PES Institute of Medical Sciences & Research
Kuppam- 517 425, Chittoor Dist., Andhra Pradesh
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NAAC Criterion 2: Teaching Learning and Evaluation

2.5 Evaluation Process and Reforms

2.5.4: The Institution provides opportunities to students for midcourse improvement of performance through specific interventions.

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Re-test and Answer sheets

PES INSTITUTE OF MEDICAL SCIENCE AND RESEARCH

DEPARTMENT OF PHYSIOLOGY

I MONTHLY TEST - I MBBS Date: 29.11.2023

Time: 4.00pm – 5.30pm **Total marks=50 M**

LONG ESSAY:

1x15=15M

1. Define HEMOSTASIS. Write the steps of hemostasis. Explain both intrinsic and extrinsic mechanism of clotting in detail. Add a note on hemophilia. (1+2+10+2)

SHORT ESSAY:

7x5=35M

2. Explain the factors affecting erythropoiesis.
3. Define IMMUNITY. Classify immunity. Explain any one type of immunity in detail (1+1+3)
4. Define Action potential. Explain all the phases of action potential in detail with a neat labelled diagram.
5. Name the body fluid compartments. Write its normal values. Explain How to measure ECF ?
6. Name the types of neuroglia. Explain the functions of neuroglia with diagrams.
7. Write about intercellular connections with neat labelled diagram
8. A 3 month old baby presented with H/O of yellowish discolouration of skin since a few days often birth. O/E the baby has severe pallor and edema and shows decreased activity, his mother is Rh-negative blood type (1+3+1)

a.What is the likely diagnosis?

b. Discuss the pathophysiology of the disease

c. How can it be prevented?

PES Institute of Medical Sciences and Research
Department of Physiology
Monthly test-II

Date-28-02-24 **Time: 4.00PM-5.30PM**

Long Essay questions (1x15=15Marks) (draw diagrams wherever necessary)

1. Define Cardiac cycle & write its normal value. Describe the events of cardiac cycle with normal values. Describe the pressure volume changes of left ventricle in detail (2+8+5)

Short Essay questions (7x5=35Marks)

2. Describe the factors affecting Cardiac output.
3. Short term regulation of blood pressure
4. Describe Conducting system of heart
5. Cardiac arrhythmias
6. Explain Lead II ECG with a neat labeled diagram
7. Heart sounds
8. A 45 year old man met with an RTA & had severe bleeding. On examination he was restless, BP was 80/50mm Hg, HR was 120 /min, RR was 18 /pm. His skin was cold and clammy & he had thready pulse
   a. What is the diagnosis? 1M
   b. Describe the stages 2M
   c. Describe the treatment for the above condition. 2M

PES Institute of Medical Sciences and Research
Department of Physiology
Monthly test-III

Date-28-03-24 **Time: 4.00PM-5.30PM**

Long Essay questions (1x15=15Marks) (draw diagrams wherever necessary)

1. Define Glomerular filtration rate & write its normal value. Describe the factors affecting GFR and how is it regulated. Add a note on measurement of GFR (3+5+4+3)

Short Essay questions (7x5=35Marks)

2. Special features of renal circulation
3. Explain the mechanism of HCl secretion
4. Deglutition reflex
5. Regulation of pancreatic secretion
6. Describe the digestion and absorption of fat
7. Types of small intestinal movements
8. A 40 year old business executive complained of severe epigastric pain since last 6 months which increased with intake of food. He had few episodes of vomiting with blood and of late his work is stressful due to which his meals are irregular.
   a. What is the likely diagnosis? 1M
   b. Describe the pathophysiology of the disease 2M
   c. Describe the treatment for the above condition. 2M

PES Institute of Medical Sciences and Research
Department of Physiology
Monthly test-IV

Date-28-05-24

Time: 4.00PM-5.30PM

Long Essay questions (1x15=15Marks) (draw diagrams wherever necessary)

1. Define synapse. Classify synapse. Explain transmission of impulse with the help of diagram. Explain any four properties of synapse. (2+3+6+4)

Short Essay questions (7x5=35Marks)

2. Corticospinal tract
3. Muscle spindle
4. Pain pathway
5. Spermatogenesis
6. Hormonal regulation of menstrual cycle
7. Types of cutaneous receptors with diagram
8. Dorsal column pathway

PES Institute of Medical Sciences and Research,Kuppam

Department of Physiology Monthly test-1 **Date**-24-01-23,Time:2.15-3.45pm

Long Essay questions (1x15=15Marks)

1.Define Erythropoiesis. Explain the steps of Erythropoiesis with a neat labeled diagram. Add a note on factors and regulation of Erythropoiesis.(2+8+5marks)

Short Essays questions (5x7=35Marks)

2.Describe the functions of Plasma proteins. Add a note on Edema

3.Draw a neat labeled diagram of Cell .Add a note on Peroxisomes.

4. Explain the stages of Phagocytosis with diagram.

5.Name the Body Fluid Compartments. Describe the methods of measurement of ECF. Add a note on characteristics of ideal substance .

6. What is RMP. Explain the Ionic basis of Resting membrane potential.

7. Define Homeostasis . Explain the types of feedback mechanisms with examples.

8. Define Anaemia. Explain with diagram Morphological classification of Anaemia.

PES INSTITUTE OF MEDICAL SCIENCE AND RESEARCH

DEPARTMENT OF PHYSIOLOGY

First -MBBS /Monthly test/ March 2023

THEORY PAPER

Time: 1hour and 30 Min

Maximum marks: 50

Draw diagrams wherever necessary

LONG ESSAY:

2X15marks=15marks

1. A young female aged 24 Years presents with complaint of drooping of eyelid and double vision especially in the evenings, weakness in the jaw muscles, upper arms. Her symptoms become worse after climbing stairs.
   - What is the probable diagnosis and give your reasoning(2)
   - How can you confirm your diagnosis(1)
   - Where is the location of the lesion? (1)
   - Explain the events that occur at the location with neat diagram (4+2)
   - Name the drugs/toxins that act on the location and describe their mechanism of action(3)
   - Name the drug that can help her condition(1)
   - Describe other diseases that occur here(1)

SHORT ESSAYS:

7X5 marks =35

3. Explain lung pressure during breathing cycle with help of diagram
4. Describe non-respiratory functions of lungs.
4. Draw and explain the diagram of Sarcomere before and after muscle contraction.
5. Explain the functions of platelets.
6. Explain isometric and isotonic contraction with diagram and give example.
7. What are divisions of autonomic nervous system? Briefly explain their functions
8. Briefly explain enterohepatic circulation

PES INSTITUTE OF MEDICAL SCIENCE AND RESEARCH

DEPARTMENT OF PHYSIOLOGY

I-MBBS 2022-2023

THIRD MONTHLY TEST

Max marks: 40

Draw diagrams wherever necessary **Time: 1hours and 30 Min**

LONG ESSAYS: **1X15marks= 15marks**

1. Explain transport of Oxygen in Blood. Draw and explain Oxygen Hemoglobin dissociation curve. What is the significance of the shape. What are the factors that affect it (3+6+2+4)

SHORT ESSAYS: **5X5marks=25marks**

2. Patient aged 45 year-old male comes with h/o breathlessness, cough and tightness of chest. On examinations rhonchi is heard bilaterally. The doctor requested spirometry to confirm diagnosis
   a. What is your probable diagnosis? (1)
   b. What Spirometry tests will help in this diagnosis(1)
   c. What do you expect to find in the spirometry results? (2)
   d. What is the principle behind the treatment?(1)
3. What is Acclimatization? Explain the changes that occur.(1+4)
4. Explain chemical regulation of respiration.(5)
5. What is CPR? When do you initiate CPR? Describe the steps of doing CPR in detail. (1+1+3)
6. What is Cyanosis? What is the cause of Cyanosis? Describe the types of Cyanosis. How do you assess Cyanosis by clinical examination?(1+1+2+1)

PES INSTITUTE OF MEDICAL SCIENCE AND RESEARCH
DEPARTMENT OF PHYSIOLOGY
IV MONTHLY TEST - I MBBS Date: 06.06.2023

Time: 4.00pm – 5.30pm **Total marks=50 M**

LONG ESSAY: **1x15=15M**

1. Define Cardiac cycle and name the events with the help of a diagram. Describe the volume and pressure changes in detail and its relationship to ECG and heart sounds (1+2+12)

SHORT ESSAY: **7x5=35M**

2. Describe the salient features of coronary circulation.
3. Explain Frank-Starling law and how does it affect cardiac output?
4. Dysbarism (Decompression sickness)
5. Define Compliance and explain factors affecting it. Name two conditions in which it is altered and give reasons? (3+2)
6. Explain neural regulation of respiration with a diagram.
7. A 45 year old office assistant one morning as she woke up late, she panicked and jumped out of the bed . she felt lightheaded and thought she might faint .She felt her heart racing . On walking she noticed her light-headedness disappeared and the rest of the day was uneventfull .

a)What is the above condition called? (1/2marks)

b) What are the sequence of events that caused this condition? (1 ½ marks)

c)Her light-headedness was transient because of a reflex . what is this reflex and explain it in detail? (3 marks)

8. Explain Triple response in detail.

PES INSTITUTE OF MEDICAL SCIENCE AND RESEARCH
DEPARTMENT OF PHYSIOLOGY
V MONTHLY TEST - I MBBS Date: 27.07.2023

Time: 4.00pm – 5.30pm **Total marks=50 M**

LONG ESSAY: **1x15=15M**

1. Define synapse. Classification of synapse. Explain the steps of transmission of synapse. Explain any four properties of synapse in detail? (1+2+4+8=15M)

SHORT ESSAY: **7x5=35M**

2. Explain Dorsal column medial lemniscal pathway with a neat labelled diagram?
3. Physiological actions of Glucocorticoids
4. Addissons disease
5. Physiological actions of parathormone and add a note on tetany
6. Explain any six properties of Receptors
7. Describe the types of pain and pain pathway in detail? (10M)

PES INSTITUTE OF MEDICAL SCIENCE AND RESEARCH

2021 - 2022

DEPARTMENT OF PHYSIOLOGY

I MONTHLY TEST

Date: 26:05:2022 **Time: 4:00 to 5:30pm**

Max marks:50M

DRAW NEAT LABELLED DIAGRAMS WHEREVER NECESSARY

LONG ESSAY : **1X15=15M**

1. Define Erythropoiesis. Explain steps of erythropoiesis in detail. Add a note on factors regulating erythropoiesis.

SHORT ESSAYS : WRITE IN BREIF **5X5=25M**

2. Role of feedback mechanisms in Homeostasis
3. Active transport mechanism
4. Classify Anaemia based on etiology and morphology. Explain Pernicious anaemia.
5. Cell mediated immunity
6. Explain the genesis of RMP

SHORT ANSWERS : WRITE IN SHORT **5X2=10M**

7. Apoptosis
8. Name the body fluid compartments and mention their normal values.
9. Functions of plasmaproteins
10. Steps involved in phagocytosis
11. Nerst equation

PES INSTITUTE OF MEDICAL SCIENCE AND RESEARCH

DEPARTMENT OF PHYSIOLOGY

II MONTHLY TEST

Date: 28.07.2022 **Time: 4:00 to 5:30pm**

Max marks:50M

DRAW NEAT LABELLED DIAGRAMS WHEREVER NECESSARY

LONG ESSAY : **1X15=15M**

1. Describe the action potentials in heart? Explain the refractory period in a cardiac muscle and its importance.

SHORT ESSAYS : WRITE IN BREIF **5X5=25M**

2. Define Hypoxia? Classify and explain the different types of Hypoxia.
3. Explain Conducting system of heart with a neat labelled diagram.
4. Compare and contrast Cardiac muscle and Skeletal muscle.
5. Define Acclimatization.Explain the changes during Acclimatization.
6. Explain Chemical regulation of Respiration.

SHORT ANSWERS : WRITE IN SHORT **5X2=10M**

7. What is AV nodal delay?What is its clinical significance.
8. Draw a labelled diagram of ECG?
9. What is PR interval.What is its importance.
10. What is cyanosis?What are its types.
11. Give the normal value of total body water.Which substance is used to measure ECF.Give 2 properties of an ideal substance.

PES INSTITUTE OF MEDICAL SCIENCE AND RESEARCH

DEPARTMENT OF PHYSIOLOGY

III MONTHLY TEST

I MBBS August 2022

Date: 25.08.2022 **Time: 4:00 to 5:30pm**

Max marks:50M

DRAW NEAT LABELLED DIAGRAMS WHEREVER NECESSARY

LONG ESSAY : **1X15=15M**

Define Cardiac cycle.Explain the pressure volime changes in the left ventrical with the help of a neat lablled diagram.Add a note on heart sounds.

SHORT ESSAYS:WRITE IN BREIF **4X4=20M**

1. Describe the factors affecting cardiac output?
2. Describe the short regulation of Blood pressure?
3. Describe the waves and intervals of typical ECG and its importance with the help of neat lablled diagram?
4. What are the phases of gastric secreation.Describe the machanisim of gastric HCL secreation in detail with diagram?

SHORT ANSWERS : WRITE IN SHORT **2X2=10M**

1.Write four pecularities of coronary circulation?

2.Describe the functions of Sliva?

VERY SHORT NOTE: **1X1=1M**

1.What is stoke's Adam's syndrome?

PES INSTITUTE OF MEDICAL SCIENCE AND RESEARCH

DEPARTMENT OF PHYSIOLOGY

I MONTHLY TEST

Date: 02:03:2021 **Time: 4:00 to 5:00 pm**

Max marks:30

Short Notes **3x5=15**

1. Describe the steps of Erythropoiesis
2. Define Action Potential, Explain nerve action potential its ionic basis.
3. Genesis of Resting Membrane Potential

Short answers **5x3=15**

4. Define Simple Diffusion, Facilitated Diffusion Osmosis
5. Function of plasma proteins
6. Define Homeostasis, Name the control mechanism involved in Homeostasis
7. Define Rheobase, Chronaxie, Utilization Time
8. Neuroglia

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DEPARTMENT OF PHYSIOLOGY

II MONTHLY TEST

Date: 30:03:2021 **Time: 4:00 to 5:00 pm**

Max marks: 30

LONG ESSAYS **2X10=20**

1. List the clotting factors. Describe the intrinsic and extrinsic pathway of clotting. Add a note on anticoagulants.
2. Draw a labeled diagram of a sarcomere and sarcotubular system. Explain in detail the molecular basis of skeletal muscle contraction.

Short answers **5x2=10**

3. Landsteiner's Law
4. Haemophilia
5. Gamma globulins
6. Motor unit
7. Starling's law of muscle contraction.

PES INSTITUTE OF MEDICAL SCIENCE AND RESEARCH

DEPARTMENT OF PHYSIOLOGY

I MBBS 2020-2021 BATCH- MONTHLY TEST - III

Time: 4.00 - 5.30 PM **Date: 28.10.2021**

Draw diagrams wherever necessary

LONG ESSAY **1X15=15**

1. Name the functional divisions of cerebellum. Describe their connections and functions. Add a note on cerebellar lesion.

SHORT ESSAY (WRITE BRIEFLY ON) **7x5=35**

2. Role of hypothalamus in the regulation of food intake.
3. Structure and functions of the muscle spindle.
4. Referred pain
5. Diseases involving Basal Ganglia
6. Non-rapid eye movement (NREM) sleep.
7. Explain a) types of Aphasia

   b) Kluver - Bucy syndrome.
8. Differences between signs of UMN and LMN lesion

PES INSTITUTE OF MEDICAL SCIENCE AND RESEARCH

DEPARTMENT OF PHYSIOLOGY

IV MONTHLY TEST

Time: 4.00 - 5.30 PM

Date: 25.11.2021

LONG ESSAY 1x15-15M

1. Describe the biosynthesis, regulation of secretion and actions of thyroid hormone. Discuss the effects of hyposecretion of thyroid hormone.

SHORT ESSAY (Write breifly) 7X5=35M

2. Effects of hypersecretion of Growth hormone.
3. Actions of ADH
4. Mechanism of action of protein/peptide hormone
5. Actions of parath hormone
6. Cushing's syndrome
7. Neuro endocrine reflex
8. Regulation of secretion of aldosterone

PES INSTITUTE OF MEDICAL SCIENCE AND RESEARCH

DEPARTMENT OF PHYSIOLOGY

I MONTHLY TEST - I MBBS Date: 27/9/2019

Time: 4.00pm – 5.30pm **Total marks=50 M**

LONG ESSAY: **1x15=15M**

1. Define HEMOSTASIS. Write the steps of hemostasis. Explain both intrinsic and extrinsic mechanism of clotting in detail. Add a note on hemophilia. (1+2+10+2)

SHORT ESSAY: **7x5=35M**

2. Explain the factors affecting erythropoiesis.
3. Define IMMUNITY. Classify immunity. Explain any one type of immunity in detail (1+1+3)
4. Define Action potential. Explain all the phases of action potential in detail with a neat labelled diagram.
5. Name the body fluid compartments. Write its normal values. Explain How to measure ECF ?
6. Name the types of neuroglia. Explain the functions of neuroglia with diagrams.
7. Write about intercellular connections with neat labelled diagram
8. A 3 month old baby presented with H/O of yellowish discolouration of skin since a few days often birth. O/E the baby has severe pallor and edema and shows decreased activity, his mother is Rh-negative blood type (1+3+1)

a.What is the likely diagnosis?

b. Discuss the pathophysiology of the disease

c. How can it be prevented?

PES Institute of Medical Sciences and Research
Department of Physiology
Monthly test-II

Date-01/11/2019 **Time:**
4.00PM-5.30PM

Long Essay questions (1x15=15Marks) (draw diagrams wherever necessary)

1. Define Cardiac cycle & write its normal value. Describe the events of cardiac cycle with normal values. Describe the pressure volume changes of left ventricle in detail (2+8+5)

Short Essay questions (7x5=35Marks)

2. Describe the factors affecting Cardiac output.
3. Short term regulation of blood pressure
4. Describe Conducting system of heart
5. Cardiac arrhythmias
6. Explain Lead II ECG with a neat labeled diagram
7. Heart sounds
8. A 45 year old man met with an RTA & had severe bleeding. On examination he was restless, BP was 80/50mm Hg, HR was 120 /min, RR was 18 /pm. His skin was cold and clammy & he had thready pulse
   a. What is the diagnosis? 1M
   b. Describe the stages 2M
   c. Describe the treatment for the above condition. 2M

PES Institute of Medical Sciences and Research
Department of Physiology
Monthly test-III

Date- 17/03/2020 **Time: 4.00PM-5.30PM**

Long Essay questions (1x15=15Marks) (draw diagrams wherever necessary)

1. Define Glomerular filtration rate & write its normal value. Describe the factors affecting GFR and how is it regulated. Add a note on measurement of GFR (3+5+4+3)

Short Essay questions (7x5=35Marks)

2. Special features of renal circulation
3. Explain the mechanism of HCl secretion
4. Deglutition reflex
5. Regulation of pancreatic secretion
6. Describe the digestion and absorption of fat
7. Types of small intestinal movements
8. A 40 year old business executive complained of severe epigastric pain since last 6 months which increased with intake of food. He had few episodes of vomiting with blood and of late his work is stressful due to which his meals are irregular.
   a. What is the likely diagnosis? 1M
   b. Describe the pathophysiology of the disease 2M
   c. Describe the treatment for the above condition. 2M

PES Institute of Medical Sciences and Research
Department of Physiology
Monthly test-IV

Date-25/6/2020 Time:
4.00PM-5.30PM

Long Essay questions (1x15=15Marks) (draw diagrams wherever necessary)

1. Define synapse. Classify synapse. Explain transmission of impulse with the help of diagram. Explain any four properties of synapse. (2+3+6+4)

Short Essay questions (7x5=35Marks)

2. Corticospinal tract
3. Muscle spindle
4. Pain pathway
5. Spermatogenesis
6. Hormonal regulation of menstrual cycle
7. Types of cutaneous receptors with diagram
8. Dorsal column pathway

Physiology test 27-07-23

1. SYNAPSE

Synapse/neurosynapse is a specialized junction between the presynaptic neuron and to the axon, soma, dendrite of post synaptic neuron.

If it is in a muscle = Neuromuscular junction

In gland = neuroglandular junction.

