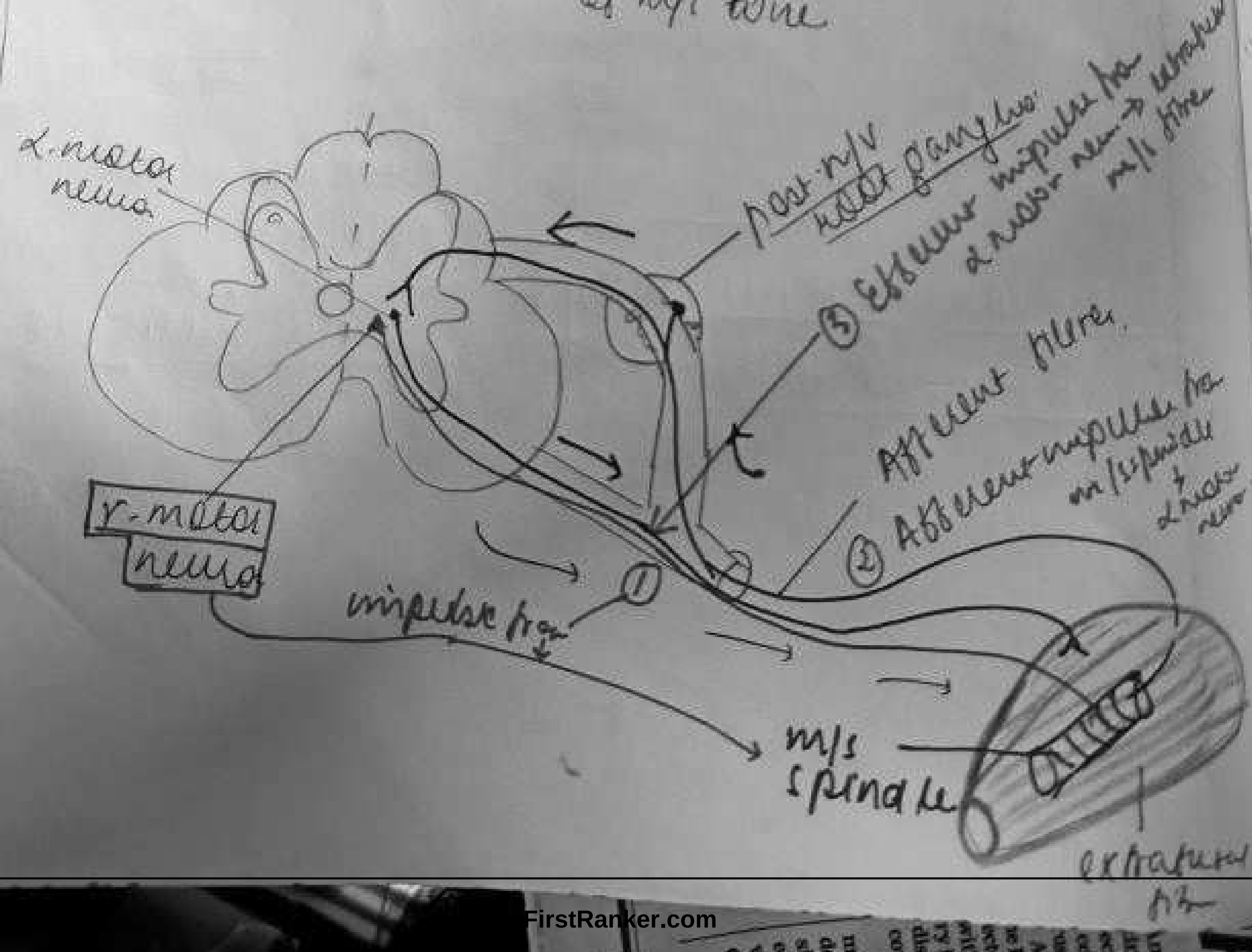
→ **Significance of m/s Tone** :-

↓ imp. for maintainance of posture

- change in m/s tone enables the movement of different parts of body.

