

REPRODUCTIVE SYSTEM ***

① SERTOLI CELLS

→ Sertolacellar cells in seminiferous tubules

Structure :- • Irregularly shaped, extending from basement membrane to the lumen of seminiferous tubule.

- Sperm are attached to the apical mem. of cells.
- At base, attached to each other by tight junction.

divide it into 2 compartments

Basal Comp → • Outer comp

- mainly consist spermatogonium

Adluminal comp → • Inner comp

- consist of 1° & 2° spermatocytes & spermatids

* Blood - Testis Barrier

The tight junction b/w Sertoli cells are too tight, they do not allow sub from the interstitial space & blood to pass through them, except steroids & proteins

⇒ This tight junction b/w Sertoli cells near the basal lamina forms Blood - testis barrier (BTB).

→ Functions

1. Protects the germ cells.

2. Prevents autoimmune disease.

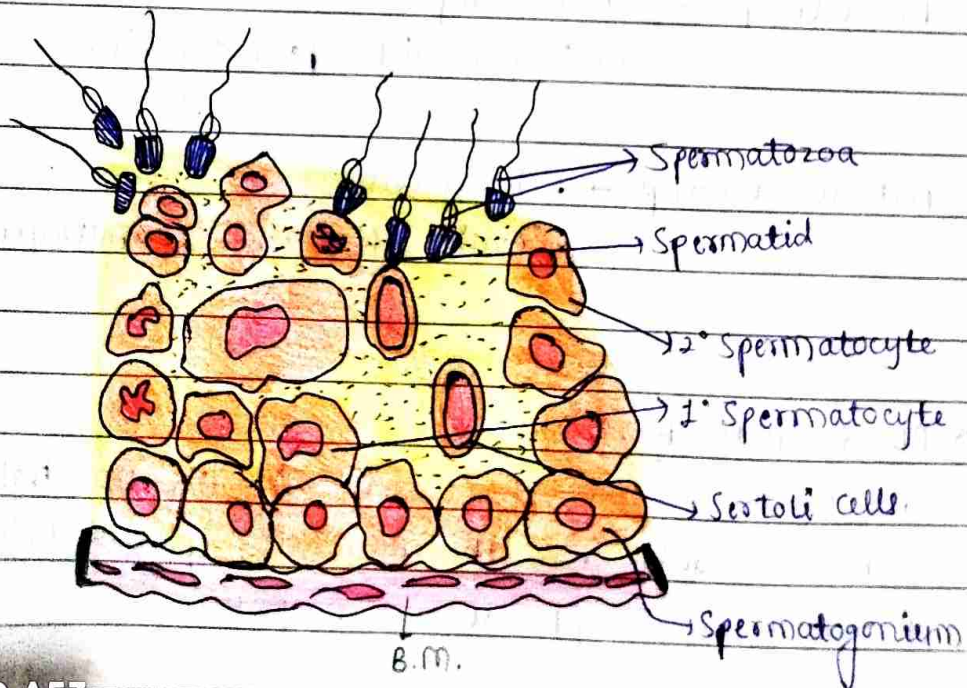
3. Maintains the composition of fluid in lumen

4. Maintains higher osmolarity of luminal content

★ Functions of Sertoli Cells

1. Support & Nourish the spermatogenic cells
2. Secrete enzyme aromatase, which converts androgen into estrogen.
3. Secrete androgen-Binding protein (ABP), essential for testosterone activity.
4. Secrete Estrogen-Binding protein (EBP).
5. Secrete Inhibin, which inhibits FSH release.
6. Secrete Mullerian-Regression factor (MRF) in fetal testes

↓
responsible for regression of mullerian duct during sex-differentiation in fetus.



② SPERMATOGENESIS

→ Process of development of male germ cells into Spermatozoa from primitive Spermatogonia.

* Steps

Divided into 3 phases :- Mitosis, Meiosis & Spermiogenesis

1. Mitosis

Spermatogonia present in basal lamina undergo mitotic division

↓
forms 1° Spermatocytes, process of Spermatocytogenesis

↓
Spermatogonia undergo many divisions & form 2 types of Spermatogonia

Spermatogonia A
Resembles original cell
& form spermatogonia
reserve pool in testis

Spermatogonia B
grows & enter adluminal comp.
where they develop into 1°
Spermatocytes.

2. Meiosis

↓
1° Spermatocytes (46)

↓
undergo 2 meiotic divisions

1st meiotic div. ↓

2° Spermatocytes [haploid 2N-DNA]

2nd meiotic div. ↓

Spermatids [haploid 1N DNA]

→ contains half the no. of chromosomes

- After undergoing, several mitotic & 2 meiotic div
↓
each spermatogonium yields 512 spermatids

3. Spermiogenesis

- Process of development of spermatids into matured spermatozoa
- Spermatids → small, round & less characteristic cells
• undergo structural changes & form spermatozoa

* Spermiation

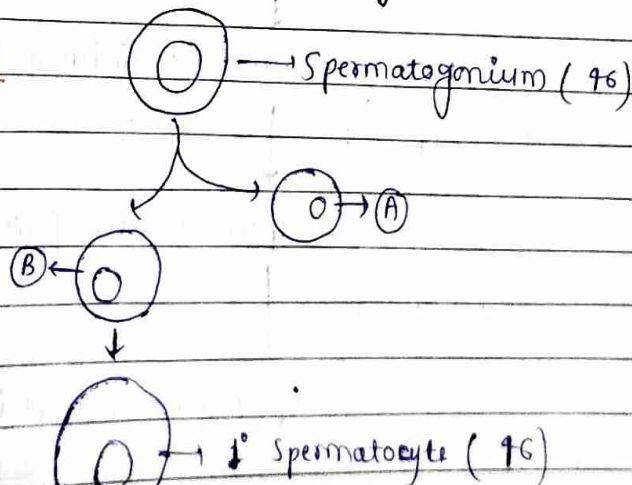
- Process of detachment of spermatozoa & their free release into luminal fluid from Sertoli cells.

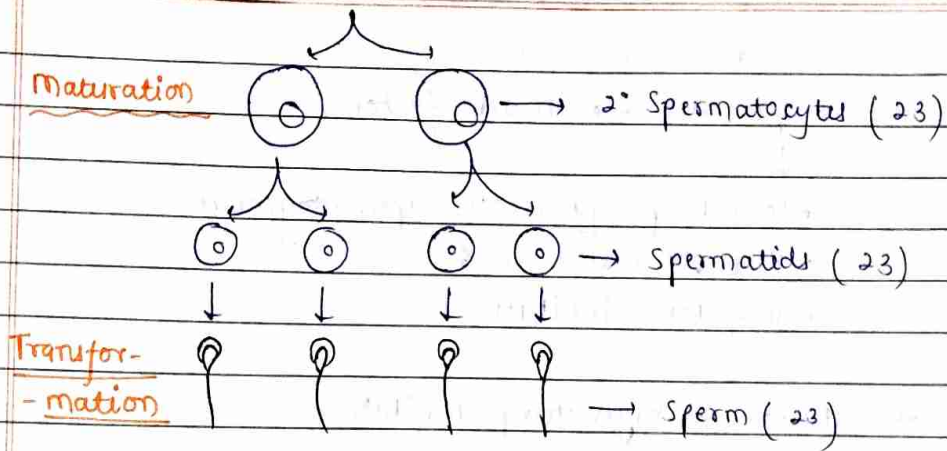
* Capacitation

When spermatozoa are released into female genital tract, they undergo further maturation c/d Capacitation.
It includes:- Inc in further motility of sperm & preparation for acrosomal rxn.

Flowchart of Spermatogenesis

Proliferation





★ Regulation of Spermatogenesis

→ Factors regulating can be divided into 2 categories :-

1. Hormonal factor

- **Androgen** → • stimulates spermatogenesis.
 - LH elevates conc. of Androgen by stimulating Leydig cells to secrete Testosterone & this **high interstitial conc. of testosterone is imp. for spermatogenesis.**
- **Estrogen** → • not in rete testis & there are estrogen receptors in rete testis.
 - It concentrates the fluid by facilitating fluid reabsorption & making spermatozoa concentrated, required for sperm maturation.
- **LH & FSH** → • helps by producing a high conc of androgen
 - FSH → maintains **gametogenic functions**
 - FSH required for initiation for spermatogenesis at puberty & then LH maintains it.

2. Environmental Factors

→ Temperature → main factor



- low temp. facilitates spermatogenesis
- Best occur at $30-35^{\circ}\text{C}$
- very low inhibits

→ whereas high temp inhibits

→ Generally, sperm count is more in comfortable winter & less in intense summer.

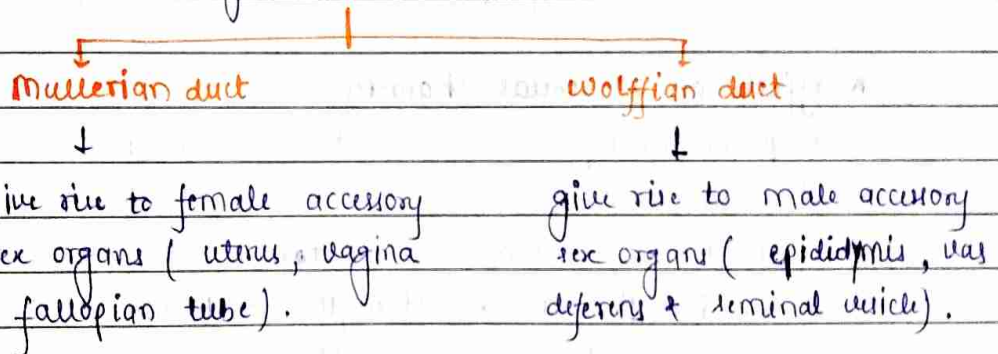
③ FUNCTIONS OF TESTOSTERONE

1. Functions in fetal life :-
1. Sex differentiation
 2. Development of accessory sex organs
 3. Descent of testis

* Sex differentiation

- Sex chromosome → responsible for determination of sex
- Testosterone → responsible for differentiation of sex.

- Fetus has 2 genital ducts



- If testosterone secretes at about 7th week → mullerian duct disappears & male sex organ develops.
- **Mullerian Regression factor (MRF)** → By sertoli cells also helps in regression.

* Development of Accessory Sex Organs

- Responsible for development of external genitalia i.e., penis & scrotum. & accessory organs i.e., ducts, seminal vesicle & prostate.

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Descent of Testis
→ Testis develops in abdominal cavity & descends in scrotum

through inguinal canal just before birth

↓
testosterone helps in this process.

* **Cryptorchidism** → failure of 1 or both testes to descent from abdomen into scrotum.

2. Functions in Adult Life :-

1. Effect on sex organs
2. Effect on 2° sexual character

* Effects on Sex organs

→ It fixes the size of penis, scrotum & testes after puberty.

* Effects on 2° Sexual character

→ helps in development of 2° sexual character

→ Characters are :-

1. Development of musculature

2. Fixes the size & strength of bones

3. Causes broadening of shoulder

4. Effects on pelvis :- male → funnel like & Narrow
Female → Broad & Round

5. Fixes the thickness & ruggedness of subcutaneous tissue of skin

6. Causes hair growth on pubis, linea alba, on face, chest & other parts.

7. Effect on voice :- low pitched with high intensity.

8. Fixes BMR

9. Fixes Na reabsorption from renal tubule

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2022/10

↓
causes rise in RBC count.

④

ESTROGEN

→ Secreted from granulosa cells of ovarian follicles, corpus luteum & placenta.

→ Not directly secreted from adrenal gland

↓

but adrenal androgen is converted to estrogen by aromatase

★ Functions

→ Major function is to promote cellular proliferation & tissue growth in sexual organs.

1. Effect on ovarian follicles

→ Promotes growth of follicles by ↑ing proliferation of follicular cells.

2. Effect on uterus

- Enlargement of uterus
- ↑ the blood supply to endometrium (E.M.)
- ↑ the glycogen & fat deposition in E.M.
- Proliferation & dilation of B.V. of E.M.
- ↑ in contractility of uterine muscles.

3. Effect on fallopian Tube

- ↑ the no. & size of ciliated epithelial cells.
- ↑ the activity of cilia
- Enhance proliferation of glandular tissue in tube.

4. Effect on vagina

- changes vaginal epi. from suboidal into stratified.

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↑ thickness of vagina
occurs to prevent vaginal infections like Gonorrheal infections

5. Effect on 2° Sexual Character

→ helps in development of character such as :-

1. Hair distribution in pubic ^{region} & axilla.
2. Skin becomes soft & smooth
3. Shoulder becomes narrow & fat deposition in breasts & buttocks.
4. Pelvis becomes broad, round/oval.
5. Voice is high pitched with low intensity

6. Effect on breast

→ Causes:-

1. Development of stroma in breast.
2. Causes development of lobules & alveoli of breast.

7. Effect on bones

- Increases osteoblastic activity
- Causes early fusion of the epiphysis with shaft.
- In old age, not secreted leads to osteoporosis.

8. Effect on Metabolism

- Induces anabolism of protein
- Causes deposition of fat in subcutaneous tissue.

9. Effect on Electrolyte Balance

Causes Na^+ & H_2O retention from renal tubules.

Receptors are on nuclear mem. of target cells & are of 2 types :- α & β estrogen receptor.

⑤

MENSTRUAL CYCLE

→ Cyclic events that takes place in rhythmic fashion during the reproductive period of a woman's life.

→ 1st cycle → Menarch → 12-14 yr.

→ last cycle → Menopause → 45-50 yr.

* Duration → about 28 days

* Phases of Cycle

→ Divided into 2 phases :- follicular & luteal phase.



separated by ovulation that occurs b/w these two.

1. Follicular Phase → • period from onset of bleeding to the day of ovulation

• Maturation of ovum with development of ovarian follicles takes place in this phase.

• Has 2 parts

↓
Menstrual Phase

↓
Proliferative phase



phase of menstrual
bleeding

phase of proliferation of uterine
endometrium

• also c/d Pre-ovulatory phase.

2. Luteal Phase → • part b/w ovulation & the onset of next menstrual bleeding.

• c/d luteal phase b/c Corpus luteum is formed in ovary.

PPO A530 c/d Secretory phase b/c endometrium is highly secretory.

22/07/15-21:42 — post-ovulatory phase

* Corpus luteum

- Glandular yellow body, developed from matured ruptured graffian follicle after release of ovum, also c/d Yellow body.

Development → • after rupture of graffian follicle & release of ovum, the follicle is filled with blood & c/d Corpus Hemorrhagicum (does not degenerate & ~~forms~~ transformed into corpus luteum).

- Blood clot is slowly replaced by serous fluid containing fibrin.

Structure → Granulosa cells & Theca interna cells are transformed into lutein cells, by process c/d luteinization

c/d Granulosa lutein & Theca lutein

↓
(lipid + yellow granules) ↓
(only lipid)

Functions

1. Secretion of Hormones

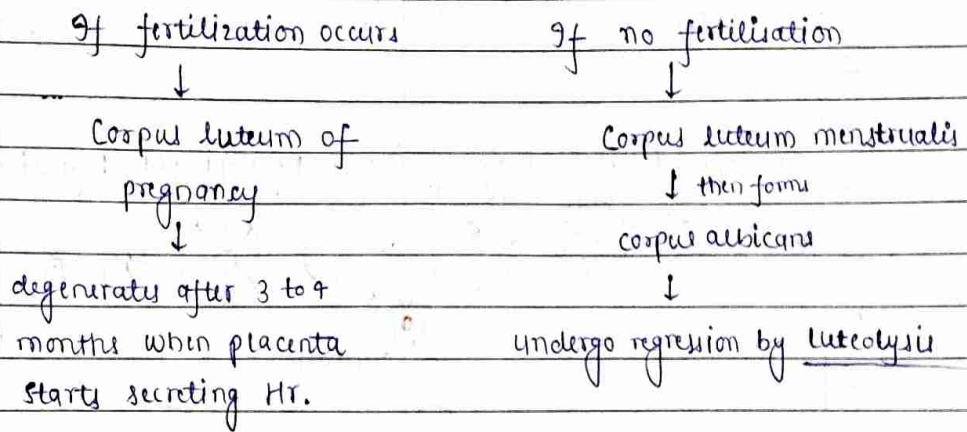
- Acts as temporary endocrine gland
- Release progesterone (large quantity)
 - Estrogen (small — " —)

2. Maintenance of Pregnancy

- If occurs, corpus luteum remains active until placenta develops.

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If abortion occurs, it becomes inactive / removed before 3rd month i.e., before placenta develops.

Fate → depends upon whether ovum is fertilised or not.



Hormonal Regulation of Menstrual Cycle

1. Hypothalamic H_r → GnRH

→ Triggers cyclic changes by stimulating secretion of FSH & LH from anterior pituitary.

2. Anterior Pituitary H_r → FSH & LH

→ Regulates ovarian & uterine changes by acting directly/indirectly via ovarian H_r.

FSH → stimulates the recruitment & growth of immature ovarian follicle.

LH → Triggers the ovulation & sustains corpus luteum.

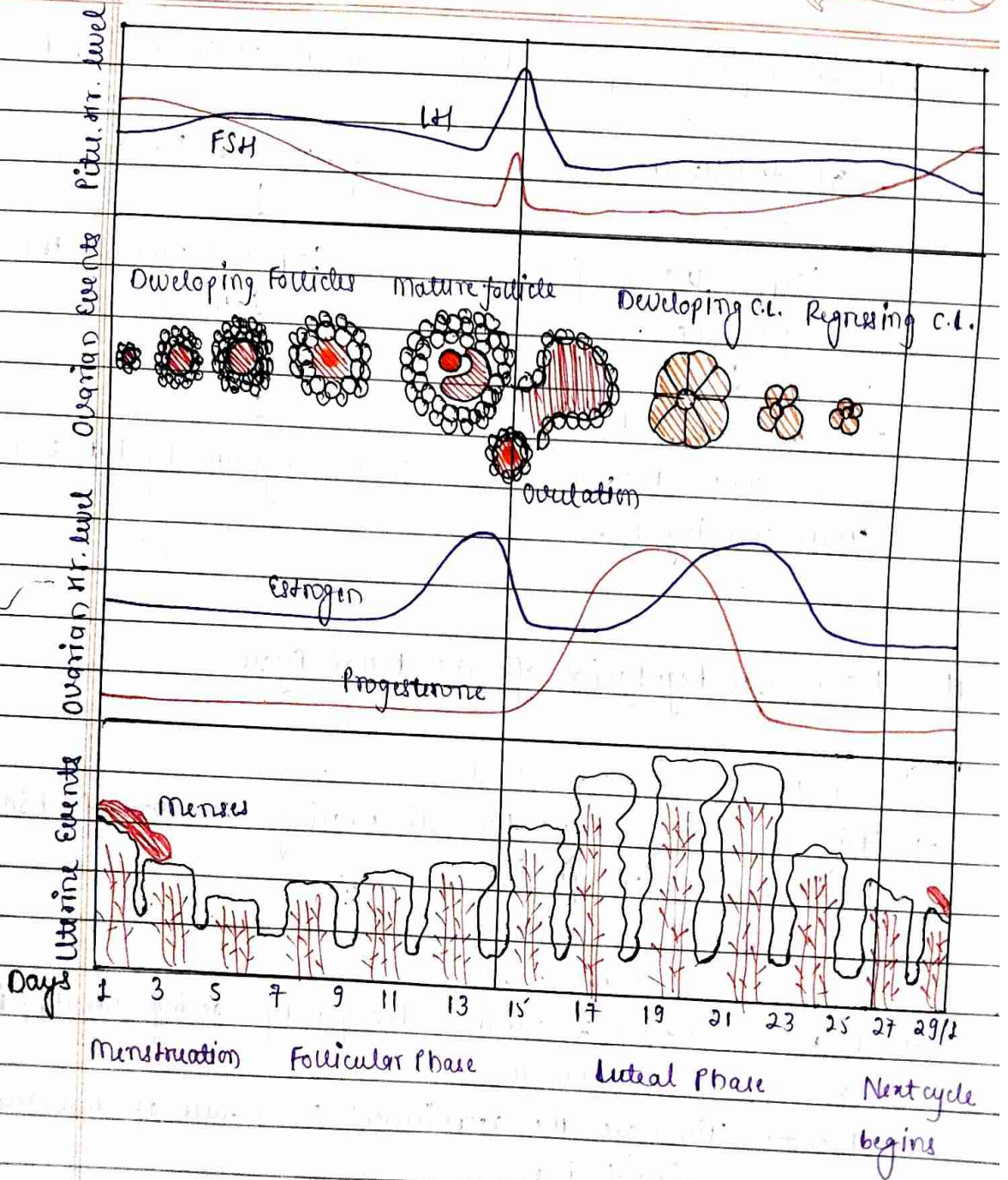
3. Ovarian H_r → Estrogen & Progesterone

→ Ovarian follicle → Estrogen

→ Corpus luteum → Progesterone

→ Estrogen secretion reaches peak twice in each cycle [Before ovulation & during luteal phase].

Progesterone → Ent during follicular phase but plays critical role during luteal phase.



⑥ OVULATION

- Process of release of ovum from ovary that results from rupture of graffian follicle, at end of follicular phase of menstrual cycle.
- Usually, occurs on 14th day in a 28 day cycle.
- If cycle length alters, occurs 14 days before the onset of menstrual bleeding as duration of luteal phase (which is 14 days) always remains const.

★ Process / Mechanism

Prior to ovulation, large amount of LH is secreted [LH-surge]

↓

this causes changes in graffian follicle & leads to ovulation

Stages:- Movement of graffian follicle to periphery of ovary

↓

form of new blood vessels in ovary

↓

Protrusion of blood vessels into follicular wall

↓

Fixed blood flow to follicle

↓

leakage of plasma into follicle

↓

Swelling of follicle

↓

Protrusion of follicle against ovarian capsule

↓

Form of stigma in capsule

↓

Activation of proteolytic enzymes by progesterone

weakening & rupture of
follicular capsule

Degeneration of
stigma

Oozing of follicular fluid

Release of ovum

Determination / Test of Ovulation

1. Rise in Basal Body Temperature
Temp rises by about 0.5°C

2. Fleeting lower abdominal pain

- With ovulation bleeding occurs into the antrum of follicle
- Small amount of blood also escapes in abdominal cavity

↓
causes fleeting lower abdominal pain

3. Vaginal Discharge (Spotting)

- there may be transitory rise in vaginal discharge during ovulation

4. Spinnbarkeit

- During ovulation uterine mucosa is thinnest & its elasticity is maximal (can be stretched as long as 10 cm or more)

↓
This elastic nature is called Spinnbarkeit

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Fern Test

2022/10

Due to estrogen, the cervical mucus before ovulation

forms an arborizing fern-like pattern, when spread on a slide.

6. Laproscopic observation

→ demonstrating ovum in the abdominal cavity by laparoscopy confirms ovulation.

7. Demonstration of LH peak

→ LH surge occurs just prior to ovulation.

Date _____
Page _____

↓

Maternal part of placenta → formed by Endometrium under-
-lining the chorion

4

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4. Integration function

Not secreted are :-

1. HCG (Human chorionic Gonadotropin)

→ Glycoprotein (chemical st. similar to that of LH).

Actions :-

- Responsible for prrenvation & secretory activity of corpus luteum

- Stimulates Leydig cells of fetal testes & causes secretion of testosterone

2. Estrogen → Similar to ovarian estrogen

Actions :-

- Causes enlargement of uterus

- ———— " ———— breast

- ———— " ———— female external genitalia .

- Relaxes pelvic ligaments & facilitates passage of fetus

3. Progesterone → Similar to ovarian progesterone

Actions :-

- Ties proliferation & development of endometrium

- Inhibits the contraction of muscles in uterus . so, prevents expulsion of fetus during pregnancy .

- Causes enlargement of breast & preparation of mammary gland for lactation .

4. Relaxin → secreted from corpus luteum, placenta & mammary gland (during labor)
→ polypeptide

5. Human Chorionic Somatomammotropin (HCS) / Human Placental lactogen (HPL)
→ Protein Hr.

→ Acts like Prolactin & Growth Hr.



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- Enhance growth of fetus by rising metabolic activities

- Causes anabolism of protein & its accumulation



in fetal tissue

- Reduces utilisation of glucose by mother leading large quantity of glucose to the fetus.
- mobilises fat from mother ~~from~~ adipose tissue
↓

so large amount FFA is available as source of energy which compensates the loss of glucose from mother.

⑧ PREGNANCY TEST

→ Test used to detect or confirm pregnancy

↓
Done to determine the presence or absence of hCG in urine of woman suspected.

* Immunological Test

→ Based on double antigen-antibody rxn

→ Commonly performed test is Gravidex test

Procedure Fresh urine sample of woman, is used for test

↓
One drop of urine + one drop of hCG-antiserum mixed

↓
T2 3h Now, one drop of latex particle is added & mixed

Result If hCG $\ominus$ → . all antibodies of antiserum are used up
↓
for agglutination.

Once of agglutination . when latex particle is added

↓
↓

woman is pregnant
↓
Not agglutinated b/c free antibody is not available.

If hCG $\ominus$ → just opp. to above

hCG Antiserum + urine

Agglutination

↓ + Latex Particle

No Agglutination

↓
Pregnancy -ve

↓
Pregnancy +ve

⑨ CONTRACEPTION

Methods

Temporary
↓

Permanent
↓

1. Barrier method

2. Natural contraception

3. IUDs

4. Steroidal contraception

Tubal
occlusion

↓
Vasectomy

★ Barrier method

1. In males → Condom is most widely used
↓

- prevents sperm to enter in vagina
- Biggest advantage → protection against STDs.

2. In ~~males~~ females → Diaphragm most commonly used
↓
Femucidal jellies are used along with it

• Another → Vaginal sponge

★ Natural Contraception

→ No device is used. So, chance of failure is high
→ following types :-

1. Periodic Abstinence

Based on fact that there will be no sexual intercourse during fertile period (10-17) b/c in this period ovulation occurs & chances of fertilisation are high.

2. Coitus interruptus / withdrawal

At the time of ejaculation penis removed from vagina to avoid insemination.

3. Temperature method

During ovulation temp of body rises by 1°C .

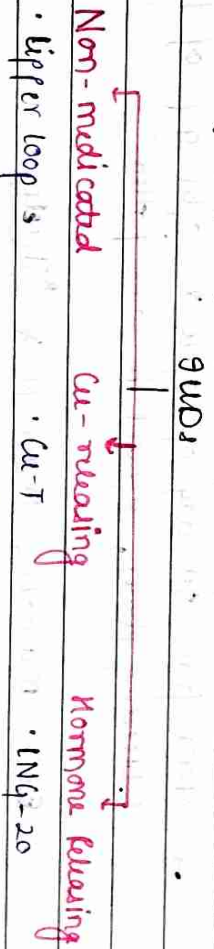
4. Lactational Amenorrhoea

Based on fact that there will be no menstrual cycle during lactation period of female.

* IUDs

→ These devices are insert inside uterus.

→ Two phagocytosis of sperm.



• Lipper loop

• Cu-T

• ING-20

• Multiload 375

• Progestant

Cu suppress movement of sperm and fertilisation capacity of sperm.

Progestone Mr. maki uterus invitable for implantation & the quantity of cervical mucus

* Implants

→ Progestone / Progestone + Estrogen combination placed below the skin.

→ Mode of action similar to pills.

* Oral Contraceptives



- Taken in the form of Pills / Tablets so there are commonly 1/4 as OCPs.
- Highly effective with least side effect
- contain Progesterone / Progesterone + estrogen combination
- Responsible for:-
 1. Prevent ovulation by +ve feedback
 2. Make uterus unsuitable for implantation
 3. Thicken cervix mucus
 4. Inhibit pituitary gonadotropin release

Preventing ovulation

- Taken daily for a period of 21 days
- starts from 5th day of cycle & after gap of 7 days (bleeding phase), repeated in same pattern.

Ex:- Mala-D, Mala-N & Shaeli

- contains Non-steroid preparation
- taken weekly
- blocks estrogen receptors on uterus & prevents implantation.

Side effects :- 1. CVS effect

Myocardial infarction, cerebral thrombosis, venous thrombosis & hypertension.

2. Carcinogenesis

(uncontrolled growth of cell)
Tied risk of cervical cancer & breast neoplasia

13. Metabolic side effects

- ↑ HDL & alter blood coagulability.
- cause glucose intolerance & insulin resistance

4. Miscellaneous

- includes cholestatic jaundice, breast tenderness, wt. gain & migraine.

* Postconceptional pills

Progesterone, Progesterone-estrogen combination or yuDs

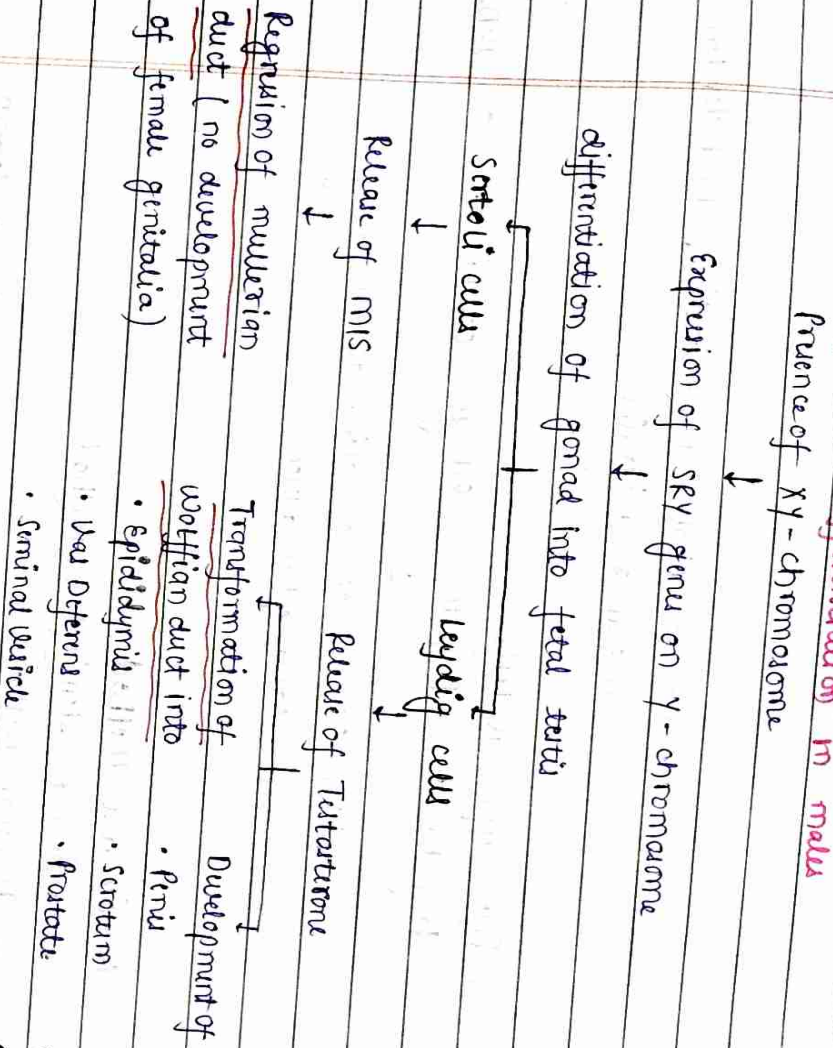
within 72 Hr. of copulation act as emergency contraceptive device.

* Surgical method

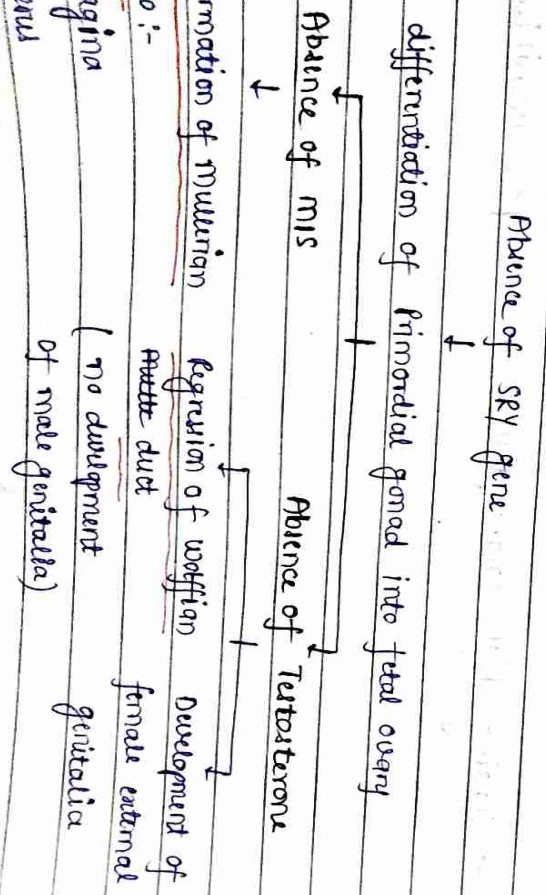
- also cld sterilisation method
- Terminal method
- Prevents gamete transport but not gamete formn
- In male → Vasectomy
- In female → Tubectomy
- Having high contraceptive value but poor reversibility

10. SEX DIFFERENTIATION & ABNORMALITIES

1. Mechanism of Sex Differentiation in males



2. Mechanism of Sex Differentiation in females



Fallopian Tube

* Abnormalities

1. Chromosomal

* Klinefelter Syndrome

- Most common ~~xx~~-chromosome abnormality
- also c/d Seminiferous tubule dysgenesis
- ~~Abnormality~~ of 3n this syndrome Triomy of x-chromosome occurs in male, i.e., the karyotype is 47XXY (44+XXY)
- Sex :- male
- Symptoms :- 1. Gynaecomastia
2. Enlarge mammary gland
3. Sterile

* Turner Syndrome

- also c/d Gonadal / ovarian dysgenesis
- 3n this syndrome, monosomy of x-chromosome occurs in female i.e., karyotype is 45XO (44+XO)
- Sex → Female
- Symptoms → 1. Sterile
2. Webbed Neck
3. Shield like chest

* Super-female

- 3n this syndrome triomy of x-chromosome occurs in female i.e., karyotype is XXX (44+XXX)
- Symptoms :- females are usually taller, heavier & stronger than normal females
- Quo sterile

* Testicular feminization Syndrome (TFS)

- Patient appears normal female externally.