It is of (3) types namely:—

(1) chemical synapse
(2) Electrical synapse
(3) Mixed synapse

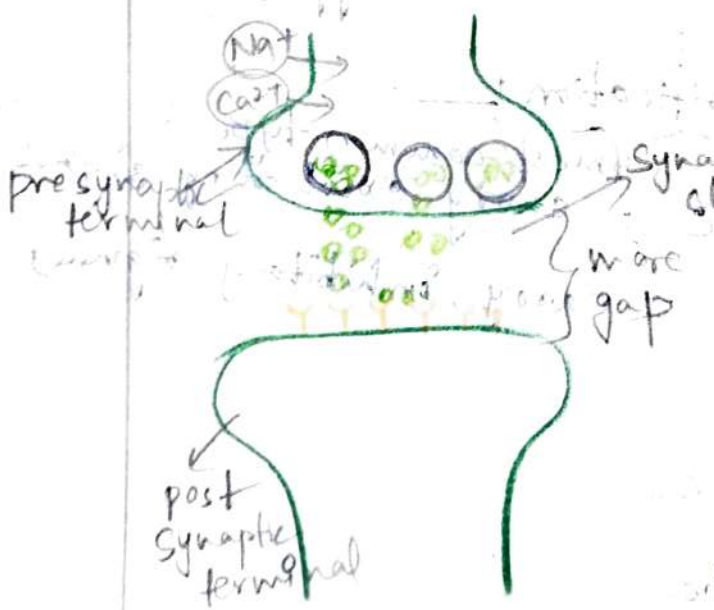
} functional classification.

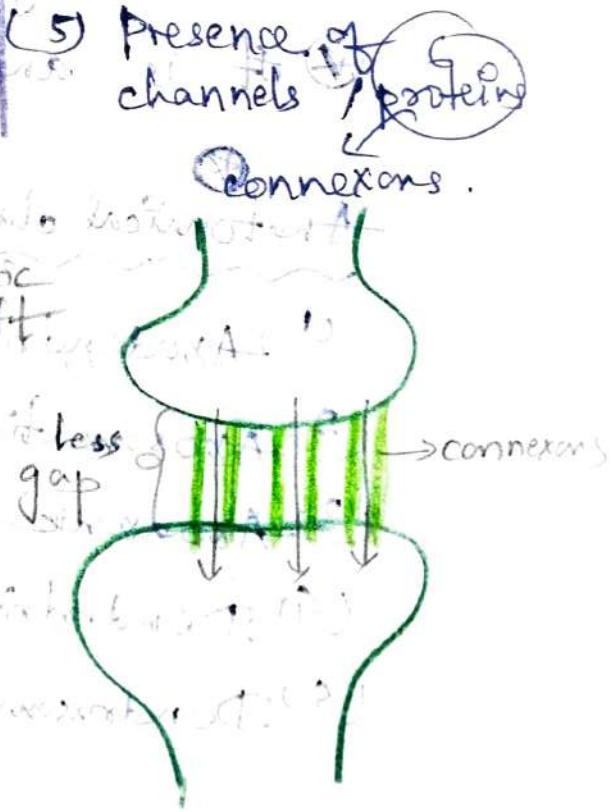
* It is anatomically & functionally of many types

Anatomical classification:—

(1) Axodendritic = (most common type) carry excitatory signals
(2) Axosomatic = carry inhibitory signals
(3) Axoaxonic
(4) Dendrodritic
(5) Dendrosomatic

chemical synapse	Electrical synapse
(1) Presence of neurotransmitters, synaptic vessicles and receptors.	(1) No synaptic vessicle and receptors
(2) Space between the pre synaptic terminal and post synaptic terminal.	(2) No space.
(3) common	(3) Rare
(4) Slow transmission of impulse	(4) fast impulse transmission.
(5) no special channels.	(5) Presence of channels / protein connexons.





Steps of transmission of synapses

on stimulation of the presynaptic terminal of a neuron

↓

opening of the Ca^{2+} channels and release of Ca^{2+} ions.

↓

This causes the opening of synaptic vesicles to release neurotransmitters.

↓

Neurotransmitters are released via Ca^{2+} mediated channels by exocytosis into the synaptic cleft.

↓

Binding of the neurotransmitters to the post synaptic receptors

↓

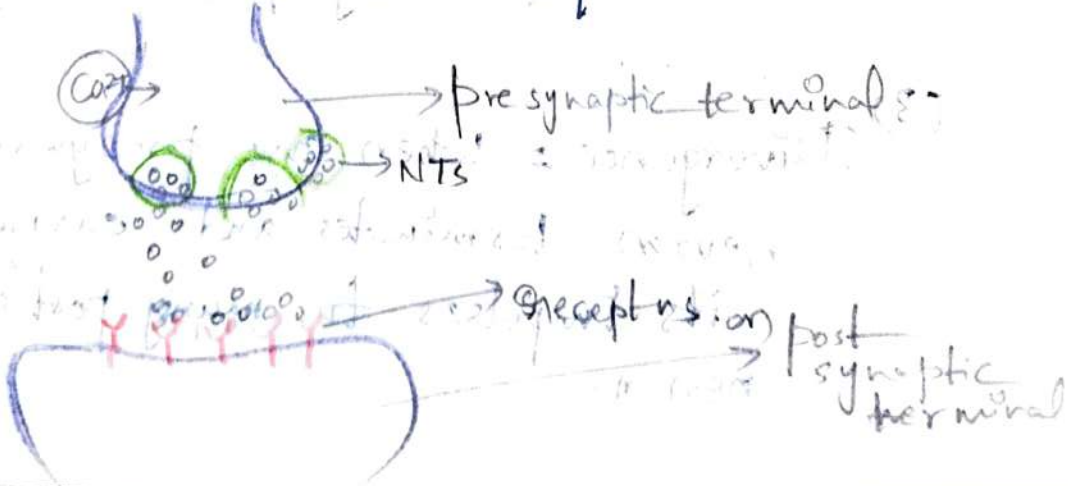
cause opening of ion channels

↓

cause generator/receptor potential

↓

cause action potentials.



Properties of Synapse are :-

(1) Mode of forward conduction =

The impulses are always trasmitted in one direction (one way direction) from pre synaptic neuron to post synaptic neuron

(orthodromic conduction)

Reason = As the Neurotransmitters are present only in the vesicles in pre synaptic neuron

(2) Synaptic delay

= There is a delay of 0.5 ms for the conduction of impulses from one neuron to other.

⇒ Only in chemical synapses

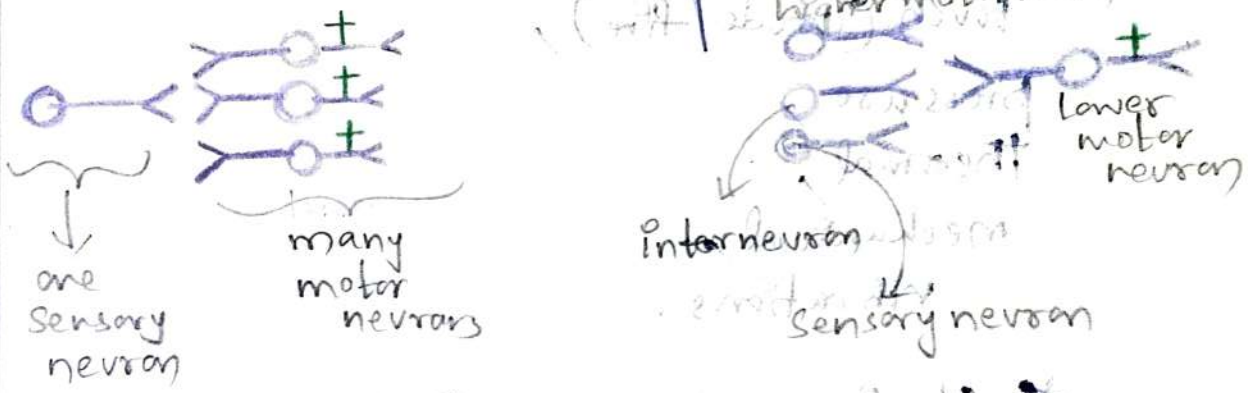
Reason = due to delay in release of NTs from synaptic vesicles.

(3) Divergence = When one presynaptic neuron terminates and carries its impulses to many post synaptic neurons.

ex = Pain receptors carrying impulses (withdrawal reflex)

(4) convergence → when many post synaptic neuron give/transmit their impulses to ~~many post sy~~ only one pre synaptic neuron.

ex → of divergence | of convergence



(5) Inhibition = It is of many types namely

(1) presynaptic inhibition = due to inhibition from inhibitory interneuron

(2) postsynatic / Reciprocal / direct inhibition = from interneuron which inhibits at post synaptic neuron level.

(3) -ve feed back / Renshaw cell inhibition.
= due to inhibitory interneuron on receiving collaterals from ~~ra~~ motor neurons, starts inhibiting the same motor neurons.

2. Dorsal column Medial lemniscal pathway.

(DCML pathway)

It is the pathway for detection of various sensations like:

touch (coude/fine),
pressure,
thermal,
mechanical,
vibrations.

* It is made up of ③ types of neurons at each level namely:

(1) 1st order neuron
(2) 2nd order neuron
(3) 3rd order neuron.

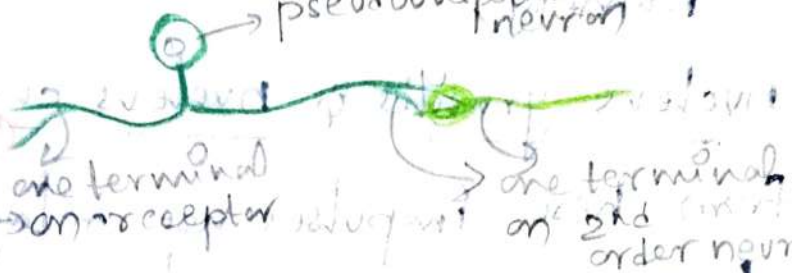
Receptor → Spinal cord → Thalamus → cortex

(1st order neuron) (2nd order neuron) (3rd order neuron)

* Also known as ascending pathway of impulse transmission.

1st order neuron = It is between the receptor and the spinal cord.

It is a pseudounipolar neuron.

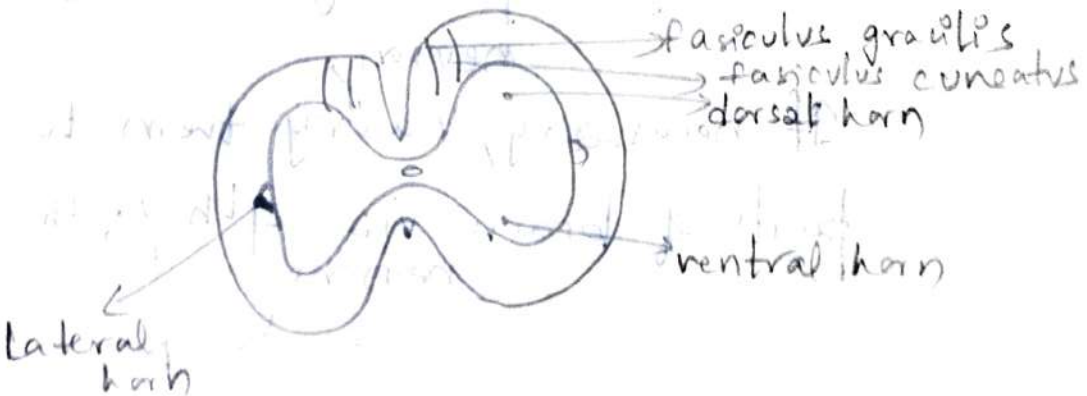


It then goes via the dorsal root ganglion which carries sensory information to the spinal cord.

It has → fasciculus gracilis
→ fasciculus cuneatus

Fasciculus gracilis = present in middle part of the spinal cord & transmits impulses to lower part of the body.

Fasciculus cuneatus = present in upper part of spinal cord & carry impulse to upper part of body.



from spinal cord, now the impulses are carried to 2nd order neuron.

It consists of (2) nucleus namely,

nucleus gracilis & nucleus cuneatus.

from here impulses are relayed to the thalamus which consists of these nucleus.

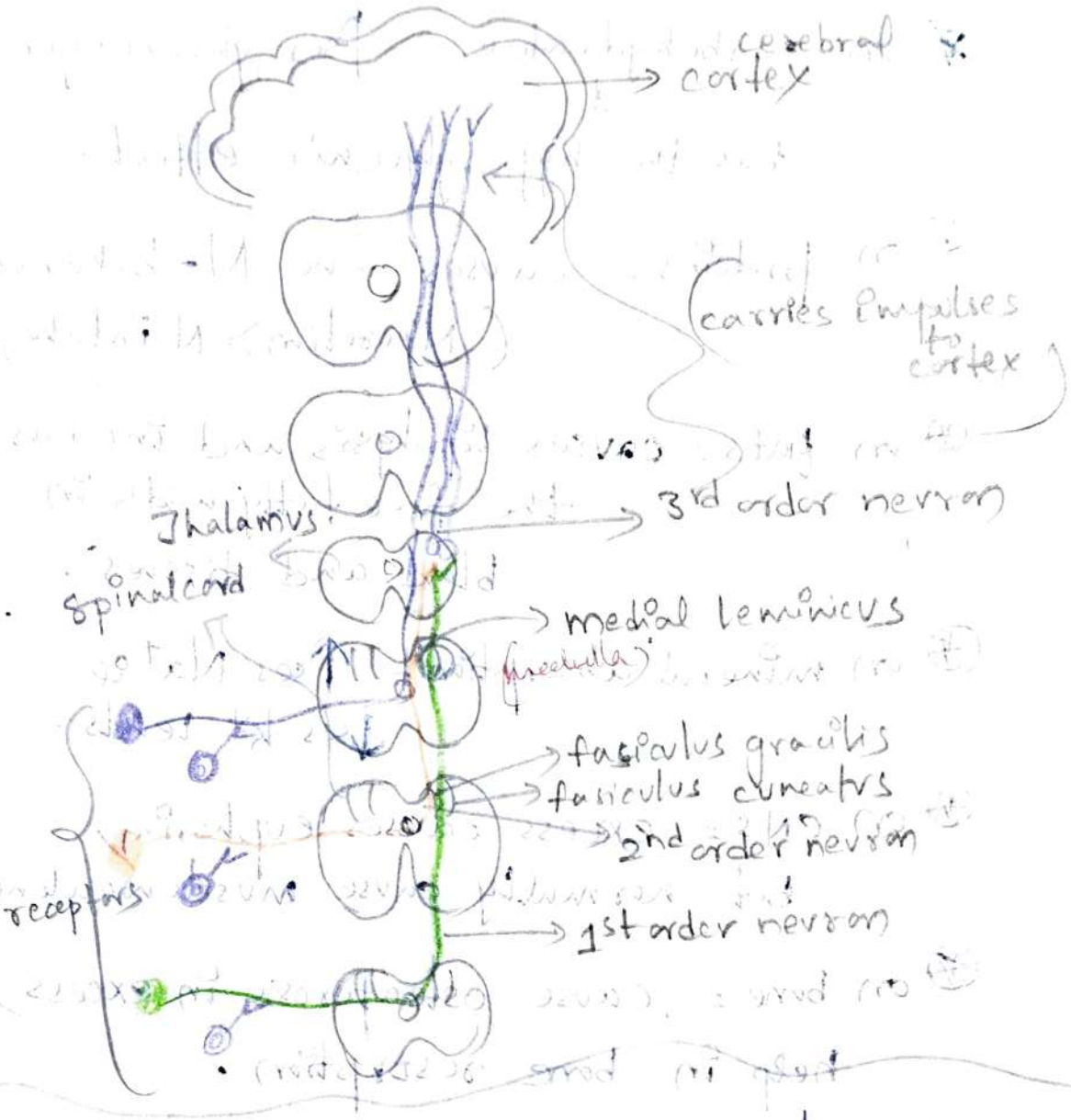
From here, they are carried through the medial Lemniscus to the 3rd order neuron consisting of Ventral Posterolateral nucleus.

It is via trigeminal nerve.

from here impulses are carried to the brain (cortex)

Sensory cortical areas are present which relays impulses of 3rd order neuron,

If necessary, carry them to other parts of brain via 4th / 5th order neurons



3. Physiological functions/actions of glucocorticoids.

glucocorticoids → are the "cortisol" / corticosterone

These are the products synthesized mainly from zonal fasciculata of adrenal cortex / of adrenal gland.

It has many functions like

- on carbohydrates = ↑ses gluconeogenesis
  ↓
  has the hyperglycemic effect.
- on proteins = causes –ve N-balance
  (N secretion > N intake)
- on fats = causes lipolysis and increases the free fatty acids in blood and tissues.
- on mineral absorption = ↑ses Na^+ re
  ↓ses K^+ levels.
- on CNS = excess causes euphoria, but normally cause muscle weakness.
- on bone = cause osteoporosis in excess, help in bone resorption.
- Anti allergic = Prevents the release of histamine from mast cells.
- Anti inflammatory = Prevents inflammation.
- Anti stress = ↓ses stress levels via hypothalamo pituitary axis.
- It increases the levels of platelet, neutrophils, RBC (polycythemia).
  ↓ lymphocytes, Eosinophils

⊛ on GIT - ↑ses the gastric acid secretion,
↓
↑ses gut motility.

↳ More production of cortical causes Peptic ulcers
↓
prevented by H2-Blockers

⊛ permissive actions = ↑ses the catecholamine entry into the blood.
↓
maintain Blood sugar levels.

⊛ Calorigenic action = ↑Heat production & ↑ses BMR.

⊛ Delays wound healing → ↓ses the collagen formation.

4. Addissons disease
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This is the condition of multiple syndromes mainly due to hyposecretion / Insufficiency of both mineralocorticoids and glucocorticoids by adrenal cortical cells.
Mainly due to glucocorticoids.

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Autoimmune disorders
Carcinomas
Tuberculosis

⊛ Effects are wide namely →

causes ↑ MSH activity and leads to hyperpigmentation of the skin & mucus membranes.

Addisonian crisis = when an individual is unable to cope up with the stress due to less levels of cortisol in blood. It causes a Addisonian crisis.

* There is also delay in the wound healing capacity due to less production of cortisol.

* It causes hyperphosphatemia and ↑se in Ca^{2+} absorption by the bones.

Other effects include:
Mental retardation, convulsions
Low muscle tone, muscle weakness
lethary, hyperpigmentation etc.

Treatment = Administration of synthetic cortisol hormones.

45

5. Physiological actions of parathormone (PTH):

* It has its effects mainly on
  ③ parts namely → small intestine
  → Bone
  → kidney.

Vitamin D / 1,25 dehydrocholecalciferol / calcitanin is absorbed mainly due to the presence of PTH.

① On small intestine = It increases the Ca^{2+} and glucose uptake by cells, also phosphate uptake by ↑ the calcium channels on basolateral membrane.

② on Bone = It causes bone reabsorption due to ↑ in osteoblastic activity and causes Ca^{2+} absorption.

③ on kidney = Cause reabsorption of Ca^{2+} & P by tubular cells of the nephron causes less excretion of Ca^{2+} through urine.

* Deficiency of PTH causes
  ↓
  Hypoparathyroidism

This condition leads to ↓

↓ ca²⁺ levels & ↑ phosphate levels

i.e

Hypocalcemia, Hypocalciuria (↓ Ca^{2+} in urine)

Hyperphosphataemia.

This causes tetany

(*) It is marked by (3) signs:

(1) Carpopedal spasm = sustained contraction of skeletal muscles, causes flexion at metacarpophalyngeal joints, extension at interphalyngeal joints & opposition of thumb.

(2) Chvostek sign = It is by tapping the facial nerve supplying the facial muscles.

(3) Trousseau's sign

= It is done by occluding the blood vessels → causes flexion of wrist and hand & extension of fingers.

6. Properties of Receptors

★ Adaptive property → rapid adaptation
→ slow adaptation.

when a required threshold stimulus is applied on the receptor, the stimulus of the receptor ↓ses its frequency and ↓ses it's firing capacity.

↓

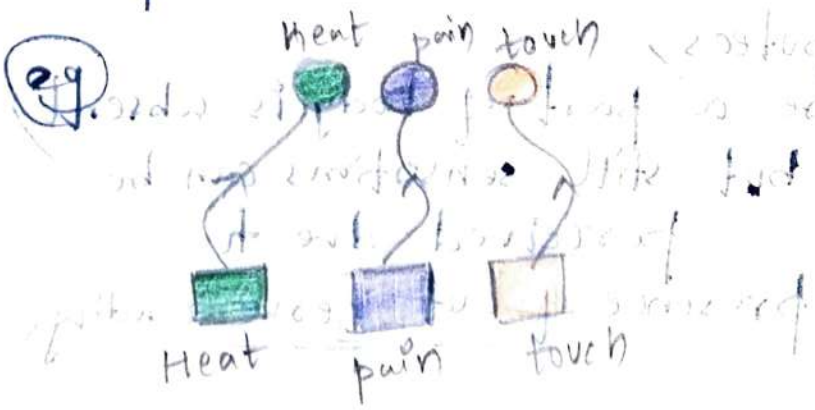
If it stops firing rapidly = Rapid adaptation, without any delay

If is stops by steady decrease in its stimulus = slow adaptation.

★ Doctrine of spefic nerve energies

↳ when a recepter is stimulated & sensations are produced, the sensations will be specific to its particular stimulations only no matter where / how the stimulations are produced.

eg.



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most prominent in less receptive / small receptive areas
like tongue than in large receptive areas like back of our body / limbs.

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In Amputees, where a part of body is absent but still sensations can be perceived due to presence of cut nerve endings

★ Intensity discrimination = The signals to brain are send via (2) pathways of Summation namely temporal & spatial summation.