→ **Developement of m/s Tone** :-

γ motor neuron & m/s spindle] responsible for the development & maintainance of m/s tone



impulses from γ-motor neurons

↓ ⊕

intrafusal m/s fibres

↓

generation of impulses

↓

Transmission of impulses to C.C. by
1° sensory nerve fibres (Afferent nerve fibres)

↓ ⊕

α-motor neurons of S.C. in ant. grey horn

↓

Transmission of impulses via
α-motor neuron fibres
(Efferent fibres)

↓ ⊕

extrafusal m/s fibre

↓

Partial contraction of m/s

↓

M/S tone

Regulation of m/s tone :-

- Though m/s tone is developed by discharges from γ-motor neuron.
- It is maintained continuously & regulated by tone supraspinal centers present in diff parts of brain

↑ tone ← → ↓ tone

supraspinal facilitatory centers.

↑ m/s tone

① Motor area (4) of cerebral cortex.

② Red nucleus

③ Cerebellum

④ vestibular nucleus

⑤ descending facilitatory reticular system.

supraspinal inhibitory centers.

↓ m/s tone.

⑥ suppressor area of cerebral cortex.

⑦ Basal ganglia.
↳ γ motor neuron.

⑧

⑨ descending inhibitory reticular system.

→ SLEEP - 2 types

→ REM sleep / Rapid Eye Movement sleep.

→ NREM sleep / Non REM sleep / Non Rapid Eye movement sleep.

	REM (Rapid Eye Movement) sleep	NREM sleep
	present	(-nt)
Dreams	present	(-nt)
Muscle twitching	present	-nt.
Heart Rate	Fluctuating	Stable.
B.P.		
Body Temp.		
Respiration		
Neurotransmitter (NT)	Acetylcholine	Serotonin

→ REM sleep :- type of sleep associated with rapid conjugate movements of eyeball which occur frequently.

① Though the eyeballs move the sleep is deep

② also called PARADOXIAL SLEEP

③ occupies 20-30% of sleeping period.

④ Dreams occurs.

⑤ REM sleep is nip : consolidation of memory occurs during the period.

EEG (Electroencephalogram)

- ~~Elect~~
- Wave of EEG :-

(Study of electrical activity of brain.

electrical activity recorded by EEG may show synchronised / desynchronised waves.

→ **Synchronised wave:**
regular
invariant wave.

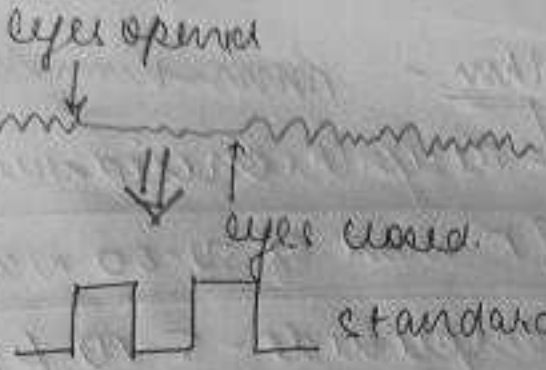
Desynchronised wave
irregular & variant wave.

→ **EEG has ④ rhythm:-**

① α wave rhythm.
② β wave rhythm.
③ δ wave rhythm.

① **α - Rhythm:-** synchronised wave.
rhythmic wave.
frequency 7 → 18 - 12 waves/sec.
amplitude 50 μV.

- α rhythm is obtained in
  - light sleep.
  - drowsiness.
  - person with closed eye.



α-block

α-block:- replacement of synchronised α wave by desynchronised ↓voltage waves in EEG when eyes are opened.

- desynchronised waves do not have specific r.

→ Desynchronisation:-

L common term used to refer for the replacement of regular α waves with irregular ↓voltage waves.

L It is due to loss of synchronised activity in neural system i.e. responsible for regular wave pattern.

→ β Rhythm:- asynchronised waves.

↑r wave of 50-60 waves/sec

↓ amplitude - 5-10 μV

recorded during - mental tension or arousal state.

- not affected by opening of eyes.

→ Delta Rhythm:- appear mostly during deep sleep.

δ waves.