- At puberty breast develops normally
- No development of uterus, gonads are testis with immature seminiferous tubule.

↓
∴ also cld TFS b/c testis are $\emptyset$ nt but spermatogenesis does not occur.

* Patient are genetic male (XY karyotype)

* True Hermaphroditism

- In this condn, both testis & ovaries are $\emptyset$ nt.
- Ovary are on one side & testis on opp. side.
- Karyotype is $46XX-XY$

2. Developmental

* Pseudohermaphroditism

- Individual with genetic constitution & gonads of one sex and external genitalia of other sex.

Female Pseudohermaphroditism

- Male external genital development occurs in genetic female
- Individual possess ∴ ovaries, oviduct but varying degree of masculine differentiation of external genitalia.

* Chromosomal sex is female

Male Pseudohermaphroditism

- Female external genitalia development in genetic male.
- Occurs due to defective testicular development.



II PARTURITION

- Expulsion / delivery of fetus from mother's body
- occurs at the end of pregnancy & the process by which delivery occurs is called labor.
- involves various activities such as :- 1. uterine contraction

2. Dilatation of cervix
3. opening of vaginal canal.

Fully Developed fetus

↓ due to stress

Fetal Cortisol

Mature Placenta

↓ Progesterone secretion ↓ Estrogen secretion

stimulates uterus to

secrete prostaglandins

Placenta rupture

↑ oxytocin release on uterine muscles

fetal ejection reflex

Constrict uterus

Constrict uterine muscles

oxytocin

Mother Pituitary

labor pain

Parturition is Neuro-endocrine Mechanism

→ Signal is given by fully developed fetus & mature placenta

causes mild contraction

↓
due to this a reflex, fetal ejection reflex originates

→ This reflex stimulates mother's pituitary to release oxytocin which causes constriction in myometrium.

↓
Due to the feedback of oxytocin

↓
level ↑ & gradually & causes further strong constriction

& ultimately leads to expulsion of fetus through birth canal.

* Stages

1. Dilation stage

- uterine contraction of labor starts.
- Vagina & Pubis symphysis relax due to relaxin H₂.
- Downward movement of fetus occurs & amnion rupture & amniotic fluid comes out of vagina.

2. Expulsion stage

- Expulsion of fetus occurs through birth canal
- Umbilical cord is cut & tied.
- Normal breathing of fetus starts.

3. After Birth

- Placenta as well as remaining part of uterus comes out through vagina.

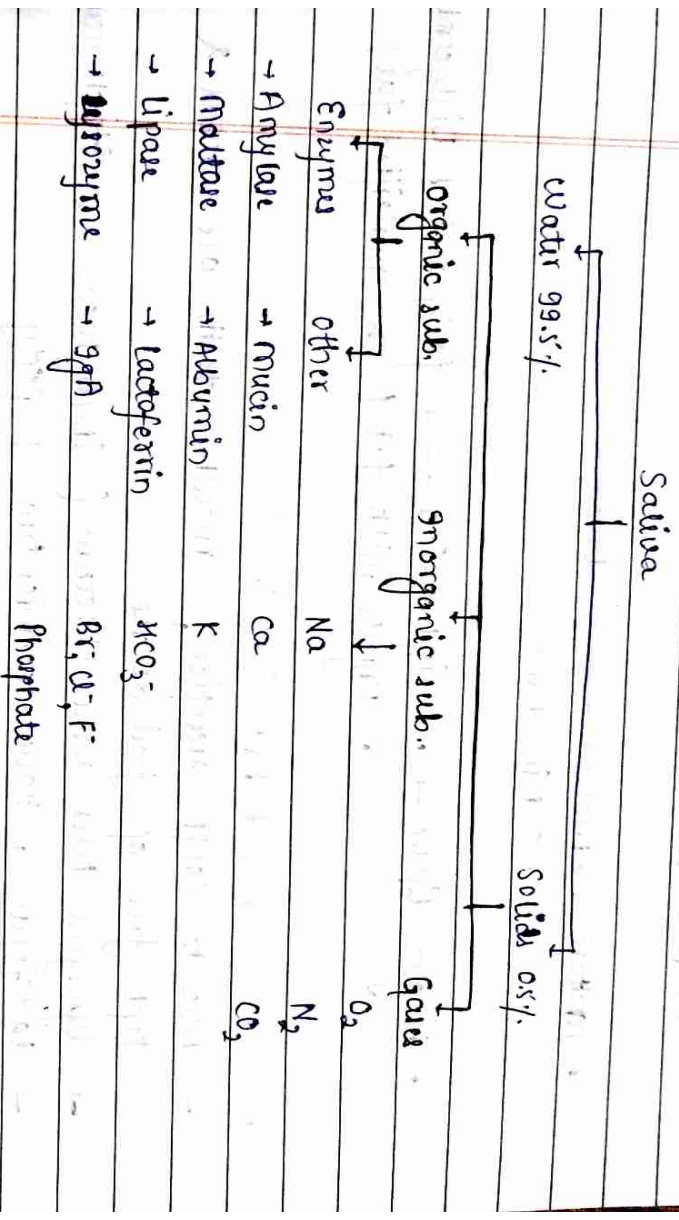
* **During Parturition, level of fetal cortisol, estrogen, proglandins, oxytocin & relaxin ↑.**

① SALIVA

★

Composition of Saliva

Contains 99.5% water & 0.5% solids.



★ Functions of Saliva

1. Preparation of food for Swallowing

- food is moistened and dissolved by saliva.
- By movement of tongue, moistened & masticated food is rolled into bolus.
- Mucin → lubricates the bolus and facilitates swallowing.

2. Appreciation of Taste

- Taste is chemical sensation.
- Saliva dissolves solid food, dissolved food stimulates taste buds that recognise the taste.

3. Digestive function

→ Has 3 digestive enzymes :-

- Salivary Amylase → cooked / amylase → Dextrin + Maltose
↓
Boiled starch
- optimum $pH = 6$
- cannot act on cellulose

- Maltase → Maltose → Glucose
- Not in trace

- Lingual lipase → Triglycerides → fatty acid + Diglycerol
- Digests milk fat (pre-emulsified fat)

4. Cleaning and Protective function

- Due to const. secretion, mouth & teeth are rinsed & kept free of food debris
- Lysozyme kills some bacteria (Streptococcus, Staphylococcus)
- Lactoferrin → have antimicrobial property
- Immunoglobulin (IgA) → have antibacterial & antiviral property.
- Protein rich protein → Neutralises toxin sub. (tanning)
- Protein rich protein + lactoferrin → stimulates enamel form

5. Role in Speech

- By moistening & lubricating soft parts of mouth & lips.

6. Excretory function

- Excretes both organic + inorganic sub.
- Excretes sub. like Hg , K , Pb & some viruses (Rabies & Mumps)
- In some pathological condn, excretes sub. which are not found in normal condn

Ex :- Glucose → in diabetes mellitus
Urea → in nephritis
Calcium → in Hyperparathyroidism

7. Regulation of Body Temperature

→ In dogs & cattle, excessive dripping during panting help in loss of heat & regulation of Temp.
→ In humans, no role in temp regulation.

8. Regulation of Water Balance

→ When body water content ↓ → salivation ↑.
↓
causes dryness of mouth, and induces thirst
↓
water is taken, thirst quenched & restore body water

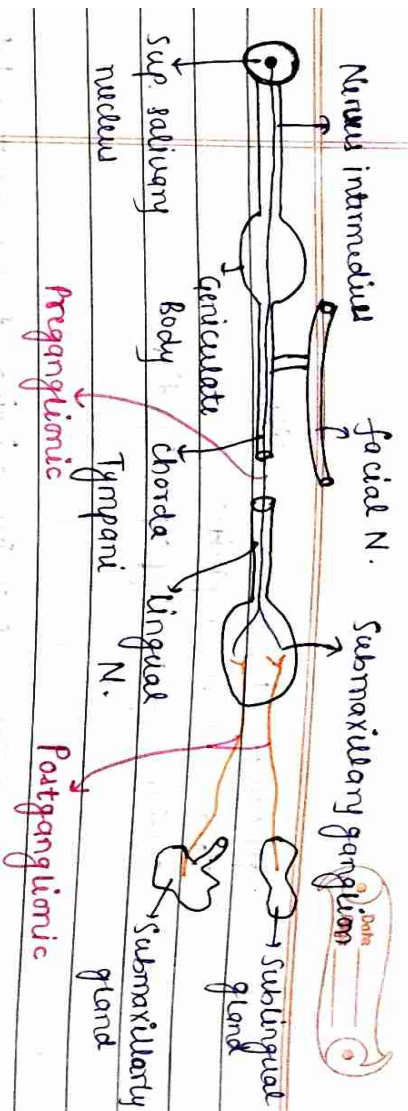
* Regulation of Salivary Secretion

Regulated only by Nervous mechanism

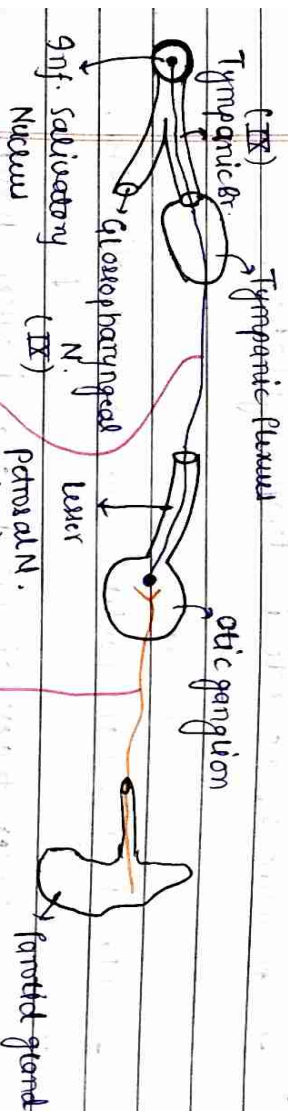
Nerve Supply → Supplied by both sympathetic + parasympathetic

• Parasympathetic

→ stimulation causes secretion of salivary amount of organ
↓
b/c activates acinar cells & dilate blood vessels of gland
→ NT is Acetylcholine



Parasympathetic N. supply to sublingual & submandibular



Parasympathetic N. supply to parotid

• Sympathetic fibers

- Preganglionic fibers arise from lateral horn of 1st & 2nd thoracic segment of spinal cord.

leave the cord through anterior root & ends in superior cervical ganglion of sympathetic chain

Postganglionic fibers arise from this ganglion & distribute to salivary gland.

→ Function → • causes secretion of saliva

- thick & rich in organic sub. such as mucus
- activates acinar cells & causes vasoconstriction
- NT is Nor-adrenalin

★ Reflex Regulation of Salivary Secretion

→ Regulated by Nervous mechanism through reflex action

1. Unconditional reflex

- Inborn but since birth
- Not need any previous experience
- Induce salivation when any sub. is present in mouth.

2. Conditional reflex

- Acquire by experience & need previous experience
- Presence of food is not necessary to elicit this reflex.
- stimuli are sight, smell, hearing or thought of food.

★ Effects of Drugs & Chemicals

1. Sub. which Tns

- Sympathomimetic drugs → Adrenaline & Ephedrine
- Parasympathomimetic drugs → Acetylcholine, muscarine
- Histamine

2. Sub. which Tns

- Sympathetic depressant → ergotamine & dibenamine
- Para ——— " ——— → Atropine & Scopolamine
- Anaesthetic → chloroform & ether (stimulate secretion)
- However deep anaesthesia Tns the secretion due to central inhibition

② STOMACH

* Phase of Gastric Secretion

- Occurs in 3 phases :-
1. Cephalic
 2. Gastric
 3. Intestinal
- In human, 4th phase i.e. interdigestive phase exists.
- Each phase regulated by nervous or hormonal mechanism or both.

or Cephalic Phase

- Stimuli arise from the head region (cephalus).
- Regulated by Nervous mechanism.
- Gastric juice secreted is called Appetite juice.
- Secretion occurs even without the presence of food in stomach.
- During this phase, 30% of total gastric juice is secreted.

(quantity less, rich in enzymes & HCl)

• Unconditional Reflex

- Inborn reflex, food placed in mouth salivation induced & simultaneously gastric secretion also occurs.

Stages :- Presence of food in mouth

↓
Stimulation of taste buds & receptors

↓
Afferent impulses pass via nerve fibres of glossopharyngeal & facial nerves to amygdala & appetite centre in hypothalamus

Then, efferent impulses pass through dorsal nucleus of vagus to the wall of stomach

↓
Vagal efferents secrete acetylcholine, which stimulates gastric secretion

- Conditional Reflex
- Acquired by previous experience.
- Presence of food is not necessary to elicit this reflex.
- Sight, smell, hearing or thought of food induces salivation and gastric secretion.

• Stages :- Impulses from special sensory organs (ear, nose & eye) pass through efferents of neural circuit

↓
To cerebral cortex

↓
Then, efferent impulses pass through dorsal nucleus of vagus & reach the stomach wall

↓
Vagal endings secrete acetylcholine, which stimulates gastric secretions.

b) Gastric Phase

- Secretion of food juice when food enters the stomach.
- Regulated by both Nervous & Hormonal mechanism.
- During this phase, 60% of total amount of gastric juice is secreted.

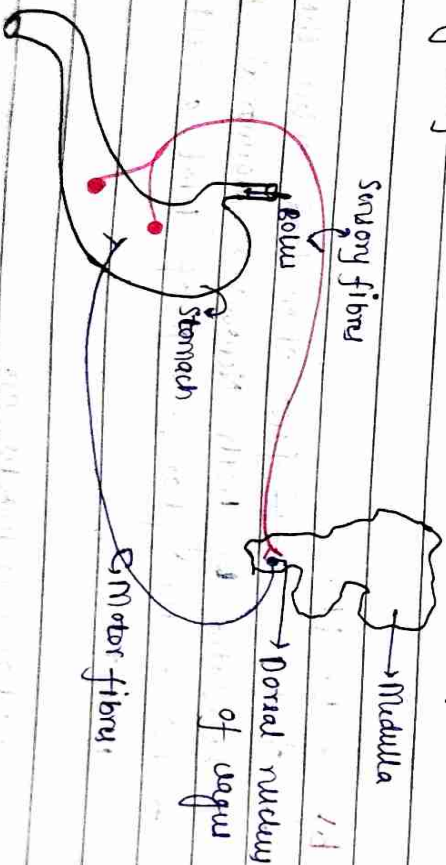
• Mechanisms involve are

1. Stimuli which initiate these 2 reflexes are :-
 - i) Distention of stomach
 - ii) Mechanical stimulation of gastric mucosa by bulk of food.
 - iii) Chemical stimulation by content of food.

Nervous Mechanism

1. Local myenteric reflex → • Elicited by stimulation of myenteric nerve plexus which are local nerve plexus present in wall of stomach:
↓
releases acetylcholine which stimulates gastric glands to secrete gastric juice.

2. Vagovagal reflex → • Involves both afferent & efferent vagal fibres.
↓
• Sensory impulses are transmitted by sensory fibres of vagus
↓
dorsal nucleus of vagus sends efferent (motor) fibres back to the stomach & causes gastric secretion.
• Both afferent & efferent impulses pass via vagus so called vagovagal reflex.



• Hormonal Mechanism (Gastrin)

- Gastrin is a GSI HT. secreted by G-cells which are present in pyloric gland of stomach.
- Released when food enters stomach.
- Mechanism involves in release may be nervous reflex or vagovagal reflex.
- Gastrin stimulates the secretion of pepsinogen & HCl.

• Intestinal phase

- Secretion of gastric juice when chyme enters the intestine.
- Regulated by both Nervous & hormonal control.
- During this phase, only 10% of gastric juice is secreted.

Initial phase → chyme that enters stimulates the duodenal mucosa to release gastrin.



then it transports to stomach via blood & GI gastric secretion.

Later stage → After initial phase, there is the or complete stage of secretion.

- Inhibited by 2 factors :-
 1. Enterogastric
 2. GI hormones

Enterogastric → • Inhibits gastric secretion & motility.

- Occurs due to distention of intestinal mucosa by chyme.
- Mediated by myenteric nerve (Auerbach) plexus.

GI Hormones → • Presence of chyme stimulates secretion of many GI HT. & all these inhibit gastric secretion & gastric motility.



→ G₉ hr. which inhibits gastric secretion are :-

1. Secretin
2. Cholecystokinin
3. Gastric Inhibitory Peptide (GIP)
4. Vasoactive Intestinal Polypeptide (VIP)
5. Peptide YY

→ On addition to these **pancreas** also secretes hr. called **somatostatin** (inhibits gastric secretion)

4. Interdigestive Phase

→ Secretion of small amount of juice in b/w the meals

→ Secretion is mainly due to gastrin.

* Factors influencing gastric secretion

- The gastric secretion is stimulated by stimulating gastric mucosa
- Factors are :-
- Alcohol
 - Caffeine

1. Cephalic Phase :- Only Nervous

• **Conditional reflex** :- Sight, Smell, Thought of food

Unconditional reflex :- Presence of food in mouth

→ **Vagus Nerve** — stimulation —→ Secretion of gastric juice

2. Gastric Phase :- Nervous & Hormonal

• Bolus in stomach

→ **Vagus Nerve**
Local Nerve
Gastrin — stimulation —→ Secretion of gastric juice

3. Intestinal Phase :- Mostly Hormonal

• Chyme in intestine

→ **Gastrin**

— stimulation —→

Secretin
CCK-PZ
Somatostatin
GIP
VIP

— inhibition —→

→ Secretion of gastric juice

★ Methods of Study

1. Experiment to study cephalic phase.

→ **Sham feeding** → in this a dog is taken as experimental animal.

• fistula is made in oesophagus of dog, when animal eat food comes out of neck through fistula (not allowed to reach stomach)

• At same time, gastric juice is collected from stomach by placing a cannula & then juice is analysed.

• Then, bilateral vagotomy is performed & gastric juice is collected after vagotomy.

• Vagotomy abolish gastric secretion during cephalic phase which proves that this phase is vagally mediated.

2. Experiment to study gastric phase

→ studied by making 5 diff types of pouches;

→ Pouches pouch, Heidenhain pouch, Bidel pouch, Farrell pouch & soy pouch.

→ Not performed now-a-days, only for academic importance

• Pouches pouch → small pouch separated from main body of stomach having intact blood & nerve supply
↓

So, help to study both neural & chemical factors of secretion

• Heidenhain pouch → denervated pouch, helps to study influence of neural factors on secretion.

★ SECRETION OF HCL

- HCl secretion is an active process that take place in canaliculi of parietal cells in gastric glands.
- Energy is derived from oxin of glucose.
- CO_2 is derived from metabolic activities of parietal cells & blood.
- $CO_2 + H_2O \xrightarrow{CA} H_2CO_3$
- $H_2CO_3 \rightarrow$ most unstable compound & immediately splits into H^+ & HCO_3^- .
- $H^+ \rightarrow$ actively pumped into canaliculus of parietal cell.
- At same time, Cl^- is also pumped into canaliculus actively.

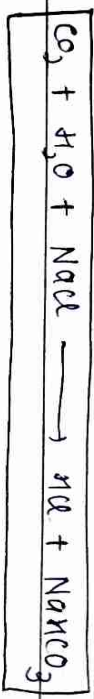
derived from NaCl from blood

Now,

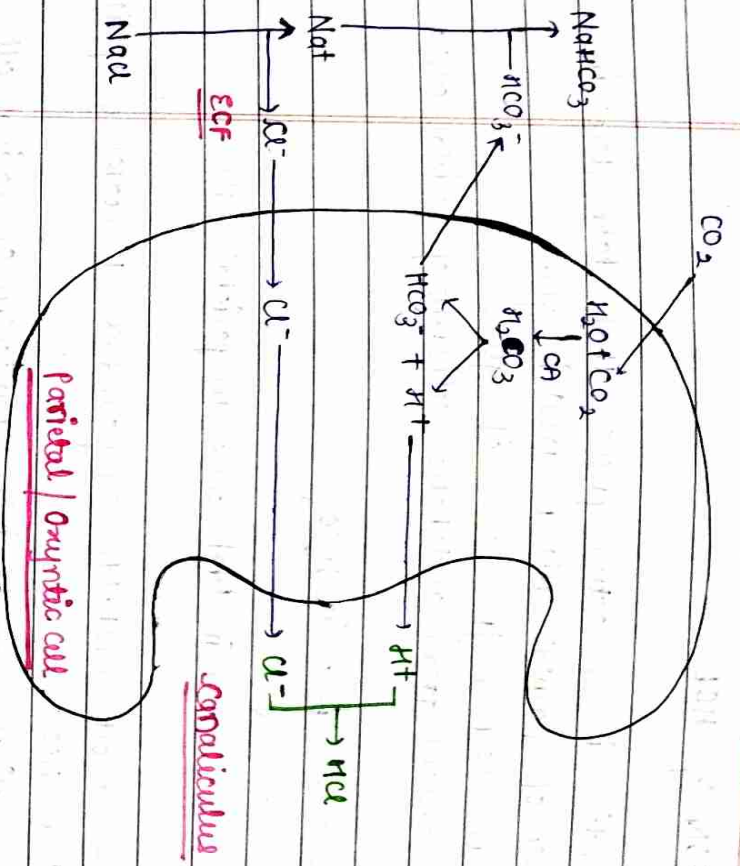


- To compensate loss of Cl^- , HCO_3^- from parietal cell enter the blood & combining with Na^+ to form $NaHCO_3$.

Summary:-



- Factors stimulating secretion
Gastrin, Histamine, Vagal stimulation.
- Factors inhibiting secretion
Secretin, GIP & Peptide YY



Mechanism of Secretion of HCl

★ Bacterial flora of large intestine synthesizes B₉, B₁₂ & Vit. C.

↓
By this contributes in erythropoietic activity & blood clotting.



★★ PEPTIC ULCER

24/5/2021

- Ulcer means erosion of surface of any organ due to shedding or sloughing of necrotic (dead) tissue that lines the organ.
- Peptic ulcer means ulcer in the wall of stomach or duodenum, caused by action of HCl gastric juice.
- It found in stomach c'd Gastric ulcer
 - " — in duodenum c'd duodenal ulcer

Cause :- 1. ↑ peptic activity due to ↑ secretion of pepsin.

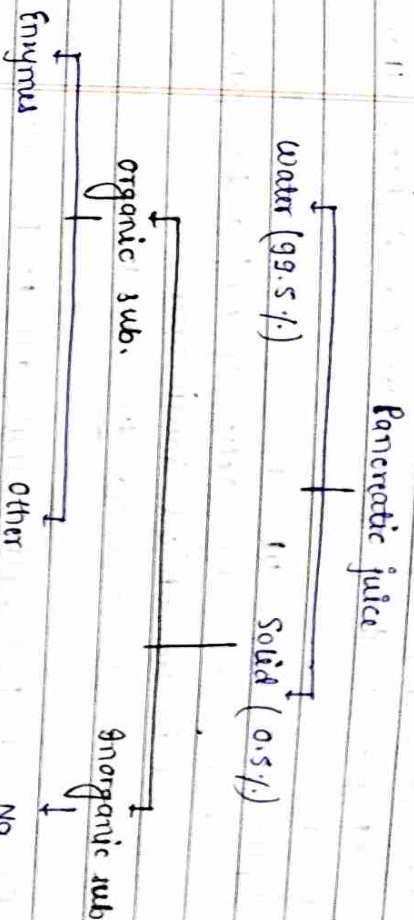
2. ↑ hyperacidity of gastric juice.
3. ↓ alkalinity of duodenal content
4. ↓ mucin content in gastric juice
5. ↑ Inflammation due to Helicobacter pylori.

Features :- 1. Severe burning pain in epigastric region (most common).

2. Nausea
3. Vomiting
4. Haematemesis (vomiting blood)
5. Heartburn
6. Loss of appetite (Anorexia)
7. Loss of wt.

③ PANCREAS

★ Composition



Proteolytic

lipolytic

→ Trypsin

→ Pancreatic

→ Chymotrypsin

lipase

→ Carboxypeptidase

→ Phospholipase A

→ Nuclease

→ Phospholipase B

→ Elastase

→ 'Bile salt - activated lipase

Amylolytic → Pancreatic Amylase

★ Functions

1. **Digestion of proteins** function of pancreatic juice.
 - Major role in digestion of protein & lipid.
 - mild action on carbohydrates.

2. Digestion of protein

Major enzymes → Trypsin & chymotrypsin
 others → Carboxypeptidase, nuclease, elastase & collagenase

• Trypsin →

Trypsinogen (inactive) $\xrightarrow{\text{Enterokinase}}$ Trypsin (active)

secreted by brush border cells

→ One activated, then get itself activated by means of autocatalytic or autoactive action

Action :- 1. Digestion of Protein

Protein $\longrightarrow$ Protease + Polypeptides

2. Curdling of milk

Casimogen $\longrightarrow$ Casein

3. Accelerate blood clotting

4. Activates other enzymes also

5. Autocatalytic action

Trypsin inhibitor $\longrightarrow$ • Activation in pancreas is inhibited by an inhibitor protein called Trypsin inhibitor

• If get activated in pancreas will damage pancreatic tissue protein

• Chymotrypsin $\longrightarrow$ Secreted as inactive chymotrypsinogen

↓
Trypsin

Active chymotrypsin

Action :- 1. Digestion of Protein

Protein $\longrightarrow$ Polypeptides

2. Digestion of milk

• Carboxypeptidase $\rightarrow$ Both A & B are activated by trypsin.

Action :- • Exopeptidase (break terminal bond of protein)
• Break protein $\rightarrow$ Amino Acids

• Nuclease $\rightarrow$ • are Ribonuclease & Deoxyribonuclease
 $\downarrow$
responsible for digestion of nucleic acid.

• Convert RNA & DNA $\rightarrow$ Mononucleotides

• Elastase $\rightarrow$ • pro-elastase trypsin, elastase
• digest collagen

3. Digestion of lipid

• Pancreatic lipase $\rightarrow$ Triglycerides $\rightarrow$ monoglycerides + fatty acids
 $\downarrow$

$\rightarrow$ Activity is accelerated in presence of bile.

$\rightarrow$ pH optimum is 7 to 9

$\rightarrow$ Require 2 factors :- Bile salt $\rightarrow$ for emulsification of fat
Colipase $\rightarrow$ coenzyme for lipase
 $\rightarrow$ Deficiency causes steatorrhea (undigested fat in feces)

• Cholesterol ester hydrolase
cholesterol $\rightarrow$ free cholesterol + fatty acid

• Phospholipase A
digest phospholipids lecithin & cephalin

$\downarrow$
lycogenin lysocephalin $\rightarrow$ convert

• Phospholipase B

lysophospholipids $\longrightarrow$ Phosphoryl choline + free F.A.
(lysophosphatidylcholine + lysophosphatidylglycerol)

• Colipase $\rightarrow$ facilitates action of pancreatic lipase.

4. Digestion of carbohydrates

Pancreatic Amylase convert, starch into Maltose + Dextrin

5. Neutralizing Action of Pancreatic Juice

- $\rightarrow$ large quantity of HCO_3^- makes pancreatic juice alkaline
- $\rightarrow$ Alkaline juice ~~makes~~ neutralises acidity of chyme & protect intestine from destructive action of acid in chyme.

★ Regulation of Secretion

1. Cephalic Phase

- $\rightarrow$ By nervous mechanism
- $\rightarrow$ 30% of total amount is secreted.
- $\rightarrow$ Same as that of gastric juice regulation

2. Gastric Phase

- $\rightarrow$ Under hormonal control (Gastrin H.P.)
- $\rightarrow$ 80% of total amount is secreted.
- $\rightarrow$ Juice rich in enzymes

3. Intestinal Phase

- $\rightarrow$ Under hormonal control
- $\rightarrow$ 70% of total amount is secreted.

Hormones stimulating secretion

1. Secretin
 2. Cholecystekinin
- (demo to action of after fat)

Hormones inhibiting secretion

1. Pancreatic Polypeptide (PP) → secreted by PP cells
2. Somatostatin → secreted by D cell
3. Peptide YY → by intestinal mucosa
4. Peptides like ghrelin & leptin

① Cephalic Phase :- Nervous

Conditional Reflex → sight, smell, thought of food

Unconditional Reflex → Presence of food

→ Vagus N. → secretion of P. juice

② Gastric Phase :- Hormonal

Bowls in stomach

Gastric secretion

→ Gastrin →

③ Intestinal Phase :- Hormonal

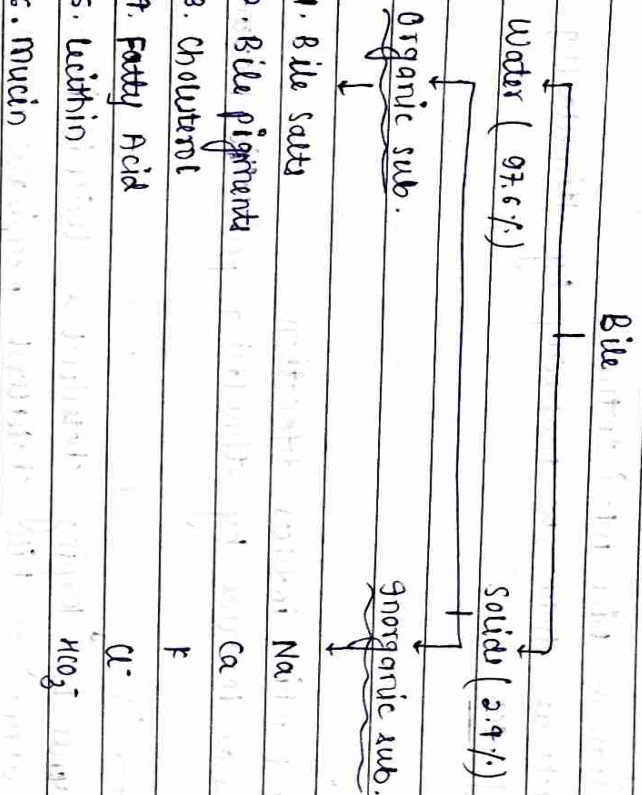
Chyme in intestine

→ Secretin → Rich in HCl

→ CCF-P2 → Rich in enzymes

④ LIVER & GALL BLADDER

* Bile composition



* Function of Bile

1. Emulsification of fat

→ Emulsification → process by which fat globules are broken down into minute droplets & made in form of milky fluid cld emulsion.

→ Bile emulsify the fat by reducing the surface tension due to their detergent action.

→ Emulsification needs the presence of lecithin

2. Absorption of fat

→ Bile combine with fat & make complexes cld micelles

→ Fat in form of micelle can be absorbed easily.

3. Choleretic Action

→ Stimulate secretion of bile from liver.

4. Cholagogue Action

- agent which causes contraction of gall bladder & release of bile into intestine
- acts as cholagogue indirectly by stimulating secretion of CK.

5. Laxative Action

- agent which induces defecation
- acts as laxative by stimulating peristaltic movements.

6. Prevention of Gallstone formation

- prevents by keeping cholesterol & lecithin in soln.
- in absence of bile cholesterol precipitates with lecithin & forms gallstone.

7. Excretory Function

- Bile Pigments → major excretory product of Bile.
- others :- 1. heavy metals (Cu & Fe)

2. Typhoid bacteria

3. Toxins

4. Cholesterol

5. Lecithin

6. Alkaline phosphatase

8. Antiseptic Action

inhibits growth of bacteria by its natural detergent action.

9. Lubrication Function

Mucin in bile salt acts as lubricant for chyme



10. Maintenance of pH in G.I.T

- Bile is highly alkaline, **it** neutralizes the chyme. Thus an optimum pH is maintained for action of digestive enzymes.

* Functions of Liver

- Liver is largest gland & one of vital organs of the body.

1. Metabolic Function occurs in liver

- Maxm metabolic rfn, such as metabolism of carbohydrate, proteins, fats, vitamins & many hr.

2. Storage Function

- Glycogen, A.A., Iron, Folic Acid (B_9), Vit. A, B_{12} & D are stored in liver.