⊛ Temporal summation = When impulse (input) is transmitted from only one pre-synaptic neuron to ~~many~~ one post synaptic neurons

⊛ Spatial summation
when inputs are carried from addition of many presynaptic neurons to only one post synaptic neurone.

It also consists of → Web Fechner's principle
⊛ (gradation in strength of stimulus is proportional to Log of stimulus strength)

7. Pain
→ It is the sensation whenever we accompany any kind of harm to our body.
→ Way of protective Mechanism.

It is mainly of (2) types :—

→ Fast pain
→ slow pain

Both are different by various ways →

- FAST PAIN = It shows fastest response.
  - (prick pain / acute pain) ✓
  - lasts for < 0.1 seconds

They sensations are carried via myelinated Aδ fibers.

They end on antero receptive pathway (Lateral pathway)

SLOW PAIN
- It shows same delayed response.
- (Chronic pain / electric pain / sharp pain)
- lasts for > 1sec

The sensations are carried via non-myelinated "C" fibers.

They end on posteroreceptive pathway (Medial pathway)

- Fast pain = neurotransmitter is glutamate
- Slow pain = " " Substance P.

The pain pathway is
- Anterolateral Spinothalamic pain pathway.

fast pain relays at lower level of thalamus
But slow pain goes upto the cortex to produce cortico-sensory signals

Physiology test 27-07-23

1. SYNAPSE

Synapse/neurosynapse is a specialized junction between the presynaptic neuron and to the axon, soma, dendrite of post synaptic neuron.

If it is in a muscle = Neuromuscular junction

In gland = neuroglandular junction.

It is of (3) types namely:—

(1) chemical synapse
(2) Electrical synapse
(3) Mixed synapse

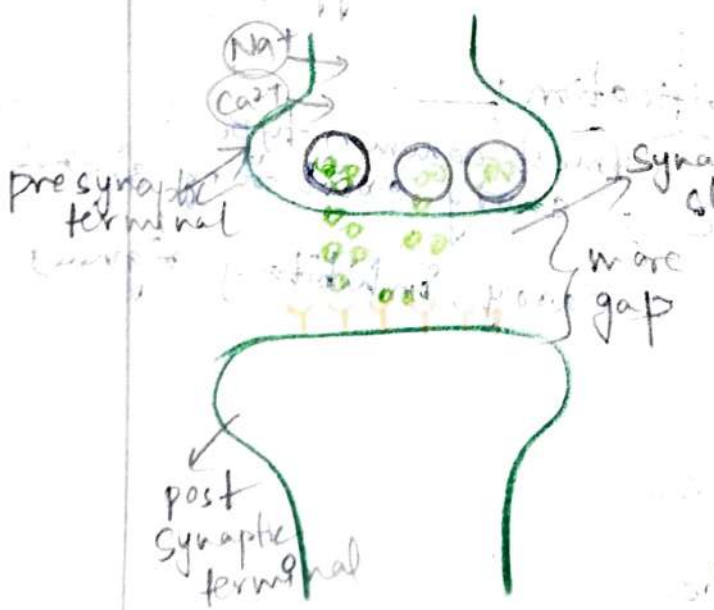
} functional classification.

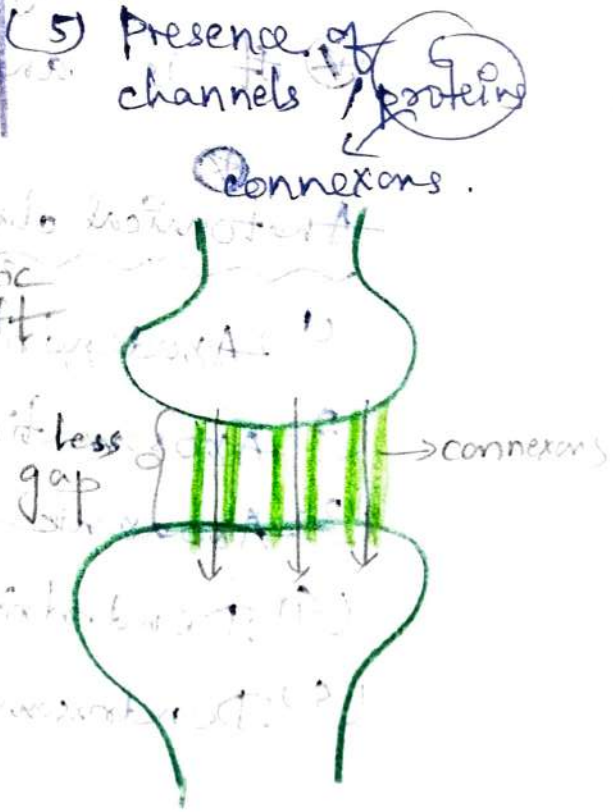
* It is anatomically & functionally of many types

Anatomical classification:—

(1) Axodendritic = (most common type) carry excitatory signals
(2) Axosomatic = carry inhibitory signals
(3) Axoaxonic
(4) Dendrodritic
(5) Dendrosomatic

chemical synapse	Electrical synapse
(1) Presence of neurotransmitters, synaptic vessicles and receptors.	(1) No synaptic vessicle and receptors
(2) Space between the pre synaptic terminal and post synaptic terminal.	(2) No space.
(3) common	(3) Rare
(4) Slow transmission of impulse	(4) fast impulse transmission.
(5) no special channels.	(5) Presence of channels / protein connexons.





Steps of transmission of synapses

on stimulation of the presynaptic terminal of a neuron

↓

opening of the Ca^{2+} channels and release of Ca^{2+} ions.

↓

This causes the opening of synaptic vesicles to release neurotransmitters.

↓

Neurotransmitters are released via Ca^{2+} mediated channels by exocytosis into the synaptic cleft.

↓

Binding of the neurotransmitters to the post synaptic receptors

↓

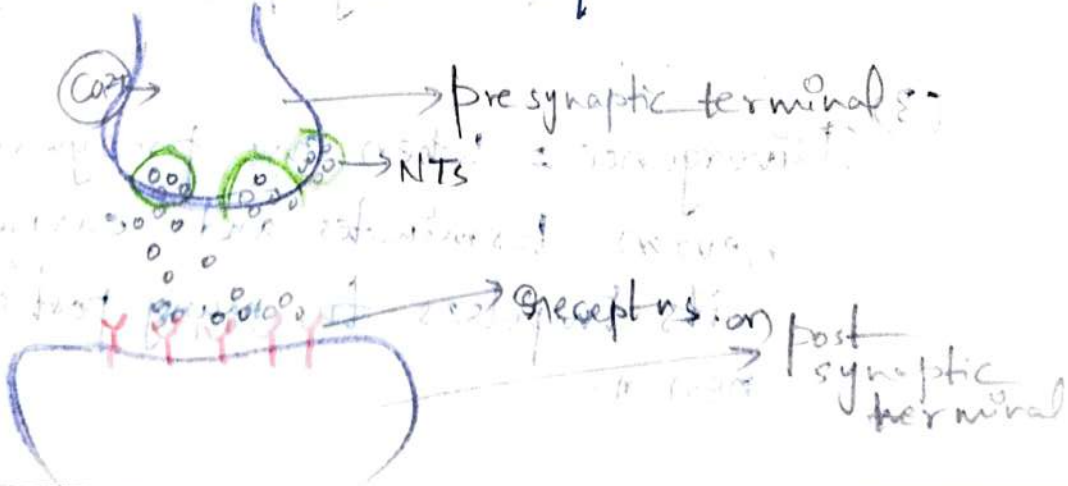
cause opening of ion channels

↓

cause generator/receptor potential

↓

cause action potentials.



Properties of Synapse are :-

(1) Mode of forward conduction =

The impulses are always trasmitted in one direction (one way direction) from pre synaptic neuron to post synaptic neuron

(orthodromic conduction)

Reason = As the Neurotransmitters are present only in the vesicles in pre synaptic neuron

(2) Synaptic delay

= There is a delay of 0.5 ms for the conduction of impulses from one neuron to other.

⇒ Only in chemical synapses

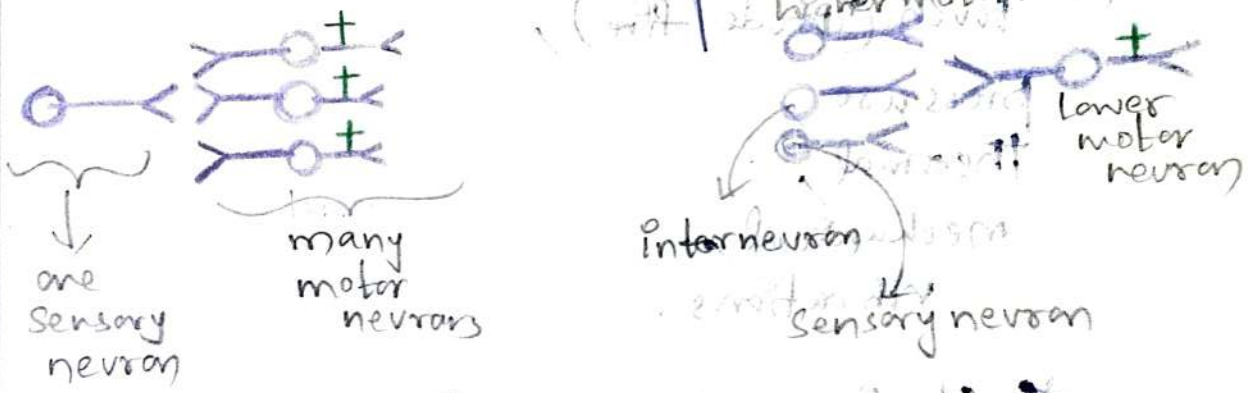
Reason = due to delay in release of NTs from synaptic vesicles.

(3) Divergence = When one presynaptic neuron terminates and carries its impulses to many post synaptic neurons.

ex = Pain receptors carrying impulses (withdrawal reflex)

(4) convergence → when many post synaptic neuron give/transmit their impulses to ~~many post sy~~ only one pre synaptic neuron.

ex → of divergence | of convergence



(5) Inhibition = It is of many types namely

(1) presynaptic inhibition = due to inhibition from inhibitory interneuron

(2) postsynatic / Reciprocal / direct inhibition = from interneuron which inhibits at post synaptic neuron level.

(3) -ve feed back / Renshaw cell inhibition.
= due to inhibitory interneuron on receiving collaterals from ~~ra~~ motor neurons, starts inhibiting the same motor neurons.

2. Dorsal column Medial lemniscal pathway.

(DCML pathway)

It is the pathway for detection of various sensations like:

touch (coude/fine),
pressure,
thermal,
mechanical,
vibrations.

* It is made up of ③ types of neurons at each level namely:

(1) 1st order neuron
(2) 2nd order neuron
(3) 3rd order neuron.

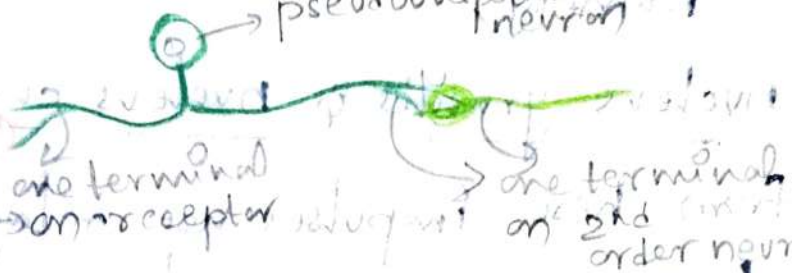
Receptor → Spinal cord → Thalamus → cortex

(1st order neuron) (2nd order neuron) (3rd order neuron)

* Also known as ascending pathway of impulse transmission.

1st order neuron = It is between the receptor and the spinal cord.

It is a pseudounipolar neuron.

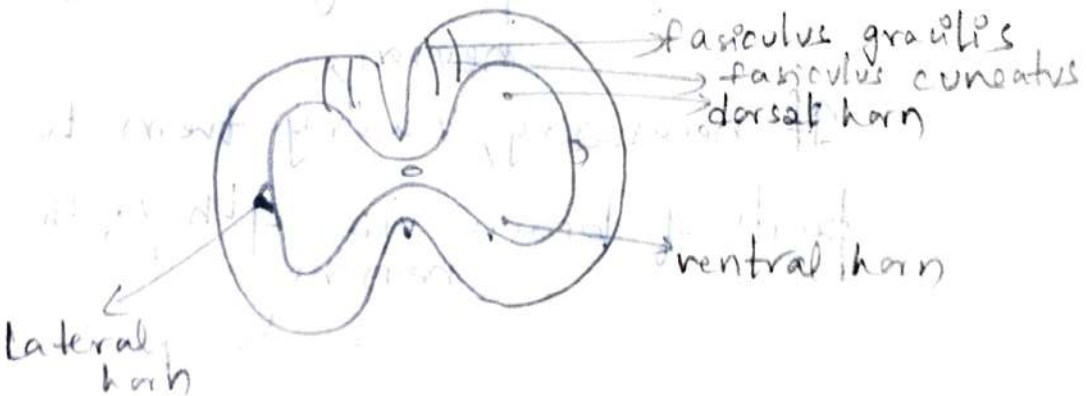


It then goes via the dorsal root ganglion which carries sensory information to the spinal cord.

It has → fasciculus gracilis
→ fasciculus cuneatus

Fasciculus gracilis = present in middle part of the spinal cord & transmits impulses to lower part of the body.

Fasciculus cuneatus = present in upper part of spinal cord & carry impulse to upper part of body.



from spinal cord, now the impulses are carried to 2nd order neuron.

It consists of (2) nucleus namely,

nucleus gracilis & nucleus cuneatus.

from here impulses are relayed to the thalamus which consists of these nucleus.

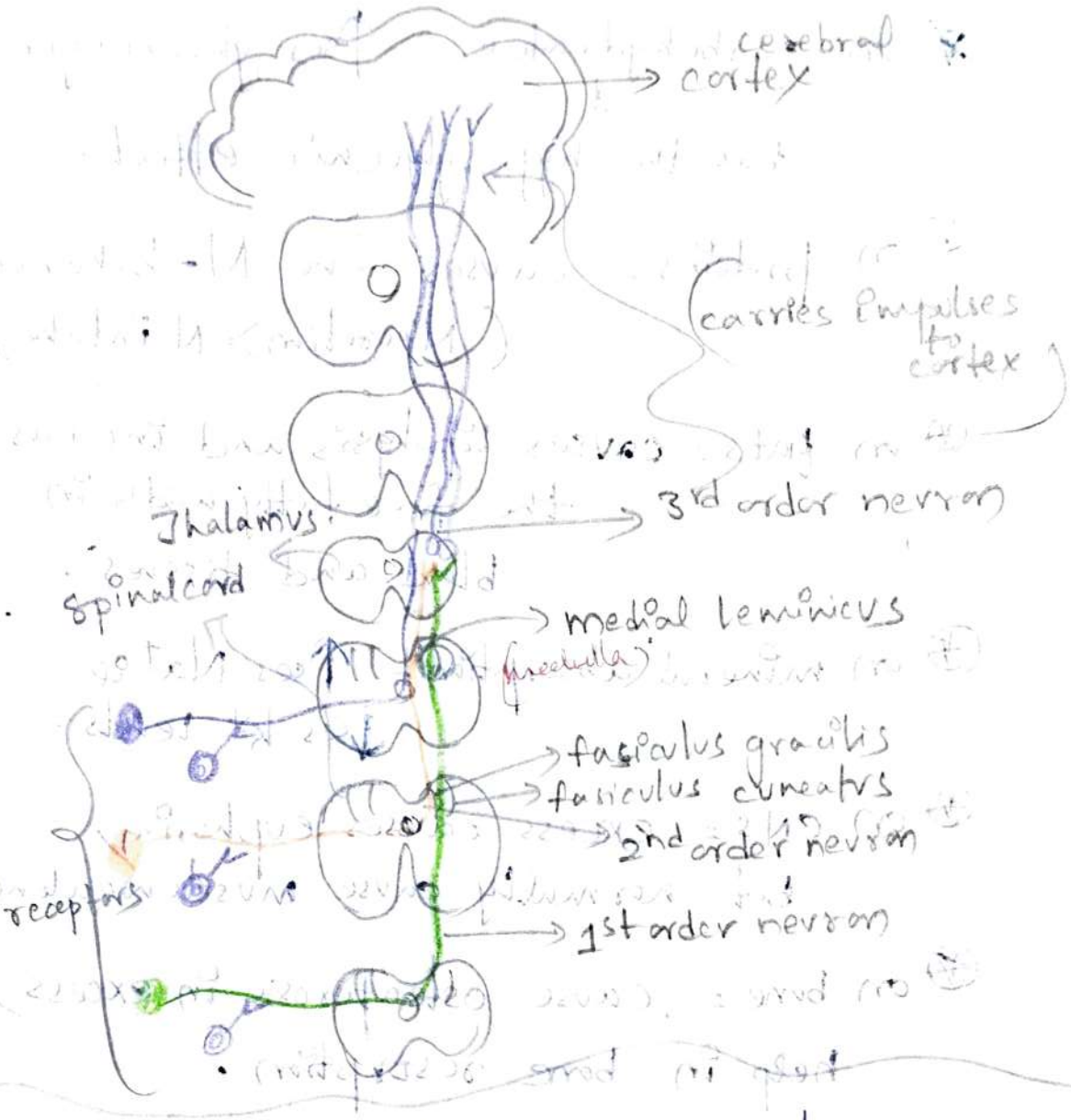
From here, they are carried through the medial Lemniscus to the 3rd order neuron consisting of Ventral Posterolateral nucleus.

It is via trigeminal nerve.

from here impulses are carried to the brain (cortex)

Sensory cortical areas are present which relays impulses of 3rd order neuron,

If necessary, carry them to other parts of brain via 4th / 5th order neurons



3. Physiological functions/actions of glucocorticoids.

glucocorticoids → are the "cortisol" / corticosterone

These are the products synthesized mainly from zonal fasciculata of adrenal cortex / of adrenal gland.

It has many functions like

- on carbohydrates = ↑ses gluconeogenesis
  ↓
  has the hyperglycemic effect.
- on proteins = causes –ve N-balance
  (N secretion > N intake)
- on fats = causes lipolysis and increases the free fatty acids in blood and tissues.
- on mineral absorption = ↑ses Na^+ re
  ↓ses K^+ levels.
- on CNS = excess causes euphoria, but normally cause muscle weakness.
- on bone = cause osteoporosis in excess, help in bone resorption.
- Anti allergic = Prevents the release of histamine from mast cells.
- Anti inflammatory = Prevents inflammation.
- Anti stress = ↓ses stress levels via hypothalamo pituitary axis.
- It increases the levels of platelet, neutrophils, RBC (polycythemia).
  ↓ lymphocytes, Eosinophils

⊛ on GIT - ↑ses the gastric acid secretion,
↓
↑ses gut motility.

↳ More production of cortical causes Peptic ulcers
↓
prevented by H2-Blockers

⊛ permissive actions = ↑ses the catecholamine entry into the blood.
↓
maintain Blood sugar levels.

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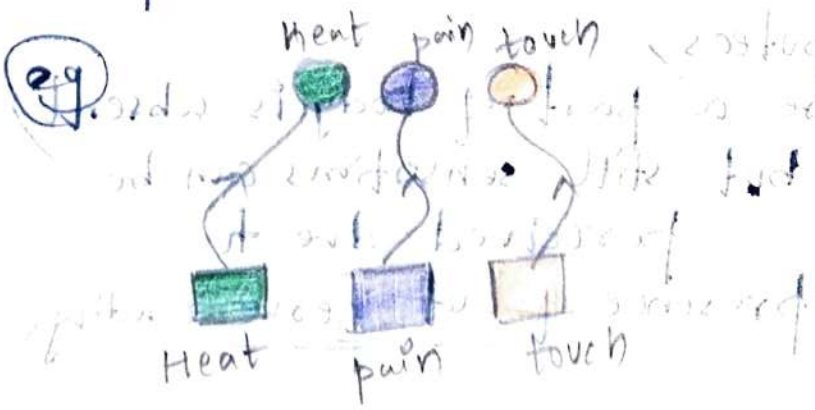

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PES INSTITUTE OF MEDICAL SCIENCE AND RESEARCH

DEPARTMENT OF PHYSIOLOGY

I MONTHLY TEST - I MBBS Date: 29.11.2023

Time: 4.00pm – 5.30pm **Total marks=50 M**

LONG ESSAY:

1x15=15M

1. Define HEMOSTASIS. Write the steps of hemostasis. Explain both intrinsic and extrinsic mechanism of clotting in detail. Add a note on hemophilia. (1+2+10+2)

SHORT ESSAY:

7x5=35M

2. Explain the factors affecting erythropoiesis.
3. Define IMMUNITY. Classify immunity. Explain any one type of immunity in detail (1+1+3)
4. Define Action potential. Explain all the phases of action potential in detail with a neat labelled diagram.
5. Name the body fluid compartments. Write its normal values. Explain How to measure ECF ?
6. Name the types of neuroglia. Explain the functions of neuroglia with diagrams.
7. Write about intercellular connections with neat labelled diagram
8. A 3 month old baby presented with H/O of yellowish discolouration of skin since a few days often birth. O/E the baby has severe pallor and edema and shows decreased activity, his mother is Rh-negative blood type (1+3+1)

a.What is the likely diagnosis?

b. Discuss the pathophysiology of the disease

c. How can it be prevented?