3. Synthetic Function

- Produces glucose by gluconeogenesis.
- It synthesise all plasma proteins & other proteins (except antibody) such as clotting factors & hr.-Binding proteins.
- Also synthesise steroids, somatomedin & thyroxin.

4. Secretion of Bile

- Secrete bile which contains bile salts, bile pigments, cholesterol, fatty acids & lecithin.

5. Excretory function

Excretes cholesterol, Bile pigments, heavy metals, toxins bacteria & virus (yellow fever)

6. Heat Production

- Enormous amount of heat is produced b/c of metabolic rxn.

7. Hematopoietic Function

- In fetus → produces blood cells.
- Stores Vit B₁₂ (necessary for erythropoiesis) from (necessary for synthesis of hb).
- Produces Thrombopoietin → promotes production of Thrombocytes

8. Hemolytic function.

- RBC are destroyed by Reticuloendothelial cells (buffer cells) of liver.

9. Inactivation of Hormones & Drugs

- Catabolism of drugs such as Gl, PTX, Cortisol, insulin, Glucagon & estrogen.
- Also inactivates drugs particularly fat soluble.

10. Defense and Detoxification function

- buffer cells plays imp. role in defense.

- perform phagocytosis of foreign bodies
- produces IL & TNF (tumour necrosis factor)

activate immunity.

- Detoxification occurs in 2 ways :-

- Total destruction → by degradation
- Conversion of toxic sub → into non-toxic material.

Jaundice → **BioC.**

⑤ MOVEMENTS OF GIT

* DEGLUTITION

→ Process by which food moves from mouth to stomach.

Stages :-

1. Oral stage → food moves from mouth → pharynx
2. Pharyngeal stage → " " pharynx → oesophagus
3. Oesophageal stage → " " oesophagus → stomach

1. Oral stage / 1st stage

- Voluntary stage.
- Bolus from mouth passes to pharynx.

→ Sequence of events

Bolus placed on postero-dorsal surface of tongue (preparatory

Ant. part of tongue retracted & depressed

(position)

Post. " " " " elevated & retracted

This pushes bolus backward into pharynx

forceful contraction of tongue against the palate
also pushes food into pharynx.

2. Pharyngeal stage / 2nd stage

- Involuntary stage.
- Bolus from pharynx passes into oesophagus
- Since, pharynx communicate with mouth, nose, larynx & oesophagus. So bolus from mouth can enter into 4 parts :-

1. Back in mouth
2. Upward in nasopharynx
3. Forward in larynx
4. Downward in esophagus.

→ Entrance is presented as follows :-

- Back in mouth :- By position of tongue against soft palate
- By intraoral pr. developed by movement of tongue.

- Upward in Nasopharynx :- By elevation of soft palate along with its extension i.e. uvula.

- Forward in larynx :- By forward & upward movement of larynx.

- By backward movement of epiglottis to seal glottis.

Deglutition Apnea → Temporary arrest of breathing during pharyngeal stage.

- Entrance in Esophagus → Since other 3 paths are closed, bolus has to pass through esophagus.

→ Occurs by :-

- upward movement of larynx
- upper 3 to 4 cm of esophagus relax
- peristaltic contraction starts in pharynx

→ whole process occurs in 1 to 2 sec & is purely involuntary

3. Esophageal stage / 3rd stage

- Involuntary stage
- Food from oesophagus passes to stomach
- Movement of oesophagus for movement of bolus is called Peristaltic waves.
- Peristalsis → means wave of contraction followed by wave of relaxation, which travel in aboral dirⁿ (away from mouth).
- 2 types of contraction are produced in esophagus:-

1° peristaltic contraction → occurs when bolus reaches upper part of esophagus.

• Initially the pr. is negative due to stretching of lower esophagus but after becomes true & prs upto 10 to 15 cm $\frac{1}{2}$ 0

2° peristaltic contraction → If 1° is unable to propel then 2° appears & pushes the bolus into stomach.

• Induced by distention of upper esophagus by bolus.

• After origin prs down like 1°

→ Role of lower Esophageal sphincter

- distal 2 to 5 cm of Esophagus acts like sphincter & remains contracted always.
- when bolus enters it relaxes and after the entry it contracts & closes the lower end of esophagus.



★ PERISTALSIS (Movement of stomach)

- when food enters stomach, the peristaltic contraction appears.
- starts from lower part of body of stomach, passes through pylorus till pyloric sphincter.
- initially the contraction appears as light / slight indentation & becomes deeper while travelling.
- Finally ends with constriction of pyloric sphincter.
- Each peristaltic wave takes about 1 min. to travel from point of origin to point of ending.
- This is called digestive peristalsis b/c it is responsible for grinding of food particles & mixing them with gastric juice for digestion.

★ Movements of small intestine

- Are of 4 types :-
 1. Mixing Movement
 1. Segmentation
 2. Pendular

by Propulsive Movement

1. Peristaltic movement
2. Peristaltic rush

or Peristalsis in fasting

- migrating motor complex

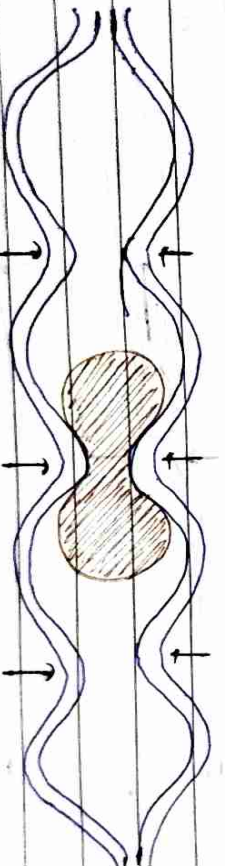
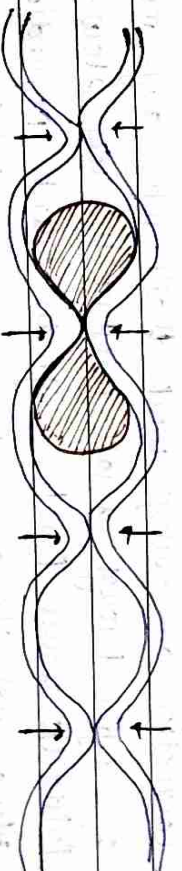
or Movement of villi

if * Segmentation Contraction

- Common type of movement of small intestine, which occurs regularly or irregularly but in a rhythmic fashion.
- also called Rhythmic Segmentation contraction
- occurs at regular spaced intervals along a section of intestine.
- Segment of intestine in b/w the contracted segment is relaxed.
- length of relaxed seg. is same as that of contracted.
- These alternate segments of contraction & relaxation give appearance of rings.
- After some time, the contracted segments are relaxed & relaxed seg. are contracted.

∴ Segmentation contraction chop the chyme many times.

This helps in mixing of chyme with juices



ii) Pendular Movement

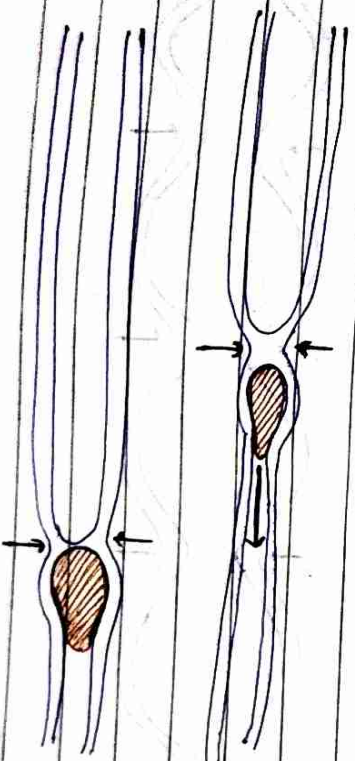
- Sweeping movement, small portion of intestine sweep forward & backward / upward and ~~down~~ downward.

is ~~is~~ Peristaltic Movement

- defined as wave of contraction followed by wave of relaxation.
- Always travel in aboral direction.
- initiated by contraction of smooth muscles.
- Travels from point of stimulation in both direction.
- But normally, contraction in oral direction is inhibited quickly & disappears.

Starting law of intestine

- Acc to law, response of intestine for local stimulus consist of contraction above and relaxation below the stimulated area.
- Contraction starts at any point and travels towards the end (anal end).
- Contraction are always weak that's why avg. movement of chyme through small intestine is very slow.
- Peristaltic waves Try to a greater extent immediately after a meal b/c of Gastroenteric Reflex (initiated by distension of stomach).



ii) Peristaltic Rush

- Powerful peristaltic contraction of small intestine.
- caused by excessive irritation of intestinal mucosa or extreme distension of intestine.
- It begins in duodenum & passes through entire small intestine & reaches caecocolic valve within few minutes, this is c/d Peristaltic Rush / Rush waves.
- It sweeps the content of intestine into the colon & relieves it off either from irritant or excessive distension.

*** Movement of large intestine

Defecation → • voiding of feces
• feces → formed in large intestine & stored in sigmoid colon.

- Defecation Reflex → • defecation occurs when some feces enters the rectum due to mass movement.
- usually defecation occurs when intrarectal pr. fcs by about 20 to 25 cm H_2O .
 - usually stimulus is → tea / coffee / water.

Act of Defecation → • preceded by voluntary efforts like assuming an appropriate posture, relaxation of ext. sphincter & compression of abdominal content by contraction of abdominal muscles.

- process involve contraction of rectum & relaxation of internal & ext. anal sphincter.

Int. Anal sphincter → • made of smooth muscles
• innervated by ^{para} sympathetic fibres
via pelvic nerve

ext. Anal sphincter → • made of skeletal muscles

• innervated by somatic N. fibres
which pass through pudendal Nerve.

always keeps sphincter closed &
can relax only when it is inhibited

Gastrocolic Reflex → Contraction of Rectum followed
by diarrhoea for defecation caused by distension of stomach
by food & mediated by intrinsic N. fibres of GI tract.
→ causes only a weak contraction of rectum but initiates
defecation reflex.

Pathway for Defecation Reflex

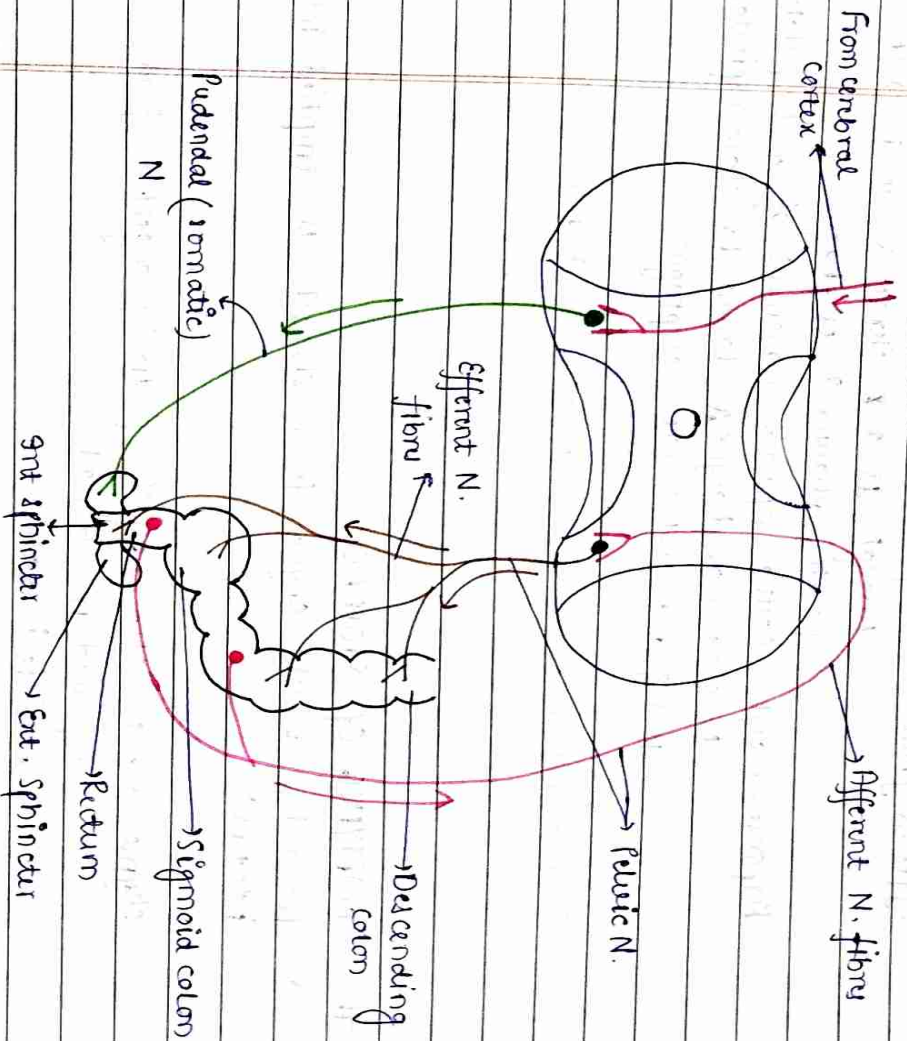
Rectum is distended due to entry of feces by mass
movement

↓
Sensory Nerve endings are stimulated

↓
Impulses are transmitted via afferent fibres of
pelvic n. to defecation center situated in sacral seg.
of spinal cord.

↓
Motor impulses from centre via efferent fibres of
pelvic n. cause strong contraction of descending colon,
sigmoid colon & rectum & relaxation of
internal sphincter

Simultaneously, voluntary relaxation of ext. sphincter occurs due to inhibition of pudendal Nerve, by impulse from cerebral cortex.



DEFECATION REFLEX

- Initially defecation is voluntary act, later becomes involuntary & is carried out by reflex action c/d Defecation reflex
- occurs during pharyngeal & esophageal stages.

Stimulus → Receptor of oropharyngeal region.

Afferent fibres → Impulse from oropharyngeal receptors pass via glossopharyngeal N. to defecation centre.

Centre → Deglutition centre is at floor of 9th ventricle in medulla.

Efferent fibres → Impulses from deglutition centre travel through IX & X Nerve & reach soft palate, pharynx & oesophagus.

Response → reflex causes upward movement of soft palate to close nasopharynx & larynx to close respiratory passage, so bolus enters oesophagus.

Swallowing of hard & solid food in babies below 6 months is prevented by gag reflex.

EMPTYING OF STOMACH

→ Process by which chyme from stomach is emptied into intestine.

Chyme → semi-solid mass of partially digested food

- formed in stomach
- acidic in nature
- it takes about 3 to 4 hr. for emptying of chyme
- slow emptying facilitates final digestion & maxm absorption of digested food from small intestine

→ Gastric emptying occurs due to peristaltic waves in body and pyloric part & simultaneous relaxation of pyloric sphincter.

* Factor Affecting

1. Vol. of gastric content
2. Consistency of gastric content
3. Chemical composition

4. Pn of gastric content
5. Osmolar conc of gastric content.

* Regulation of Gastric Emptying
→ Regulated by Nervous & hormonal factor.

1. Nervous factors
Regulated by Entero gastric reflex

inhibits gastric emptying

Mechanism :- 1. Presence of chyme generates impulses in duodenum which inhibits emptying in stomach
2. Some impulses from duodenum travel through afferent vagal fibres to brainstem & thereby inhibit emptying
[normally stimulates emptying]

2. Hormonal factors

chyme enters duodenum

↓

duodenum mucosa releases hr. which enter stomach &

inhibits motility of stomach.

hr. inhibiting gastric motility & emptying

1. Vasovactive intestinal peptide (VIP)
2. Gastric inhibitory peptide (GIP)
3. Secretin

4. CCK

5. Somatostatin

6. Peptide YY

⑥ GASTROINTESTINAL HORMONES

* GASTRIN

- Secreted mainly by **G-cells** of **pyloric gland** of stomach.
- also secreted by **T_g cells** in stomach, duodenum & jejunum.
- Secreted from stomach → during gastric phase
intestine → during intestinal phase

→ Stimulants for secretion

1. Presence of food in stomach.
2. Stimulation of local nervous plexuses
3. **Vagovagal** reflex during gastric phase

Gastrin-Releasing Polypeptide is released at vagal nerve ending & it causes secretion of gastrin by stimulating the G-cells.

→ Actions

1. Stimulates gastric gland to secrete gastric juice.
2. Accelerates gastric motility
3. Promote growth of gastric mucosa
4. Stimulates secretion of pancreatic juice (rich in enzyme)
5. Stimulates growth of pancreas to release pancreatic H₂O.

* SECRETIN

- 1st H₂O. to be discovered.
- Peptide H₂O. with 27 A.A. residues
- Secreted by S-cells of duodenum, jejunum & ileum.
- Prosecretin $\xrightarrow{\text{acidity of chyme}}$ Secretin (active)
(inactive)

→ Stimulants for secretion

- Stimulant is acid chyme
- Product of protein digestion also stimulates.

→ Actions

- Stimulates exocrine pancreatic secretion.
- Act on pancreatic duct via cyclic AMP & cause secretion of large amount of watery juice & high content of HCO_3^- .
- other actions :- 1. Inhibits secretion of gastric juice
- 2. Inhibits motility of stomach
- 3. Causes constriction of pyloric sphincter.
- 4. The potency of action of CCK on pancreas

3. CHOLECYSTOKININ

→ Same H₂O has action on both Pancreas as well as gall bladder. So, named cholecystotinin - pancreozymin (CCK-PZ) or cholecystokinin.

→ Secreted by G-cells in mucosa of duodenum & jejunum, small quantity is secreted in ileum also.

→ Stimulants :- chyme containing digestive product of fat & protein.

→ Actions :- • constrict gall bladder

- Stimulates exocrine pancreatic secretion

↓
activates acinar cells via 2nd messenger (cAMP)

↓
causes secretion of pancreatic juice with enzymes



- others :-
 1. Accelerates activity of secretion
 2. The secretion of enterokinase
 3. Inhibits gastric motility
 4. Increase intestine motility
 5. Suppresses hunger
 6. Induces drug tolerance to opioids.
- 1. Glucose dependent mu opioid receptor / GqR $\rightarrow$ stimulates mu opioid secretion
 - inhibits gastric secretion & motility
- 2. Vasopressin antidiuretic hormone (VH) $\rightarrow$ inhibits HCl secretion, The secretion of succus entericus, The insulin secretion
- 3. Glucagon like polypeptide-1 (GLP-1) $\rightarrow$ The insulin secretion, inhibits gastric motility
- 4. GLP-1 $\rightarrow$ Suppresses Appetite
- 5. Somatostatin $\rightarrow$ inhibits secretion of GH, pancreatic juice, Gq hormones, gastric secretion & motility.
- 6. Peptide YY $\rightarrow$ inhibits gastric & intestinal secretion & motility, suppresses appetite.
- 7. Substance P $\rightarrow$ The movement of small intestine
- 8. Ghrelin $\rightarrow$ Promotes GH release, induces appetite & The gastric emptying.

⑦ DIGESTION AND ABSORPTION OF FAT

→ Digested by lipolytic enzymes

DIGESTION

- In mouth
- Saliva contains lingual lipase
- secreted by lingual glands of mouth & swallowed along with saliva. so digestion does not occur in mouth.
- In stomach
- Gastric lipase → Tributyrate
ent in gastric juice
- In intestine
- Almost all lipid digest in small intestine b/c of bile salts, pancreatic & intestinal juice which contains lipase.

Role of Bile salt → function of bile salt ~~important~~ !

* **Pancreatic lipase is most imp. enzyme for digestion of fat**

Final Products of digestion ⇒ fatty acids, cholesterol, monoglycerides

ABSORPTION

→ Monoglycerides, cholesterol, fatty acids from micelle enter the cell of intestinal mucosa by diffusion.

In mucosal cell, monoglycerides convert to Triglycerides

• also formed by ~~new~~ re-esterification of fatty acids with some cholesterol also esterified.

↓

Triglycerides + cholesterol → coated with layer of protein, cholesterol & phospho-

-lipid

↓
form **chylomicrons**

- chylomicrons → cannot pass through membrane of capillaries due to large size
- so enter lymph & transferred into blood via lymph.
- F.A. containing less than 10-12 C enter portal blood & transported as free F.A.
- Most of fat is absorbed in upper part of small intestine
- Bile is essential for absorption.

Area	Juice	Enzyme	Substrate	End Product
Mouth	Saliva	Lingual lipase	Triglycerides	Fatty Acid Diacylglycerol
Stomach	Gastric juice	Gastric lipase	- " -	F.A. Glycerol
Small intestine	Pancreatic juice	Pancreatic lipase	- " -	F.A., Monoglyceride cholesterol
		Cholesterol ester hydrolase	cholesterol ester	F.A. cholesterol
		Phospholipase A	Phospholipids	lysophospholipids
		- " - B	lysophospholipids	Free F.A.

Succus entericus	antutinal lipase	Triglycerides	F.A. Glycerol
------------------	---------------------	---------------	------------------

Chart of digestion of lipid

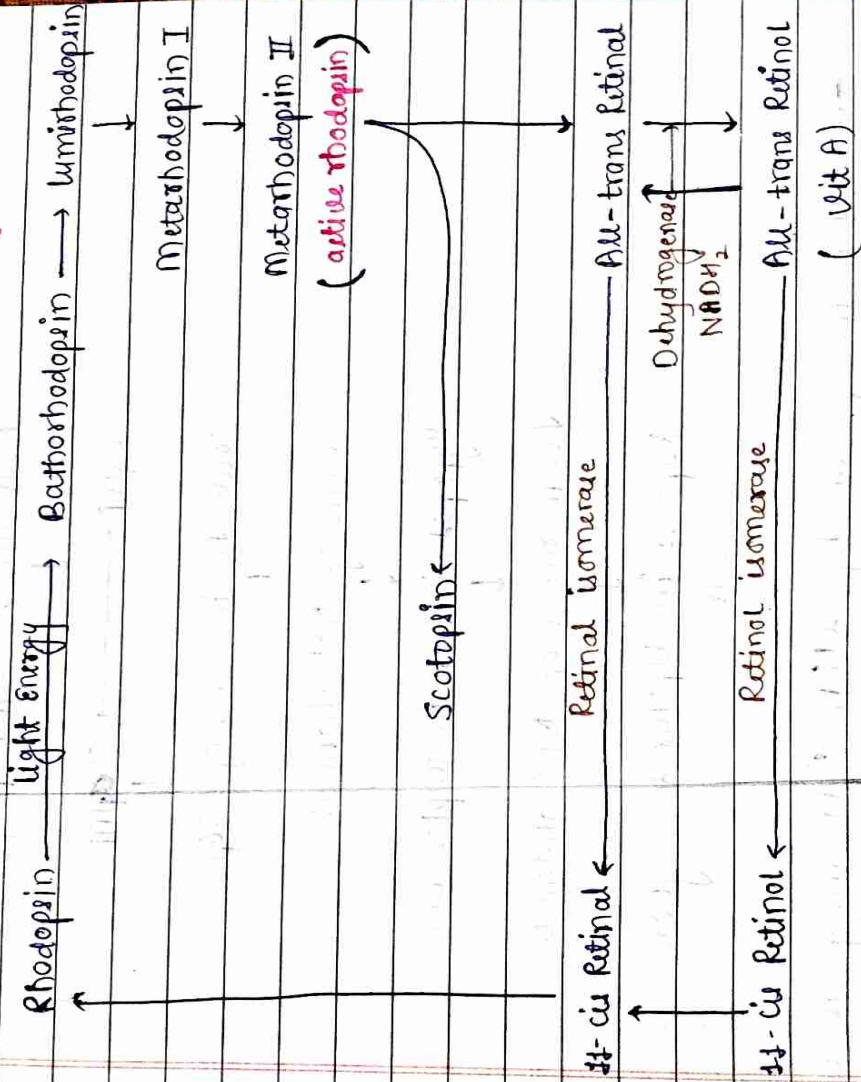
SPECIAL SENSES

VISUAL PROCESS

→ Chemical Basis of Visual Process | Photochemical changes & resynthesis of rhodopsin

Rhynthesis

Photochemical changes



→ Photochemical change in visual receptor cells are called

Wald's visual cycle

→ Rhodopsin / Visual Purple is the photosensitive pigment of rod cells, found in membranous disc located in outer seg. of rod cells.

→ Phototransduction

It is - Retinal

↓
Rhodopsin II

↓
Activation of transducin
(G-protein)

↓
Activation of cGMP phosphodiesterase

cGMP → $\downarrow$ cGMP

↓
causes the inc. of cGMP

↓
closure of Na^+ channels

↓
Hyperpolarization in receptor cells

↓
inc. in release of NT (glutamate)

↓
Response in bipolar & ganglionic cells.

→ It is the process by which light energy is converted into receptor potential in visual receptors.

→ EMF in other sensory cells is usually -70 to -90 mV. However, during darkness (-)itivity is reduced & EMF becomes -70 mV b/c of influx of Na^+ ions.

→ Dark Adaptation Time (DAT)

- Process by which person is able to see the objects in dim light.
- If a person enters a dim-lighted room from a bright-lighted area, he is blind for sometime. After some time his eyes get adapted & he starts seeing the object.
- Duration of DAT = 20 minutes

★ Causes for DAT

1. Red insensitivity of rods as a result of regeneration of rhodopsin

- In bright light, rhodopsin is bleached (broken down)
- In dim light, it requires some time for regeneration of rhodopsin, which is imp. for optimal rod functioning.
- ★ DAT occurs in cones only

2. Dilation of Pupil

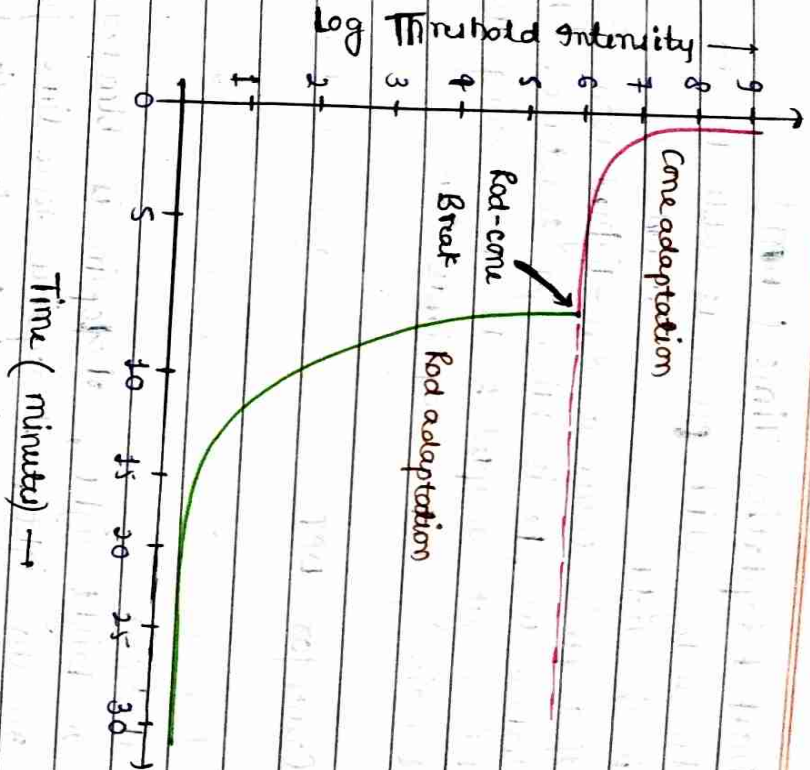
- Dilation allows more light to enter eye.

★ DAT curve

- Demonstrates the relationship b/w threshold of light & time spent in dark.

Parts of DAT curve

- Curve is biphasic
- 1st part → represents threshold of photopic vision which indicates the cone adaptation.
- 2nd part → represents threshold of scotopic vision which indicates the rod adaptation.



② VISUAL PATHWAY

→ Rods & cones are in the retina of eye form the visual receptors.

1st order Neurons → Bipolar cell in Retina

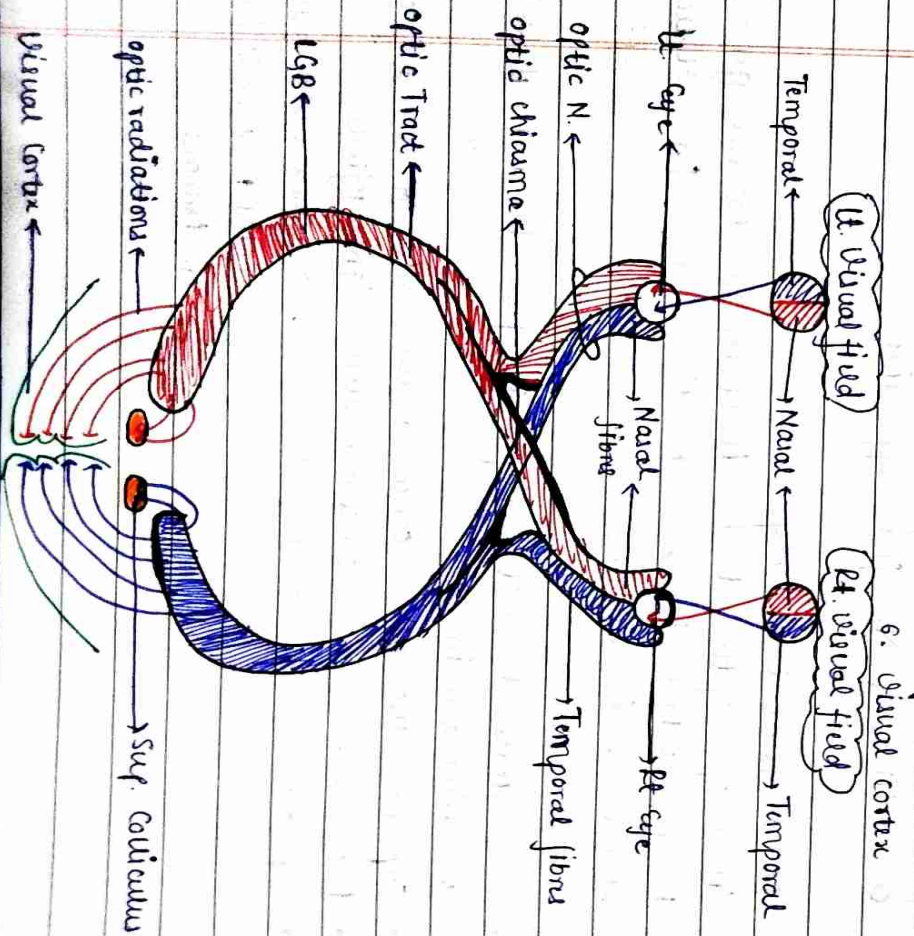
2nd order Neurons → Ganglionic cell in ganglionic cell layer of retina.

3rd order Neurons → Lateral geniculate body.

* Course :-

Consist of 6 components :-

1. optic Nerve.
2. optic chiasma.
3. optic tract
4. lateral geniculate body
5. optic radiation.
6. visual cortex



1. optic N.

- formed by the axons of ganglionic cells.
- leave the eye through optic disc.
- fibres from temporal part of retina are in lateral part of nerve & carry the impulses from nasal half of visual field.
- fibres from nasal part of retina are in medial part of nerve & carry the impulses from temporal half of visual field of same eye.

2. optic chiasma

The area of crossing of medial fibres of each optic N. is called optic chiasma.

3. optic Tract

formed by uncrossed fibres of N. on same side and crossed fibres of N. from opp. side.

1. lateral geniculate body

- fibres of optic tract terminate in lateral geniculate body, which forms subcortical center for visual sensation. From here, geniculocalcarine tract / optic radiation arises.

→ Some fibres do not synapse & pass through it only & terminate in :-

- **Superior Colliculus** → concerned with reflex movement of eyes.
- **Pretectal Nucleus** → concerned with light reflex.
- **Supraoptic Nucleus of hypothalamus** → concerned with retinal control in animals (not in humans).

control in animals (not in humans)

5. Optic Radiations

- fibres from L. geniculate body pass through internal capsule & form optic radiation.
- Ends in visual cortex.

6. Visual Cortex

- located in medial surface of occipital lobe.
- Area of cortex & function.
- 1st visual area (Area 17) → perception of visual impulses
- 2nd — " — (Area 18) → interpretation — " —
- occipital eye field (Area 19) → movements of eyes.

Applied

Anopia → loss of vision in one visual field.

• hemianopia → — " — — half of visual field.

↓
homonymous H. → loss of vision in same halves of both visual field.

heteronymous H. → — " — — opp. — " —



Lt. Homonymous H.



Lt. Homonymous H.



Bitemporal heteronymous H.



Binasal — " —

Representation of Visual Pathway

Visual receptors in Retina



1st order neurons → bipolar cells in inner nuclear layer



2nd order neurons → ganglionic cells in ganglionic cell layer



optic Nerve



optic chiasma



optic Tract



3rd order neurons → lateral geniculate body



optic Radiations



Visual Cortex

Effect of lesions at different levels

1. Lesion of optic N.

- causes total blindness / anopia.
- occurs due to ↑ed Intracranial pressure.