PES Institute of Medical Sciences and Research
Department of Physiology
Monthly test-II

Date-28-02-24 **Time: 4.00PM-5.30PM**

Long Essay questions (1x15=15Marks) (draw diagrams wherever necessary)

1. Define Cardiac cycle & write its normal value. Describe the events of cardiac cycle with normal values. Describe the pressure volume changes of left ventricle in detail (2+8+5)

Short Essay questions (7x5=35Marks)

2. Describe the factors affecting Cardiac output.
3. Short term regulation of blood pressure
4. Describe Conducting system of heart
5. Cardiac arrhythmias
6. Explain Lead II ECG with a neat labeled diagram
7. Heart sounds
8. A 45 year old man met with an RTA & had severe bleeding. On examination he was restless, BP was 80/50mm Hg, HR was 120 /min, RR was 18 /pm. His skin was cold and clammy & he had thready pulse
   a. What is the diagnosis? 1M
   b. Describe the stages 2M
   c. Describe the treatment for the above condition. 2M

PES Institute of Medical Sciences and Research
Department of Physiology
Monthly test-III

Date-28-03-24 **Time: 4.00PM-5.30PM**

Long Essay questions (1x15=15Marks) (draw diagrams wherever necessary)

1. Define Glomerular filtration rate & write its normal value. Describe the factors affecting GFR and how is it regulated. Add a note on measurement of GFR (3+5+4+3)

Short Essay questions (7x5=35Marks)

2. Special features of renal circulation
3. Explain the mechanism of HCl secretion
4. Deglutition reflex
5. Regulation of pancreatic secretion
6. Describe the digestion and absorption of fat
7. Types of small intestinal movements
8. A 40 year old business executive complained of severe epigastric pain since last 6 months which increased with intake of food. He had few episodes of vomiting with blood and of late his work is stressful due to which his meals are irregular.
   a. What is the likely diagnosis? 1M
   b. Describe the pathophysiology of the disease 2M
   c. Describe the treatment for the above condition. 2M

PES Institute of Medical Sciences and Research
Department of Physiology
Monthly test-IV

Date-28-05-24

Time: 4.00PM-5.30PM

Long Essay questions (1x15=15Marks) (draw diagrams wherever necessary)

1. Define synapse. Classify synapse. Explain transmission of impulse with the help of diagram. Explain any four properties of synapse. (2+3+6+4)

Short Essay questions (7x5=35Marks)

2. Corticospinal tract
3. Muscle spindle
4. Pain pathway
5. Spermatogenesis
6. Hormonal regulation of menstrual cycle
7. Types of cutaneous receptors with diagram
8. Dorsal column pathway

PES Institute of Medical Sciences and Research,Kuppam

Department of Physiology Monthly test-1 **Date**-24-01-23,Time:2.15-3.45pm

Long Essay questions (1x15=15Marks)

1.Define Erythropoiesis. Explain the steps of Erythropoiesis with a neat labeled diagram. Add a note on factors and regulation of Erythropoiesis.(2+8+5marks)

Short Essays questions (5x7=35Marks)

2.Describe the functions of Plasma proteins. Add a note on Edema

3.Draw a neat labeled diagram of Cell .Add a note on Peroxisomes.

4. Explain the stages of Phagocytosis with diagram.

5.Name the Body Fluid Compartments. Describe the methods of measurement of ECF. Add a note on characteristics of ideal substance .

6. What is RMP. Explain the Ionic basis of Resting membrane potential.

7. Define Homeostasis . Explain the types of feedback mechanisms with examples.

8. Define Anaemia. Explain with diagram Morphological classification of Anaemia.

PES INSTITUTE OF MEDICAL SCIENCE AND RESEARCH

DEPARTMENT OF PHYSIOLOGY

First -MBBS /Monthly test/ March 2023

THEORY PAPER

Time: 1hour and 30 Min

Maximum marks: 50

Draw diagrams wherever necessary

LONG ESSAY:

2X15marks=15marks

1. A young female aged 24 Years presents with complaint of drooping of eyelid and double vision especially in the evenings, weakness in the jaw muscles, upper arms. Her symptoms become worse after climbing stairs.
   - What is the probable diagnosis and give your reasoning(2)
   - How can you confirm your diagnosis(1)
   - Where is the location of the lesion? (1)
   - Explain the events that occur at the location with neat diagram (4+2)
   - Name the drugs/toxins that act on the location and describe their mechanism of action(3)
   - Name the drug that can help her condition(1)
   - Describe other diseases that occur here(1)

SHORT ESSAYS:

7X5 marks =35

3. Explain lung pressure during breathing cycle with help of diagram
4. Describe non-respiratory functions of lungs.
4. Draw and explain the diagram of Sarcomere before and after muscle contraction.
5. Explain the functions of platelets.
6. Explain isometric and isotonic contraction with diagram and give example.
7. What are divisions of autonomic nervous system? Briefly explain their functions
8. Briefly explain enterohepatic circulation

PES INSTITUTE OF MEDICAL SCIENCE AND RESEARCH

DEPARTMENT OF PHYSIOLOGY

I-MBBS 2022-2023

THIRD MONTHLY TEST

Max marks: 40

Draw diagrams wherever necessary **Time: 1hours and 30 Min**

LONG ESSAYS: **1X15marks= 15marks**

1. Explain transport of Oxygen in Blood. Draw and explain Oxygen Hemoglobin dissociation curve. What is the significance of the shape. What are the factors that affect it (3+6+2+4)

SHORT ESSAYS: **5X5marks=25marks**

2. Patient aged 45 year-old male comes with h/o breathlessness, cough and tightness of chest. On examinations rhonchi is heard bilaterally. The doctor requested spirometry to confirm diagnosis
   a. What is your probable diagnosis? (1)
   b. What Spirometry tests will help in this diagnosis(1)
   c. What do you expect to find in the spirometry results? (2)
   d. What is the principle behind the treatment?(1)
3. What is Acclimatization? Explain the changes that occur.(1+4)
4. Explain chemical regulation of respiration.(5)
5. What is CPR? When do you initiate CPR? Describe the steps of doing CPR in detail. (1+1+3)
6. What is Cyanosis? What is the cause of Cyanosis? Describe the types of Cyanosis. How do you assess Cyanosis by clinical examination?(1+1+2+1)

PES INSTITUTE OF MEDICAL SCIENCE AND RESEARCH
DEPARTMENT OF PHYSIOLOGY
IV MONTHLY TEST - I MBBS Date: 06.06.2023

Time: 4.00pm – 5.30pm **Total marks=50 M**

LONG ESSAY: **1x15=15M**

1. Define Cardiac cycle and name the events with the help of a diagram. Describe the volume and pressure changes in detail and its relationship to ECG and heart sounds (1+2+12)

SHORT ESSAY: **7x5=35M**

2. Describe the salient features of coronary circulation.
3. Explain Frank-Starling law and how does it affect cardiac output?
4. Dysbarism (Decompression sickness)
5. Define Compliance and explain factors affecting it. Name two conditions in which it is altered and give reasons? (3+2)
6. Explain neural regulation of respiration with a diagram.
7. A 45 year old office assistant one morning as she woke up late, she panicked and jumped out of the bed . she felt lightheaded and thought she might faint .She felt her heart racing . On walking she noticed her light-headedness disappeared and the rest of the day was uneventfull .

a)What is the above condition called? (1/2marks)

b) What are the sequence of events that caused this condition? (1 ½ marks)

c)Her light-headedness was transient because of a reflex . what is this reflex and explain it in detail? (3 marks)

8. Explain Triple response in detail.

PES INSTITUTE OF MEDICAL SCIENCE AND RESEARCH
DEPARTMENT OF PHYSIOLOGY
V MONTHLY TEST - I MBBS Date: 27.07.2023

Time: 4.00pm – 5.30pm **Total marks=50 M**

LONG ESSAY: **1x15=15M**

1. Define synapse. Classification of synapse. Explain the steps of transmission of synapse. Explain any four properties of synapse in detail? (1+2+4+8=15M)

SHORT ESSAY: **7x5=35M**

2. Explain Dorsal column medial lemniscal pathway with a neat labelled diagram?
3. Physiological actions of Glucocorticoids
4. Addissons disease
5. Physiological actions of parathormone and add a note on tetany
6. Explain any six properties of Receptors
7. Describe the types of pain and pain pathway in detail? (10M)

PES INSTITUTE OF MEDICAL SCIENCE AND RESEARCH

2021 - 2022

DEPARTMENT OF PHYSIOLOGY

I MONTHLY TEST

Date: 26:05:2022 **Time: 4:00 to 5:30pm**

Max marks:50M

DRAW NEAT LABELLED DIAGRAMS WHEREVER NECESSARY

LONG ESSAY : **1X15=15M**

1. Define Erythropoiesis. Explain steps of erythropoiesis in detail. Add a note on factors regulating erythropoiesis.

SHORT ESSAYS : WRITE IN BREIF **5X5=25M**

2. Role of feedback mechanisms in Homeostasis
3. Active transport mechanism
4. Classify Anaemia based on etiology and morphology. Explain Pernicious anaemia.
5. Cell mediated immunity
6. Explain the genesis of RMP

SHORT ANSWERS : WRITE IN SHORT **5X2=10M**

7. Apoptosis
8. Name the body fluid compartments and mention their normal values.
9. Functions of plasmaproteins
10. Steps involved in phagocytosis
11. Nerst equation

PES INSTITUTE OF MEDICAL SCIENCE AND RESEARCH

DEPARTMENT OF PHYSIOLOGY

II MONTHLY TEST

Date: 28.07.2022 **Time: 4:00 to 5:30pm**

Max marks:50M

DRAW NEAT LABELLED DIAGRAMS WHEREVER NECESSARY

LONG ESSAY : **1X15=15M**

1. Describe the action potentials in heart? Explain the refractory period in a cardiac muscle and its importance.

SHORT ESSAYS : WRITE IN BREIF **5X5=25M**

2. Define Hypoxia? Classify and explain the different types of Hypoxia.
3. Explain Conducting system of heart with a neat labelled diagram.
4. Compare and contrast Cardiac muscle and Skeletal muscle.
5. Define Acclimatization.Explain the changes during Acclimatization.
6. Explain Chemical regulation of Respiration.

SHORT ANSWERS : WRITE IN SHORT **5X2=10M**

7. What is AV nodal delay?What is its clinical significance.
8. Draw a labelled diagram of ECG?
9. What is PR interval.What is its importance.
10. What is cyanosis?What are its types.
11. Give the normal value of total body water.Which substance is used to measure ECF.Give 2 properties of an ideal substance.

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III MONTHLY TEST

I MBBS August 2022

Date: 25.08.2022 **Time: 4:00 to 5:30pm**

Max marks:50M

DRAW NEAT LABELLED DIAGRAMS WHEREVER NECESSARY

LONG ESSAY : **1X15=15M**

Define Cardiac cycle.Explain the pressure volime changes in the left ventrical with the help of a neat lablled diagram.Add a note on heart sounds.

SHORT ESSAYS:WRITE IN BREIF **4X4=20M**

1. Describe the factors affecting cardiac output?
2. Describe the short regulation of Blood pressure?
3. Describe the waves and intervals of typical ECG and its importance with the help of neat lablled diagram?
4. What are the phases of gastric secreation.Describe the machanisim of gastric HCL secreation in detail with diagram?

SHORT ANSWERS : WRITE IN SHORT **2X2=10M**

1.Write four pecularities of coronary circulation?

2.Describe the functions of Sliva?

VERY SHORT NOTE: **1X1=1M**

1.What is stoke's Adam's syndrome?

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DEPARTMENT OF PHYSIOLOGY

I MONTHLY TEST

Date: 02:03:2021 **Time: 4:00 to 5:00 pm**

Max marks:30

Short Notes **3x5=15**

1. Describe the steps of Erythropoiesis
2. Define Action Potential, Explain nerve action potential its ionic basis.
3. Genesis of Resting Membrane Potential

Short answers **5x3=15**

4. Define Simple Diffusion, Facilitated Diffusion Osmosis
5. Function of plasma proteins
6. Define Homeostasis, Name the control mechanism involved in Homeostasis
7. Define Rheobase, Chronaxie, Utilization Time
8. Neuroglia

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II MONTHLY TEST

Date: 30:03:2021 **Time: 4:00 to 5:00 pm**

Max marks: 30

LONG ESSAYS **2X10=20**

1. List the clotting factors. Describe the intrinsic and extrinsic pathway of clotting. Add a note on anticoagulants.
2. Draw a labeled diagram of a sarcomere and sarcotubular system. Explain in detail the molecular basis of skeletal muscle contraction.

Short answers **5x2=10**

3. Landsteiner's Law
4. Haemophilia
5. Gamma globulins
6. Motor unit
7. Starling's law of muscle contraction.

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DEPARTMENT OF PHYSIOLOGY

I MBBS 2020-2021 BATCH- MONTHLY TEST - III

Time: 4.00 - 5.30 PM **Date: 28.10.2021**

Draw diagrams wherever necessary

LONG ESSAY **1X15=15**

1. Name the functional divisions of cerebellum. Describe their connections and functions. Add a note on cerebellar lesion.

SHORT ESSAY (WRITE BRIEFLY ON) **7x5=35**

2. Role of hypothalamus in the regulation of food intake.
3. Structure and functions of the muscle spindle.
4. Referred pain
5. Diseases involving Basal Ganglia
6. Non-rapid eye movement (NREM) sleep.
7. Explain a) types of Aphasia

   b) Kluver - Bucy syndrome.
8. Differences between signs of UMN and LMN lesion

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DEPARTMENT OF PHYSIOLOGY

IV MONTHLY TEST

Time: 4.00 - 5.30 PM

Date: 25.11.2021

LONG ESSAY 1x15-15M

1. Describe the biosynthesis, regulation of secretion and actions of thyroid hormone. Discuss the effects of hyposecretion of thyroid hormone.

SHORT ESSAY (Write breifly) 7X5=35M

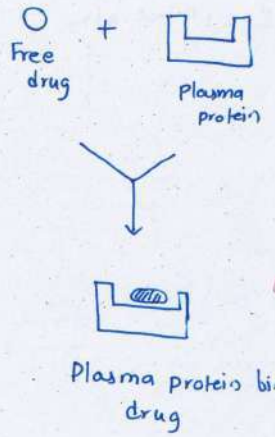
2. Effects of hypersecretion of Growth hormone.
3. Actions of ADH
4. Mechanism of action of protein/peptide hormone
5. Actions of parath hormone
6. Cushing's syndrome
7. Neuro endocrine reflex
8. Regulation of secretion of aldosterone

① Plasma Protein Binding

1) Acidic drug ⟷ Albumin

Basic drug ⟷ α_1 glycoprotein

2) Mechanism:-



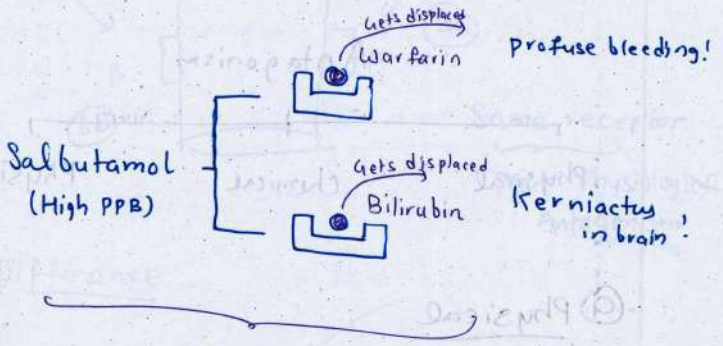
* Long Acting
* Tissue storage.

Note:-

When a drug get plasma binded it will be not available for

ⓐ it's action
ⓑ it's distribution
ⓒ it's metabolism
ⓓ it's excretion (GFR↓)

2) Clinical consequences



* Drugs with high PPB

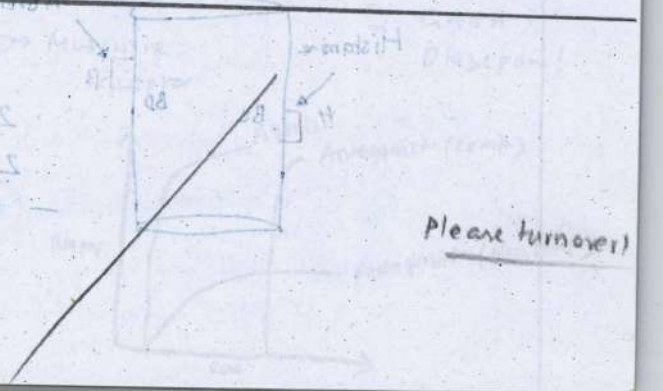
ⓐ Warfarin

ⓑ Tolubutamine (Tolbutamide)

ⓒ Bzd's (Diazepam) etc.

In case of hypoalbuminuria, Furosemide will be given in low dose. (Low Albumin)

In case of MI, Ligocanine will be given in higher dose. (High α-1 GP)



Please turnover!

② Drug Antagonism (1+1 = <2)

Antagonism

Physical — Chemical — Physiological.

① Physical

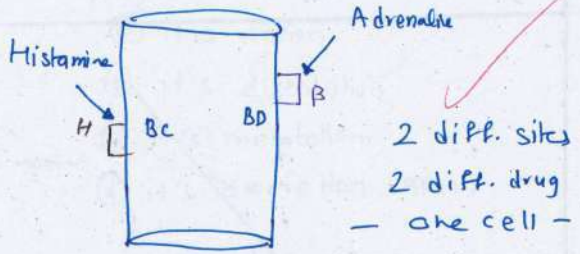
Posion + Activated charcol
(Univirsel antidote)

② Chemical

HCl + Mg(OH)$_2$ → Neutrilisation
(Stomach)

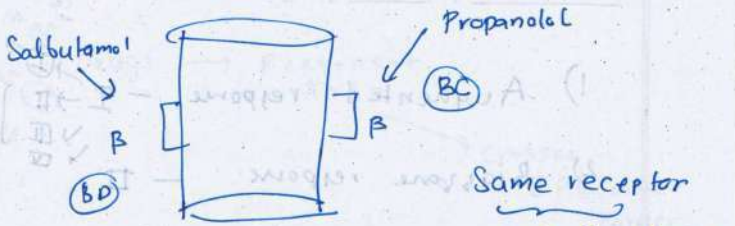
Antacid MOA. (Mode of Action)

③ Physiological



2 diff. sites
2 diff. drug
— one cell —

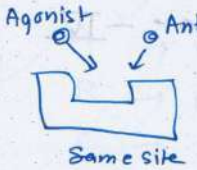
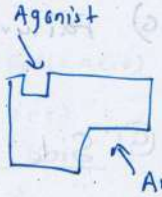
Note:-



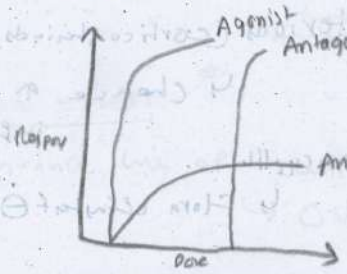
Same receptor

Not Physiological Antagonism.

(d) Difference

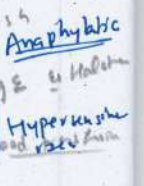
Competive	Non competitive
	 (Allosteric Site)
When dose is ↑ Respones ε Efficacy will be Same.	Response ↓ Efficacy will be ↓
Potency remain ↓	Potency remain Same
Eg Ach Atropin → Muscurie receptor	Eg GABA Diazepam!

Graph



③ Adverse Drug Reactions

1) Augmented response — I
2) Biazzare reponse — II
3) Chronic reponse — III
4) Delayed effect — IV
5) End of drug effect — V
6) Failure of drug effect — IV.



⑤ Side effects

Phenramine → Rx. Alleray
↳ Neurological manifestation. Also!

⑥ Secondary effects

* NSAID (Nonsteriodal antiinflammatory drug)
↓
Gastritis / peptic ulcer

* Steriods (corticosteriods)
↳ Chance ↑ for opportunistic infection

* Tetracyclin
↳ Flora of intest ⊖ → Diarrhea.

© Teratogenicity

Drugs → Pregnent lady → Crosses B Placental Barrier → Effect plac. Foetus!

USFDA

↳ Index for Drug to be used in pregnant women!

(Low Vd) (Slow Hng acting)

3/2

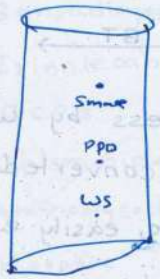
A → Very Safe (Eg. Folic acid)

B → Moderate Safe (Eg. PCT)

C → Safe (Medium)

D → Safe (Low)

X → Toxic (Eg. warfain)



DOC in pregancy

Malaria prophylaxis

↳ Mefloquine.

Anticoagulant

↳ Heparin

Hypothyroidism

↳ Propyl thiouracil

(D) Delayed effect

Chronic use of corticosteroids

↳ Osteoporosis!

© Withdrawl Syndrome! → Therapy: Hospitalisation, Psychological, General, Substitution

A person feel that his well being is dependent on the intake of drug. If he/she suddenly stops the drug they undergo opp. symptoms to which their body is adapted to.

ⓐ Physiological ⓑ Physical. (Tremors; sweating)

⑦ Toxicity

ⓐ Acute ⇒ PCT → Hepatotoxicity

ⓑ Chronic ⇒ ↑Aspirin → Gastritis.

---

④ Biotransformation

* LS —BT→ WS

* The process by which lipid soluble drug is converted into water soluble drug (i.e, easily excretable) is known as "BT/Drug metabolism"

* Sites:-

* 1° site ⇒ Liver → Microsomal enzyme, Macrosomal enzyme

* other → Placenta, Intestine

BT

Phase - I (Non-conjugation)	Phase - II (conjugation) (synthetic)
1) Oxidation	1) Glucouronide conjugation
2) Reduction	2) Glutathione
3) Cyclization	3) Glycine
4) Decyclization	4) Aspi Sulfate
	5) Acetylation
	6) Methylation

* All are mediated by "transferases".

4½

(a) Glucouronide conjugation

Enzyme:- UDP-glucouronyl transferase

Drugs:-

1) Benzodiazepen
2) Irinotecan
3) OCP's.
4) Chloramiplenco)
5) Aminoglycosides / Atravastain / Amiodarone.
6) Morphie
5) PCT.

(b) Glutathione :-

Eg. PCT (Glutathione transferase)

(c) Sulfate :-

(d) Glycine

→ Aspirin

(e) Acetylation

1) Sulfonamide
2) Hydralazine
3) Procainamide

(f) Methylation

Histamine $\xrightarrow{+CH_3}$ Methyl Histamine

Nor Adrenalin $\xrightarrow{+CH_3}$ Adrenaline.

---

(5) Prolonging Drug Action

(A) Oral route

1) Sustained release tablets.
   - ↳ Longer acting
   - ↳ Need not be repeated.

Eg. Diclofenac (Normal = 12 hrs)
(SR = 24 hrs)

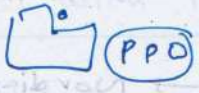
2) Adrenaline + Local Anaesthetic (Eg. Lignocaine)
   - ↓
   - Vasoconstriction
   - Local action
   - Toxicity will be prevented.

4) Implants Eg. Oral contraceptive's.

5) In proteamine Insulin (Drug + Protein)

6) Pencillin G + Procaine
   pencillin G + Benethine } Prolonged action than Normal penicillin G.

(B) ↑ PPB



* Drug goes into storage
* It cannot be excreted
* Stays for longer time.

t 1/2

(C) ↓ Metabolism

* By ↓ metabolism of drug makes drug to stay long in body by preventing it's conversion into water excretable form.

(d) ↓ Excretion

* Low will be excretion
↑ drug will be retained in body.

Note:

Enterohepatic recycling

Eg. OCP's

Drug ⟶ Metabolism ⟶ Intestine
(Glucouronide transformer)
↓
Excretion

Fate

* Active Drug ⟶ Inactive

* Inactive drug (Prodrugs ⟶ Active drug.

Eg. Levadopa ⟶ Dopamine

* Active drug ⟶ Active metabolite

Eg. Diazepam ⟶ Nordiazepam

Hydroxine ⟶ Cetrizine.

* Active drug ⟶ Active toxic

Eg. PCT ⟶ NAPQI ⟹ Mercaptoi uricaal ↓ Excret

Alcohol ⟶ Haptotoxicity.

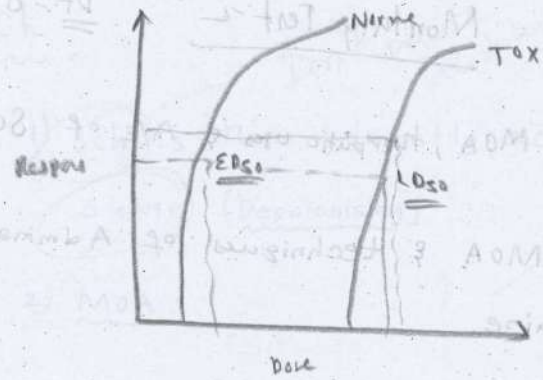
⑥ Threpatic index

↳ Index of drug Safety

Formula

$$TI = \frac{LD_{50}}{ED_{50}}$$

* Drugs with narrow / Low TI will need



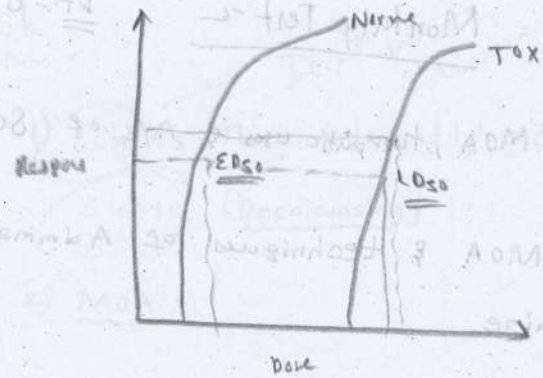
Drugs with Narrow TI

1) Lithium
2) Aminoglycosides
3) Theophyllie
4) Digioxin
5) Morphine / Methotrexale
6) Calcineurin inhibitors.

* They need to monitored because their drug concentration will coincide with pt. respons!

Eg. Digioxin ⇒ 0.4 to 1.4 ng/ml.

* To prevent Adverse effect.



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Eg. Digioxin ⇒ 0.4 to 1.4 ng/ml.

* To prevent Adverse effect.

Monthly Test-2

Dt:- 07/05/23

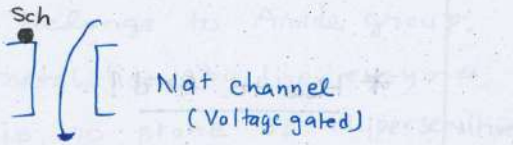
1. Discuss MOA, therputic uses & A/E of Succinylcol.
2. Discuss MOA & techniques of Adminstration of lignocaine.
3. Compare & Contrast Homatropine with Phenylephrin
4. Classify Anticholinergic drugs & discuss their uses of Atropine substitutes. with Suitable examples.
5. Compare & Contrast Dopamine with Dobutamin
6. Enumerate Cardioselective β blockers & discuss their uses & their Advantages over non-selecti β #'s. Enumerate the C/I for the use of β #'s

① Sch

Don't write abbreviations

1) Sch is the indirectly acting peripheral SkMR (Depolarising)

2) MOA



Nat

↓

Muscle contraction

↓

Intial fasiculations → Hyperkalemia

↓

Nm recepter undergoes RAPID DESENSITIZATION

↓

FLACCID PARALYSIS

3) Uses

- For intubation,

* Sch —Typical Pseudochε→ Inactive [Normal patient]

↓

SA
(Duration = 5min)
(onset = 1 to 1.5min)

* Patient with Typical Pseudochε defeciency,

* Sch $\xrightarrow[\text{Pseudochε}]{\text{Atypical}}$ ~~Inactive~~

LONG ACTING!

Sch - Induced Apnea!

(Genetic)

* How to find?

Dibucaine Number

(N)	Heterozygous	Homozygous
80	50-60	20-30

Rx
- Rocuronium
- Ventilator Support
- Fresh Frozen plas
- Recombinant typic Pseudochε.

4) A/ε

i) Hyperkalemia (c/I in Muscle & Nerve i

ii) Malignanet Hyperthermia

iii) Muscle ~~Apnea~~ Soreness after recovery.

iv) ↑ Intragastric Pressure

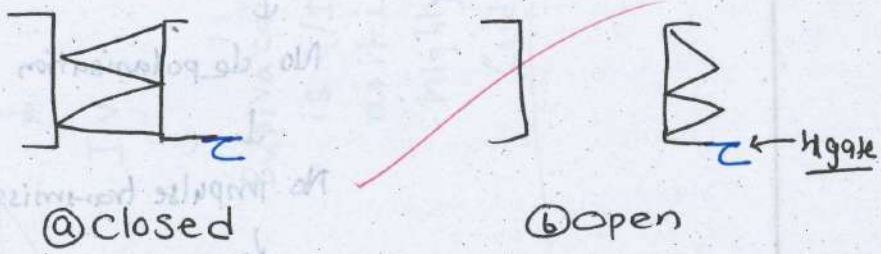
② MOA of Lignocaine

1) Lignocaine is a commonly used Local anesthesia that belongs to amide group.

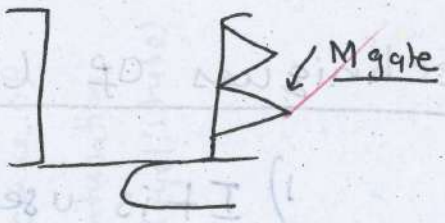
2) As it belongs to Amide group, it is metabolised by liver enzymes, there is no prone of Hypersensitive reactions & the drug has α glyco protein binding.

3) It acts by blocking Voltage-gated Nat channel from inside.

Nat channel types



ⓐ closed ⓑ open



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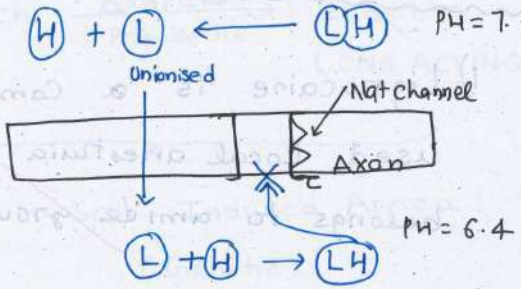


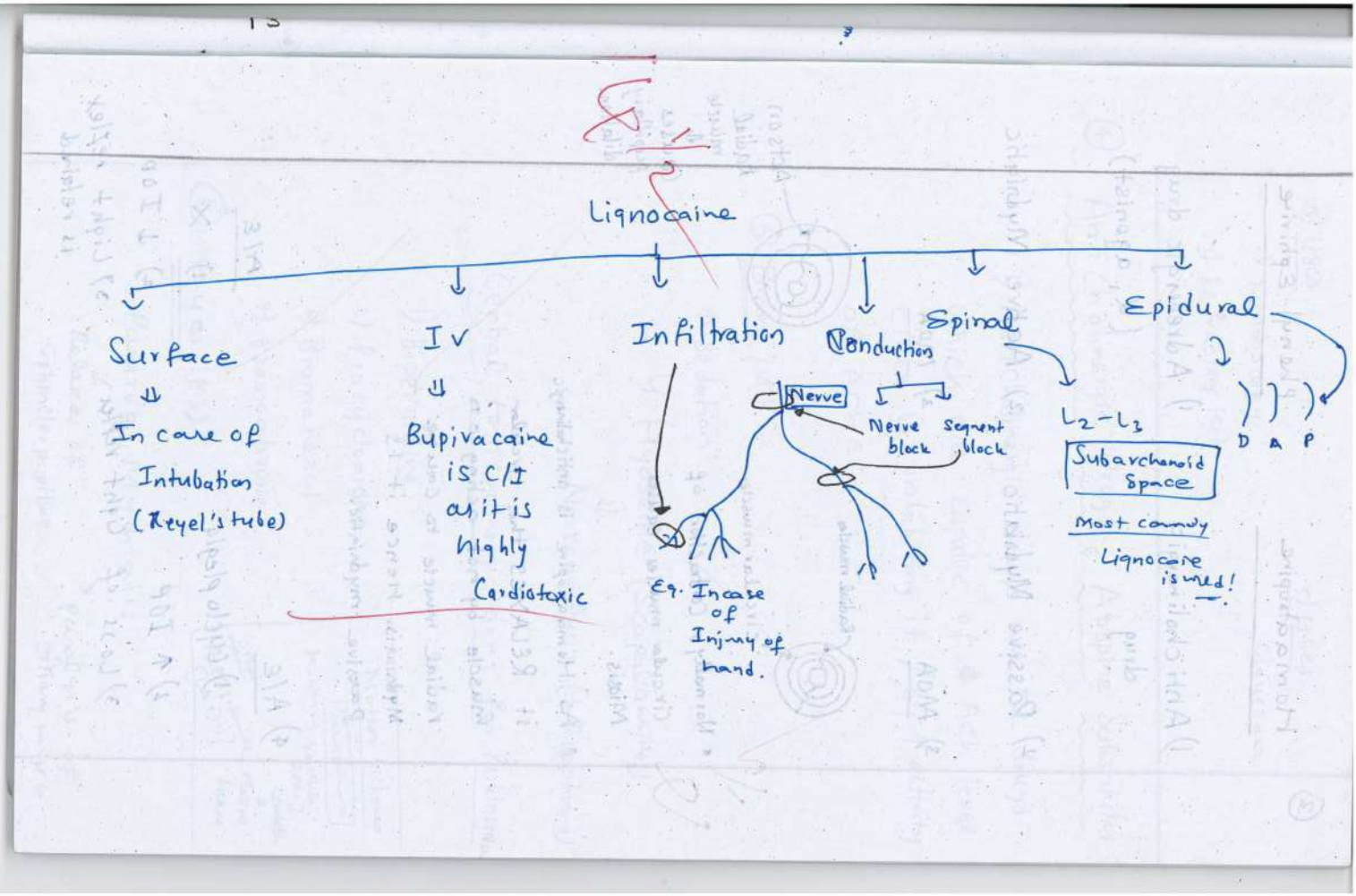
* Lignocaine is a basic drug. Hence, it remains unionised in Alkali medium & becomes ionised in slightly acidic medi

'L' refers to lignocaine

Lignocaine
↓
Block Na+ Channel
↓
No Na+ Entry
↓
No depolarisation
↓
No impulse transmission
↓
No muscle contraction.

Techniques of Lignocaine Adminstrat

1) It is used as class IB antiarrythmic drug.


Lignocaine
Surface
In case of Intubation (Reyel's tube)
IV
Bupivacaine is C/I as it is highly Cardiotoxic
Infiltration
Eg. Incase of Injury of hand.
Conduction
Nerve
Nerve block
Segment block
Spinal
L2-L3
Subarchanoid Space
Most commonly Lignocaine is used!
Epidural
D
A
P

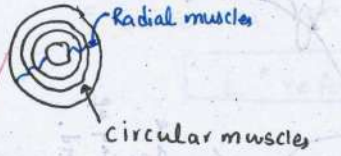
③

Homaatropine

1) Anti cholinergic drug

2) Passive Mydriatic

3) MOA



- Normally, Contraction of circular muscles causes Miosis.
- As Homaatropine is anticholinergic it RELAXES the circular muscle by not acting on radial muscle to cause Mydriasis. Hence it is passive mydriatic.

4) A/E

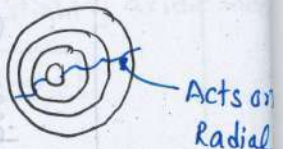
1) Cycloplegia
2) ↑ IOP

Phenyl Ephirine

1) Adrenergic drug (α_1 agonist)

2) Active Mydriatic

3) MOA



A/E

1) ×
2) ↓ IOP

5) Uses

1) Fundoscopy
2) Refractory test

5) Uses

Glaucoma.

④ Anticholinergic Drugs & Atropine Substitutes

1) Anticholinergic drugs are the drugs which are capable of ↓ Ach level either by inhibiting it / by ↑ activity of AchE!

Ⓐ Brain (M1)

↑ Sedation

↳ Hyoscine (Scopolamine)

⇓

Narcoanalysis (+ Thiopentanone)

Central Anticholinergic drug for Parkinsonism

1) Bipernidine
2) Procyclonidine
3) Bromohexol
4) Bromoatropine.

Motion sickness

Scopolamine

Motion → Vestibular app. → cerebellum → Motion Sickness

other Acetazolamide (LA ⊖'s)

Ⓑ Eyes (M3)

Paralysis Mydriasis

✱ Atropine (7-10days) Long acting
Homatropine
Cyclopentolate
Tropicamide (Short acting)

✱ Uses
- Fundoscopy
- Refractory testing.

✱ Ars
Blurring of vision.

© Lungs (M3)
1) Ipratropium Br
2) Tiotropium Br
] BD → COPD

Ⓑ β (M2)
↓ Tachycardia Eg. Atropine
Rx. Bradycar
AV bloc

Ⓔ Stomach (M3)
Telenzepine
Pirenzepine Rx · PUD.
Peptic ulcer d

Ⓗ Intestine (M3) (Anti spasmodic)

④ Urinary bladder (M3)

* Festerodine
* Tolterodine
* solifenian
* Doripeniain.
* oxybutynin.

Rx. overactive bladder.

⑪ Glycopyrollate

↳ preanesthetic

↓ secretion

↓ Reflex Bradycardia

⑫ valethmate BR

↳ cervical ripening

↓

Induction of Labour.

⑤ Dobutamine

1) Exogenous catecholamine

2) Acts on β_1, β_2 & α_1 receptor.

β_1 >> β_2 > α_1 → VC (Mild)

β_1 → Nodal tissue >> Muscle

↑ HR ↑ CO

β_2 → VD

Hence C/I in Hypotension

④ Urinary bladder (M3)

* Festerodine
* Tolterodine
* solifenian
* Dorifeniain.
* Oxybutynin.

Rx. overactive bladder.

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β_1 → Nodal tissue >> Muscle

↑ HR ↑ CO

β_2 → VD

Hence C/I in Hypotension

* Cardiogenic shock with maintained
* Acute CHF

Low dose

* Cardias stress testing

High dose.

Dopamine

1) Dosage dependent action.

2) Endogenous Catecholamine.

* Low dose (2 µg/kg/min)

D_1 recepter (VD)

Renal → GFR ↑

Mesentic → BP ↓

* Moderate dose (2-10 µg/kg/min)

D_1 & β_1 → HR ↑↑

Use

Cardiogenic shock

* High dose (>10 µg/kg/min)

D_1 + β_1 + α_1 → VC

Use

Cardiogenic shock + Hypotension.

Note:-

Hypovolemic shock (15 µg/min/kg body wt)

⑥ Cardioselective β #'s

Rx.

1) Atenolol
   Acebutolol
   } Rx. HTN

2) Bisprolol → chronic CHF
   Betaxolol → Glaucoma

3) Celiprolol → ↑ HDL

4) Esmolol (SA = 9 min ↳ esterase)
   ↳ HTN + Arrythmia.

5) Metapropolol
   ↳ HTN + Arrythmia + Chronic CHC

6) Nebivolol

8) Labetalol
- HTN + Pregnancy
- HTN + Emergency

9) Crevedilol
- Antioxidant
- Chronic CHF
- Has both α + β action.

Non selective β #s

- Propanolol
- Timolol
- Sotalol
- Nodalol

Causes & crosses BOB
→ Dyslip
→ erec dy
↓ Performance anxiety
Sedation

C/I

1) Prinzmetal Angia
2) Diabetes Mellitus
3) Bronchial Asthma / COPD
4) Acute CHF

Other uses of CS B#'s

1) Glaucoma (Betaxolol)

2) Migrane prophylaxis

3) Infantile Hemiangiomas

4) Thyrotoxicosis

5) Portal HTN

6) Performance Anxiety.

tensa.
ile
function

Monthly Test - 3 Dt:- 02/08/23

① Pharmacotherapy of Severe Migraine (or)

Acute Attack of Migrane

Migrane

Vascular theory ⇓ Vasoconstrition followed by Vasodilation

Neurogenic theory ⇓ Inflammation of Trigeminal Nerve

Management of Severe migraine (Prophy

1) TCAs
2) Ona botulinium toxin
3) Anti epileptics (Eg. Gabapentine)
4) β blockers (Eg. Propanolol ; Metapro
5) Candesartan
   CCBs (verapamil) → To counteract initial vasoconstrictio
   Fremanezumab (CGRP Antagonist)

Management of Acute attack of Migraine

1) Non-steriodal Anti inflammatory drugs.
   * Diclofenac
   * Aceclofenac

2) Anti-~~epile~~Emetic drugs
   * Metaclopromide
   * Ondansetrone

3) Sumatriptan (5HT, D & E Agonist)
   (Drug of choice)
   - ⊕ Vasoconstriction (C/I Ischemic Heart ds.)
   - ⊖ Neurogenic inflammation

4) Ergots
   * Ergotamine → Hydroxy Ergotamine
   * Acts as upon α # ; Vasoconstrictor; Dopamine receptor.
   * VC >>> α # (Action)
   * A/E
     - Gangerene
     - Fibrosis
   * C/I
     - Ischamic ds
     - coronary vasospasm.