2. Lesion of optic chiasma

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Nature of defect depends upon the fibres involved.

i) Pr. on uncrossed lateral fibres by aneurysmal dilation of carotid Ar. causes **left or Right nasal hemianopia**

ii) If lat. fibres of both sides are affected causes **binasal hemianopia**

iii) Compression of crossed fibres by pituitary tumour causes **bitemporal hemianopia**

3. Lesion of optic Tract, lateral Geniculate Body and optic Radiations

→ causes **homonymous hemianopia**

4. Lesion of visual field / cortex

→ Lesion of upper / lower part of visual cortex leads to inferior / superior homonymous hemianopia.

★ Macular Sparing

→ In all condⁿ total blindness does not occur because, the macular vision is not lost & this is k/n as

Macular Sparing

→ Occurs b/c of following reasons :-

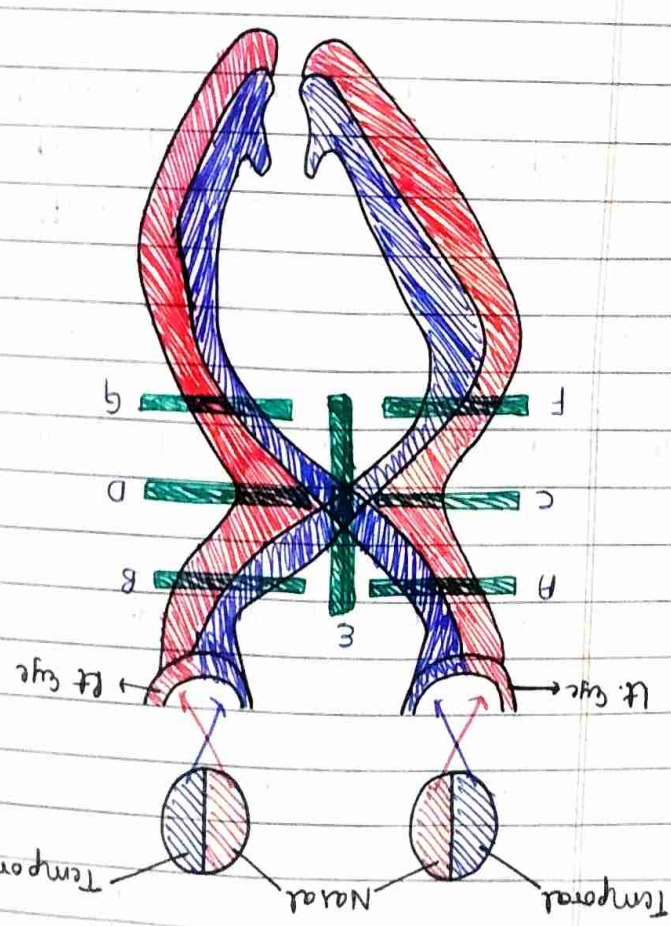
i) Fibres from macula project in visual cortex of both sides.

OPPO A53 are projected into both ant. & post. part of each

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- A → Union of Lt. optic N. (Anopia of Lt. eye)
- B → " " " " (Rt. eye)
- C → Union of lat. fibre of Lt. optic chiasma (Lt. Nasal hemianopia)
- D → " " " " (Rt. eye)
- C+D → Binasal hemianopia
- E → Union of medial fibres (Bitemporal hemianopia)
- F → Union of Lt. optic radiation (Lt. homonymous HA)
- G → " " " " (Rt. eye)

- A
- B
- C
- D
- C+D
- E
- F
- G



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③ PUPILLARY REFLEXES

* ACCOMODATION

- Adjustment of eye to see either near or distant object clearly.
- Process by which light rays from near/distant ob. are brought to a focus on sensitive part of retina.
- Achieved by various adjustments made in eyeball.

** Accommodation Reflex

- Accommodation is a reflex action. When a person looks at a near ob. after seeing a far ob., three adjustments are made in eye ball.

1. Convergence of eye ball due to contraction of medial recti.
2. Constriction of pupil due to contraction of constrictor pupillae of iris.
3. Incr. in ant. curvature of lens due to contraction of ciliary muscles.

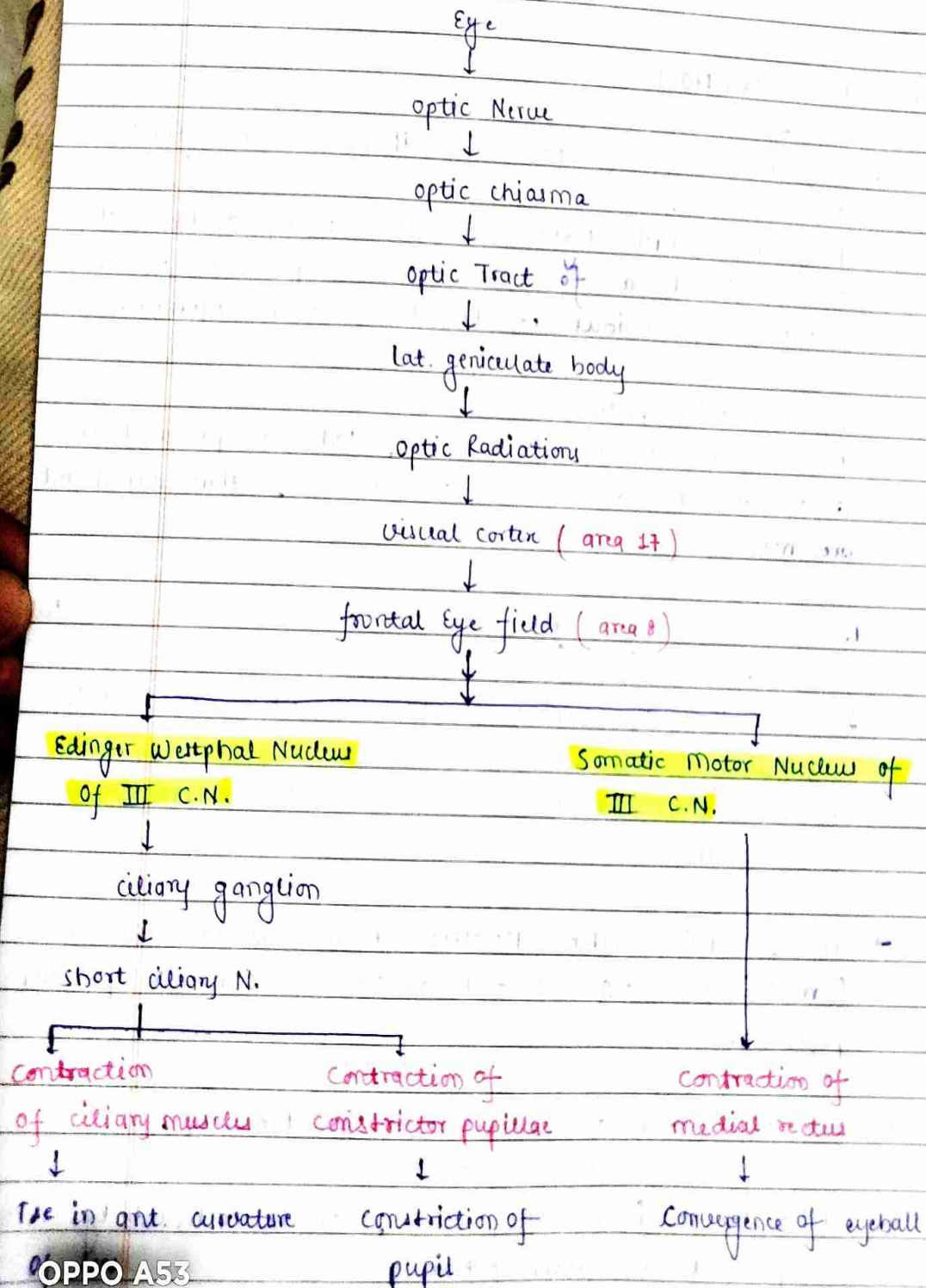
- Thus, this reflex involves both skeletal muscle (medial recti) & smooth muscle (ciliary & sphincter pupillae).

- All adjustments are carried out simultaneously.

- Although it is a reflex action, can be controlled by **OPPO A53** to a certain extent.

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Pathway for Reflex



④ COLOUR VISION

- Human eye is sensitive to light rays in visible spectrum.
- There are 7 colors in visible spectrum c/d spectral colours.
They are: **VIBGYOR**



with wavelength ranging from 400 nm to 750 nm.

- Colours produced by mixing of 2 or more spectral colours are kn as Extra spectral colors.

★ characteristic of colours

3 characteristics:- Intensity, Hue & Saturation

1: Colors

- Red, Green, Blue
- when mixed in equal proportions produce white color.

Complementary Colors

- Any color mixed with its complementary color, mixture appears white.

Ex:- Purple & Green

Orange & Blue

Yellow & dark blue

Effect of illumination

- Brightest part of spectrum shifts to lt. (green) during scotopic vision & to the rt. (yellow) during photopic vision.

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The shift to maxm sensitivity with change in illumination is Purkinje shift Phenomena.

Effect of Intensity

For same λ of light, when intensity of color is fixed / and diffn shades of color are perceived.

Receptors :- Cones are receptor for color vision

THEORIES of COLOR VISION

1. Pigment Theory

→ k/n as Young-Helmholtz Theory, also k/n as Retinal or Trichromatic Theory

1. There are 3 types of cones & each has a pigment with diffn absorbance spectrum.
2. Cones and cone pigments are called Red, Green & Blue.
3. Each type of cone pigment can absorb a wide range of λ .
4. Visual cortex compares the relative D of action potentials in activated cone pathways.
5. At least 2 types of cones are required for color vision.
6. The color vision is due to presence of 3 types of cones each maximally sensitive to one of 3 primary color.
7. Fovea mediates color vision as it contains cones

Genes :- for Rhodopsin → ch. 3

for blue-sensitive cone → ch. 7

for red-sensitive & green sensitive → q arm of ch. X

2. Opponent Theory

- also kn as **Hering's theory of color vision**.
- Based on observation that there is no greenish-red or bluish-yellow color.
- Red and green oppose each other and blue & yellow are opposed.
- Ganglionic cells, cells of LGN, neurons of visual cortex show color-opponent property.

Single opponent Property

Ganglionic cells & cells of LGN



Single opponent type

Double opponent Property

Cells of visual cortex



double opponent type

COLOR BLINDNESS

- Total loss of color vision → **Achromatopia**
- failure to appreciate one or more color.
- It is an **inherited sex-linked recessive character**.
- Common in 8% (males) & 0.4% (females).
- also occurs due to acquired condⁿ ~~also~~ such as ocular disease / injury or disease of Retina.

Cause / Etiology

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injury to eye due to accidents / stroke.

2. Chronic disease

Degeneration of macula of eye.

Sickle cell Anemia

Leukemia

Diabetes

Retinitis

3. Drugs

Antibiotics

Antihypertensive drug

Antituberculosis drug

Barbiturates

4. Toxins

Fertilizers

CO

CS₂

chemicals with high Pb content

5. Chronic alcoholism

6. Aging

* Classification

1. Trichromats

→ Most common type

→ Individual with all the 3-cone systems but one of the systems is off defective.

Ability to recognize one of 1st color is lost.

2. Dichromats

- Individual with 2-cones system & 3rd cone system is not.

3. Monochromats

- Individual with only 1-cone system
- Cannot appreciate any color b/c minm 2-cones are required to appreciate color.
- See only black and white & shades of gray.

* Test

- Ichihara chart
- Edridge - Green Lantern
- Holmgren's wool - matching

⑤ ERRORS OF REFRACTION

1. MYOPIA / SHORT SIGHTEDNESS

- Eye defect characterised by inability to see the distant object.
- c/d shortsightedness b/c person can see near ob. clearly but not the distant ob.
- Near vision is normal but far point is not infinite i.e., it is at a definite distance.
- In extreme condition, it may be only a few cm away from eyes.

Cause →

- Anteroposterior diameter of eyeball is long.
- Image is brought to focus a little in front of Retina.
- Light after coming to focus, disperses again so, blurred image is formed on retina.

Correction →

- Light rays entering the eye must be divergent & not parallel.

↓

thus corrected by using a Biconcave lens

↓

Light rays are diverged by concave lens before entering the eye.

2. HYPERMETROPIA / LONG SIGHTEDNESS

- Eye defect characterised by inability to see near ob.
- c/d longsightedness b/c person can see distant ob. but not near.

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→ 2022/07/11 11:52 Vision is normal but near one is affected.

Lele Page
Cause $\rightarrow$ ↓ $\downarrow$ anteroposterior diameter of eyeball

↓
light rays are not converged enough to form a clear image on retina i.e., brought to focus behind retina

↓
cause blurred image of near object

- $\rightarrow$ Occurs in childhood, if eyeball fails to develop the correct size.
- $\rightarrow$ Common in old age also.

Correction $\rightarrow$ By using Biconvex lens

↓
light rays are converged by convex lens before entering the eye.

3. **ASTIGMATISM** (A=not, stigma=point)

- $\rightarrow$ Condⁿ in which light rays are not brought to sharp point upon retina.
- $\rightarrow$ Common optical defect
- $\rightarrow$ when moderate Hn as Physiological Astigmatism
- $\rightarrow$ Ex:-

Stars appears as small dots of light to person with normal eye. But appears as radiating short lines of light to astigmatic.

* Types

1. Regular

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* Regular $\rightarrow$ Refractive power is unequal in diffⁿ meridians b/c of alternation of curvature in $\pm$ meridians.

* Irregular $\rightarrow$ Refractive power is unequal not only in diffⁿ meridians but unequal in diffⁿ point of same meridians.

Corrections $\rightarrow$ By using Cylindrical lens

4. PRESBYOPIA

- $\rightarrow$ characterised by progressive diminished ability of eye to focus on near ob. with age.
- $\rightarrow$ Due to gradual reduction of amplitude with age.
- $\rightarrow$ Distant vision is unaffected, only near vision is affected b/c near point is away from eyes.
- $\rightarrow$ Ant. curvature of lens does not $\uparrow$ during near vision. So, light rays from near ob. are not brought to focus on retina.

Cause:-

- $\downarrow$ Elasticity of lens
- $\downarrow$ convergence of eyeball

Correction :- By using Biconvex lens

⑥ STRUCTURE OF EAR

★ FUNCTIONS OF MIDDLE EAR

1. Receives sound waves from external ear by the tympanic membrane.
2. Transmission of sound to inner ear through tympanic mem. and ossicular chain.

3. Amplification of sound waves

force of sound $\uparrow$ several times as it reaches the footplate of stapes & oval window due to :-

- i) • S.A. of Tympanic mem $\gg$ S.A. of oval window (17:1)
• Total force of sound wave is transmitted to oval window but due to $\downarrow$ S.A. pr. $\uparrow$ 17 times. $[IP = \frac{F}{A}]$
- ii) • The ossicular chain acts as a lever while transmitting the vibration. (Enhance the force 1.3 times)

Thus, total amplification of sound occurs by about 22 times. In this manner, middle ear performs the job of impedance matching

4. Equilization of Air Pressure through the eustachian tube.
5. Protects against prolonged, loud sound by activation of tympanic reflex.

★ ORGAN OF CORTI

- Receptor organ for hearing.
- Neuro-epithelial st. in cochlea.
- When sound wave reaches organ of Corti, it acts as a transducer that converts the mechanical form of energy into action potentials in the cochlear nerve.

→ Situation and Extent

- Rests upon the lip of osseous spiral lamina & basilar mem.
- Roof is formed by gelatinous tectorial membrane.

→ Structure

- Made up of sensory elements i.e. Hair cells & various supporting cells.
- All the cells are arranged in order from center towards the periphery of cochlea.
- Cells are :-

1. Border cells
2. Inner Hair cells
3. Inner Phalangeal cells
4. Inner Pillar cells
5. Outer Pillar cells
6. Outer Phalangeal cells
7. Outer Hair cells
8. Cells of Hensen
9. Cells of Claudius
10. Tectorial membrane & lamina Reticularis

1. Border cells

- Slender columnar cells.

- Arranged in a single layer on tympanic lip along

inner side of inner hair cells.

→ Surface have cuticle

2. Inner Hair Cell

→ flask shaped & broader than outer hair cells.

→ Rounded base rest on adjacent supporting cells of inner phalangeal cell.

→ Surface bears cuticular plate & no. of stiff hairs of stereocilia. One of stereocilia is larger & f/n as kinocilium

→ Cilia are in contact with tectorial membrane.

→ Inner H.C. + Outer H.C. ⇒ Receptor Cells

→ Sensory nerve fibres are distributed around H.C.

3. Inner phalangeal cells

→ Supporting cells of inner H.C.

→ Base rest of on Basilar mem.

→ Cuticular plate looks like phalanx (finger bones)

4.5 Inner & Outer Pillar Cells (Rods of Corti)

→ Each has broader base, elongated body, head at the tip.

→ Pillars of inner & outer cells converge towards each other & their heads articulate

→ Pillars form series of arches, which enclose a tunnel of inner Tunnel / Tunnel of Corti.

6. Outer phalangeal cells

→ Supporting cells of outer hair cells.

→ B/w the innermost outer phalangeal cell & outer pillar, is a fluid space f/n as Space of Nuel.

7. Outer Hair cell

- Columnar cell
- Base is supported by outer phalangeal cell.
- St. is similar to that of inner hair cells.

8. Cells of Hensen

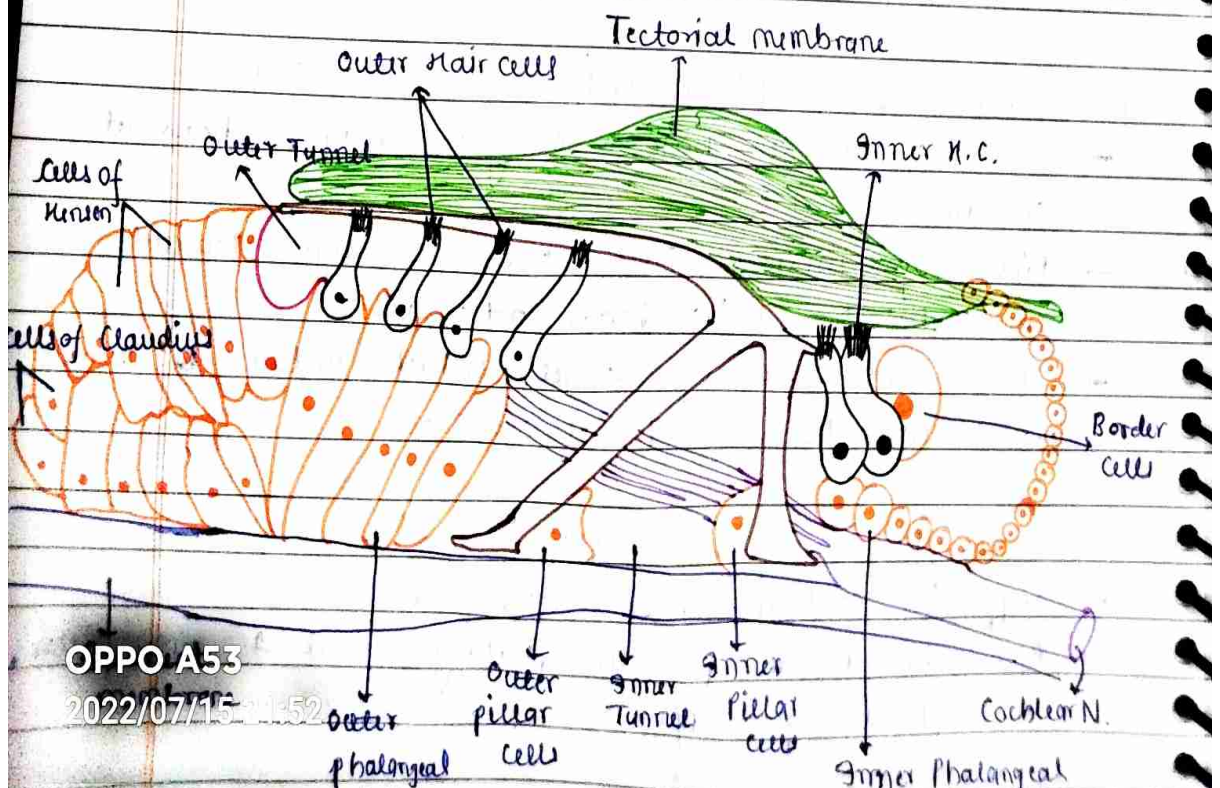
- Tall columnar cells forms outer border of costi.
- Space b/w outer phalangeal cell & cell of Hensen is called outer tunnel

9. Cells of Claudius

- Cuboidal in nature.

10. Tectorial Membrane

- forms roof of costi
- Processes of hair cells are stimulated by movement of tectorial membrane, in relation to vibration in endolymph.



⑦

AUDITORY PATHWAY

→

Have 5 orders of neurons

1. 1st order neuron → Starts from hair cell & project to ipsilateral dorsal & ventral, nucleus.
Cochlear
2. 2nd order neurons → Starts from cochlear nu. & project to sup. olivary nu. & nu. of trapezoid body.
3. 3rd order neurons → from sup. olivary nuclei to inf. colliculus of midbrain.
4. 4th order neurons → from inf. colliculus to medial geniculate body of thalamus.
5. 5th order neurons → from MGB to 2^o auditory cortex (area 41 & 42).

Schematic Representation

Hair cells



Spiral Ganglion



Cochlear Nucleus



Sup. olivary Nuclei.



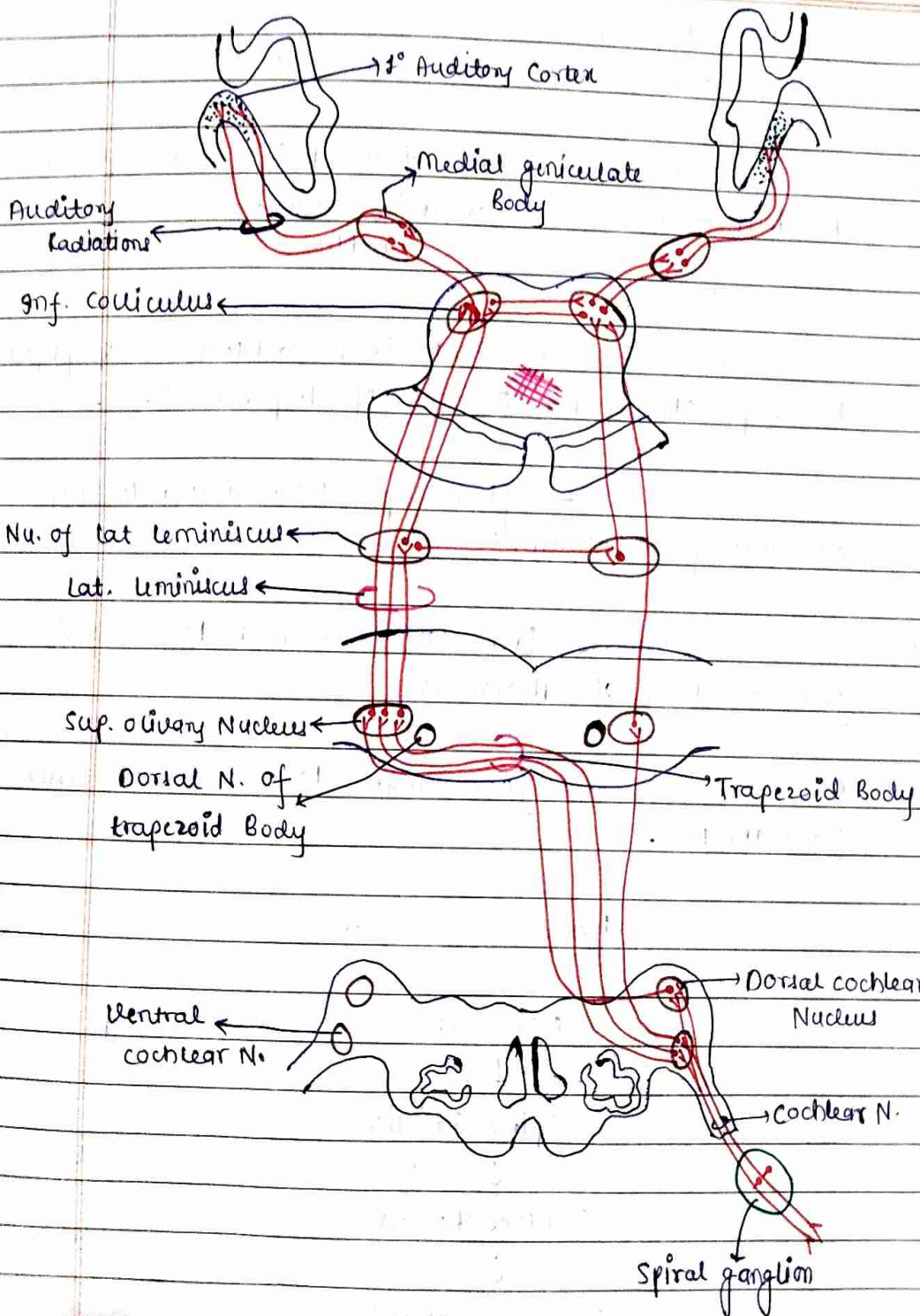
Inf. Colliculi



MGB



2^o auditory cortex



⑧ MECHANISM OF HEARING

→ Sound wave travel through ext. auditory meatus & produce vibration in tympanic mem.



These vib. travels through malleus & incus & reaches the stapes & causes movement of stapes



This produces movement in fluid of cochlea



These vib. stimulates ~~cochlea~~ Hair Cells in organ of corti



This causes generation of Action Potential in auditory N. fibres



when auditory impulse reaches central cortex



perception of hearing occurs

∴ During hearing, Ear converts energy of sound waves into A.P. in auditory N. fibres



This is Sound Transduction

Role of Ext. Ear

- Direct sound wave towards the tympanic mem.
- In man, extrinsic & intrinsic muscles of auricle are rudimentary & movement is not possible.

Role of Middle Ear

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1. Role of Tympanic Mem.

- Acts as a resonator that reproduces the vibration of sound.

2. Role of Auditory Ossicles

- Ossicles act like a lever system so that stapes exerts a greater force against the cochlear fluid.



This feed force is very essential to set up vibrations in cochlear fluid.

Impedance Matching is most imp. function of middle ear.

- Process by which tympanic mem. & auditory ossicles convert sound energy into mechanical vibrations in cochlear fluid with min^m loss of energy.
- without this conductive deafness occurs.

3. Role of Eustachian Tube

Responsible for equalizing the pr. on either side of tympanic membrane.

Role of Inner Ear

1. Travelling Wave

Movement of footplate of stapes against oval window



movement of perilymph in scala vestibuli



fluid does not move all the way & immediately

hits vestibular mem. & causes movement of Endolymph in scala media



this causes bulging of Basilar mem. (BM)



bulging ↑s elastic tension in that portion of BM



This tension initiates a wave which travels along Basilar mem. towards helicotrema



c/d Travelling wave.

2. Excitation of Hair cells

when travelling wave causes vibration of basilar mem.



it causes movement of stereocilia leading to excitement of hair cells and



generation of receptor potential.

Electrical Events During Process of Hearing

1. Sound Transduction

→ type of sensory transduction in hair cell in organ of corti by which sound energy is converted into action potential in auditory N. fibre.

→ Events during transduction are :-

1. Receptor Potential

2. Endocochlear P.

3. Action P.

1. Receptor Potential / Cochlear microphonic Potential

- It is mild depolarization that is developed in the hair cell of cochlea when sound wave are transmitted to internal ear.
- RMP of hair cells is -60 mV .

* Role of hair cells

1. Inner H.C.

- Responsible for sound transduction
- Are 1st sensory cells
- causes generation of action potential in auditory nerve fibre.

2. Outer H.C.

- get short during depolarization & elongated during hyperpolarization.
- This is called Electromotility
- facilitates movement of basilar mem. & Increases amplitude & sharpness of sound
- collectively called Cochlear Amplifier

2. ** Endocochlear Potential / Endolymphatic Potential

Electrical potential developed in fluids outside the hair cells.

Cochlear fluid

Extracellular fluids in inner ear.

Perilymph → Scala vestibuli & Scala tympani are filled with this.

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Composition is same as that of ECF with high Na^+ ion conc.

Endolymph → • Scala media is filled with this!

- Contains high conc. of K^+ & low conc. of Na^+

* Electrical Potential

- Diff in K^+ conc. is responsible for development of an electrical potential diff'n b/w Endo. & Perilymph
- Potential in Endolymph is (+)ve upto +80 mV.

* Significance of Endocochlear Potential

- Endolymph has (+)ve potential of +80 mV.
- So, inside H.C. the electrical potential is -60 mV in comparison to that of perilymph.
- and -170 mV in comparison to that of endolymph.



This high P.D. sensitizes the Hair cells (H.C.)



so excitability of H.C. ↑



It also ↑ response of H.C. even to slight movement of stereocilia.

3. A.P. in auditory Nerve fibre

- obeys All or None law
- has definite threshold & refractory period.
- Generated by cochlear microphone potential & appears 0.5 to 1 msec after ——— " ———

⑨

AUDITORY DEFECTS

1. Conduction Deafness

→ Type of deafness that occurs due to impairment in transmission of sound waves in ext. ear or middle ear.

- Causes :-
1. obstruction of ext. auditory meatus with dry wax
 2. Thickening of tympanic mem. due to repeated middle ear infection.
 3. Perforation of tympanic mem. due to inequality of pr. on either side.
 4. Otitis media (inflammation of middle ear)
 5. Otosclerosis (fixation of footplate of stapes against oval window).

2. Nerve Deafness

→ deafness caused by damage of any st. in cochlea, such as hair cell, organ of corti, basilar mem., lesions in auditory pathway.

- Causes :-
1. Degeneration of hair cells due to some antibiotics like streptomycin & gentamycin.
 2. Damage of cochlea by exposure to loud noise.
 3. Tumor affecting VIII C.N.

* Treatment of Deafness

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Hearing Aids

→ useful to restore the hearing capacity in case

of deafness of due to partial $\downarrow$ in function of middle ear / cochlea.

→ Electronic devices attached to ear that amplifies the sound wave

2. Cochlear Implants

→ Tiny electronic devices implanted under the skin over mastoid process / bone.

→ Used for deafness due to disease of hair cells of organ of Corti.

* Test for Hearing

1. Rinne Test

→ Normally air conduction is better than bone conduction

→ Bone condⁿ is tested by placing the tuning fork on the mastoid process.

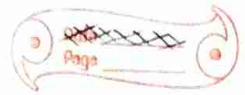
→ Air condⁿ is tested by holding the tuning fork near the ear & a normal person continues to hear sound for some time.

→ In conductive deafness BC is more than AC.

2. ** Weber Test

→ The base of a vibrating tuning fork is placed at the centre of forehead, a normal person will hear it equally in both ears.

→ But in conductive deafness, will hear louder in diseased ear b/c the environmental noise does not reach the inner ear of that side.



→ In neural deafness, will hear louder in normal side.

3. Schwabach Test

→ Same as that of Weber's test

Principle is

→ Bone condⁿ of patient is compared with that of normal person.

→ In conductive deafness, bone condⁿ is prolonged.

→ In neural ———, ——— shortened.

(10) SENSATION OF TASTE

★ TASTE BUDS

→ Sense organ for taste / gustatory sensation are the taste buds.

→ Most of the taste buds are on papillae of tongue :-

1. Filiform Papillae

→ Small, conical shaped situated over dorsum of tongue, contains less taste buds.

2. Fungiform Papillae

→ Round in shape, situated over ant. surface of tongue near tip, contains moderate taste buds.

3. Circumvallate Papillae

→ Large, on post. part of tongue & are many in no., contains large taste buds.

★ St. of Taste Bud

→ Taste bud is a bundle of taste receptor cells.

→ with supporting cells embedded in epithelial covering of papillae.