5) CGRP antagonist
   * Ubrogebpant

6) Lasmiditan (5HT1 F Agonist)
   ↳ Pt with Ischemic Heart ds/cardiac problems →

② 2nd Generation Antihistaminics

1) Drugs

* Terfenadine → Fexofenadine (Active)
  (Active)
  → QT prolongation! (A/E)
* Ebastine
* Rupatadine (Anti Platelet activating factor)
* Loratadine

2) Advantages

* Donot cross blood brain barrier
* Less / No anticholinergic properties
* Less sedative

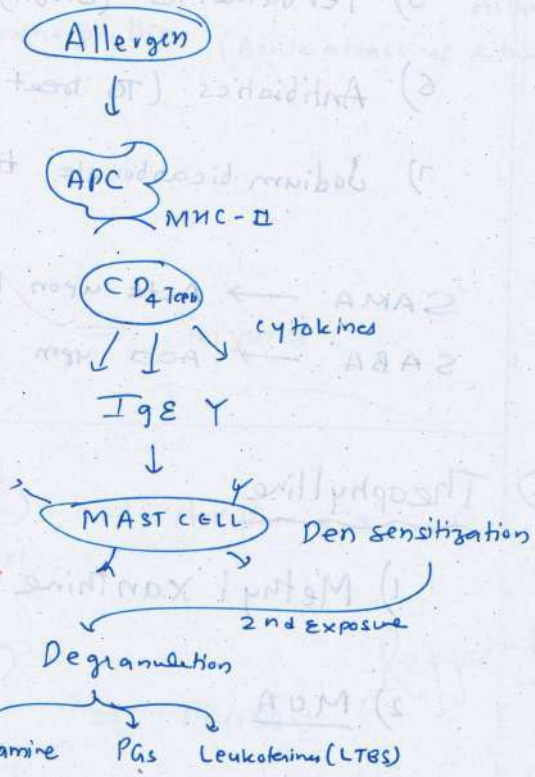
3) Rx.

Allergic Rhinitis.

Topical 2nd gen Antihistaminics

1) Azelastine
2) Olopatadine → Drops / Spray

(3) Management of Status Asthmaticus



* Sudden onset of Asthma is "Status Asthmaticus" (It requires immediate Support).

1) Salbutamol (β_2 agonist) (SABA)
2) 100% Humidified O_2
   (Dry air irritates Bronchi)
3) Hydrocortisone (IV)
   (Steriod)

4) Ipratropium Bromide (SAMA)

5) Terbutaline (SABA)

6) Antibiotics (TO treat infection)

7) Sodium bicarbonate to treat acido

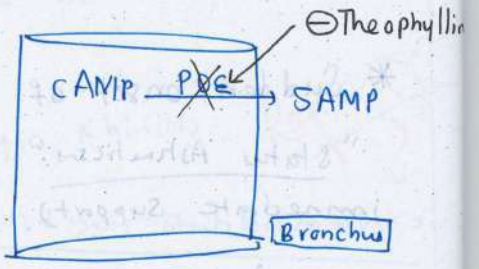
SAMA → Acts upon Large bronchiole → COPD

SABA → Acts upon Small bronchiol → Asthma

④ Theophylline

1) Methyl xanthine (Eg. Caffine)

2) MOA



* ⊖ PDE
* ⊖ Adenosine Receptors
* Promotes histone deacetylation
* Activates anti-inflammatory interleukin (IL-10)

3) Rx.

Theophylline (Severe to peristant Asthma)

Amino phyllin (Acute attack of Asthma)

4) A/E

⇒ Seizures

⇒ Arrythmia's

⇒ Diuresis

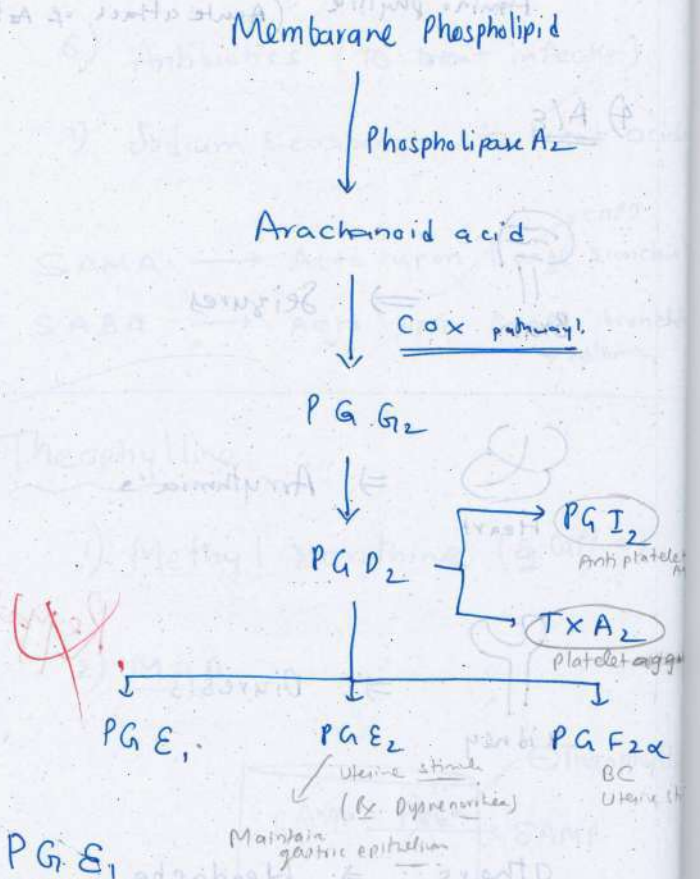
Repeating?

Others ⇒ Headache

⇒ Nausea

→

⑤ Prostaglandin Analogues



PG E_1

1) **Misprostol**
   - * Cervical priming
   - * Medical termination of Pregnan
   - * Peptic Ulcer ds.

2) **Gemaprost**
   - * Cervical priming

3) Alprostadil

* To open patent ductus arteriosus

why:

As flow is already established Sudden closure may leads to death until surgery. So it kept open using Alprostadil until surgery.

* Erectile dysfunction.

Don't use penis!

PGE_2

* Dinoprostone

→ Mid-term abortions.

→ Induction of labour

→ PPH.

$PGF_{2\alpha}$

1) Unoprost
2) Carboprost

} • PPH Rx
• Mid term abortions.

C/I in Bronchial Asthma

3) Latanoprost
Tarvoprost
Bimatoprost

} - open angle glaucoma. Rx.

Topical hence can be used in Bronchial Asthma

PGI_2

* Iloprost

Rx Pulmonary HTN

→

⑥ Expectorants

1) Used in productive Cough.

2) MOA

↑ Cough to pull out Sputum.

3) Drugs

* Ammonium chloride
* Guaiphenesin

Regarding?

4) others

* Mucolytics
  - Bromohexine
  - Ambroxol

Thick Sputum —Depolymerisation→ Thin & wat

(Mucopoly" saccaride)

* Carbocystine & Acetylcysteine

→ Break Sulphide bonds in Thick Sputum & makes it Thin & watery.

Monthly Test - 1

20 March 24

1) Fixed Dose Combination:-

Definition:-

Combination of two (or) more drugs in a fixed dose ratio into a single formulation is called as the fixed dose combination.

Advantages:-

→ Fixed dose combination increases the patient compliance.

→ Fixed dose combination increases efficacy

→ Reduces the cost of drugs

→ Reduces the side effects

→ Have syngeristic effect

→ prevents the development of the antimicrobial resistant diseases like - Tuberculosis, AIDS etc., by a single drug usage instead of multiple drugs.

Disadvantages:-

- Inflexible fixed dose ratio
- Increased Toxicity due to the inappropriate combinations.
- In case of adverse effects the component of FDC causing it can't be known.
- Physicians & pharmacologists tend to ignore the compositions.
- Drug is not compatible if any one of compound is contraindicating other.

Examples:-

1. Levodopa + carbiDopa
   ↓
   Treatment of parkinsonism
2. Ferrous sulphate + Folic acid
   ↓
   Treatment of anemia in pregnancy.

③ Oestrogen and progresterone

↓

used as an oral contraceptive

④ Probenecid + Ampicillin / penicillin

↓

prolongation of drug action.

2) ADR:-

• ADR - Adverse Drug reaction.

Definition:-

According to WHO -

"Any response to the drug that is noxious and unintended and occurs at the doses given to the humans for therapy (or) diagnosis of a disease (or) for modification of a physiological function".

Classification of ADR:-

1. Based on
   - Onset of Event
   - Severity
2. Rawling-Thompson classification
3. Wills and Brown classification.

Onset of Event:

1. Acute - occured less than 2hours after taking drug
2. Subacute - 2hrs-6hrs
3. lethal - more than 2days.

Severity:

1. Minor
2. Moderate
3. Severe
4. lethal.

Rawling-Thompson classification:-

Type A Type B

Type A :- Augmented

- It is non-immunological
- Commonly termed as Intolerance.

Type B :- Bizzare

- It is immunological
- commonly termed as Allergic reactions

Wills and Brown classification:-

- ✓ Type A – Augmented
- ✓ Type B – Bizzare
- ✓ Type C – Continous / prolonged
- ✓ Type D – Delayed
- ✓ Type E – End of Therapy
- ✓ Type F – Failure of Therapy
- ✓ Type G – Genotoxicity
- ✓ Type H – Hypeosensitivity
- ✓ Type U – Unclassified.

Type A:- Augmented.

predictable reactions.

Reactions that can be predicted by known knowledge of pharmacology of drug.

Eg:-

- ✓ Hypoglycemia due to insulin
- ✓ Hypotension due to antihypestensives

Type B: Bizzare

Unpredictable reactions

Reactions can't be predicted by the known pharmacology of drug.

Eg:- ✓ penicillin : ⟶ Anaphylaxis
causing

✓ Anaphylaxis – Death due to respiratory / cardiovascular depression.

Type C: Continous

Reactions due to prolonged use of drugs

Eg:- ✓ Immunosupression
↓
Due to Costicosteroids

✓ parkinsonism

↓

Due to antipsychotic Drugs.

Type D:- Delayed

- ~~Orexo~~ - Mutagenicity
- Carcinogenicity
- Tetragonecity.

Type E :- End of Therapy

- Drugs of addiction

morphine phenobarbitones

Type F :- Failure of Therapy

- Oral contraceptives Failure

↓

Due to Enzyme induction of certain microsomal Enzymes.

Type G → Genotoxicity

Type H → Hypersensitivity

↓

- Usage of certain drugs
  Penicillin, Aspirin etc.,
  ↓
- Release of IgE - fixed to mastcells
  ↓
- Exposure to drug again
  ↓
- Antigen - Antibody reaction occurs
  ↓
- Release of
  - Histamine
  - Prostagladins
  - Leukotreins
  ↓
- Causes
  - Hypotension
  - Bronchospasm
  - Anaphylactic reaction.

Managment of Anaphylactic reaction:-

① Adrenaline
  ↓
- main route of treatment
- 0.3 - 0.5ml (1:1000)
- Intramuscular

- Same dosage is given after 10-20min if required.

② Hydrocortisone

↓

100mg through IV Every 6th hourly

↓

(intravenous)

③ α_1 antihistamines.

↓

chlorpheniramine malate

↓

10-20mg through slow IV.

3) Methods of prolongation of drug Action:

1) By retarding absorption of drug
2) By increasing the plasma protein-binding
3) By decreasing the metabolism of drug
4) By retarding the renal Excretion.

1) By retarding absorption of drug:-

a) Oral:

Drugs are coated with resin (or) substances that release / dissolve at different intervals of time (sustained - release)

Eg:- Diclofenac has duration of action 12hrs.

Sustained release of diclofenac

↓

Duration of action → 24hrs.

b) parenteral:

① Drugs given by subcutaneous (or) intramuscular administration in insoluble form (or) as oilysolution

Eg:- insoluble form → procain in combination with penicillin

oilysolution → Depots.

② By including a vasoconstrictor along with the drug.

Eg:- Adrenaline + local anesthetics.

c) Transdermal Drug delivery system:-

Drug administered in form of the adhesive patches / strips

Eg:- GTN.

d) By increasing plasma protein binding:-

Highly plasma Protein bounded drugs

↓

Pharmacologically inactive

↓

Temporary storage of drugs

↓

Delays absorption.

Eg:- Sulphodiazine

↓

low plasmaproteinbound

↓

Duration - 6hrs

Sulphodoxine

↓

High plasma ptn bound

↓

24hrs.

3) By Decreasing metabolism of drug:-

a) Small chemical modifications

Eg:- Addition of Ethinyl group to Estradiol

↓

prolong duration of oral contraceptives

b) Inactivation/ Inhibition of specific Enzyme by one drug that prolongs duration of another drug

Eg:- Allopurinol prevent degeneration of 6-mercaptopurine.

4) By retarding renal Excretion:-

Done by supressing tubular secretion of drug.

Eg:- probenicid with penicillin/ ampenicillin/amoxillin.

4) ## Drug Antagonism:-

Definition:-

In antagonism, "Effect of one drug is decreased (or) abolished due to the presence of another drug".

Types:-

1. physical antagonism
2. chemical antagonism
3. physiological antagonism
4. Receptor antagonism
   - competitive
   - non competitive

1. physical antagonism:-

The opposing action of drugs are due to their physical property

Eg:- Adsorption of alkaloid by activated charcol ↓ used in "Alkaloid poisioning".

② Chemical antagonism:-

The opposing action of the drug is due to chemical property.

Eg:- Antacids are weak bases
↓
neutralize the gastric juices
↓
used in "peptic Ulcers".

③ physiological antagonism:-

There, two drugs acts on two different receptors (or) different mechanism but on same physiological system showing opposite effects.

Eg:- insulin & Glucagon on blood glucose

Histamine & Adrenaline
↓ ↓
Broncho constriction Bronchodilation

④ Receptor antagonism :-

Antagonist acts on the receptor same as agonist and inhibits its effect.

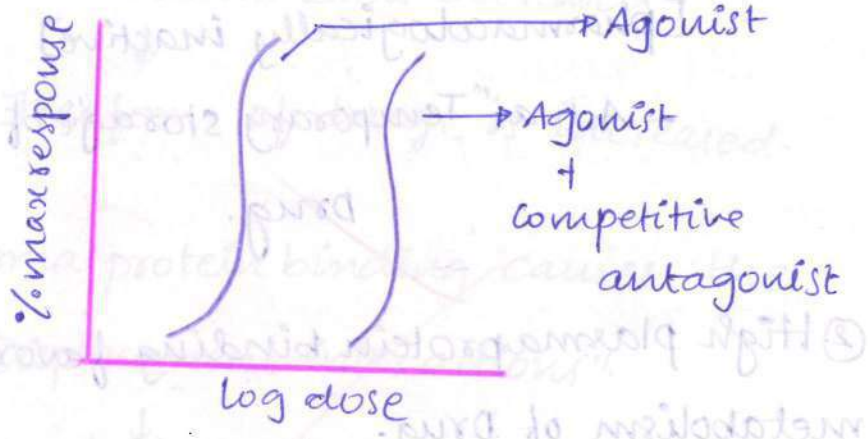
a) Competitive antagonism :

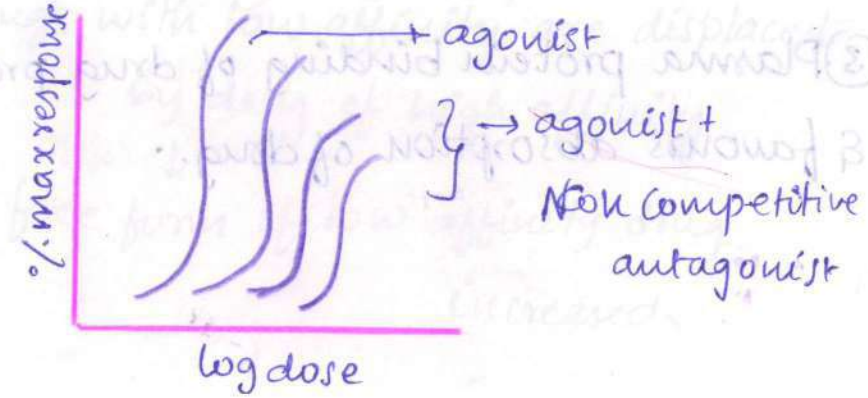
Antagonist binds on same site of receptor as that of agonist.

b) Non Competitive antagonism :-

Agonist & Antagonist bind for different sites of receptor but the effect of Agonist is effected.

Examples





6) First order Kinetics :-

Def :- Constant fraction of drug is eliminated from body per unit time.

- Rate of elimination of drug is directly proportional to the plasma concentration of drug.
- plasma half life of drug following the first order kinetics is constant.
- Most of drug is eliminated at the end of 4-5 $t_{1/2}$

Eg :- Most of drugs follows this kinetics.

Eg :- Lets assume a drug 'A' with $t_{1/2}$ of 1hr with a plasma concentration 100 µg/ml.

Then rate of elimination,

100 µg/ml $\xrightarrow[t_{1/2}]{1hr}$ 50 µg/ml

$t_{1/2}$ ↓ 1hr

25 µg/ml.

"Very good presentation of the answers- keep it up"

Zero Order Kinetics :-

Def :- Constant amount of drug is Eliminated from body per unit time.

- Rate of elimination is independent of the plasma concentration.

Eg :- Ethyl alcohol Eliminated at rate of 10 µg/hr.

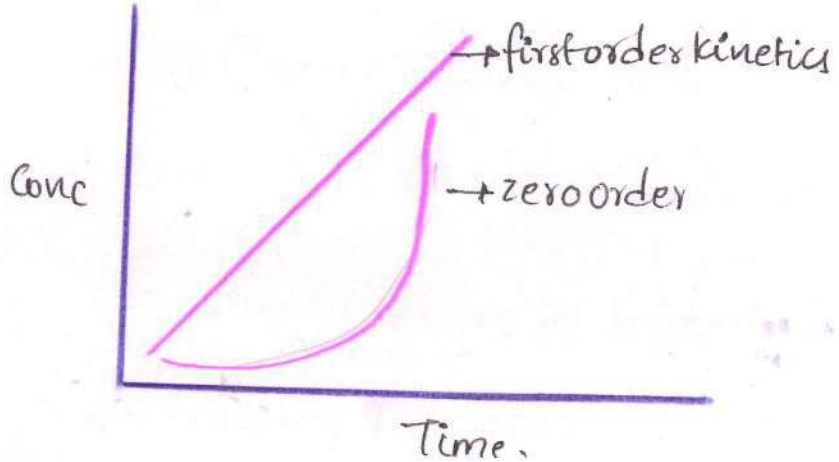
- Plasma half life of drug isn't constant.

Eg :- Drug 'B' with initial conc of 200 µg/ml & rate of elimination 10 µg/hr, then,

- 200 µg/ml $\xrightarrow[10\ \mu g/hr]{t_{1/2}}$ 190 µg/ml

$\downarrow t_{1/2}$

180 µg/ml.



1) Nitrates:-

- Organic nitrates are prodrugs
- They release nitric oxide (NO)
- Mainly venodilators, but sometimes also arteriolar dilators

↓

∴ Decreases / Preload
\ Afterload.

Anginapectoris:-

- chest pain due to myocardial ischemia that occurs as a result of imbalance in myocardial O_2 supply & O_2 demand.

Mechanism of Action:-

1) Molecular mechanism of Action:-

Nitrates

↓ Dinitrated in cardiac muscle cell

Release of active free radical NO

↓

Increase in cytosolic Guanylcyclase

↓

Increase in CGMP

↓

Activation of protein kinase

↓

Dephosphorylation of myosin light

↓

Inhibition of myosin

↓

NO binding with Actin

↓

Relaxation

2) cellular mechanism of nitrates:-

Nitrates → ↑ formation of NO

↓

↑ Guanylcyclase

↓

↑ cGMP

↓

relaxation of Cardiac / Vascular smooth muscle.

Therauptic Uses:-

1) Anginapectoris

- used in both Variant / classical Angina.
- Acute anginal attack - 0.5mg SL
- prophylaxis of Angina
- 6-8 Drug free hours.

2) Myocardial Infarction

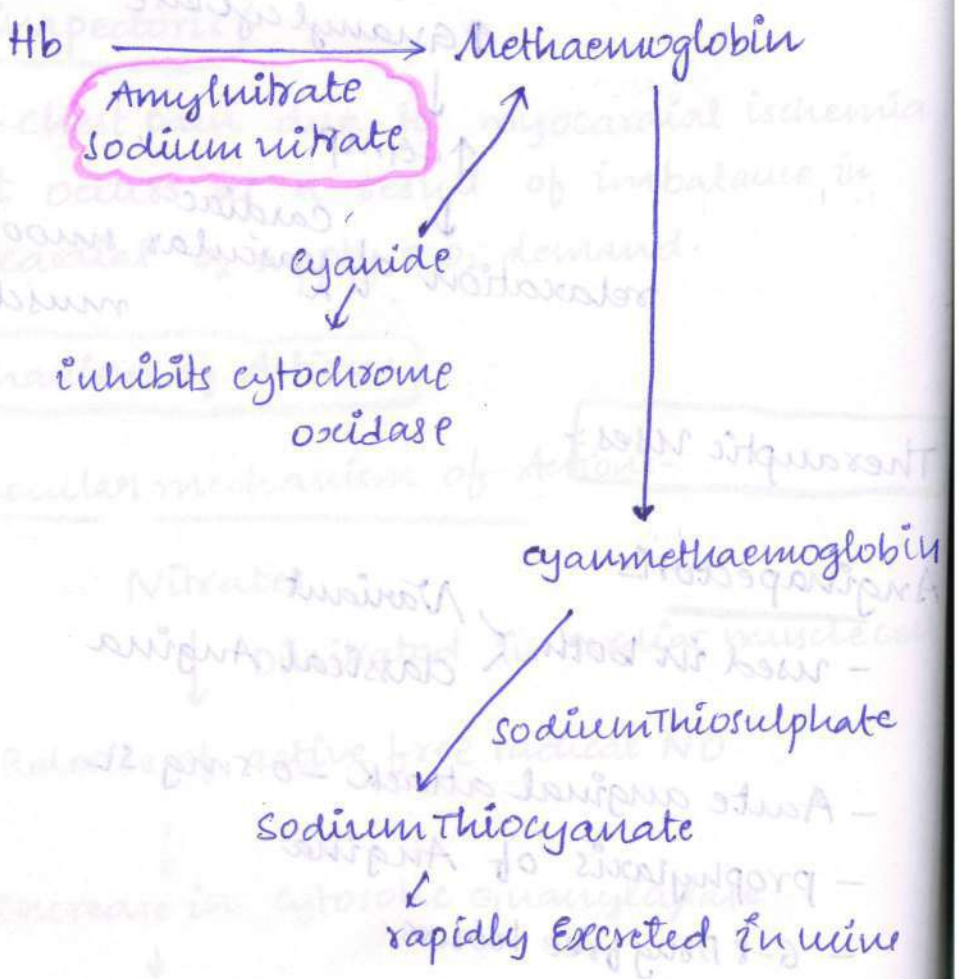
- GlycesylTrinitrate - IV

3) Congestive heart failure / left ventricular failure
   - nitroglycerin - IV infusion

4) Interventional cardiac procedures

5) Hypertensive Emergencies
   - nitroglycerine (IV)

6) cyanide poisioning



7) Biliary colic - nitroglycerine IV)

8) Oesophageal spasm

2) Management of Myocardial Infarction:-

1) Bedrest

2) O_2 inhalation (100%)

3) **Analgesic**

- Inj. Morphine – 5mg (IV)

[~ 1mg/min]

4) **Antiplatelet Agent**

- Tab. Aspirin – 100-300mg O.D

- If patient allergic to ~~Aspirin~~

↙

clopridogrel – 300mg.

5) **Anticoagulant**

- Inj. Heparin – 5000U bolus.

[~ 10000U/hr]

6) **Thrombolytic Therapy.**

- Alteplase – 100mg

- 15mg – IV bolus
- 50mg – over 30min IV infusion
- 35mg – over 60min IV infusion.

7) **Nitrates**

- Inj Nitroglycerin - for peristant & recurrent pain & LVF
- Tab Nitroglycerin - 0.5mg sublingual
  - max: 3 DOSES.

8) **ACE inhibitors**

- Tab Enalapril - 5mg - OD.

9) **β blockers:-**

- Administered only after 24hrs if there are no contraindications.

- Inj Metaprolol - 5mg - IV
- Tab Metapropol - 50mg/day - after 2nd day.

10) **statins:-**

- Atorvastatin - 40mg - OD.

11) **Left ventricular failure:-**

- Inj furosemide - 40mg - IV

12) **Pump failure:-**

- Dopamine - 2μg/kg/min - IV

13) Ventricular fibrillation:-

- Inj Lignocaine - 1mg/min - IV

14) Lifestyle modification.

3) Therauptic Uses of Digoxin:-

- Digoxin - cardiac glycoside
- source - Digitalis lanata.
- MOA - partial inhibition of Na+-K+ ATPase
  - calcium induced calcium release from sarcoplasmic reticulum.
- Result - Increase in strength of contraction
- Therauptic Use:- Atrial fibrillation
  - AF is one of the commenest cardiac arrythmia
  - In AF - heast beats at rate of - 350-600/min

- Digoxin
  ↓
  Has both direct & in direct Effect on Avnode (vagomimetic)
  ↓
  ↑ Effective refractory period
  ↓ conductionvelocity
  ↓
  ↓ ventricular rate.

- preparation:- 0.25mg Tab

Drugs Used in Heast failure :-

Heast failure

① Aldosterone Antagonists
- Spironolactone
- Eplerenone

② β-blockers
- Bisoprolol
- Carvediol
- Metoprolol
- Nebivolol

③ calcium sensitiser
- Levosemindan

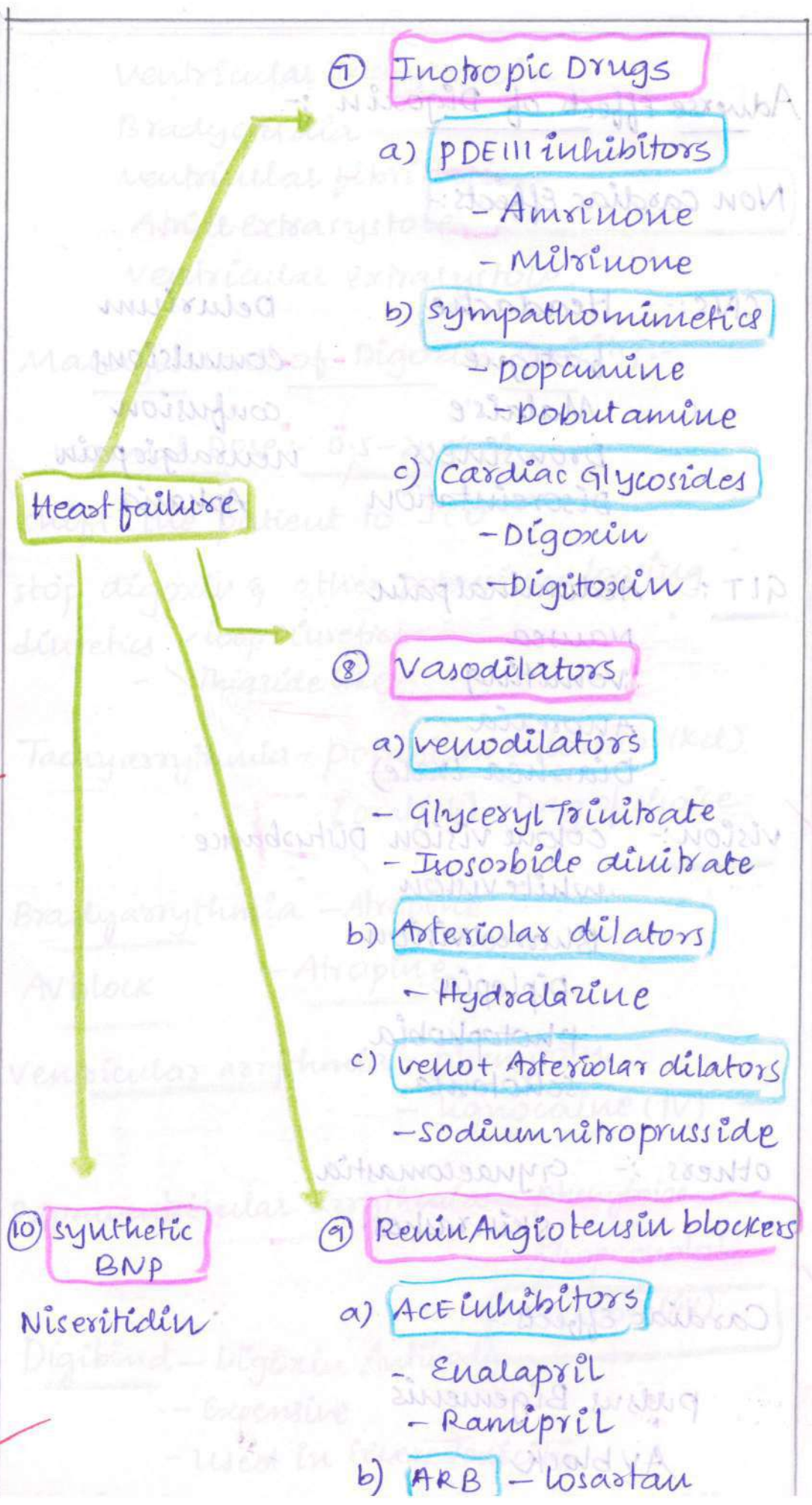
④ Diuretics

a) High ceiling diuretics
- Furosemide
- Torsemide

b) Thiazides
- Hydrochloro Thiazide
- Xipemide

⑤ Neprilysin inhibitor
- Sacubutril

⑥ Newer Drugs
- Sacubitril + Valsartan


Heart failure
① Inotropic Drugs
a) PDE III inhibitors
- Amrinone
- Milrinone
b) Sympathomimetics
- Dopamine
- Dobutamine
c) Cardiac Glycosides
- Digoxin
- Digitoxin
⑧ Vasodilators
a) Venodilators
- Glyceryl Trinitrate
- Isosorbide dinitrate
b) Arteriolar dilators
- Hydralazine
c) Veno + Arteriolar dilators
- Sodiumnitroprusside
⑨ Renin Angiotensin blockers
a) ACE inhibitors
- Enalapril
- Ramipril
b) ARB - Losartan
⑩ Synthetic BNP
Niseritidin

4) Adverse Effects of Digoxin :-

Non cardiac Effects :-

CNS :- Headache
fatigue
Malaise
Drowsiness
Disorientation
Delurium
convulsions
confusion
neuralgic pain
Aphasia

GIT :- Abdominal pain
Nausea
Vomiting
Anorexia
Diarrhea (rare)

Vision :- colour vision Disturbance
white vision
Blurred vision
Diplopia
photophobia
schotoma

Others :- Gynaecomastia
skin rashes.

Cardiac Effects :-

Pulsus Bigeminis
AV block

Ventricular Tachycardia
Bradycardia
Ventricular fibrillation
Atrial extrasystole
Ventricular extrasystole.

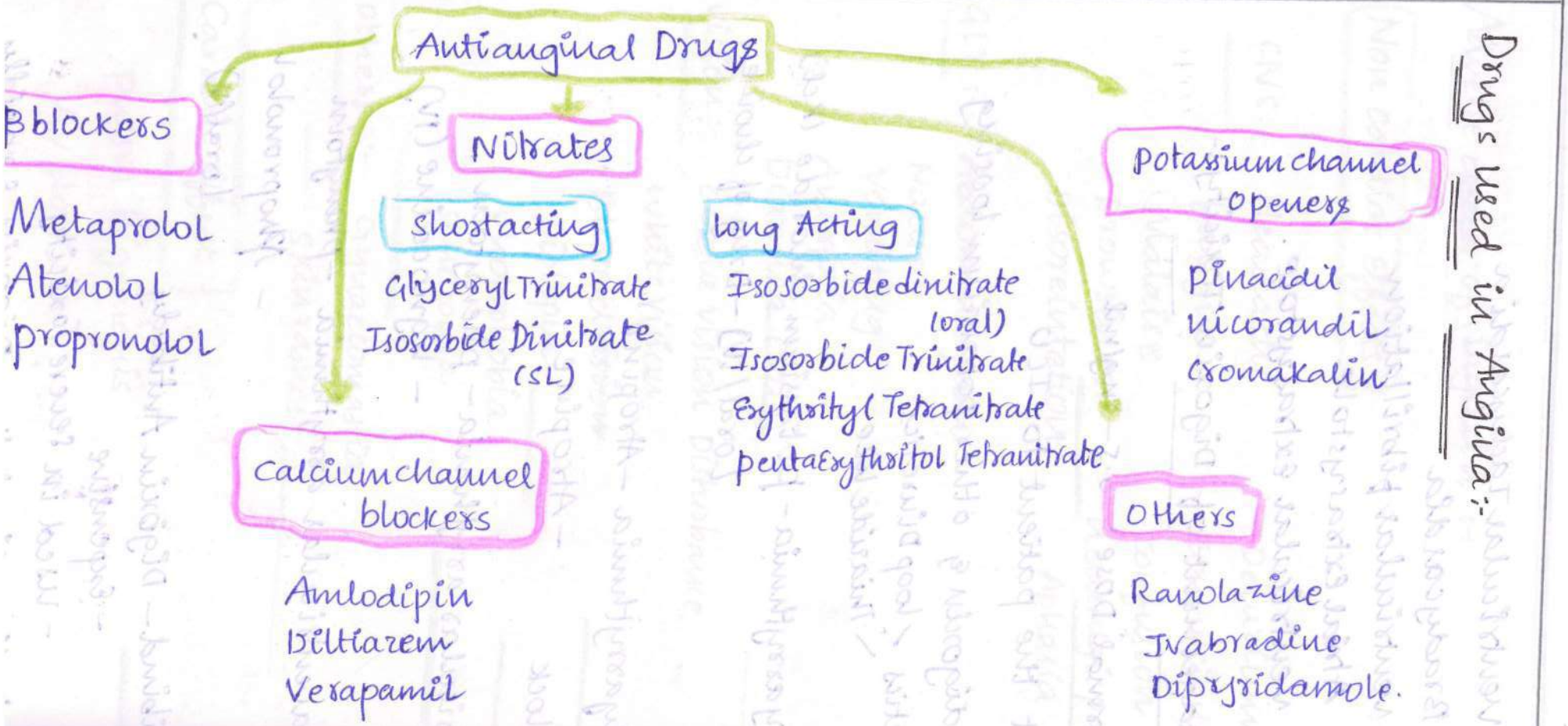
Management of Digoxin Toxicity:-

Normal Dose :- 0.5-2ng/ml

1) shift the patient to ICU
2) stop digoxin & other potassium loosing diuretics - loop Diuretics
   - Thiazide like.
3) Tachyarrythmia - potassium chloride (Kcl) [oral/IV] - Drug of choice
4) Bradyarrythmia - Atropine
5) AV block - Atropine
6) Ventricular arrythmia - phenytoin
   - lignocaine (IV)
7) Supraventricular arrythmia - phenytoin
   - Propranolol (oral/iv)
8) Digibind - Digoxin Antibody
   - Expensive
   - used in severe Toxicity

5)

Drugs used in Angina:-



Nitrates – used prophylactically in Angina.

MOA:-

Molecular mechanism:-

Nitrates

↓ Denitrated in Cardiac muscle cell

Release of active free radical NO

↓

Increased Guanylcyclase

↓

↑ cGMP

↓

Activation of protein kinase

↓

Dephosphorylation of myosin light chain kinase

↓

Inhibition of myosin

↓

NO binding with actin

↓

Relaxation

↓

of Cardiac Vasular smooth muscle.

9) Ranolazine :-

↓

Metabolic modifier

MOA :-

- Spares Fatty acid oxidation & shifts the ATP production to O_2 efficient carbohydrate oxidation.
- Inhibits "Late Na^+ currents" in myocardium

↓

Indirectly ~~inhibits~~ facilitates Ca^{2+} Entry

- Also reduces / prevents Ca^{2+} overload on myocardium during Ischemia

↓

Cardioprotective role of Ranolazine

Preperation :- Tab 500mg ER

- Dose :- 0.5 - 1g b.i.d

- Use :- Used in Acute Angina pectoris patient who isn't responding to standard Angina Therapy.

6) Rationale of combining nitrates with β blockers:-

1) nitrates x β blockers (propronolol)

↓

used in Exestional Angina

↙

because of Increased effectiveness & reduced adverse effects

2) β-blockers

↓

propronol

↓

↓ Heastrate

↓ cardiac output

↓ Force of contraction

↓

↑ LV Enddiastolic Volume & pressure

Nitrates

↓

venodilators

↓

↓ EDV & pressure.

∴ Nitrates can decrease the Left ventricular End diastolic Volume & pressure caused by β blockers.

3) Nitrates

↓

Arteriolar dilators

↓

↑ pulmonary vascular resistance

↓

Reflex Tachycardia

βblockers can counteract the reflex tachy caused by nitrates.

4) Blockade of β_2 receptors

↓

$\alpha_1\beta_2$ → coronary artery

↓

Increased action of α_1

↓

Coronary spasm.

• Nitrates can reduce the coronary spas caused by βblockers.

5) calcium channel blockers

↑ AV conduction
↑ HR
↑ ventricular rate

] → reduced b nitrat

1) Succinyl Choline:

Succinylcholine is a depolarising blocker.

Mechanism of Action:

It acts in two phases:

Phase I

Binds to Nicotinic receptors.

↓

~~opening of Na+ channels~~.

causes depolarisation. → twitching / spasticity.

Phase II

prolonged use of Sch.

↓

↓ depolarisation

Therapeutic uses of Succinyl Choline:

- used for the intubation of tracheal tube.
- provides excellent condition by relaxing the jaw and muscles.
- used in epylepticus and electroconvulsive thrapy.
- used as adjunct to general anaesthetic
- used in tetanus.

Adverse effects of Succinyl Choline:

- Succinylcholine Apnoea: In patients with atypical pseudocholine esterase, the succinyl choline dosent break down which cause paralysis of

2) ~~Mechanism of Action~~

Lignocaine is a local Anaesthetic.

It ~~acts~~ is an intermediate acting anaesth

Mechanism of Action

Lignocaine binds to the
receptors on nerve endings
↓
persistant depolarisation of the membrane
↓
the membrane
potential is raised
↓
No stimulus, can stimulate

tic.

Technique of Administration:

⇒ Surface Anaesthetia:

This is applied topically and the sensory nerve ending of the upper layer is blocked.

Numbness is felt

Used for minor procedures.

⇒ Infiltrative Anaesthetia:

A dilute solution is injected and the cutaneous nerve ending are blocked.

⇒ Epidural Anaesthetia

It is given in the dural space where the [illegible] through the fat and.

⇒ conduction block:

They are of 2 types:

⊙ field block: The LA is given a beginning of the plexus and the whole region is blocked from sensation.

⊙ nerve block: when a specific nerve given LA and a particular region blocked.

⇒ Spinal Anaesthesia:

It is given in L_2-L_3 or L_3-L_4 sub space. It is mainly given during pe

3) Homatropine vs phenylepriine.

	phenylepriine	Homatropine
Synthesis	Synthetic sympathomimetic agonist	Synthetic Anticholinergic
Receptor affinity	$\alpha_1 > \alpha_2$	Muscranic receptor.
Actions	produces vasoconstriction, Increases peripheral resistance.	Mydriasis & cycloplegia.
Administration	oral, opthalmic, Injection	opthalmic
Therapeutic uses	Allergic rhinitis, eustachian tube block	Refractive testing of eye., ocular

Anticholinergic drugs.

- Natural Alkaloids
  - Atropine
  - Hyoscine
- Semisynthetic derivates.
  - Hyoscine butyl bromide
  - Hyoscine methyl bromide
  - Atropine methonitrate
  - Homatropine
  - Homatropine methyl bromide
- Synthetic compounds.
  - Antisecretory Antispasmodic
    - Quaternary compounds
      - glycopyrolate
      - clinidium
    - Tertiary Amines
      - Dicyclomine
      - Valethamine
  - Mydriatics
    - Tropicamide
    - Cyclopentolate
  - Vasicoselective
    - Darifenacin
    - Solifenacin
  - Anti parkinsonian
    - Benzhexol.

Therapeutic uses of Atropine substitutes.

⊙ ~~Antispasmodic~~ secretory effects.

⇒ preanaesthetic medication

- used before giving irritant general Anaesthetics
- ~~Reduce~~ prevents laryngospasm.

⇒ pulmonary embolism

- prevents secretions evoked during pulmonary embolism.

⊙ Antispasmodic effects.

⇒ In spastic constipation - relieves abdominal discomfort.

⇒ drug induced diarrhoea to some extent.

⇒ pyloruospasm

⇒ gastritis ⇒ Abdominal pain

⊙ ~~Mydriatics~~ Mydriatics.

⇒ for refractive testing: Homatropine used ~~in~~ to induce cyclopegia and Mydriasis.

⇒ for treatment: used in corneal ulcer, i
iridiitis.

⊙ Parkinsonism

⇒ galantamine, donezepil

⊙ Motion sickness:

⇒ Hyoscine prophylactically given.

⊙ ~~Basic selective~~

1/2

5) Dopamine vs Dobutamine.

↳ can be given in low dose, medium dose, high dose

Dopamine	Dobutamine
⊙ Naturally occuring	Synthetic
⊙ Acts on β_1 & α_1 receptors & Dopaminergic receptors $D\alpha_1, D_2 > \beta_1 > \alpha_1$	$\beta_1 > \alpha_1$, acts only on β_1 receptors, very less affinity to α_1 receptors.
⊙ used in cardiac shock, Hypotension, Acute ~~CCF~~ Heart failure.	After surgery of MI, congestive Heart failure.
⊙ Acts on α_1 receptors when given in high doses (vasoconstriction)	No vascular effects.
⊙ Acts on β_1 recpts when given in low doses. (ionotropic effect)	ADR: tolerance developed.
⊙ Increases Renal blood flow by acting on D_1 & D_2 receptors.	

b) Cardioselective β Blockers:

⇒ Metaprolol

⇒ Atenolol

⇒ Nibevelol.