Types of cells in Taste Buds

→ Type I / Sustentacular Cell

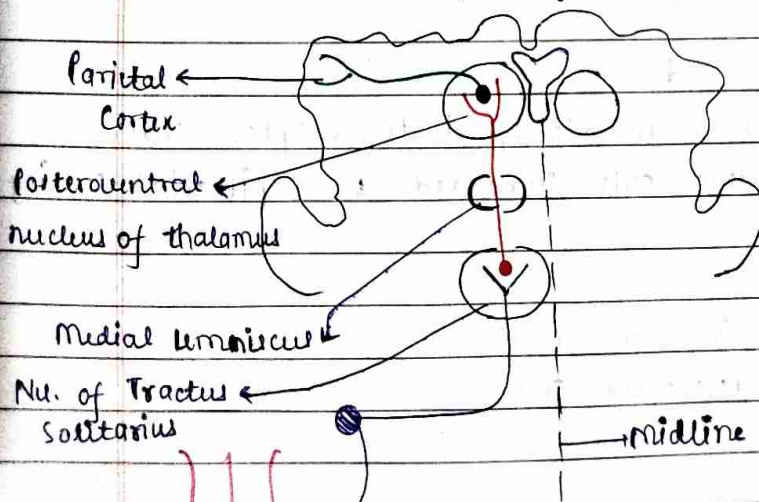
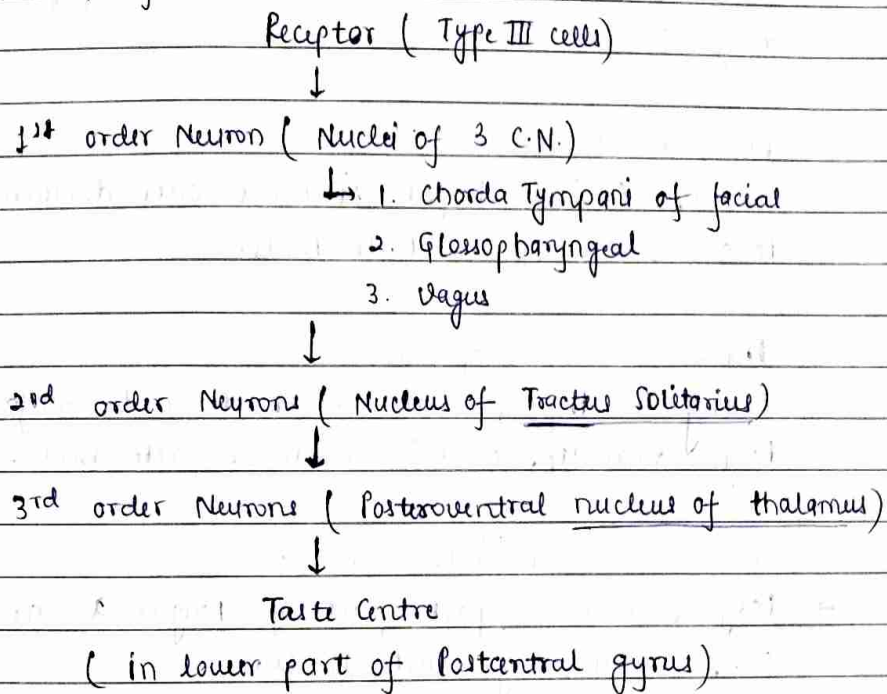
→ Type II

→ Type III

→ Type IV / Basal cell

- I & II are supporting cells.
- III are taste receptor cells.
- I, II & III have microvilli

PATHWAY of TASTE



#** TASTE SENSATION

- Divided into 5 types :-
1. Sweet
 2. Salt
 3. Sour
 4. Bitter
 5. Umami

- These are 1st fundamental taste sensation
→ others are combination of two / more of these.

* Discrimination of Taste Sensation

- All areas of tongue give response to all types of taste sensation
→ In low concn of taste sub., each taste bud give rise to one 1st taste stimuli.
→ In high concn of taste sub., each taste bud give response to more than 1 type of taste stimuli.

* Taste Sensation & Chemical Constituents

1. Sweet Taste

- Produced mainly by organic sub. like monosaccharide, glycerol & ketones, etc.

2. Salt Taste

Produce by chlorides of Na, K & NH_4^+ , Nitrates of Na & K, Sulphates, Bromides & Iodides.

3. Sour Taste

Produce by H^+ ion in acid & acid salts.

4. Bitter Taste

- Produced by organic sub. like quinine, morphine, picric acids, etc.
- Bitterness of taste is due to cations.

5. Umami

- Recently recognized
- Receptor of this sensation respond to glutamate, particularly **Monosodium Glutamate (MSG)**, which is common ingredient in asian food.

- Excess MSG consumption cause Chinese Restaurant Syndrome in people taking chinese food regularly.

Symptoms :-

1. Headache

2. Sweating

3. Chest pain

Threshold → • Bitter has low & Sweet has high

11 SENSATION OF SMELL

* Olfactory Receptor

→ Situated in olfactory mucous membrane.

↓
lies in upper part of nostril

→ Mucosa also contains Bowman's gland

↓
• continuously lines mucous mem.

→ Receptor cell is a bipolar neuron.

* Olfactory pathway

Axons of bipolar olfactory receptors

↓
pieces cribriform plate of ethmoid

↓
each olfactory bulb.

↓
Synapse with dendrites of mitral cells

↓
forms olfactory glomeruli

~~olfactory bulb~~

↓
mitral cells leaves olfactory bulb & forms olf. tract

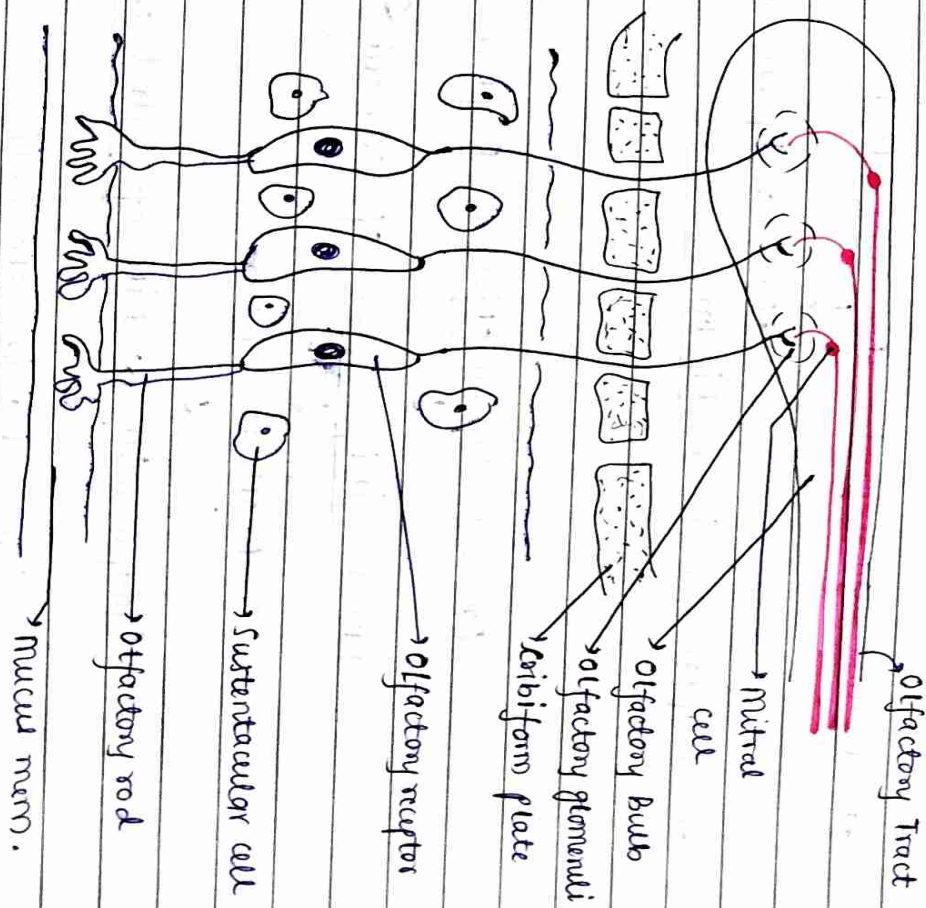
↓
Tract ends in olf. cortex

↓
through intermedial & lat. olfactory stria.

→

Cortex includes st. forming limbic system. It are :-

1. Ant olfactory Nucleus
2. Prepyriform Cortex
3. Olfactory Tubercle
4. Amygdala



Receptor cells & olfactory pathway

NERVOUS SYSTEM

1) CLASSIFICATION OF NERVE FIBRE

→ classified by 6 diffⁿ methods :-

1. Depending upon structure

Myelinated → that are covered by myelin sheath.

Non-myelinated → not covered by myelin sheath.

2. Depending upon Distribution

Somatic → supply the skeletal muscles of body.

Visceral → supply the visceral muscles or various internal organs of body.

3. Depending upon origin

Cranial → arises from brain

Spinal → arises from spinal cord

4. Depending upon function

Sensory → carry sensory impulse from diffⁿ parts of body to CNS, also kn as Afferent N. fibres

Motor → carry motor impulses from CNS to diffⁿ parts of body, also kn as Efferent N. fibres.

5. Depending upon secretion of Neurotransmitter

Adrenergic → secretes NOR-adrenaline

cholinergic → secretes acetylcholine

c. Depending upon diameter and conduction of impulse
[ERLANGER & GASSER CLASSIFICATION]

→ classified N. fibres into 3 major types, on the basis of diameter, velocity of conduction of impulse

- Type A N. fibres

- " B "

- " C "

- Type A → thickest

- Type C → thinnest, also known as type IV

- Except Type C all N. fibres are myelinated.

Type	Structure	function
A-alpha	70 to 120 large with thick myelin	• Sensory fibres from proprioceptors & touch receptors
A-Beta	Medium with thick myelin	• Motor fibres to skeletal muscle
A-Gamma	Medium with thick myelin	• Sensory fibres from proprioceptors, & touch & pr. receptors.
A-Delta	thin myelin	• Motor to muscle spindle
B	Small with thin myelin	• Sensory f. from pain & temp. receptors
C	Small without myelin	• Preganglionic autonomic
		• Sensory from pain & temp. receptors



③ PROPERTIES OF NERVE FIBRES

1. Excitability

* Action Potential / Nerve Impulse

→ Similar to that of muscle, except for some differences

Events	Nerve fibres	Skeletal muscle fibres
RMP	-70 mV	-90 mV
firing level	-55 mV	-75 mV
End of depolarisation	+35 mV	+55 mV

→ A.P. starts in initial segment of N. fiber.

→ Properties of A.P.

1. Electrotonic / local Potential

→ Non-propagated local response that develops in the nerve fiber when a subliminal stimulus is applied.

→ Subliminal stimulus does not produce A.P. but alters RMP & produce slight depolarization for about 7 mV.
↓

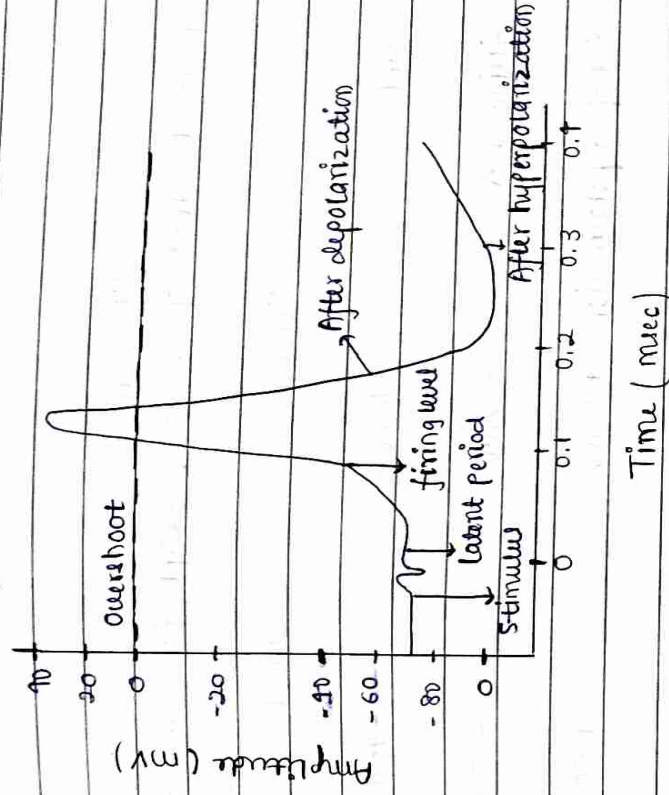
This state is called electrotonic potential.

→ It does not obey all-or-none law.

2. Voltage Clamping

→ Experimental method that uses electrodes to alter & control the membrane potential.

→ Used to measure the ionic current across the membrane of N. fiber by fixing the membrane potential at desired voltage.



2. Conductivity

★ Saltatory Conduction

→ form of conduction of N. impulse in which impulse jumps from 1 node to another. in

→ Conduction of impulse through myelinated N. fibre is 50 times faster than through a non-myelinated.

→ In it A.P. jumps from 1 node to another node of Ranvier instead of travelling through the entire N. fiber.

• Mechanism

→ Myelin sheath is not permeable to ions

Entry of Na^+ ion from ECF occurs only through node of Ranvier, where myelin is cont.

↓
this causes depolarization in node & not in internode

↓
thus depolarization occurs at successive nodes

↓
so, A.P. jumps from 1 node to another.

↓
Hence, called Saltatory conduction

3. Refractory Period

→ Period at which N. does not give any response to stimulus

→ 2 types → Absolute & Relative

4. Summation

If 2 or more subliminal stimulus is applied within a short interval of about 0.5 msec. response is produced.

↓
b/c they are summed up together to become strong enough to produce the response.

5. Adaptation

while stimulating N. fiber continuously, excitability of N. fiber is greater in beginning. Later response ↓ & slowly & finally it does not show any response. This is adaptation/ accommodation.

6. Infatigability

→ N. fiber cannot be fatigued, b/c it can conduct only 1 A.P. at a time. At that time, it is completely refractory and

does not conduct another A.P.

7. All or None Law

→ when a Nerve is stimulated, it gives maximum response or does not give response at all.

DEGENERATION AND REGENERATION OF NERVE FIBERS

When N. fiber is injured, various changes occur in it & nerve cell body. All these changes are together called Degenerative changes.

* Degenerative changes in the Neuron

→ degeneration refers to deterioration | impairment | pathological changes of an injured tissue.

→ when a peripheral N. fiber is injured, degenerative changes occur in N. fiber of same neuron and adjoining neuron.

→ classified in 3 types :-

1. Wallerian Degeneration

2. Retrograde

3. Transneuronal

1. * Wallerian Degeneration | Orthograde Degeneration

→ Pathological change that occurs in the distal cut end of N. fiber (axon).

→ starts within 24 hrs. of injury

→ occurs throughout the length of axon simultaneously.

* changes in Nerve

→ Axis cylinder swells & breaks up into small pieces.

→ Myelin sheath slowly disintegrated into small pieces of fat

→ Neurilemmal sheath is unaffected but the Schwann cells multiply rapidly.

→ Macrophages invade from outside & remove the debris of axis cylinder and fat droplets of disintegrated myelin sheath.

↓
So, neurilemmal tube becomes empty



later filled by cytoplasm of Schwann cell

* All these changes take place for about 2 months from the day of injury.

2. Retrograde Degeneration

→ Pathological changes which occurs in nerve cell body and axon proximal to cut end.

* Changes in cell body

- Nissl granules disintegrate into fragments by chromatolysis
- Golgi apparatus disintegrates
- N. cell body swells due to fluid & becomes round.
- Nucleus displaced towards periphery



Sometimes, Nucleus extruded out of cell



Death of cell occurs & regeneration of injured N. is not possible.

* Changes in axon proximal to cut end

- changes occurs only upto 1st node of Ranvier from site of injury.
- changes are similar to those which occurs in distal cut end of N. fiber.

3. Transneuronal Degeneration

→ If afferent N. fiber is cut, degenerative changes occurs in neuron with which afferent N. fiber synapse.

Ex :- 1. chromatolysis in cells of lat. geniculate body occurs due to sectioning of optic N.

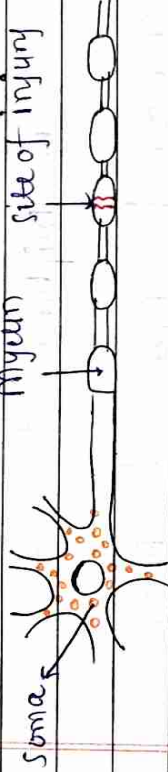
2. Degeneration of dorsal horn of spinal cord when post. N. root is cut.

3. ————— " ————— " —————
there is tumor in cerebral cortex.

★ ★ Regeneration of N. fiber

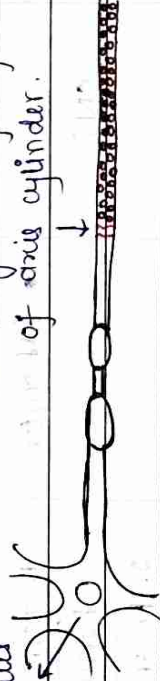
- refers to Regrowth of lost / destroyed part of tissue.
- injured / degenerated N. fiber can regenerate.
- Starts as early as 4th day after injury & becomes effective after 30 days & completes in 80 days.

Myelin site of injury

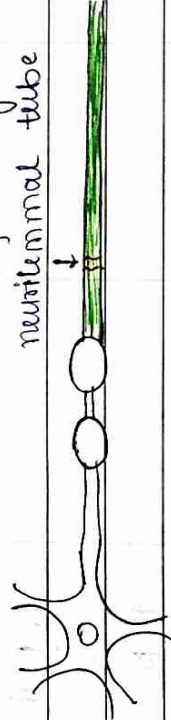


disintegration of myelin & breakdown of axis cylinder.

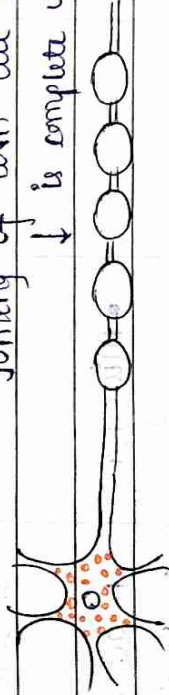
Nissl granules disappear



injured N. fiber regenerating into neurilemmal tube



Joining of both cut ends & regeneration
↓
is complete with myelin & Nissl granules.



Degeneration & Regeneration of N. fiber

④

NEUROGLIA

- Supporting cells of the nervous system
- Non-excitabile & do not transmit N. impulse. So, old
- Non-Neural cells / Glial cells
- No. is 10-15 greater than the neurons.
- Plays an imp. role in IP of nerve during infection
- Constitute the site of tumors in nervous system

* Classification :-

Distributed in CNS as well as PNS. So, classified in 2 types :-

- Central Neuroglial cells
- Peripheral ———

Classification	Situation	Type	Functions
Central Neuroglia	CNS	• Astrocytes	1. form blood-brain barrier 2. Provide supporting network 3. Maintain ECF status 4. Regulate Ca & F level in ECF 5. Secrete neurotrophins
		• Microglia	1. act as Macrophages 2. Provide immune & inflammatory response
		• Oligodendrocytes	1. Provides myelination 2. Form supporting network

Peripheral Neuroglia	PNS	• Schwann Cells	1. Provide myelination 2. Help in regeneration of injured N. fibers 3. Perform phagocytosis
		• Satellite Cell	1. Provide supporting network 2. Maintain ECF status

RECEPTORS

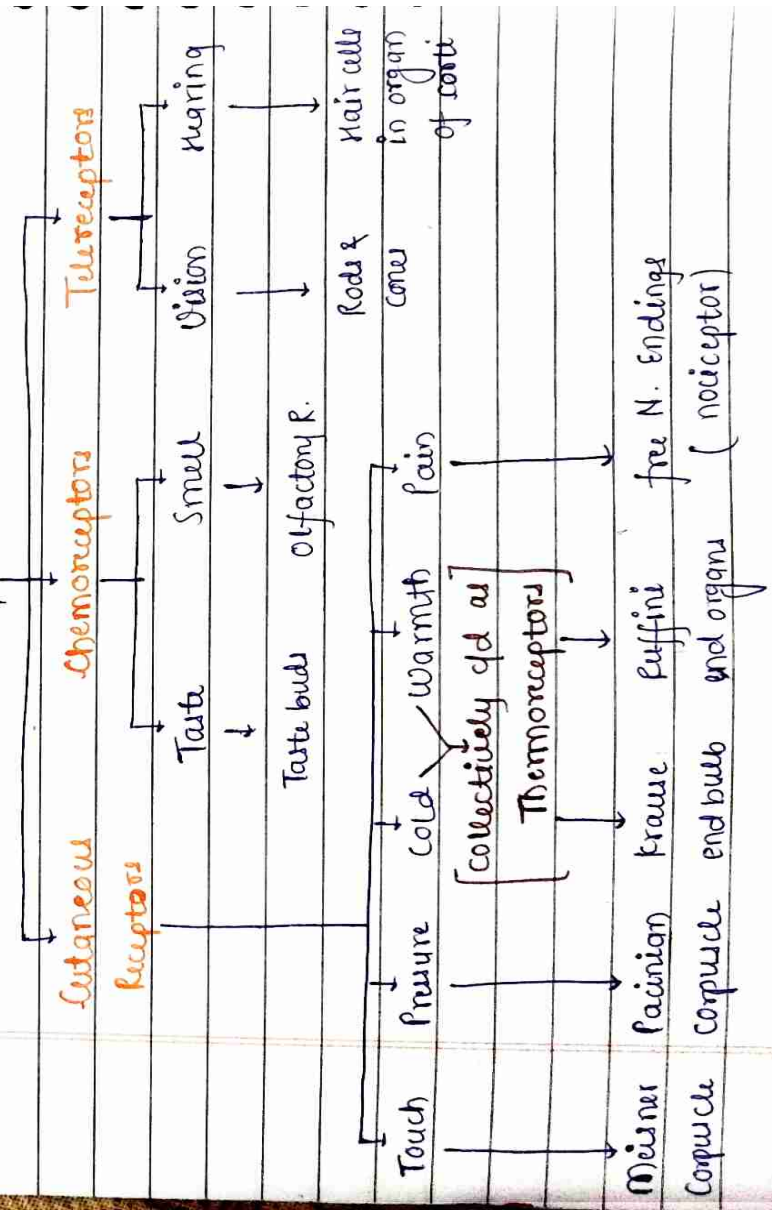
Sensory N. endings that terminate in periphery as bare 'unmyelinated endings' or in form of encapsulated structures. Give response to stimulus

when stimulated produces a series of impulses, which are transmitted through afferent Nerves.

→ functions like a **Biological Transducer** & convert various forms of energy (stimuli) in environment into action potentials in N. fiber.

* Classification of Receptors

1. Enteroceptors



• **Enteroceptors** → give response to stimuli arising outside the body.

1. Cutaneous / Mechanoreceptors

→ Receptors situated in skin

→ also c/d Mechanoreceptors b/c of their response to Mechanical stimuli such as touch, pain & pr.

2. Chemoreceptors

→ give response to chemical stimuli.

3. Thermoreceptors

→ give response to stimuli arising away from body.

→ also c/d Distance receptors.

2. **Interoceptors**

Visceroreceptors

Proprioceptors

[situated in viscera] [response to change in position of diffⁿ parts of body]

Receptor	Situation	Receptors	Situation
1. Stretch R.	Heart	1. Muscle Spindle	Muscle
2. Baroreceptor	Blood vessels	2. Golgi Tendon Organ	Tendon
3. Chemoreceptor	GI Tract	3. Pacinian corpuscle	Ligaments
4. Osmoreceptor	Urinary Tract	4. Free nerve endings	Face, joints
	Brain	5. Hair cells	Vestibular Apparatus

• **Interoceptors** → give response to stimuli arising from within in the body.



Property of Receptors

Specificity

- Refers to response given by a particular type of receptor to a specific sensation.
- Ex:- Pain receptors give response only to pain sensation.

2. Adaptation

- Decline in discharge of sensory impulses when a receptor is stimulated continuously with const. strength.
- also cld Sensory Adaptation / Oversatization.
- Depending upon adaptation time, receptors are of 2 types:-

Phasic → • get adapted rapidly.

- Ex:- Touch & Pressure receptors

Tonic → • get adapted slowly

- Ex:- Muscle spindle, Pain & Cold Receptor.

3. Weber Fechner Law

- Response to I_{sc} in strength of stimulus.
- Magnitude of sensation felt is proportional to log of intensity of stimulus.

$$R = K \log S$$

R = Intensity of response (sensation)

K = const.

S = intensity of stimulus

Intensity

- when stimulus is applied, receptors discharge depending on st. of stimulus.
- when st. of stimulus is less
 - receptors close to site of stimulus discharge
- when st. of stimulus is more
 - activated neurons fire at more rate
 - receptors that are ~~out~~ somewhat distance away from stimulus also discharge

5. Acuity

- Precision of stimulus localization.
- Depends on no. of receptors ~~out~~ in area where stimulus is applied.

6. Law of Projection

- when a sensory pathway from receptors to cerebral cortex is stimulated on any particular site along its course, sensation caused by stimulus is always felt at location of receptor
↓
irrespective of site stimulated.

7. Doctrine of specific Nerve Energy

- sensation evoked by a stimulus that generate impulse in specific sensory pathway depends on
↓
precise area of brain that is eventually activated by stimulus.

Adrenergic & Cholinergic receptors

Classification of sensory receptor cells

SYNAPSE

Junction b/w 2 neurons

Not an anatomical continuation but only a physio-logical continuity b/w 2 nerve cells.

★ Classification

Anatomical

1. Anaxonic S.
2. Anodendritic S.
3. Anaxomatic S.

Functional

1. Electrical S. (gap junction)
2. Chemical S. (synaptic cleft)

★ Structure of Chemical Synapse

Axon Terminal

Synaptic vesicle

Mitochondria

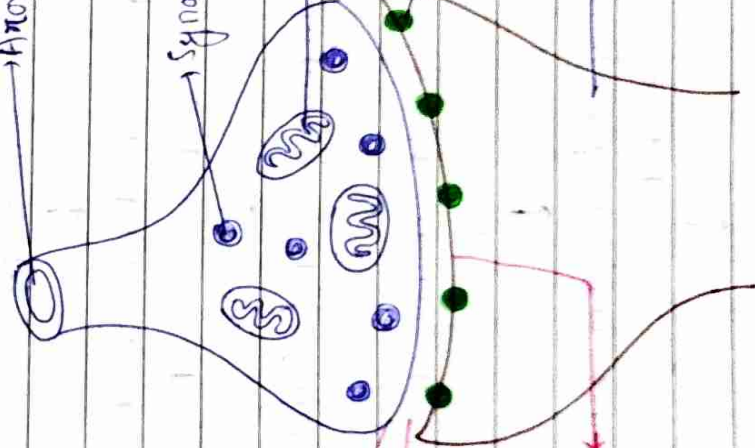
Receptor protein

Post synaptic neuron

• Pre-synaptic Mem.

• Synaptic cleft

• Post-synaptic Mem.



Sequence of Events during synaptic Transmission

Pre-Synaptic Neuron

- Arrival of A.P. in axon Terminal
- ↓
- Opening of Ca- Channels in pre-synaptic membrane
- ↓
- Influx of Ca ions from ECF into the axon terminal
- ↓
- Opening of vesicle & release of ACh
- ↓
- Passage of Acetylcholine (ACh) through synaptic cleft
- ↓
- Form of ACh-receptor complex
- ↓
- Opening of Na- channels & influx of Na ions from ECF
- ↓
- Development of EPSP (Excitatory Post-synaptic Pot.)
- ↓
- Opening of Na- channel in initial seg. of axon
- ↓
- Influx of Na ion from ECF & development of A.P.
- ↓
- Spread of A.P. through axon of Postsynaptic neuron

Synaptic Inhibition

classified into 5 types :-

1. Postsynaptic | Direct
2. Presynaptic | Indirect
3. Negative feedback | Renshaw cell inhibition $\rightarrow$ occurs in spinal cord
4. Feedforward $\rightarrow$ occurs in cerebellum
5. Reciprocal $\rightarrow$ inhibition of antagonistic muscles.

1. Presynaptic | Indirect

- $\rightarrow$ Occurs due to failure of pre-synaptic axon terminal to release sufficient quantity of excitatory NT.
- $\rightarrow$ Mediated by axoaxonic synapse.
- $\rightarrow$ Prominent in spinal cord & regulates the propagation of inf. to higher centres.

$\rightarrow$ Normally $\rightarrow$ A.P. reaching pre-synaptic neuron produces development of EPSP in post-synaptic neuron.

$\rightarrow$ In spinal cord $\rightarrow$ modulator neuron or pre-synaptic inhibitory neuron forms an axoaxonic synapse with pre-synaptic neuron.

$\downarrow$

inhibits the pre-synaptic neuron & thus the mag. of A.P. in pre-synaptic neuron.

$\downarrow$

smaller A.P. induces Ca influx

$\downarrow$

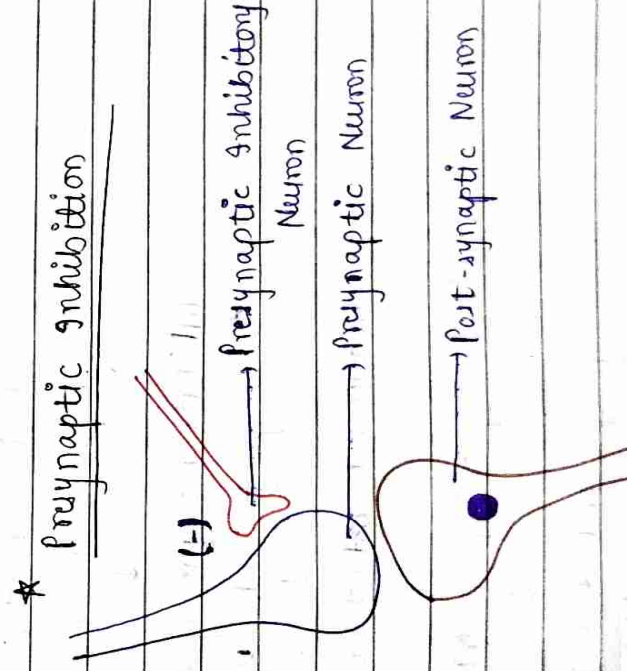
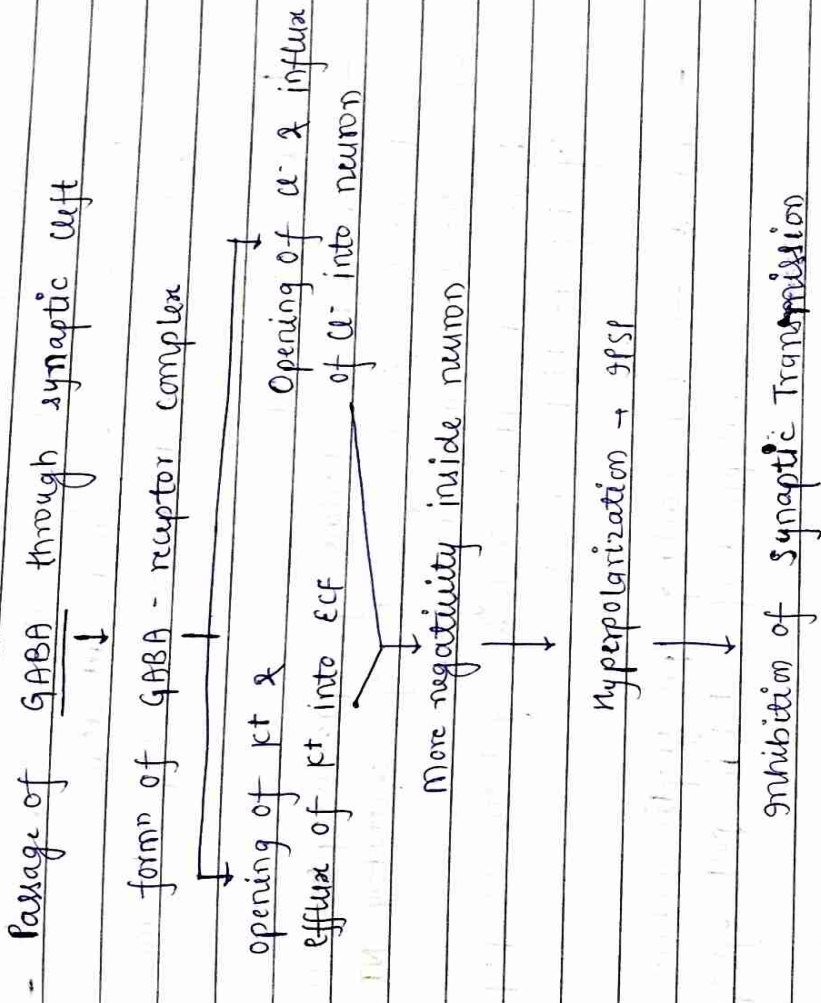
This $\downarrow$ quantity of NT released

$\downarrow$

So, mag. of EPSP $\downarrow$ resulting in synaptic inhibition

2. Postsynaptic

Presynaptic events
(same as synapse)



Properties of Synapse

1. One way Conduction (Bell Magendie law)
Impulse are transmitted only in 1 dirn in synapse i.e., from pre-synaptic neuron to post-synaptic neuron.

2. Synaptic Delay

→ Short delay that occurs during the transmission of impulse through synapse.
→ It is due to :-

1. Release of NT
2. Passage of NT from axon terminal to post-SM.
3. Action of NT to open ionic channels in post-SM.

Significance → we can find out whether the pathway is mono. or polysynaptic by determining delay.

3. Fatigue

→ during continuous muscular activity synapse becomes fatigued along with Betz cells in motor area of frontal lobe.

→ fatigue is due to depletion of NT

→ Depletion of NT is due to :-

1. Ach is destroyed by Ach-esterase.
2. New Ach is not synthesized

4. Summation

Mechanism by which EPSPs and IPSPs are integrated at post-SM neuron level.

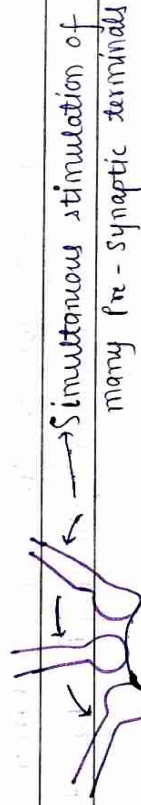
Spatial $\rightarrow$ A.P. produced by fusion of EPSPs developed from many pre-synaptic terminals.

- All the EPSPs develop simultaneously.
- occurs when many pre-synaptic terminals are stimulated simultaneously.

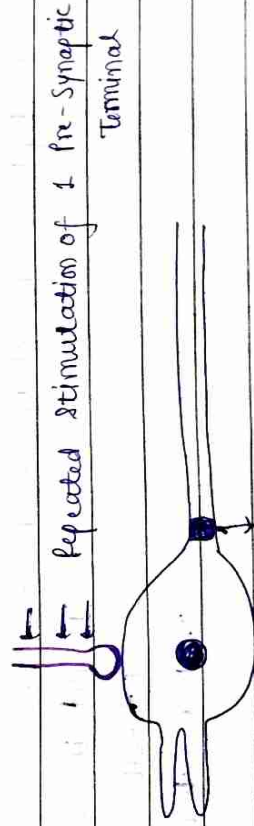
ii) Temporal $\rightarrow$ A.P. produced by fusion of many EPSPs from a single pre-synaptic terminal.

- EPSPs develop one after another.
- All the EPSPs overlap & summate with each other, so A.P. is generated in Post-SN.
- occurs when one pre-synaptic neuron terminal is stimulated repeatedly.

Spatial



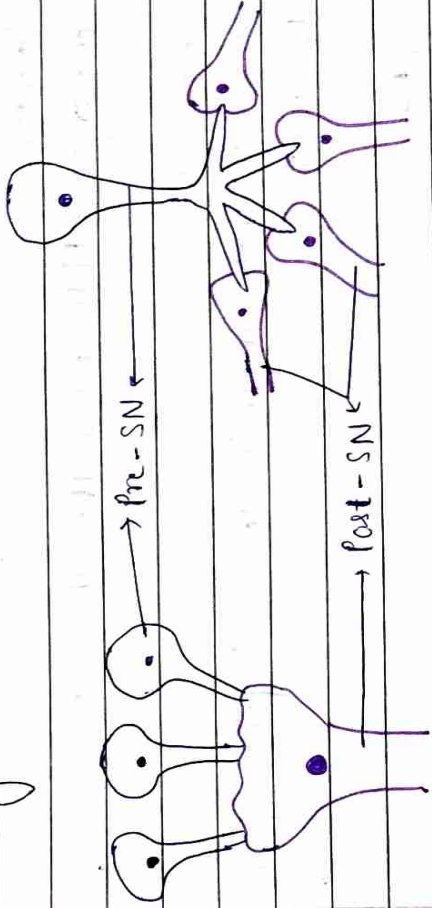
Temporal



5. Convergence and Divergence

→ Process by which many pre-SN terminate on a single post-SN. → Convergence

→ Process by which 1 pre-SN terminates on many post-SN
Divergence



6. Postsynaptic Potentials

→ If potential of post-SN carried towards depolarization
↓ c/d

Excitatory Post-Synaptic Potential (EPSP)

→ _____ " _____ → Hyperpolarization

↓ c/d

Inhibitory Post-SN

SPINAL REFLEXES

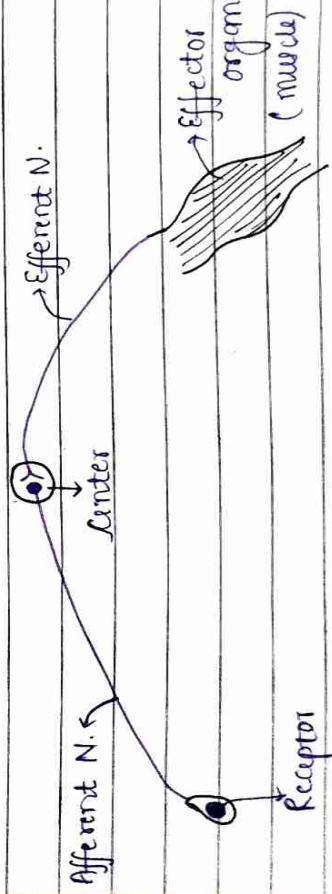
REFLEX ARC

anatomical nervous pathway for a reflex action.
simple reflex arc includes 5 components :-

1. **Receptor**
→ End organ which receives the stimulus.
2. **Afferent Nerve**
→ Sensory N. that transmits sensory impulses from receptor to center.
3. **Center**
→ Receives the sensory impulses via afferent N. & generates appropriate motor impulses.
→ Center located in Brain & Spinal Cord.
4. **Efferent N.**
→ Motor N. that transmits motor impulses from center to effector organ.

5. Effector Organ

→ St. such as muscle / gland where the activity occurs in response to stimulus.



Type

1. Stretch Reflex

simplest manifestation of muscle spindle function -
is the muscle stretch reflex (monosynaptic)
→ whenever a muscle is stretched suddenly, excitation of spindles causes reflex contraction of the large skeletal muscle fibers of the stretched muscle & also of closely allied synergistic muscles.

Stimulus → Stretch

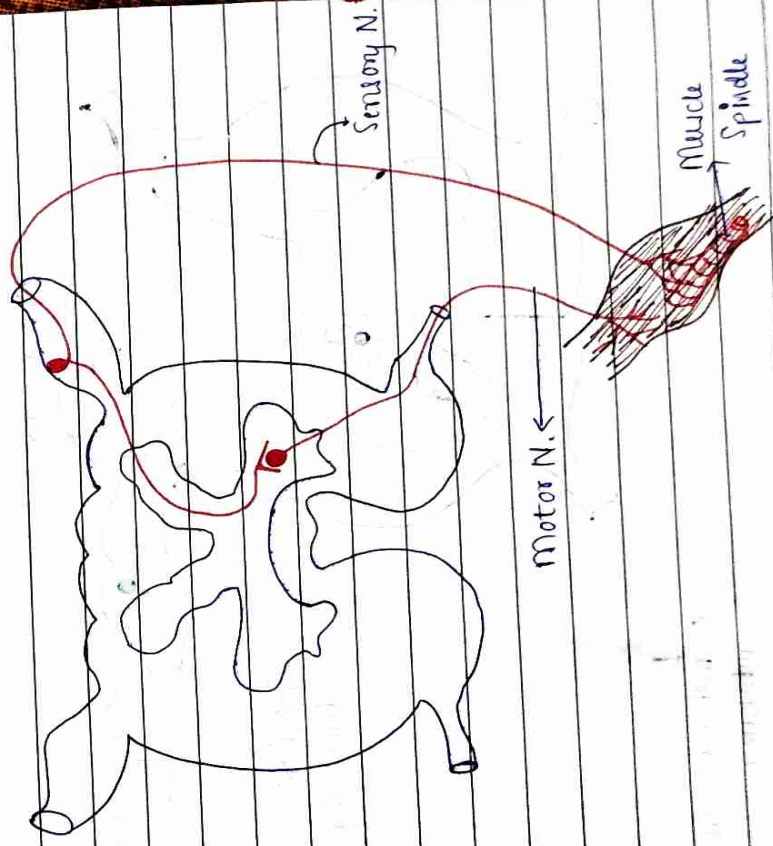
Receptor → Muscle spindle

Afferent → Ia Afferent fibers

Center → Spinal cord

Efferent → Motor Neurons

Effector organ / Response → Contraction of skeletal muscle.



Inverse Stretch Reflex

Relaxation of muscle in response to a strong stretch is cld GSR.

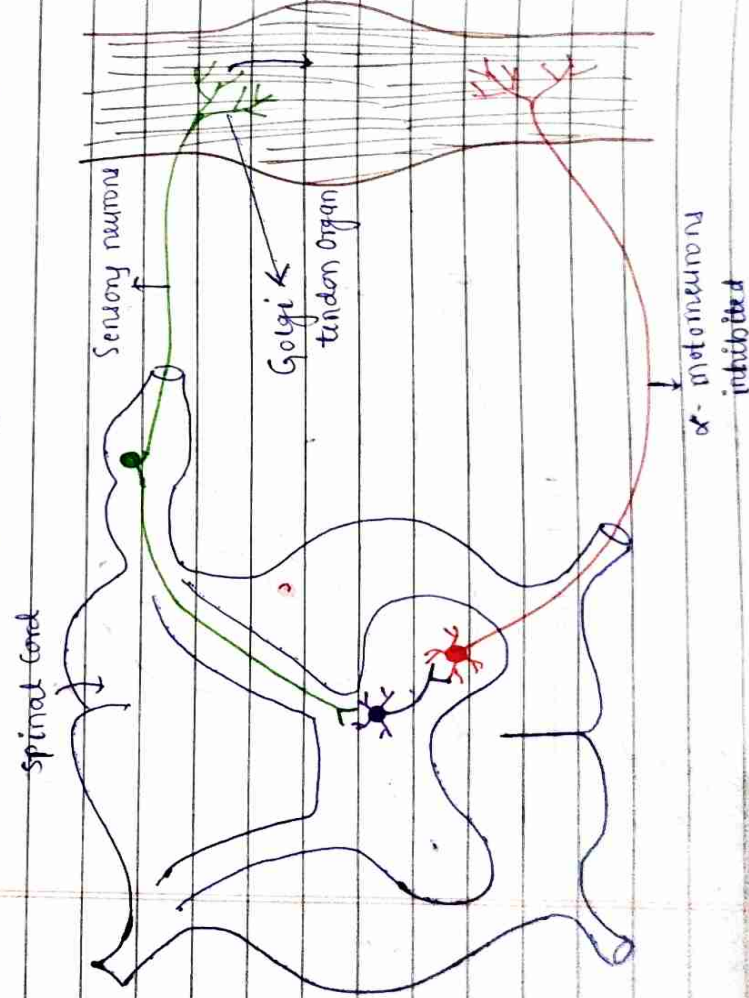
Diynaptic Reflex

Stimulus $\rightarrow$ Strong Stretch
 Receptor $\rightarrow$ Golgi Tendon organs
 Afferent $\rightarrow$ 1b afferent fibers
 Center $\rightarrow$ Spinal Cord
 Efferent $\rightarrow$ Motor N.
 Response $\rightarrow$ relaxation of muscle

Nociceptors $\rightarrow$ • pain receptor

• Stimulated due to mechanical / temp. threat.

*** By this reflex rupture of muscle can be prevented.

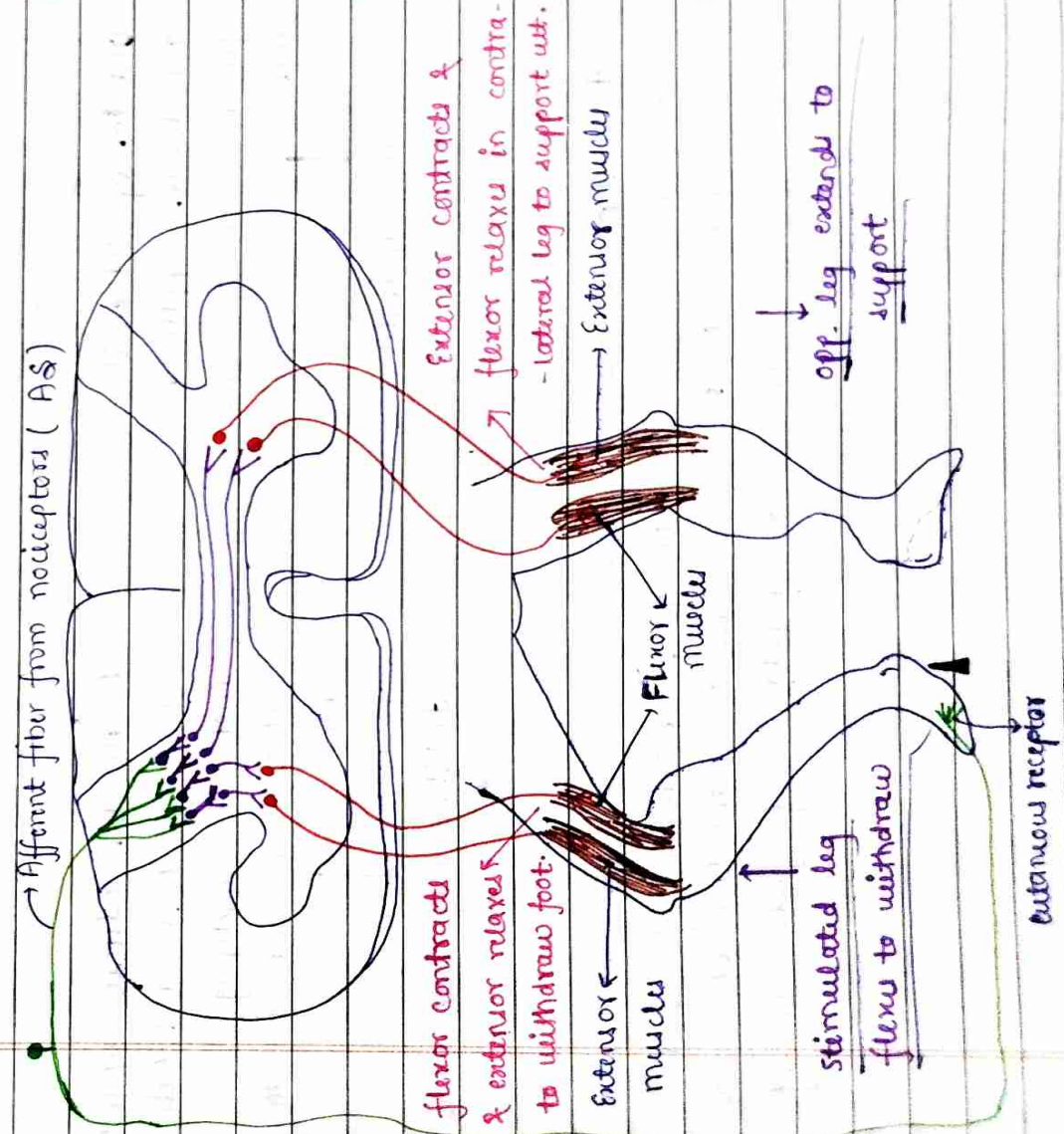


Withdrawal Reflex / Flexor Reflex

Polysynaptic spinal reflex

old flexor withdrawal reflex b/c it causes withdrawal of the body parts by flexing the limbs.

Receptor → pain receptor in skin, muscle tendon
Stimulus → Noxious / painful stimulation of skin, muscle
Afferent → flexor reflex afferents (FRA)
Center → Spinal cord
Efferent → Motor N.
Response → flexion of ipsilateral limb





Mechanism

Stimulation of pain receptor of skin, muscle, tendon, joint



Sensation carried by flexor reflex afferents (FRA)



Afferent fibres entering the spinal cord divided into 2 brs. to terminate on 2 separate sets of interneurons (IN)



One br. of IN excites

a motor N. supplying

flexor muscles of same

side.



flexion of ipsilateral limb

Another br. activates separate

set of IN that inhibits a

motor N. supplying the

extensor muscle of same

side.



Inhibition of extensor muscle

4. Crossed Extensor Reflex

- If stimulus is stronger, extension of contralateral limb occurs due to activation of extensor gr. of muscles and inhibition of flexor gr. of muscles.

(8) ASCENDING TRACT (Sensory Tracts)

- 3 major sensory tracts :-
1. Post. Column Tracts
 2. Spinothalamic Tracts
 3. Spinocerebellar Tracts

of Posterior (Dorsal) Column Pathway

1st order neuron enters the CNS at the dorsal root & the sensory root of cranial nerve.



ascend up on same side (without relay & decussating in dorsal horn)



Relay in medulla (cuneatus & gracilis nuclei) so 2nd order neuron arises



2nd order neuron decussate in medulla & reaches on opp. side of medulla



Ascend up to thalamus & 3rd order neuron arises from thalamus

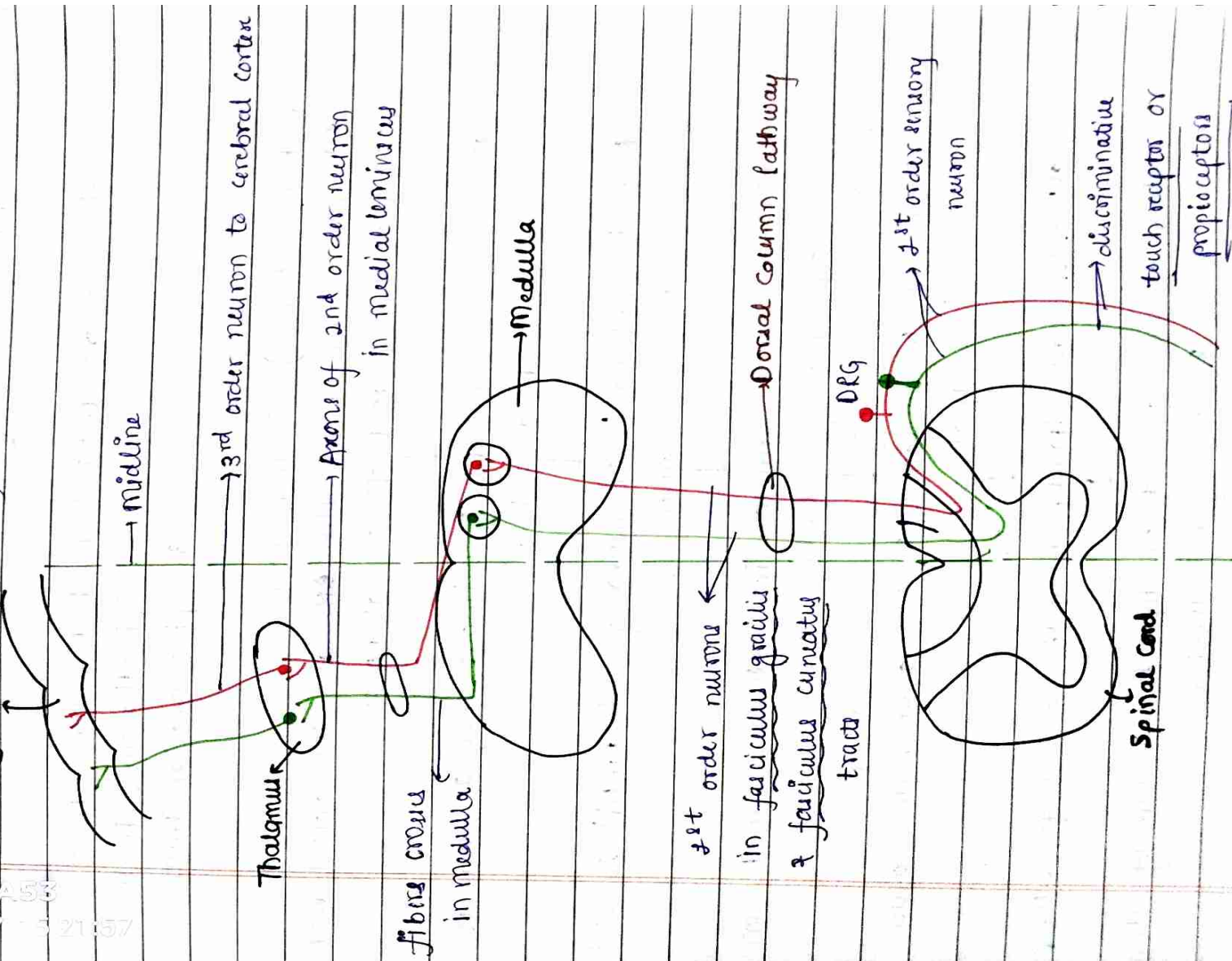


3rd order neuron finally relay in 1st sensory cortex,



where stimuli are stored by their signature of stimulus & region of body involved

1st Sensory cortex (Area 3, 1, 2)



Spino cerebellar Pathway

Inf. about muscle, tendon & joint position from the spine to cerebellum.

Inf. is Subconscious

- 1st order neuron → synapse in dorsal horn
- 2nd order neuron → ascend via ant. & post. spino cerebellar tract to cerebellar cortex.
- 3rd order neuron → Cerebellum
- Used to → coordinate movements

Spinothalamic Pathway

- Located Lat. & Ventral to ventral horn.
- Carry impulse concerned with :-
Pain & Temp. → Lat. Spinothalamic
- Crude Touch & Pr. → Ant. — " —
- Inf. is sent to 1st sensory cortex on opp. side of body.

1st order neuron synapse with 2nd in post. gray horn at level of entrance



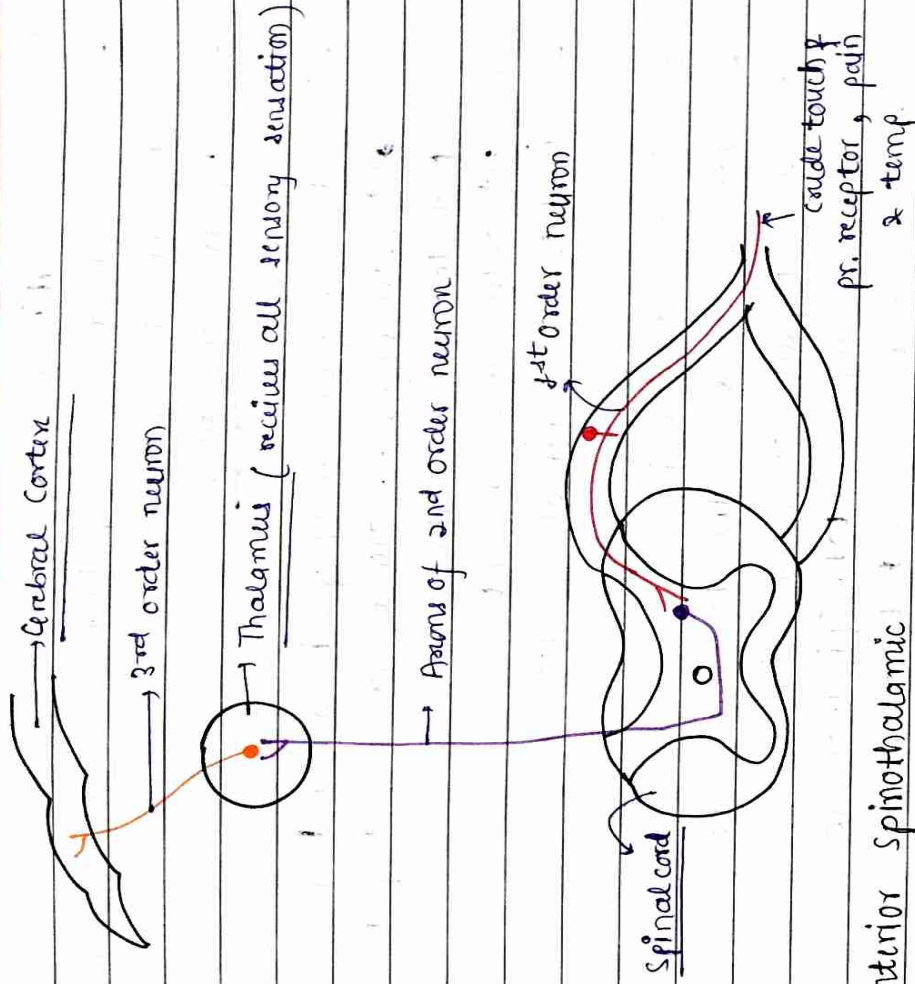
2nd order cross to opp. side at spinal cord level before ascending up to thalamus



Goes upward to thalamus & synapse with thalamic neurons



3rd order synapse at level of 1st somatosensory cortex.



★ Anterior Spinothalamic

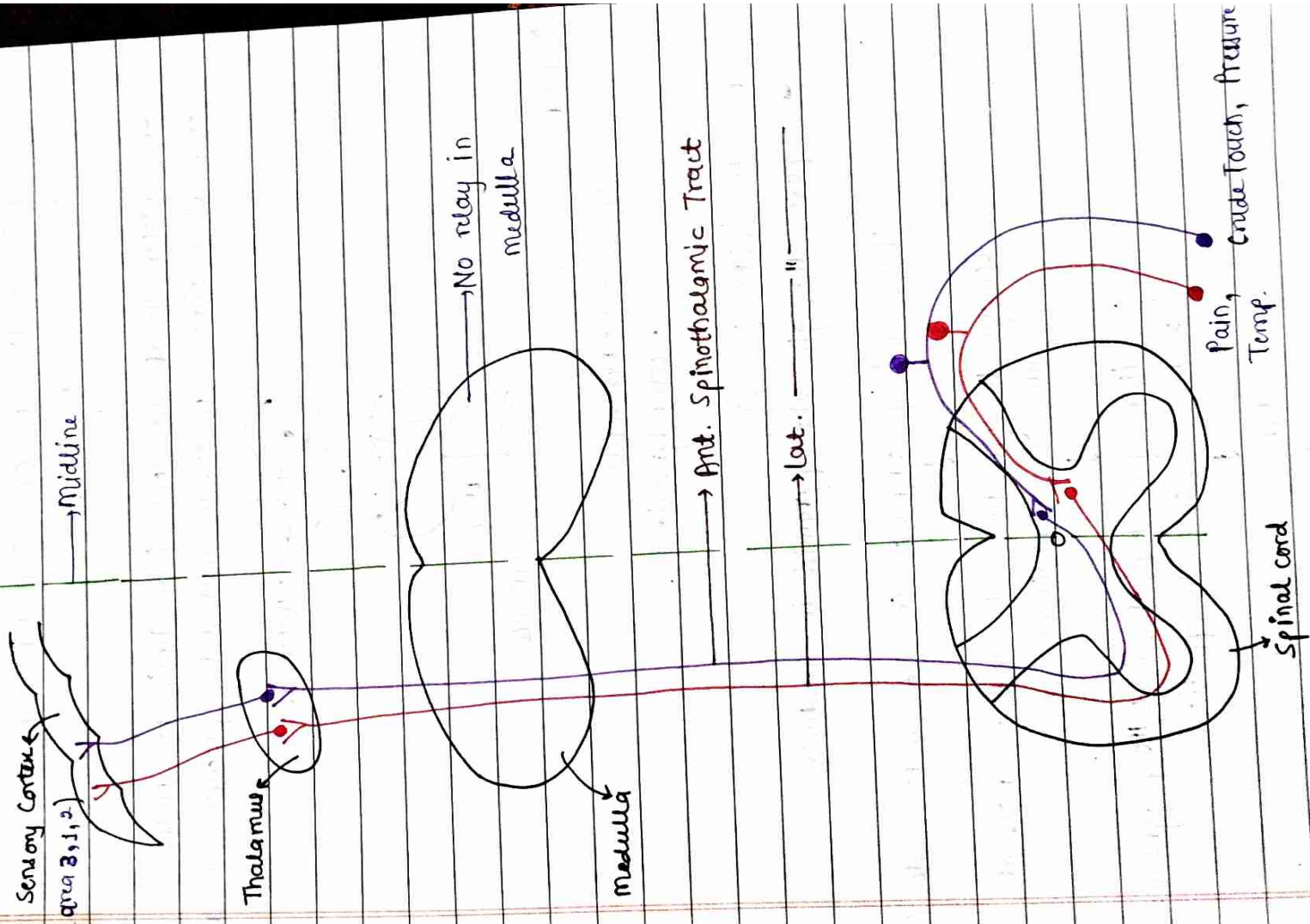
→ for crude touch & pressure.

→ carry same $\frac{1}{2}$ neural tract at T2E, at 2nd point at spinal cord to ant. white column level $\frac{1}{2}$.

★ Lateral Spinothalamic

→ for pain & Temperature

→ all same, the only change is $\therefore$ at spinal cord to lat. white column level.

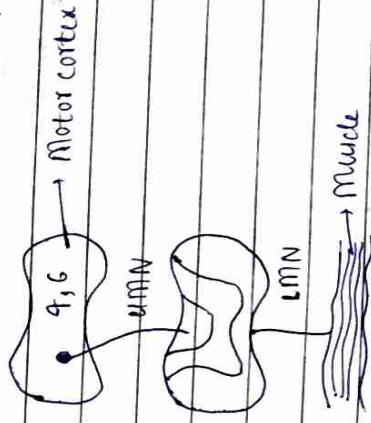


Q9

DESCENDING TRACTS [Motor Tracts]

* Motor Area (4,6)

- Motor signals are sent from brain to LMN.
- LMN directly innervates muscles to produce movement.



* Clinical Classification

- 2 Types →
- Pyramidal
 - Extra Pyramidal

1. Pyramidal (80%)

- pass through the pyramid in medulla

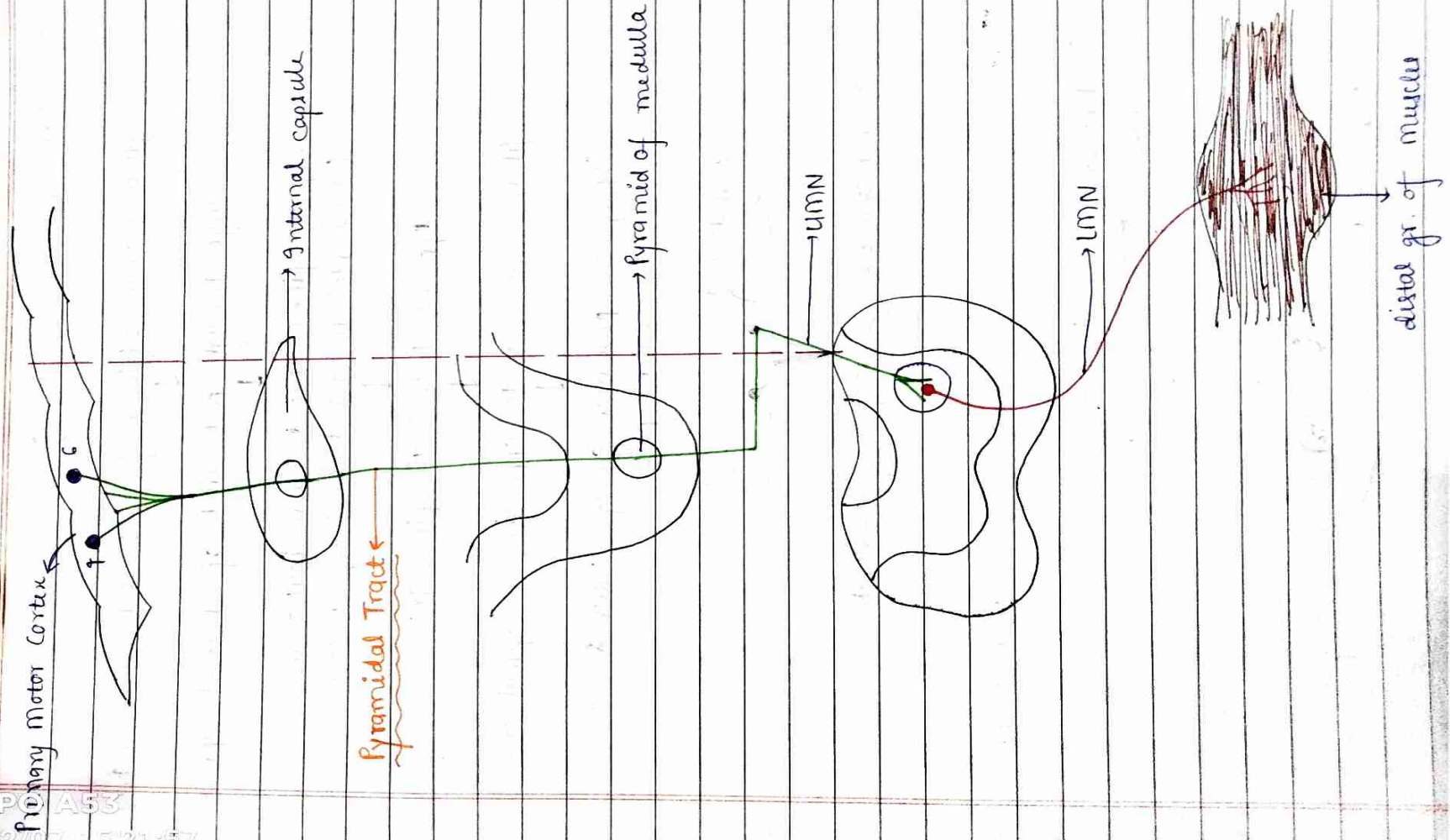
fibres mainly originate from 4° motor cortex (4,6)

↓
converge through the post. limb of internal capsule (corona radiata)

↓
80% of fibres pass through medullary pyramid & then cross over the opp. side

↓
known as **Lat. Corticospinal Tract**

↓
supply → distal gr. of muscles & control skilled voluntary movements



Extra Pyramidal (20%)

fibres mainly originate from 1° motor cortex (9,6)

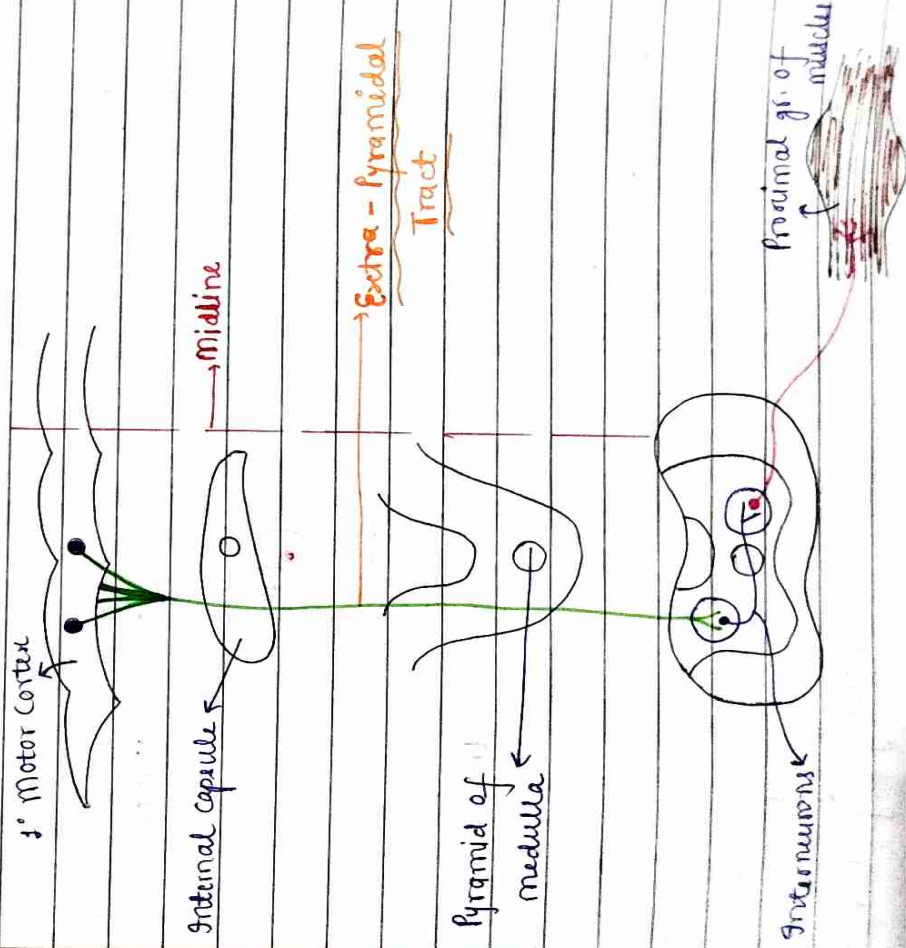
↓
converge through post. limb of internal capsule

↓
20% of fibres descend down ipsilaterally
(same side) in ant. horn of spinal cord

↓
1° as Ant. Corticospinal Tract

↓
fibres cross over to opp. side of spinal cord

↓
Supply :- proximal g.i. of muscles & control posture



Pyramidal	Extra Pyramidal
K/n as Lat. Corticospinal Tract.	K/n as Ant. Corticospinal Tract.
2. 30% fibers arise from pre-motor cortex & 70% from somatosensory area (S1 & S2)	arise from midbrain, pons & medulla.
3. fibers supplies distal gr. of muscles.	supplies proximal gr. of muscles.
4. Control fine & skilled movements.	Control body posture, tone & Egm.
5. Lesion causes <u>spasticity</u>	Lesion causes Rigidity.
6. Axon pass without relaying spinal seg. level & forms synapse directly with motor neuron.	Have many synapse in their descending pathway.

Physiological Classification

Lat. System Pathway	Medial System Pathway
• descends down in lat. column of spinal cord. • Includes :- • Lat. Corticospinal Tract • Rubrospinal Tract	• descend down in ant. & medial column of spinal cord. • Includes :- • Ant. Corticospinal Tract • Reticulospinal • Vestibulospinal • Tectospinal

Corticospinal Tract, Pyramidal Tract, UMN,
used interchangeably



UMN Paralysis

Lesion of descending fibers b/w their origin from motor area & their termination on ant. horn cells in spinal cord.

→ Pyramidal (Corticospinal) tract lesion is UMN as UMN lesion / paralysis

• features

1. Fed muscle tone → Spasticity
2. Muscles are affected in groups
3. Tendon reflexes are exaggerated
4. Superficial reflexes are lost
5. Babinski's Sign (+ve)
6. Nerve nerve conduction.

• Subrospinal → • originate from Red nucleus of midbrain
↓
• descends in lat. column of spinal cord
control skilled voluntary & terminate in lat. gr. of motor neurons
movements that supply distal gr. of muscles.

• Vertibulospinal → Lat. → • from lat. vestibular nucleus
↓
(Deiter's nucleus) of brainstem
Control posture (Lat.) • T. → in medial gr. of motor &
adjust head position in response to interneurons in ventral horn of
angular accn. (med.) spinal cord.

Medial → • from medial vestibular nucleus
T. → on medial gr. of motor & interneurons.

• Reticulospinal → Pontine → • from nucleus reticularis
pontis oralis & nucleus reticularis
pontis caudalis.



T. → medial gr. of motor & interneurons
↓

supply proximal & axial gr. of muscles.

- **Excites** proximal **extensor muscles** & involve in regulation of posture.

Medullary → O. → from nucleus gigantocellularis in reticular form of medulla.

T. → same as pontine

- **inhibits** extensor neurons

• Tectospinal → O. → from tectum / sup. colliculus

• crossover to opp. side

• T. → on medial gr. of motor & interneurons.

- Receives visual inputs

- Regulates contralateral movement of head in response to visual stimuli.

★ Lat. vestibulospinal control posture & reflex inputs from semicircular canals & otolith organs.

→ PARAPLEGIA

- Paralysis of both lower limbs
- Usually occurs due to spinal cord injury | disease that causes Transection of the cord.
- Rarely disease of motor cortex, Cauda equina or peripheral nerves causes paraplegia.

TRANSECTION OF SPINAL CORD

Complete Transsection of Spinal Cord

Occurs due to :-

- Bullet Injury → cause dislocation of spinal cord
- Accidents → Cause ——— / occlusion of blood vessels.

→ Causes immediate loss of sensation & voluntary movement below the level of lesion.

→ Patients ~~remain~~ unaffected but the ~~sp~~ Patient feels as if his lower part of body below the injury does not exist b/c higher centers remain unaffected but spinal centers below the level of injury lose the function.

→ Effect occurs in 3 stages :- 1. Stage of spinal shock.

2. Stage of Reflex activity

3. Stage of Reflex failure

2. Hemisection of Spinal Cord [BROWN SEQUARD SYNDROME]

→ Lesion involving 1 lateral half of spinal cord is c/d hemisection

→ Occurs due to injury in accidents.

Brown Sequard Syndrome

→ occurs due to hemisection of spinal cord.

→ Seen in injury to the spinal cord or in tumors of spinal cord that affects only half of the cord

Sensory Deficit :-

On same side of ~~limb~~ pain & Thermal sensation are lost

- Fine-touch sensation
- Proprioceptive sensation (sensation from tendons, muscles, joints and vibration sense)
- Tactile discrimination are lost.

2. On opp. side of lesion

→ Pain & Thermal sensation are lost

★ Occurs b/c sensation for fine-touch, proprioception & 2 point discrimination ascend up in dorsal column of same-side.

whereas sensation for pain & temperature ascend up in antrolateral system in the opp. side of spinal cord.

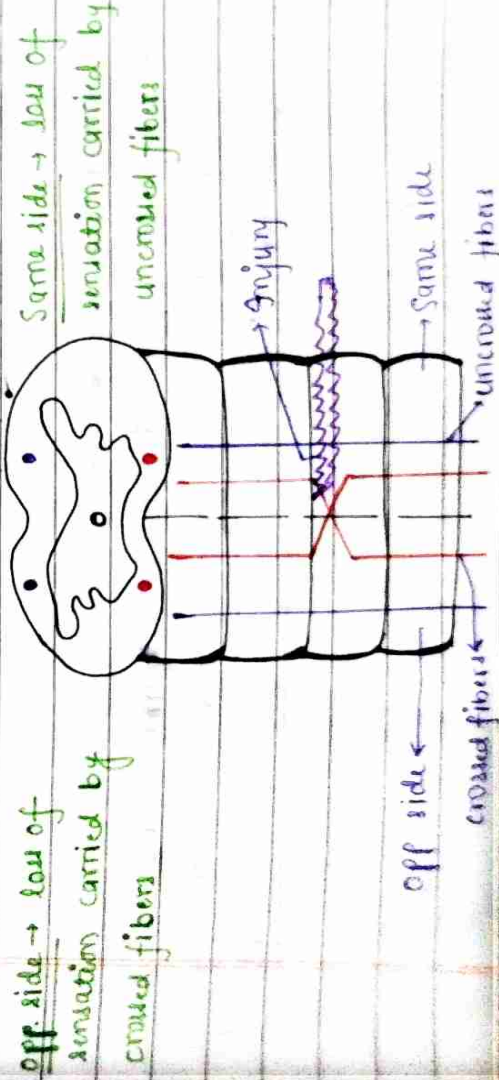
Motor Defect :-

Damage to corticospinal tract on same side of the hemilection



this causes paresis (muscle weakness)

• Spasticity of muscle of same side.



UPPER AND LOWER MOTOR NEURONS

★ Upper Motor Neurons

- Neurons in higher centers of brain.
- control LMN.
- 3 types :-

1. Motor Neuron in cerebral cortex

2. ————— " ————— Basal ganglia & Brainstem

3. ————— " ————— Cerebellum

→ Neurons in cerebral cortex → give origin to pyramidal system

→ Remaining → to extrapyramidal system

★ Lower Motor Neuron

- Are the ant. gray horn cells in spinal cord & motor neurons of cranial nerves nuclei, situated in brainstem & innervate muscles directly.

↓

This LMN constitute final 'common pathway' of motor system

→ work under influence of UMN

★ Effect of lesion of UMN & LMN



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Effects	UMN lesion	LMN lesion
Muscle Tone	Hypertonia	Hypotonia
Paralysis	Spastic type of paralysis	flaccid type of paralysis
3. Wastage of Muscle	wastage of muscle occurs	wastage of muscle occurs
4. Superficial Reflexes	Lost	lost
5. Plantar Reflexes	Abnormal plantar Reflex $\rightarrow$ Babinski sign	Ent
6. Deep Reflexes	Exaggerated	lost
7. Clonus	Ent	Ent
8. Muscle affected	Gr. of muscle is affected	Individual muscle is affected
9. Fascicular twitch in EMG	Ent	Ent
10. electrical activity	Nrml	Ent



PHYSIOLOGY OF PAIN

Pain is an unpleasant sensory & emotional experience associated with actual or potential tissue damage or described in terms of such damage.

★ Types :-

Slow Pain	Fast Pain
1. C-fibers	1. Aδ-fibers
2. Non specific nuclei of thalamus.	2. Specific Nuclei / VPL Nu.
3. Paleospinothalamic	3. Neospinothalamic
4. Non-myelinated fibers	4. Myelinated fibers
5. Most sensitive to pr.	5. Most sensitive to analgesia
6. less velocity of fibers	6. More velocity
7. Conduct impulse only to painful stimuli	7. only to thermal and mechanical stimuli
8. NT → Substance P	8. NT → Glutamic acid
9. less sensitive to electrical stimulation	9. More sensitive to electrical stimulation.

gmk ★ Pain Pathways

2 types :- 1. Paleospinothalamic
2. Neospinothalamic

1. Paleospinothalamic

- oldest pathway
- mainly carries the sensation of slow pain.
- slow conducting multineuron system that mediates poorly



Localized pain from deep somatic & visceral structures are C-fibers

1st order Neurons → Enter spinal cord & terminate in Lamina II of dorsal horn.

2nd order Neurons → decussate & ascend up in

Contralateral spinothalamic pathway

• fibers project to 3 major nuclear gr. forming 3 subsystems :-

1. At medulla (Reticular form)
2. At midbrain (periaqueductal gray)
3. At hypothalamus

3rd order Neurons → In thalamus, fiber terminate mainly in medial nuclear group also old non-specific nuclei



from here 3rd order neuron generates and projects to diffⁿ areas of cortex including limbic & cortical areas.

2. Neospinothalamic

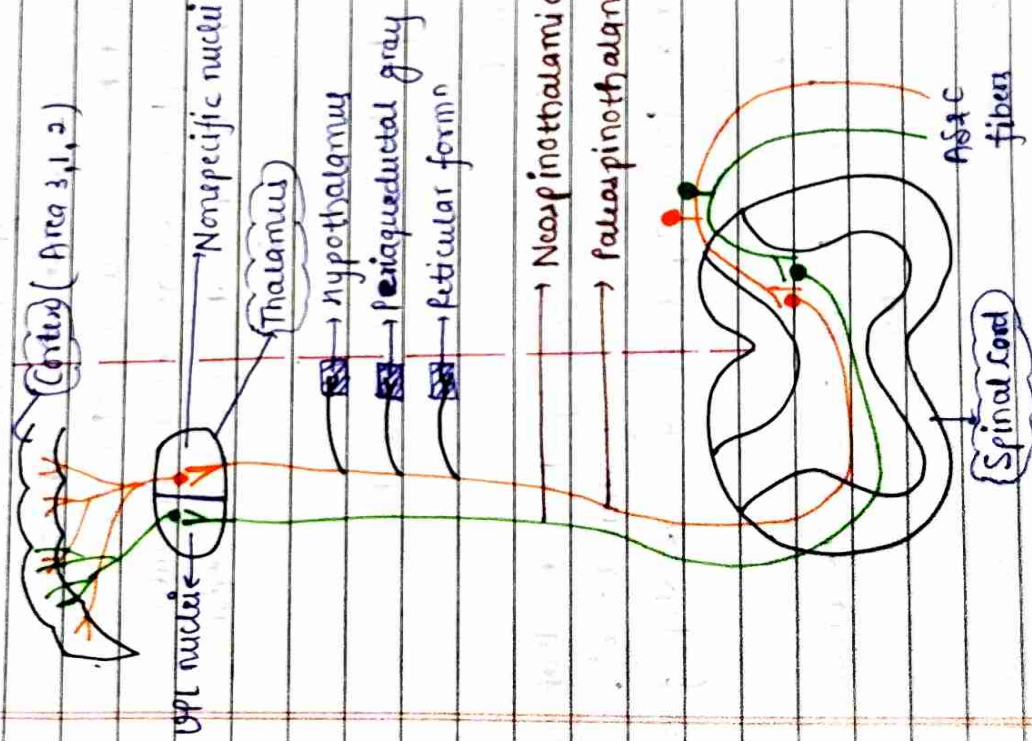
- 1 Most developed in primates
- 1 Carries the sensation of fast pain.
- 1 Mostly AS-fibers

1st order Neurons → Terminate in Lamina I & V in dorsal horn.

2nd order Neurons → Cross over to opp side in same seg. of spinal cord & ascend in **lat. cord** & **spinothalamic tract** in spinal cord there is **topographic organization**

3rd order Neurons → originate from specific thalamic gr. also c/d VPL nuclei / specific nuclei. of thalamus

where 2nd order neuron terminate



Referred Pain

Pain that is perceived at a site adjacent or away from the site of origin.

Deep pain & some visceral pains are referred to other areas but superficial pain is not

* Examples :-

1. Cardiac pain $\rightarrow$ felt in inner part of lt. arm & lt. shoulder.
2. Pain in ovary $\rightarrow$ referred to umbilicus
3. Pain in testis $\rightarrow$ felt in abdomen
4. Pain in diaphragm $\rightarrow$ referred to shoulder
5. Pain in gall bladder $\rightarrow$ to epigastric region
6. Renal pain $\rightarrow$ to loin

* Mechanism /- Theories of Referred Pain

1. Dermatome Theory

- $\rightarrow$ Visceral pain is referred usually to it. that develop from the same embryonic seg. (dermatome).
- $\rightarrow$ Ex:- Hurt & inner aspect of arm develop from same dermatome



$\therefore$ Pain of acute MG radiates to ulnar border of lt. arm.

2. Convergence Theory

- $\rightarrow$ visceral as well as somatic afferent fibers converge on 2nd order neuron in spinothalamic tract.

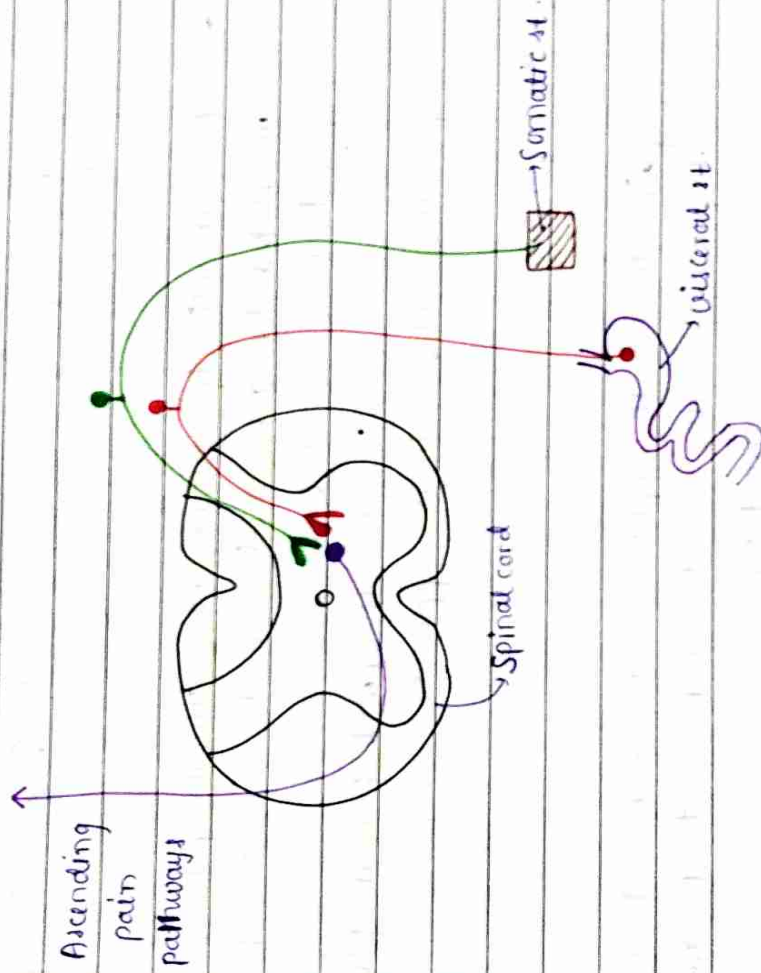


$\therefore$ fiber carrying pain sensation from somatic it also carries sensation from visceral it.

So, cortex sometimes cannot differentiate the site of origin of pain sensation.



∴ signal conveyed by brain for perception is also referred to somatic area in addition to viscera.



Convergence Theory

3. Facilitation Theory

- Collaterals arising from visceral afferents project to spinothalamic neurons that receive afferent from somatic st.

Analgesia System

Endogenous Pain Control Mechanism

[GATE CONTROL THEORY OF PAIN]

Acc. to this, pain can be modulated by peripheral mechanism, especially gating the impulse in spinal cord.

→ Pain stimuli transmitted by afferent pain fibres are blocked by gate mechanism located at post. gray horn of spinal cord.

If gate is opened pain is felt, if gate is closed, pain is suppressed.

* Mechanism

pain stimuli is applied on any part of body, receptors of other sensation such as touch are also stimulated

↓

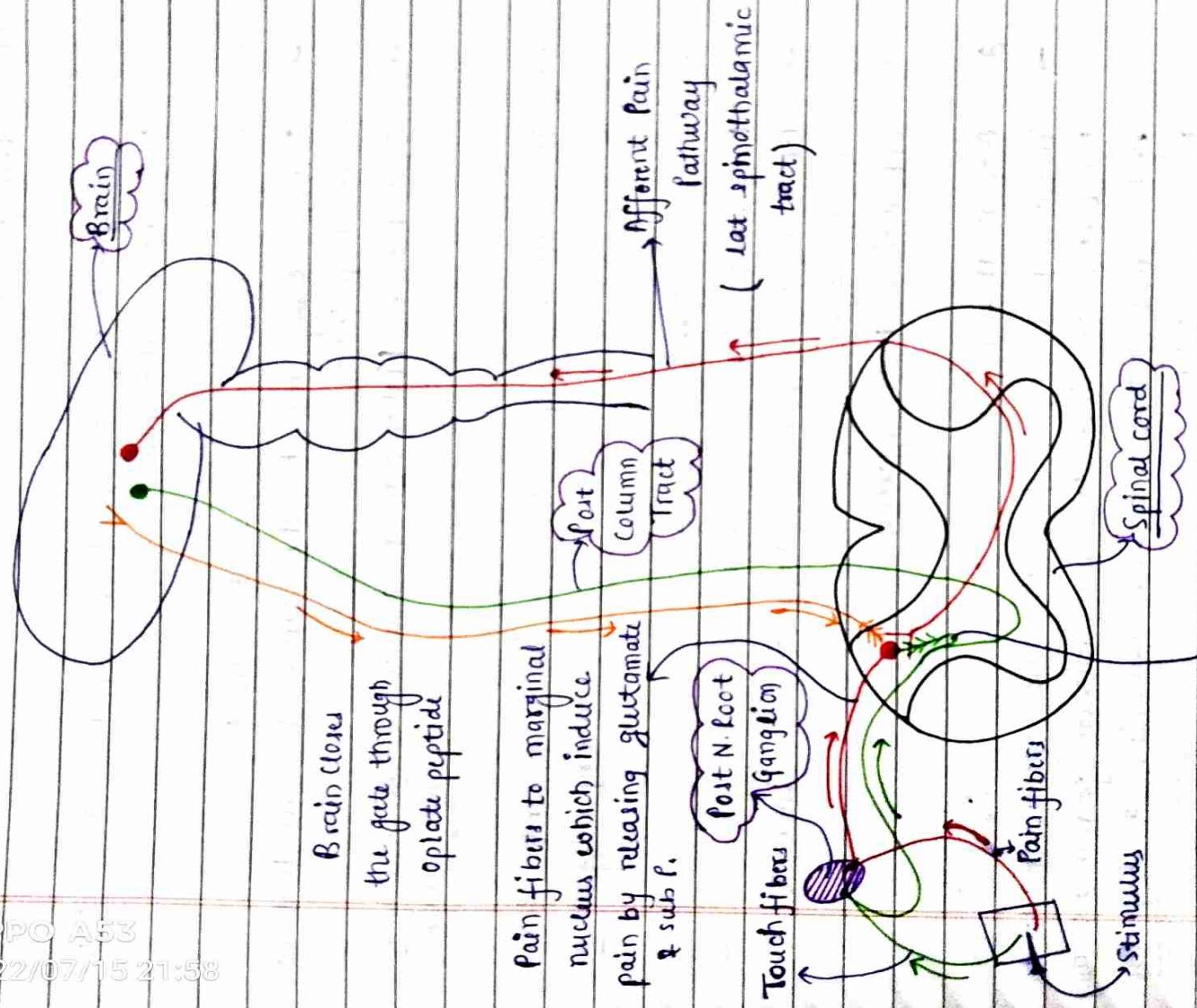
impulse reach spinal cord through post. root, fibres of touch sensation send collaterals to neurons of pain pathway

↓

impulses passing through these collaterals inhibits release of glutamate & Substance P from pain fibres

↓

This closes gate & pain transmission is blocked.



Collateral from post column tract block pain transmission by inhibiting the release of Glutamate by sub. p.

THALAMUS

All afferent impulses on their way to sensory cortex terminate in thalamus.

1. Relay station for all somatic sensations

→ Convey sensory info to the cortex through thalamo-cortical projections.

→ In human & higher order of animals → it is major relay center.

→ In lower order of animals → it is a center for sensory integration.

2. Relay of Special Sensation

→ Relay center for all special sensation except olfaction.

→ lat. & Medial geniculate body receives visual & auditory afferents.

→ VPL receives taste afferents.

3. Arousal Mechanism

→ Thalamocortical ^{fibers} projects from the non-specific nuclei to the cortex contribute to the reticular form activating system that activates arousal mechanism & keeps the individual awake.

4. Subcortical Perception of Sensations

→ Subcortical perception of pain, temp & pr. occurs to some extent in thalamus.

→ Thalamus is not only relay station but also the integration center for these sensation.

5. Motor Functions

It is an integral part of motor loop of the brain

through motor loop. It influences postural movements

- influences planning & programming of movements

6. Memory and Emotions

→ Ant. thalamus receives inputs from mammillary body of the limbic system via mammillothalamic tract

↓

through this, it is concerned with recent memory & emotions

7. Synchronization of EEG

→ Stimulation of intralaminar thalamic nuclei at low d
causes synchronization of EEG waves

↓

old Recruiting response

8. Role in Sleep

→ Ckt linking thalamus & cortex is imp. in generating the pattern of brain activity in sleep-wave cycle.

9. Sensory-motor Coordination

→ Receives all sensory inputs from the body & closely interacts with basal ganglia, cerebellum & motor cortex

↓

∴ It is one of the major pt. in brain for coordination b/w sensory & motor functions



Language and Speech

Dorsal lat. nucleus of thalamus is reciprocally connected with parietal lobe of brain & therefore with language & speech.

15) FUNCTIONS OF HYPOTHALAMUS

1. Endocrine function

- Perform this function via its connection with pituitary gland.

* Control of Ant. Pituitary Functions

- Control secretion of all ant. pituitary hr. by secreting releasing & inhibiting hr.
- Secretes 7 hr :- GHRH, GHRP, GHRH, TRH, CRH, GnRH, PIH

* Control of Post. Pituitary functions

- Hypothalamus secretes 2 hr :- ADH & oxytocin.
- Synthesised by neurons of supraoptic & para-ventricular nuclei of hypothalamus.
- Transported by means of axonic flow through Hypothalamohypophyseal tract.

2. Autonomic functions

- Sympathetic division is regulated by → Post. & lateral nuclei of Hypothalamus.
- Parasympathetic is regulated by → Ant. gr. of nuclei.

3. Circadian functions

- Regular recurrence of physiological processes or activities which occurs in cycle of 24 hr.
- also c/d diurnal Rhythm
- occurs in response to recurring daylight & darkness.
- Cyclic changes taking place in various 'physiological processes are set by means of a hypothetical int. clock c/d Biological clock.

Suprachiasmatic nucleus of Hypothalamus sets the biological clock by its connection with retina via retinohypothalamic fibers.



maintain circadian rhythm of sleep, hormonal secretion, thirst, hunger, appetite.

4. Temperature Regulation

- Regulation of body temp depends on balance b/w the mechanism that controls heat loss & heat gain.
- Hypothalamus plays key role in central mechanisms of temp. regulation.

* Role of Ant. Hypothalamus

- Detect body temp by sensing the temp. of blood & by receiving inputs from thermoreceptors.
- If temp. is above the set point → Ant. Hypothalamus activates mechanism that promotes heat loss by cutaneous vasodilation & sweating.



∴ heat loss ↓ body Temp.

* Role of Post. Hypothalamus

- Activates mechanism that ↑ heat production & promote heat gain.
- Depends mainly on info. provided by cutaneous receptors.
- If temp. is low, post. hypothalamus activates heat conservation by cutaneous vasoconstriction and piloerection
- also ↑ release of catecholamines from adrenal medulla

Many endogenous pyrogens like cytokines act on preoptic area of HT (Hypothalamus) to produce fever

↓

b/c cytokine interleukins & the release of prostaglandins

↓

that produces fever

5. Regulation of food intake (i) Neural Factor

→ Regulated by 2 centers in hypothalamus :-
Feeding and Satiety Center

* Feeding Center → is in lat. HT nucleus

* stimulation leads to uncontrolled hunger & the food intake [Hyperphagia]

• Destruction leads to loss of appetite [anorexia]

* Normally, always active.

* Satiety Center → is in ventromedial nucleus of hypothalamus.

* stimulation causes loss of appetite & cessation of food intake.

• Destruction leads to hyperphagia & animal becomes obese (Hypothalamic obesity)

* Plays an imp. role in regulation of food intake by temporary inhibition of feeding center after food intake.

ole of agust → Provides sensory signals regarding filling of stomach to hypothalamic regulatory centers.

→ ∴ stretching of stomach wall by accumulation of food is immediate factor to inhibit feeding

Date _____
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cii) Hormonal factors

Most of are either secreted from HT or acts on HT to influence food intake

a) Hr. that ↑ food intake

1. Neuropeptide Y
2. Orexin
3. Ghrelin
4. Galanin
5. GHRH

b) Hr. that ↓ food intake

1. Estrogen
2. Dopamine
3. Gut Hr.
4. Oxytocin
5. Peptide YY
6. Leptin

ciii) Metabolic factors

- Plasma glucose conc. is important metabolic factor for regulation of feeding.
- Discharge of ventromedian HT (satiety center) partly depends on its glucose utilization.

6. Regulation of water intake

- HT plays an imp. role in water intake. Two major factors that affects water intake are:-

- Plasma Osmolality & Extracellular fluid volume.

- HT respond to change in both these factors.

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Thus, HT is major thirst center in brain.

→ OLT & SFO → HT area that respond to thirst

7. Control of Reproductive Functions

- **★** **HT** secretes gonadotropin-releasing **hr.** (**GnRH**) that plays a crucial role in reproductive functions & sexual behaviour.
- **GnRH** secreting neurons are located in Preoptic Area
- **HT** also control sexual behaviour in experimental animals.

8. Other functions

★ Influence on Emotions

- ★ The behaviour of animal & humans mostly affected by 2 responding system in HT & other st. of limbic system.

The responding systems are concerned with whether the sensations are pleasant or painful. These 2 qualities are called Reward (Pleasant / Satisfaction) and punishment (aversion / avoiding).

↓

these 2 systems work opp. to each other.

★ Role in Sleep

- 3 subcortical regions that induce slow wave sleep
- Diencephalic sleep zone is one → major part of this zone is post. HT.
- Preoptic Area of HT also induces sleep.
- By regulating circadian rhythm, HT controls sleep.

★ Role in Immunity

- influence immunity by controlling secretion of ACTH

(16)

CEREBELLUM

- largest part of hind brain, consist of 2 lat lobes / parts of cerebellar hemispheres connected in the midline by a narrow central region of the vermis.

* Divisions of Cerebellum

1. Anatomical Division

- Ant. lobe → Includes lingula, central lobe & culmen
- Post. lobe → Includes lobulus simplex, declive, tuber, pyramid, uvula, paraflocculi
- Flocculonodular lobe → Includes nodules & lat. extensions on either side of flocculus.

2. Phylogenetic Division

- Archicerebellum → oldest part to develop
- Paleocerebellum → next part to appear
- Neocerebellum → latest part to develop

3. Functional Division

- * Vestibulocerebellum → Includes :- 1. flocculonodular lobe
2. vestibular connections

- Nucleus fastigial → effector nucleus
- Concerned with body posture & Egm

- * Spinocerebellum → Includes :- 1. Parts forming paleocerebellum

- Nucleus globosus & nucleus emboliformis i.e., Nucleus interpositus → effector nucleus

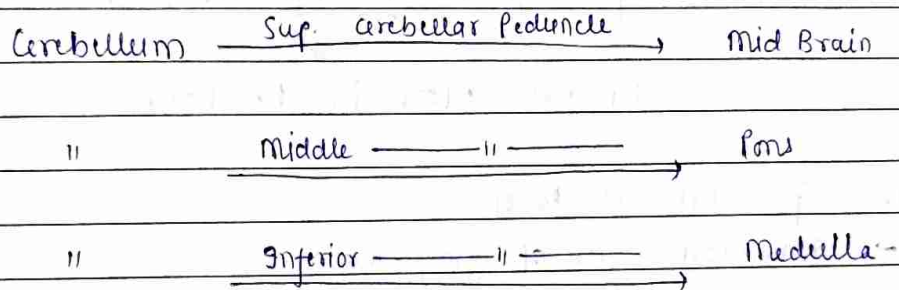
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• receives proprioceptive inputs from spinal cord.

- Concerned with control of axial (trunk) & limb muscles postural reflex.

★ Corticocerebellum → Includes :- 1. whole post. lobe except pyramis & uvula (parts of vermis)

- Nucleus dentatus → effector Nucleus
- Receives input from cerebral cortex & pons.
- Concerned with smooth performance of highly skilled voluntary movements.

★ Cerebellar Peduncle



★ Functional Anatomy of Cerebellum

- Made up of :-
1. Outer gray matter / Cerebellar Cortex
 2. Inner white matter
 3. Gray mass of Cerebellar nuclei

↓
located within white matter

★ Gray Matter

→ Made up of st. arranged in 3 layers :-

1. Molecular / Plexiform layer
2. Purkinje cell layer
3. Granular cell layer

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1. Molecular / plexiform layer
→ Outermost layer of cortex



→ Consist of :- 1. Stellate cells (star-shaped)
2. Basket cells

→ Also contains :- 1. Parallel fibers → axons of granule cells
2. Climbing fibers
3. Dendrites of Purkinje cells & Golgi cells.

2. Purkinje cell layer

→ Situated in b/w and is the thinnest layer.
→ Have a single layer of Purkinje cells (flask shaped).

↓
Biggest neuron in the body

3. Granular cell layer

→ Innermost layer of gray matter
→ In b/w Purkinje cell layer & cerebellar white matter.
→ Consist of :- 1. Granule cells
2. Golgi cells] Interneurons

** Afferent fibers to Cerebellar Cortex

→ Receives inputs from other parts of brain through
2 types of nerve fibers :-

1. Climbing fibers
2. Mossy fibers

1. Climbing fibers [Aspartate → Excitatory]

→ arise from the neurons of Inferior olivary Nucleus

↓

situated in medulla & reach the cerebellum

↓

through olivocerebellar tract

→ After reaching cerebellum, climbing fibers ascend into molecular layer and terminate on dendrites of Purkinje cells.

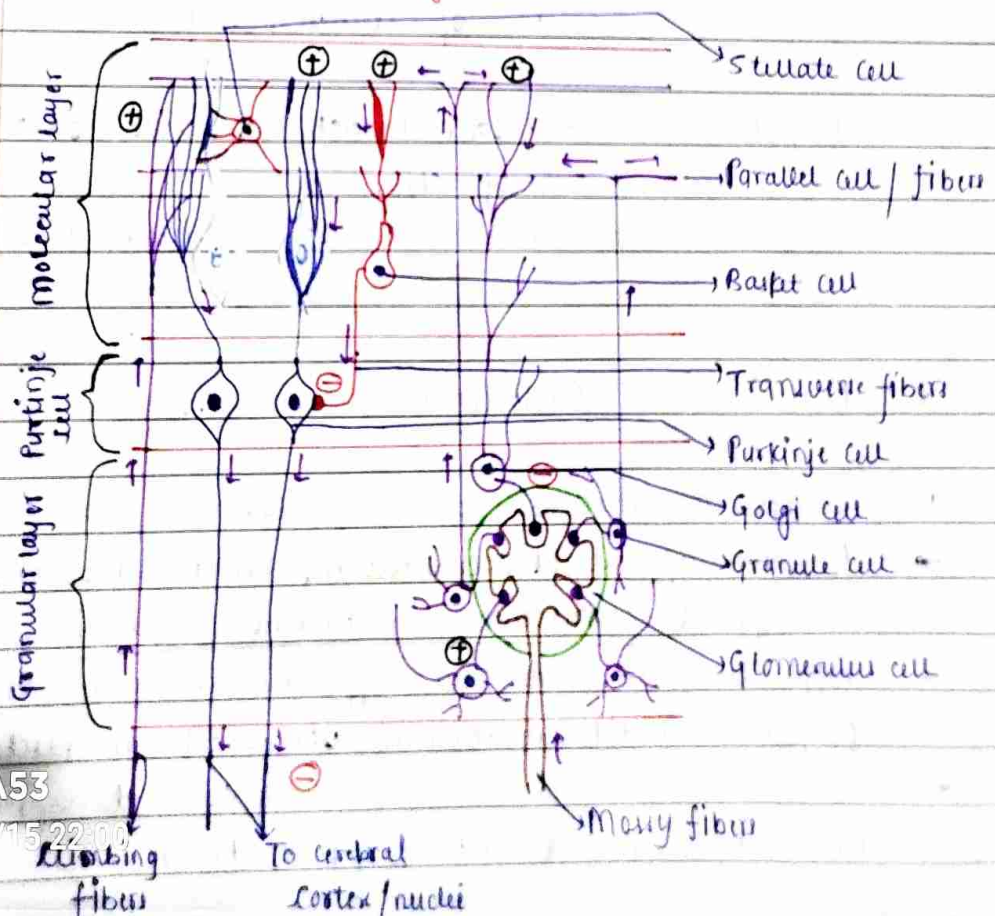
2. Mossy fibers [Glutamate → excitatory]

→ Arise from motor area of cerebral cortex, pons, medulla, spinal cord & send collaterals to cerebellar nuclei before reaching the cortex.

→ Dendrites of granule cell & axon and dendrites of Golgi cells synapse on mossy fibers.

* Only Granule cell is excitatory → release Glutamate (NT)

* Other 4 are inhibitory → release GABA (NT)
(Stellate, Basket, Golgi, Purkinje)



★ Cerebellar Nuclei

1. Fastigial N.
2. Globosus N.
3. Emboliform N.
4. Dentate N. / N. Dentatus → **Largest cerebellar Nucleus**

★ White Matter

→ formed by afferent & efferent N. fibers.

classified in 3 groups :-

1. Association fibers → Connect diffⁿ regions of same cerebellar Hemisphere.
2. Commissural fibers → Connect the areas of both halves of cerebellar cortex.
3. Projection fibers → • Connect cerebellum with other parts of CNS.
• all 3 Peduncles (already done)

★ Functions of Cerebellum

1. Control of Postural Balance & Eqm

→ **function of vestibulocerebellum**, which has extensive & reciprocal connection with vestibular nuclei

→ Afferents from vestibular apparatus in inner ear project to vestibulocerebellum via vestibular nuclei.

→ Vestibulocerebellum in turn send signals to spinal cord via vestibulospinal & reticulospinal tracts.

2. Vestibulo Ocular Reflex →

- Vestibuloocular is concerned with learning induced changes in vestibulo-ocular reflex.

3. Smoothing and Coordination of movements

- function of spinocerebellum that receives proprioceptive and sensory inputs from all body parts.
- By comparing Plan with Performance, it smoothen & coordinate movements.

4. Control of Posture

- Vermal area of spinocerebellum has profound influence on posture.

5. Control of Skilled Voluntary Movements

- Paravermal area of spinocerebellum.

★ Cerebellum controls all aspects of movements from rate, range, force and direction to termination of movement.

6. Planning and Programming of movements

- function of Neocerebellum
- Planning of each sequential movement is done by ~~each~~ hemispheric part of cerebellum.

7. Control of muscle tone & Stretch reflex

- Cerebellum influences the activity of the ~~major descending motor system~~ through its output from fastigial nucleus, especially vestibulospinal and reticulospinal.

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8. Control of movement of same side of the body

- Motor cortex of one side is connected to cerebellar hemisphere

of opp side through a closed feedback ckt, k/n as Cerebral-Cerebellar-Cerebral ckt.

→ Thus, each cerebellar hemisphere influence output of opp cerebral cortex.

↓

but cerebral cortex via corticospinal tract ^{that} decussates to opp. side just after passing medullary pyramid control function of contralateral half of body.

↓

∴ due to double decussation, each cerebellar hemisphere control movements of its own side of body.

9. Learning & Improvements of Motor Skills

→ Plays a critical role of comparing inf. of the ongoing movements & the changes required to improve performance.

→ For every activity, cerebellum improves learning & performance.

→ also controls long-term adjustment of motor skills.

10. Prevents overshoot & damps movements

→ There is tendency to overshoot, when a movement is done faster.

→ But, cerebellum prevents the overshoot & tremor.

11. Eyeball Movements.

→ Paraflocculus & Pyramis are concerned especially in upward movement of eyeball.

12. Vestibular functions

Cerebellum controls all vestibular functions like balance during movement,

★ Cerebellar lesions

- May be due to tumor, abscess or an injury
- Excess alcohol intake also causes lesion.
- During lesion there is disturbance in posture, eqm & movements.
- In unilateral lesion, symptoms appears on affected side b/c cerebellum controls the same (ipsilateral) side of body.
- Symptoms / features are :-

1. No paralysis but disturbance in smoothness of movement.
2. Pendular knee jerk is elicited
3. No sensory deficit

4. Hypotonia → reduction of tone in muscle

Hallmark of disorder 5. Ataxia → lack of coordination of movement.

It manifest in following forms :- Drunken gait, Scanning speech, Dysmetria, Intention tremors, Rebound phenomena, Adiadochokinesia, Decomposition of movements.

6. Inability to carry out long term adjustments

7. Nystagmus → to & fro involuntary oscillatory movement of eye.

8. Charcot's Triad → Nystagmus + Intention Tremors + Scanning speech.

★ Cerebellar function Test

1. Test for coordination :- Upper limb → finger-nose test
Lower limb → Knee-heel test

2. Test for postural stability → stand erect with eyes opened

3. Gait Test

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1. Adiadochokinesia → unable to perform rapid supination & pronation of forearm.

3. Past pointing → movement goes beyond the intended point (dysmetria).

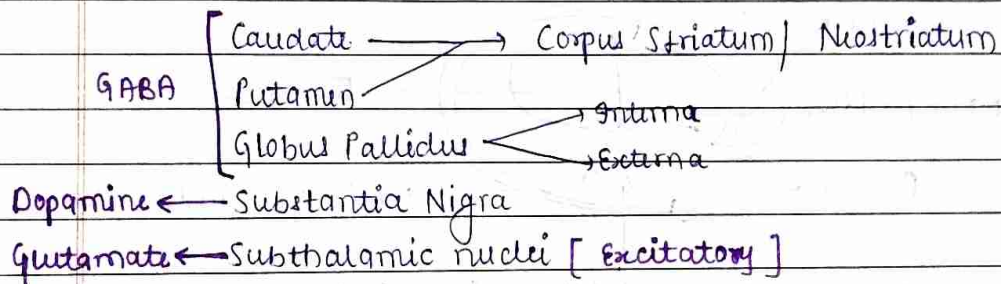
⑪ BASAL GANGLIA

- Scattered mass of grey matter submerged in subcortical substance of cerebral hemisphere. Basal ganglia form the part of extrapyramidal tract, which is concerned with motor activities.

* Components

1. Corpus Striatum
2. Substantia nigra
3. Subthalamic nucleus

Can Parkinson Get NrmL Suddenly



1. Corpus Striatum

- Situated at base of cerebral hemisphere in relation to thalamus

→ 2 parts :-

- a) Caudate N. →
- lies Medial to int. capsule
 - Related to lat. ventricle throughout its length.
 - Has Head & Tail

b) Lenticular N. →

- lies lat. to int. capsule

- 2 portions :-
- 1. Outer putamen
- 2. Inner globus pallidus.

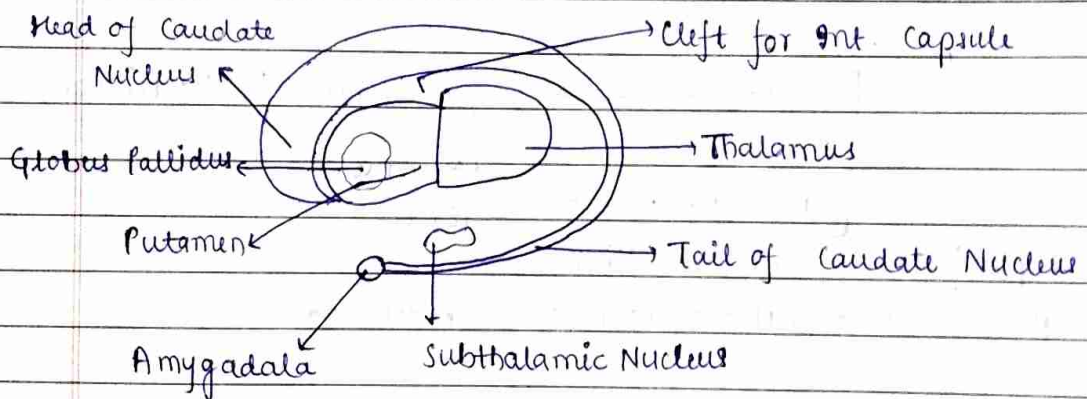
- Putamen + Caudate Nucleus $\rightarrow$ Neostriatum
- Globus Pallidus $\rightarrow$ Paleostriatum.

2. Substantia Nigra

- Situated below red nucleus
- Made of large pigmented & small non-pigmented cells.

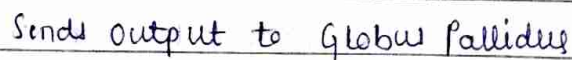
3. Subthalamic Nucleus of lvs

- Situated lat. to red nucleus & dorsal to nigra.



★★ feedback ckt b/w Cerebral Cortex & Basal Ganglia

c. Striatum receives input from cortex & thalamus

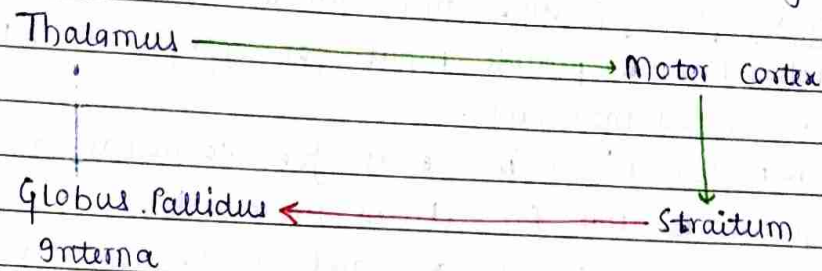


Project to Thalamus

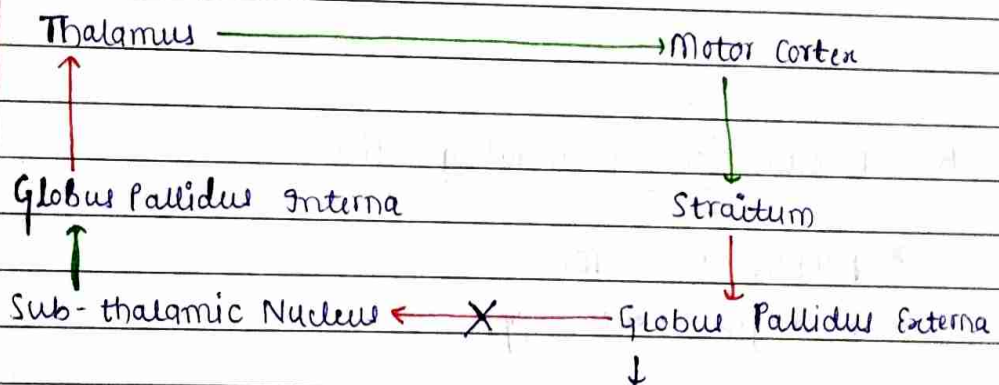
53 Inf. received by thalamus is converted back to cortex completing the ckt.

★ Pathways

1. Direct [To move initiate Striatum] → facilitate Movement



2. Indirect [To move initiate Striatum ; Enough, Stop Now]
↓
Inhibit Movement



Normally inhibits the nucleus but now is itself inhibited so cannot inhibit the nucleus & ∴ nucleus excites GPI & GPI inhibits thalamus to stop movement.

Imp.

★ Functions

1. Involved in Planning & Programming of motor activities
2. Control Posture so disease causes profound postural abnormalities.
3. Inhibits stretch reflexes by stimulation of caudate nucleus.

4. Neostriatum regulates subconscious gross movements.
5. Caudate Nucleus plays role in cognitive functions.
6. Lesions of caudate nucleus is associated with dysarthric aphasia (difficulty in articulating words).
7. Globus Pallidus provide input for appropriate muscle tone for skilled movement.
8. Substantia nigra is center for coordination of impulses essential for skilled movements.
9. Basal ganglia controls associated movements that occur normally with various body movements.
10. Lesion of BG in animal not lead to motor deficit. However, disease in human results in severe motor dysfunctions.

(functions इस वही copy से भी read करना है।)

★ Dysfunctions of Basal Ganglia

★ PARKINSON'S DISEASE

→ Read from other copy

★ HUNTINGTON COREA

→ Read from other copy

⑮ FUNCTION of LIMBIC SYSTEM

Primarily involved in Motivation, Addiction, Emotions of rage & fear, sexual behaviour & autonomic responses.

1. Motivation

- Diffⁿ areas in brain that on stimulation produces either pleasurable feeling (rewarding) or unpleasant (aversive) feeling.
- System that produce feeling of pleasure → Reward system
 " displeasure → Avoidance system

- * **Reward System** → • Mainly consist dopaminergic pathways.
- Nucleus accumbens → major reward system in which dopamine is major NT.

- * **Avoidance System** → • Includes lat. portion of post. Hypo-thalamus, dorsal midbrain & cortex.
- feelings on stimulation range from fear to terror.

∴ also c/d Punishment System

2. Addiction

- Compulsion to repeatedly use a sub. in spite of knowing the true impact of sub. on health.
- Commonly occurs to opiates such as Morphine & Heroin.
- Major mechanism of addiction is to rise in dopamine conc. in reward system of brain.

3. Emotions of Fear, Anxiety, Rage & Aggression

- * Fear → • Center is in Hypothalamus (HT) * Amygdala.
in humans, lt. amygdala is activated by looking at
ugly faces that evoke fear.

Date _____
Page _____

★ Anxiety → • Nrmal emotional reaction to a stimulus in which there is uncertainty / doubt about future and apprehension for the unknown.

• Center is frontal part of temporal lobe.

★ Rage & Placidity → • Rage is anger r/n in which person loses his temper in response to minor stimulus.

• Placidity is emotional state in which individual is not disturbed even in response to major irritating stimulus.

★ Aggression → • Violent form of rage

• Testosterone is k/n to produce aggressive behaviour.

7. Sexual Activity

• Includes Sexual reflexes & behaviour.

↓
Coordinator mainly in spinal cord & brainstem

• Sexual Behaviour

↓
coordinated primarily by limbic system.

In males → • Lesion of neocortex inhibits sexual behaviour which shows that connection of limbic system with cortex is required for integration of same.

★ • Bilateral lesion of limbic system + Destruction of amygdala

↓
Intense sexual activity (Hypersexuality)

In females → • Sexual appetite rises in pre-ovulatory phase through the basal desire for mating remains throughout the menstrual cycle.

• lesion of HT abolish sexual activity in females.

5. Autonomic functions / Responses

- HT → major role in autonomic functions so called Head ganglion of ANS.
- Innular & Prefrontal area of cortex → involved in regulation of Autonomic functions.
- ★ Part of limbic system that projects to brainstem & spinal cord have major influence on autonomic functions.

(19) RETICULAR FORMATION

→ Reticular formⁿ is diffused mass of neurons and nerve fibers, which forms an ill-defined meshwork of reticulum in central portion of brainstem.

* Situation of Reticular formⁿ

→ R.F. is situated in Brainstem.

→ Extends downwards into spinal cord & upward up to thalamus & subthalamus.

* Organization

→ Constituted by 5 gr. of nuclei, all are structurally & functionally distinct.

1. Raphe Group

→ Situated along the midline of brainstem.

→ Secrete Serotonin, which is an inhibitory NT.

2. Paramedian Group

→ Includes nucleus reticularis paramedianus & pontine

→ concerned with motor functions

3. Lateral Group

→ Situated in lat. one third of tegmentum.

→ Neurons of these nuclei receive sensory signals from C.N., cerebellum & spinal cord.

4. Medial Group

→ Situated in medial 2/3rd of tegmentum.

→ Associated with motor functions.

5. Intermediate Group

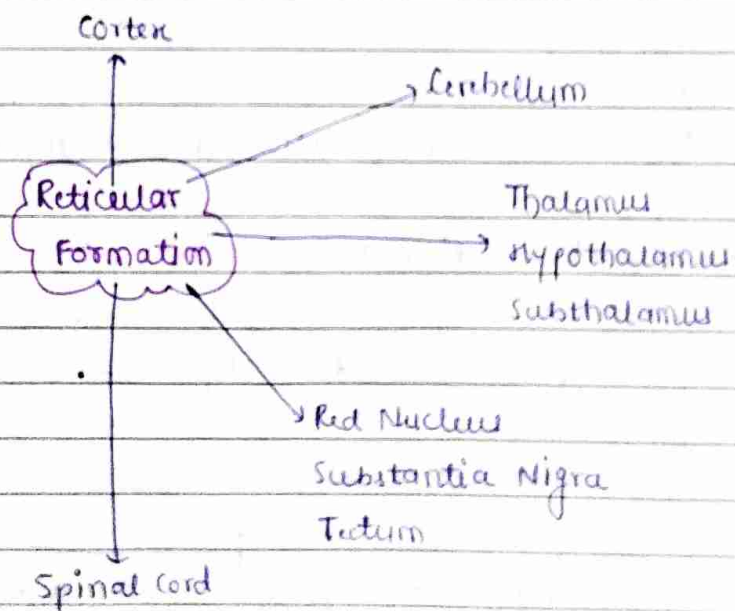
- Ant only in medulla.
- Situated in b/w the lat & medial group.
- Concerned with autonomic regulation of respiration, H.F. & B.P.

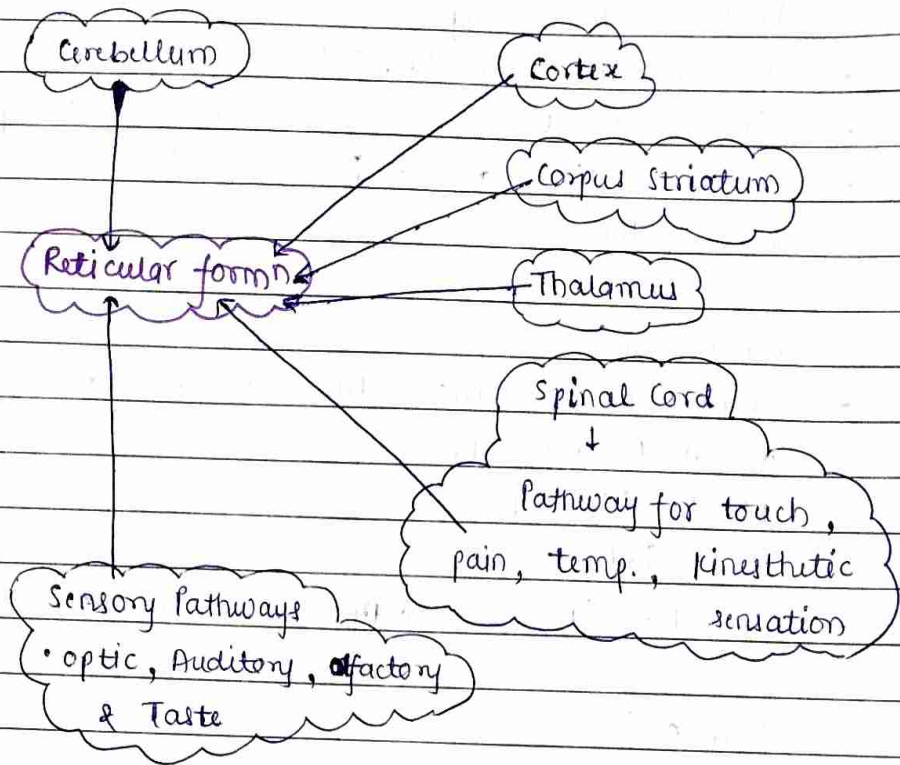
* Divisions

- Divided in 3 divisions based on location of brainstem :-
 - a) Medullary R.F.
 - b) Pontine R.F.
 - c) Midbrain R.F.
- Each has its own collection of nuclei

* Connections

- Sends fiber to following parts of CNS :-





Efferent Connections of Reticular formation

★ Functions

→ Based on function, divided into 2 gr/ system :-

- Ascending Reticular Activating System (ARAS)
Concerned with arousal phenomena, alertness, maintain-
-ance of attention & wakefulness.

• Descending Reticular System

Descending facilitatory

- Facilitation of somatomotor activities.

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" — vegetative functions.

Descending Inhibitory

- Control of somatomotor activities
- Control of vegetative functions.

②① MUSCLE SPINDLE

→ Read from other copy

②① SLEEP

→ Read from other copy

②② MEMORY

→ defined as retention of learned information & experiences. The stored inf. should be retrieved and utilized at anytime in life whenever needed.

→ Memory has 4 stages :-

1. Registration of memory that includes proper perception and attention → failure of learning & memory occurs due to impaired perception & attention b/c the material to be learned is never registered & assimilated.
2. Integration & Retention → Learned experiences are processed & integrated by various st. in CNS & then retained at appropriate place in brain in form of short-term / long-term memory.
3. Recognition & Recall → At appropriate time & place, memory is recalled for proper use.
4. Reutilization → Utilized for improvement of further learning.

★ Types

1. Short Term Memory

→ Memory that lasts for seconds to hours.



during this processing in hippocampus & elsewhere lays down long-term changes in synaptic strength.

→ Working Memory → form of short-term memory that makes inf. available for a brief period.

→ By repeated training, it can be turned into long term, which depends on a process of consolidation.

→ During this, the memory traces & subject to disruption by trauma & various drugs.

2. Long-Term Memory

→ Stores inf. for years together & sometimes for life.

→ Traces are remarkably resistant to disruption.

→ Broadly divided into explicit & implicit memory. Some forms of implicit memory also involve short-term & long term memory.

imp.

★ Conditioned Reflexes

→ It is the acquired reflex that requires learning, memory & recall of previous experience.

→ Acquired after birth & it forms the basis of learning.

* Classification

→ Classified into 2 types :-

- a) Classical Conditioned Reflexes
- b) Instrumental — " —

a) Classical Conditioned Reflexes

→ Those which are established by a conditioned stimulus, followed by an unconditioned stimulus.

→ Type & Properties

- i) Positive / Excitatory Conditioned reflexes
- ii) Negative Conditioned reflexes

• Positive C.R. → 3 types

1° → reflex developed by one unconditioned stimulus & one conditioned stimulus.

2° → reflex developed with one unconditioned stimulus and 2 unconditioned stimuli.

3° → on this 3rd conditioned stimulus is added & the reflex is established.

Negative C.R. → 2 types

• Ext. | Indirect Inhibition

→ If the CS is presented indefinitely without UCS, the response decreases & eventually stops.

externally just after the application of UCS.

b) Instrumental / Operant Conditioned Reflex

→ Those reflex in which the behaviour of the person is instrumental.

→ This type of reflex is developed by the conditioned stimulus, followed by a reward or a punishment.

→ also cld operant / Skinner conditioning

→ During development of this type of reflex, animal is taught to perform some task, to obtain a reward or to avoid a punishment.

→ Accordingly, it is of several types :-

1. Conditioned Avoidance R.

2. Food — " —

3. Conditioned Reward R.

• Conditioned Avoidance R.

→ Reflex by which animal is trained to avoid an electric shock, by pressing a bar.

• Food Avoidance R.

→ An animal is given tasty food along with injection of a drug which produces nausea & vomiting / sickness, animal starts avoiding / hating that food

• Conditioned Reward R.

→ If animal is rewarded by banana by pressing a bar, animal repeatedly presses the bar.

* Instrumental reflex plays an imp. role during the learning process of a child & also responsible for behaviour pattern of an individual.

- * Physiological Basis of Conditioned Reflexes
learning and Memory forms the physiological basis of conditioned reflexes.

APPLIED

* Dementia

- Consisting of several intellectual inability.
- 1. Deficit occurs for many cognitive functions including learning & memory.
- 2. Occurs in many condⁿ that affect cortical functions.
- 3. Most common is senile dementia
- 4. Pathological dementia → seen in Alzheimer's disease -
- 5. Drug induced & Alcoholic dementia → uncommon

- * Dementia is progressive deterioration of intellect, emotional control, social behaviour & motivation associated with loss of memory.

* Alzheimer's Disease

- Common degenerative disease of brain characterized by premature & progressive dementia.

• Etiology

- Occurs due to degeneration, loss of functions & death of neurons in many parts of brain, particularly cerebral hemispheres, hippocampus & pons.
- There is reduction in synthesis of most of NT, especially acetylcholine due to lack of enzyme choline acetyltransferase.

- Pathology

1. Presence of neurofibrillary "tangles" in nerve cells cytoplasm → cytopathologic hallmark of disease
2. Appearance of neuritic plaques in cerebral cortex.
3. Granulovacuolar degeneration.

- Features

1. Usually patient is above 50 yr.
2. Progressive development of forgetfulness
3. Starts with loss of short-term memory & later followed by loss of other cognitive functions.
4. Dyarnomia → forgetting words especially names, usually occurs.

- Treatment

- Cerebral vasodilators, High dose of Vit B, C & E.
- Trials of oral choline, lecithin & cholinergic precursors.

(23) AUTONOMIC NERVOUS SYSTEM

* SYMPATHETIC DIVISION

→ also c/d Thoracolumbar outflow

↓
b/c preganglionic neurons are situated in lat. gray horns of 12 thoracic & 1st two lumbar.

→ leave the spinal cord through ant. nerve root & white rami communicantes

↓
terminate in post-ganglionic neurons (situated in sympathetic ganglion).

→ Effect of sympathetic division on stimulation

1. Eye → ciliary muscles → Pupil → Relaxation
Dilatation

2. lacrimal glands → ↑ in secretion

3. Salivary glands → ↓ in secretion & vasoconstriction

4. GIT → motility Inhibition
Secretion ↓ & ↑

sphincters Constriction
Smooth muscles Relaxation

5. Gall bladder → Relaxation

6. Urinary bladder → Detrusor muscle Relaxation
ant. sphincter Constriction

7. Sweat Glands →	Is in secretion
8. heart - rate & force →	Is
9. Blood Vessels →	Constriction of all except of heart & skeletal muscle
10. Bronchioles →	Dilation

* FIGHT - OR - FLIGHT RESPONSE

- Typical widespread response of sympathetic activation.
- Occurs in initial situations of life when I has to either fight the situation or flee from situation
- Many component of response are due to direct effect of sympathetic stimulation, the secretion of catecholamines from adrenal medulla contributes

→ Effects are :-

1. Sympathetic stimulation of CNS T₁₂ & L₁.
2. → → Salivary gland
Via salivation
3. → → Sweat gland
cause secretion of sweat
4. Fixed exchange of blood gases at lungs
5. Supply of metabolic substrate T₁₂
6. cutaneous vasoconstriction occurs
7. Piloerection occurs
8. Pupillary dilation occurs
9. Bowel & Bladder temporarily ceases
10. Stimulation of Reticular system makes the individual maximally alert.

★ PARASYMPATHETIC DIVISION

→ also cld Craniosacral outflow

b/c fibres of this division start from brain & sacral seg. of spinal cord.

Effect of parasympathetic division on stimulation

Eye → ciliary muscles	Contraction
Pupil	Contraction
Lacrimal glands	Inc in secretion
Salivary glands	Inc in secretion
GIT → Motility	Acceleration
secretion	Inc
Sphincter	Relaxation
Smooth muscles	Contraction
Gall Bladder	Contraction
→ Detrusor muscles	Contraction
9th sphincter	Relaxation
Sweat glands	—
Heart - rate & force	↓
Blood vessels	Dilation
Bronchioles	Constriction

★ Neurotransmitters of ANS

→ Sympathetic fibers → • Preganglionic fibers :- Ach

• Postganglionic noradrenergic fibers :- Noradrenaline

Parasympathetic fibers → • Preganglionic fibers :- Ach

• Postganglionic fibers :- Ach

★ Parasympathetic ganglion are located either close to the organ or embedded in the organ. Therefore, preganglionic neurons are much longer than postganglionic.



(24)

ELECTROENCEPHALOGRAPH (EEG)

- It is the study of electrical activity of brain.
- It is the graphical recording of electrical activity of brain.

* Significance

- Useful in the diagnosis of neurological disorders & sleep disorders
- Pattern alters in following disorders :-
 - Epilepsy → excessive discharge of impulse from cerebral cortex
 - Disorders of midbrain → affects ascending reticular activating system
 - Subdural Hematoma → collection of blood in sub-dural space.

* Method of Recording

- Electroencephalograph → instrument used to record EEG.
- electrode clip scalp electrodes from the instrument are placed over skull / over brain by piercing.
- electrodes are → unipolar & bipolar

when bipolar is used → both the terminals are placed in diffⁿ part of brain.

when unipolar is used → active electrode is placed over cortex & indiffⁿ is kept in some part of body away from cortex.

* waves

- May have synchronized / desynchronized waves.
 - ↓
 - ↳ irregular & variant
 - regular & invariant

* Normal EEG

An normal person EEG has 3 D bands :-

- α -rhythm
- β -rhythm
- S-rhythm

→ EEG in children also shows theta wave

1. α (Alpha) Rhythm

- Consist of rhythmical waves, so are synchronized waves.
- Obtained in inattentive brain / mind as in drowsiness, light sleep or narcosis & diminished by opening of eyes
- Abolished by visual stimuli or any other type of by mental effort.
- Are most marked in parieto-occipital area.

2. β (Beta) Rhythm

- Are asynchronous waves.
- Recorded during mental activity or mental tension or arousal state. So, not affected by opening of eyes.

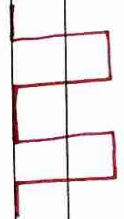
3. Delta Rhythm

- Common in early childhood during waking hours.
- In adult, occurs mostly during deep sleep.
- Not affected by opening of eyes.
- Frequent in adult in condition other than sleep indicates tumor, epilepsy, Fred intra-cranial pr. & mental deficiency / depression.

4. Theta Waves

- Obtained generally in children below 9 yr. of age.

Standard
100 μ V



Alpha block

Alpha waves

↓ eyes opened ↓ eyes closed

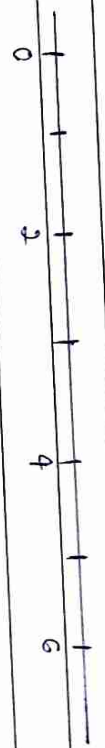
Beta waves



Theta waves



Delta waves



Time (sec) →



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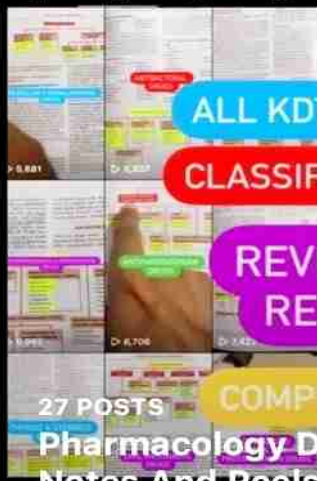
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