⇒ Acebutolol.

⇒ Bisoprolol.

⇒ Esmolol.

Therapeutic uses:

⇒ Hypertension: Acts on β1 receptors

⇒ Angina:

⇒ Myocardial Infarction

- for secondary prophylaxis
- for supression of MI during evolution

⇒ Aortic Aneurysm.

Advantages:

Acts only on β_1 receptors. No major effect on β_2 receptors.

⇒ It can be given in bronchospasm and Asthmatic patients as it dosent cause broncho-constriction.

⇒ dosent alter glucose metabolism in diabetic patients.

⇒ Hypoglycaemia induced tachycardia dosent occur.

⇒ hands and feet dont turn cold.

⇒ No deleterious effect on lipid profile.

⇒ More capacity towards exercise.

Contraindications

- Bradycardia
- Congestive heart failure.
- Depression
- Asthma.
- Diabetes mellitus

22/30

PHARMACOLOGY Date: 2/08/2023.

MONTHLY TEST - 3

1) Pharmacotherapy of severe migraine.

Severe migraine is when there is throbbing headache that occurs more than 3 times a month.

Pharmacotherapy.

Severe migraine cannot be cured with simple Analgesics such as paracetamol or aspirin.

The patient needs to be advised. $5HT_{1D/1B}$ agonists such as sumatriptan, Zolmitriptan, Naratriptan etc.

Anti Emetics can be given parallely if its associated with vomiting. Eg) promethazine.

There also needs to be prophylactic therapy for such patients. It can be given by.

⇒ β Blockers - propanolol.

⇒ Tricyclic Antidepressants.

⇒ calcium channel blockers.

Triptans are the first line of drug given severe migraine.

Sumatriptan:

MOA: It inhibits the vasodilating peptides the presynaptic ~~trace~~ ~~ending~~ nerve ending trigeminal nerve.

The main adverse effect is: it causes coronary spasm.

2) ⊛ Some 2nd generation Antihistaminics are:

- ⊙ fexofenadine
- ⊙ loratadine
- ⊙ desloratadine
- ⊙ Citrezine
- ⊙ Levocitrezine
- ⊙ Azelastine

Remember.

Advantages of Second generation Antihistaminics are:

⇒ They are selective H_1 blockers.

⇒ They do not cause CNS depression - does not cross blood brain barrier.

⇒ They also have additional anti allergic action - by inhibiting other mediators.

Therapeutic uses:

⇒ used in Allergic reactions.

- ⊙ Type 1 hypersensitivity reactions
- ⊙ Allergic rhinitis

- allergic conjuctivitis
- urticaria
- pollinosis
- skin rashes.
- Adrenaline + i.V SGA used in lar edema.

They do not suppress the AB- AG reactions inhibits the inflammatory chemical media

other uses are:

- Insect bites and ivy poisoning.
- they are also used during saline / bl

3) Management of Status Asthmaticus.

Status asthamaticus is when an asthma patient has the potential to develop severe acute asthma.

Treatment / Management:

1) Hydrocortisone hemisuccinate 100mg. (or) any other glucocorticoid with equivalent action.

2) Nebulised Salbutamol 5mg + ipratropium bromide 0.5mg. cause bronchodilation.

3) Inhalation of high flow humidified Oxygen

4) Salbutamol / terbutaline 0.4mg. im/sc is given to dilate the terminal bronchioles.

5) Antibiotics to relieve chest infection

6) If very severe the patient may require intubation

7) Na. bicarbonate / saline is given to reduce risk of acidosis.

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4) Theophylline - MOA & Adverse effects:

Theophylline is a methyl xanthine.

Mechanism of Action:

1) It causes release of Ca^{2+} from sarcoplasm reticulum that cause contraction of skeletal and cardiac muscle.

2) It cause inhibition of Phosphodiesterase that ~~dosent~~ causes rise in cAMP levels which causes bronchodilation

3) blockade of Adenosine receptors.

Theophylline is a ~~too~~ s: drug that has lo safety margin. It can ~~cause~~ cross the pl barrier and can be ejected through milk.

There are many adverse effects of theop

⇒ They cause gastric irritation

⇒ they are CNS stimulants, causes, restlessness insomnia, convulsents, and even death

⇒ they also cause nausea, vomiting, headaches

They are mainly used in Respiratory ?

* COPD
* bronchial asthma.
* Infantile apnoea.

5) Prostaglandin Analouges:

⇒ Carboprostacyclin

⇒ prosta cyclin

⇒ ~~Misoprostone~~. Metapriostal

⇒ Alpaprost

⇒ Mifepristone.

Therapeutic uses:

1) Abortion:

Termination of pregnancy within 7 weeks done using prostaglandin analouges.

600mg metapriostat is given orally 2 days a single dose of 400 µg Mifepristone.

This is equal to doing a suction - evacuation remove the conceptus.

PGE_2 analouges are also used in ~~to~~ in mi Abortion, molar pregnancies etc.

PGE_2 analouges cause oxytocin resistant uterus to oxytocin responsive uterus and causes abortion of mid term pregnancy.

2) Augmentation of labour.

PGE_2 analouges are used in patients who do respond to oxytocin or is toxaemic and has renal failure.

3) Ripning of the Cervix:

PGE_2 creams are used intravaginally to soften the cervix and help in dilatation

4) Post partum haemorrhage:

Carboprost (15-methyl $PGF_{2\alpha}$) is used in patients with uterine atony and helps control PPH.

5) Glaucoma - Latanoprost or topical prostaglandin analouges are used in wide angle glaucoma.

6) Peptic Ulcer: $PGIE_1$ analouges are used.

7) to maintain patency of ductus arteriosus $PGIE_1$ analouge (Alpaprost) is used.

8) to inhibit platelet damage: $PGII_2$ (prosta analouges are used.

9) Pulmonary hypertension: $PGII_2$ analouges used that helps in dilating pulmonary vessels.

10) Impotence: $PGII_2$ is used and is inje into the penis. erection occurs for 1-2

6) Expectorants:

Examples:

* Pot. citrate
* Sod. citrate.
* Bromohexine.

Recycling?

MOA

They ~~help~~ ~~is~~ thin the mucus secretions and cause expulsion of the mucus by increasing cough reflex by salt action.

They break the bonds present in the mucus to thin the consistency.

They also improve muco ciliary action to help in expulsion.

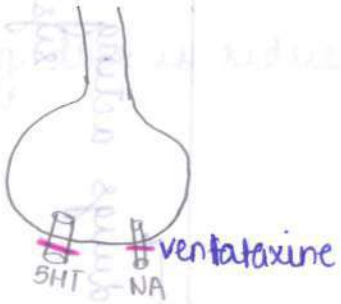
Monthly test VI 13/09/23

1) Venlafaxine : It is a serotonin Noradrenaline reuptake inhibitor

Mechanism of Action:

⊙ ↑ses 5HT & NA

⊙ Inhibits the reuptake of 5HT & NA by the nerve endings.



Therapeutic Uses:

Mood swings

Hot flashes in menstrual bleeding.

Social Anxiety

Eating disorder.

Endogenous depression

Psychotic depression.

ADR

Nausea
vomitting
diziness
Impotence.

Antiparkinsonism drugs.

drugs acting on dopaminergic system

- Dopamine precursor
  - Levodopa
- Dopamine Agonist
  - → Bromocriptine
- MOA-B inhibitor
  - → Selegiline
  - → Rasagiline
- Glutamate antagonist
  - → Amantidine
- peripheral decarboxylase inhibitor
  - → Carbidopa.
  - → ~~Benserazide~~
  - → Benserazide.
- COMT inhibitor
  - → Entacapone
  - → Tolcapone

drugs acting on cholinergic system.

- Systemic Anticholinergics
  - → procyclidine.
  - → ~~Benperidone~~.
  - → Biperiden
- Antihistaminics
  - → promethazine
  - → ~~orphenadrine~~.

Advantages of Carbidopa + Levodopa.

It is known as Cocareldopa.

* $t_{1/2}$ of levodopa is increased, and drug dose is reduced to 1/4th.
* Cardiac complications minimised.
* Vomitting and Nausea minimised.
* Systemic dopamine is reduced.
* Pyridoxine effect ~~effect~~ reversal effect of levodopa is not there.
* Drug actions is faster.
* 'on-off' effect is minimised due to better dopamine sustained in brain.
* patients show better improvement, especially patients ~~assist~~ who do not respond to levodopa.
* More drug crosses BBB and reaches site of action.

Opiod Analgesics.

NATURAL ALKALOIDS

⇒ Morphine

⇒ Codiene.

SEMISYNTHETIC OPIODS

⇒ ethyl morphine

⇒ Diacetyl morphine

⇒ phol codiene.

SYNTHETIC OPIODS

⇒ pethidine

⇒ fentanyl

⇒ Remifentanyl.

⇒ Tramodol.

Therapeutic uses of Morphine

* Analgesic: given in severe pain.
  ⇒ pre and post op analgesia
  ⇒ Visceral pain, ischemic pain, renal colic, MI.

* pre anaesthetic medication.

* Surgical Analgesia
  Important component of intraoperative anaesthesia.

* Anti-Anxiety and surgical analgesia.

* Acute left ventricular failure and acute pulmonary edema.
  ⇒ Relieves chest congestion
  ⇒ reduces preload on heart.
  ⇒ ~~Reduces~~

Contraindications:

* pregnancy.

* It is a drug of addiction

4) Levateracetam

Topiramate

~~Zonisamide~~

MOA of TOPIRAMATE:

⇒ prolongs Na^+ channel inactivation

⇒ ↑ses GABA

⇒ ↓ses Glutamate.

5)

Diazepam	Phenobarbitone.
* Benzodiazepine	* Barbiturate.
* MOA: Increase GABA ↓ Anxiolytic action & Sedation	* MOA: Increases action of GABA receptor. ↓ Anxiolytic action and muscle relaxation.
* no enzyme induction	* causes enzyme induction
* dosent cause drug automatism	* causes drug automatism
* less drug abuse	* drug of Abuse.
* GABA facilitatory action	* GABA mimetic action
* Incase of toxicity Antidote is present (Flumazenil)	* Antidote not present Forced Alkaline diuresis.

Antipsychotics

- enothiazines
  - ...hatic side chain
    - ⇒ promazine
    - ⇒ trimeprazine
  - Piperidine side chain
    - ⇒ trifluoperazine
  - Piperazine side chain.
    - ⇒ trifluoperazine
    - ⇒ phenthiazine
- Butyrophenones.
  - ⇒ Haloperidol.
  - ⇒ penfluridol
- Thioxanthines
  - ⇒ ~~Haloperidol~~.
  - ⇒ ~~penfluridol~~.
- Other heterocyclic compounds.
  - ⇒ levosulpiride
  - ⇒ loxapine.
- Atypical Antipsychotics
  - ⇒ Amisulpiride
  - ⇒ Zotepine
  - ⇒ Olanzapine

ADR of chlorpromazine :

due to its pharmacological actions.

CNS :

- lethargy, drowsiness
- ↑eating & weight gain.
- Seizures.

CVS :

- QT prolongation, palpitation
- Cardiac arrhythmias.

Endocrine :

- Hyperprolactinemia

Anticholinergics :

- constipation,
- urinary hesitancy in Males.

Extrapyramidal side effects :

- Tardive dyskinesia
- parkinsonism
- Akathisia
- Malignant Neuroleptic syndrome.

Hypersensitivity

- Rashes
- Urticaria
- Myocarditis

Therapeutic uses:

- Schizophrenia.
- ADHD.

25-3-24
Monday

Monthly Test - 1

18

1. Necrosis is defined as death of tissue due to intracellular proteins & enzymatic digestion of lethally injured cells, usually accompanied with inflammatory re

Causes

- Hypoxia
- physical agents
- Chemical agents
- Micrebial
- Immunological

Types of Necrosis:

Necrosis is of 5 types:

- Coagulative
- Caseous
- Liquefactive
- Fibrinoid
- Fat
- Gangrene

Morphology →

→ Inflammatory rxns always present

→ Death of many adjacent cells

→ Cell swelling initially.

→ The death of tissue occurs due to membrane disruption.

→ Nuclear disruption

→ phagocytosis

- Coagulative Necrosis :

- Architecture of dead tissue is preserved
- Firm texture
- Denaturation of structural proteins & enzymatic proteins
- Dead cells removed by phagocytosis
- Ischemic due to obstruction leads to this necrosis.
- Area k/as infarct, common in heart, kidney, spleen.

Caseous Necrosis :

- Occurs in tuberculous infections.
- → Cheese like appearance.
- → Combine features of both coagulative & liquifactive necrosis
- → Microscopically, structureless lysis occurs due to the cells collection.
  Inflammatory border - granuloma
- → Common sites - lungs, lymph nodes, intestines.

Liquefactive Necrosis

- → Dead cells digested
- → Seen as ischemic injury in CNS, fungal infections
- → Creamy yellow - dead leukocytes - PUS
- → Microscopically - Structureless lysed cells collection, amorphous debris enclosed
- → Gliosis, inflammatory cells & fibroblasts

- **Fibrinoid Necrosis**

→ Seen in immune rxns involved in blood vessels

→ Ag-Ab complex lodged in wall of arteries

→ Fibrin leaks out forms bright pink amorphous stain in H & E staining

→ microscopically, hyaline like deposit on wall, nuclear debris of Neutrophils.

- **Fat Necrosis**

- Focal areas of fat destruction.
- Seen in acute pancreatitis
- microscopically,

→ Necropsied fat cells are cloudy, enclosed by inflammatory cells.

→ Ca soaps - amorphous, granular, basophilic.

Activated pancreatic lipase in pancreas & peritoneal cavity

↓

the enzymes liquify fat in peritoneum

↓

Split the triglycerides into fatty acids.

↓

fatty acids combine with calcium

↓

saponification

Traumatic fat Necrosis:

- Necrosis that occurs in fatty tissue
- Unlike pancreatic fat necrosis, it is not enzyme mediated.

2) Granuloma formation :-

- It is a chronic inflammation.

Cellular attempt to contain offending agent

↓

activation of T lymphocytes

↓

macrophage activation

↓

formation of giant cells.

Pathogenesis :

- It is a type IV granulomatous hypersensitivity reaction.

- A ~~proctc~~ protective defence rx^n by the host but causes tissue destruction bcoz of persistence of poorly digested antigen.

Cell Injury

↓

Failure of digest agent

↓

Weak acute inflammatory response.

↓

Engulfment by macrophages

↓

Persistance of infurious agent

↙ ↘

T cell mediated immune response

poorly digestive agent.

↘ ↙

- Activation of CD4 + T cells
  growth factors IFN-γ & TNF-α
- monocyte chemotactic factor.

↓

- Accumulation of tissue macrophages
- proliferation of T cells

↓

Macrophages activated by IFN-γ

↘

↙

Transformation to epithelioid cells, giant cells

Secretion of fibroblastic, proliferating cytokines

Granuloma.

3)

Dystrophic calcification	Metastatic Calcification
• Deposition in dead or degenerated tissue	• Deposition in normal tissue.
• Normal calcium metabolism	• Deranged
• Generally irreversible	• Hypercalcemia
• Necrosis, infarcts, thrombosis, hematoma, old scar	• Reversible upon correction of metabolic disorder
	• Hyperparathyroidism, Bony destructive lesions, hypervitaminosis D.
Pathogenisis. Increased binding of phosphates with necrotic tissue which in turn binds to calcium & thus forming calcium phosphate precipitates.	Increased precipitation of calcium phosphate due to hypercalemia at certain sites such as lungs, stomach etc.
Diagnosis: Generally better prognosis due to little septicaemia	• Generally profound prognosis due to profound toxaemia.

27-5-24

Monthly Test - II

13

1. Systemic Lupus Erythematosus

2

It is an autoimmune disease involving multiple organs, characterized by vast array of auto-antibodies particularly antinuclear antibodies (ANAs)

It is of two types:

Systemic form

Discoid form

Etiopathogenisis:

- It is an autoimmune disease
- It is a defect in self tolerance.

Genetic factor:

→ Family members have high risk of developing SLE

high rate concordance in monozygotic twins.

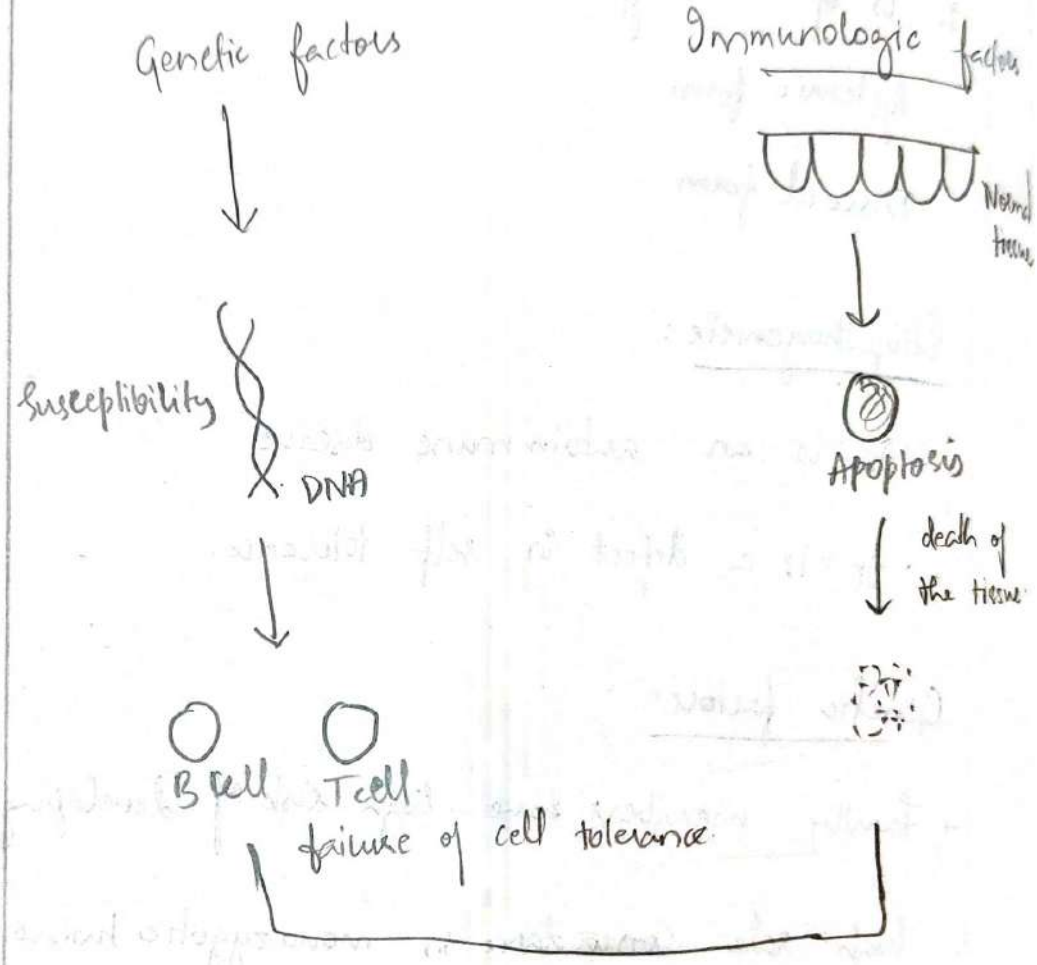
→ Alleles of HLA-DQ locus have been linked

Immunologic factors:

- Failure of self tolerance to B cells.
- CD 4^+ helper cells may escape tolerance and produce pathogenic autoantibodies

- TLR engagement by nuclear DNA & RNA
- Type I interferons ~~pad~~ play a role in lymphocyte activation of B cells.

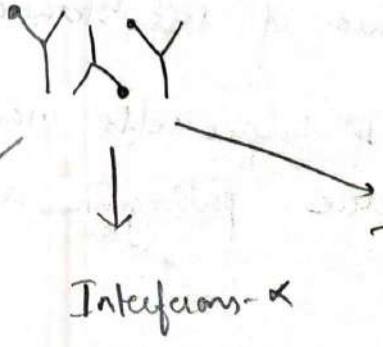
Schematic diagram



Autonuclear antibodies

3

Immune complex



↓

plasma cell

c) Morphological patterns of renal lesions in the above mentioned SLE are -

Upto 50 % of SLE patients have renal involvement.

Six patterns of glomerular disease are seen.

- Class I ~~Minimal~~ Minimum mesangial lupus nephritis.
- Class II
- Class III
- Class IV
- Class V
- Class VI.

(2)

Class I - Very Uncommon.

class II - proliferation of mesangial cells.

class III - less than <50% of the glomeruli is involved.

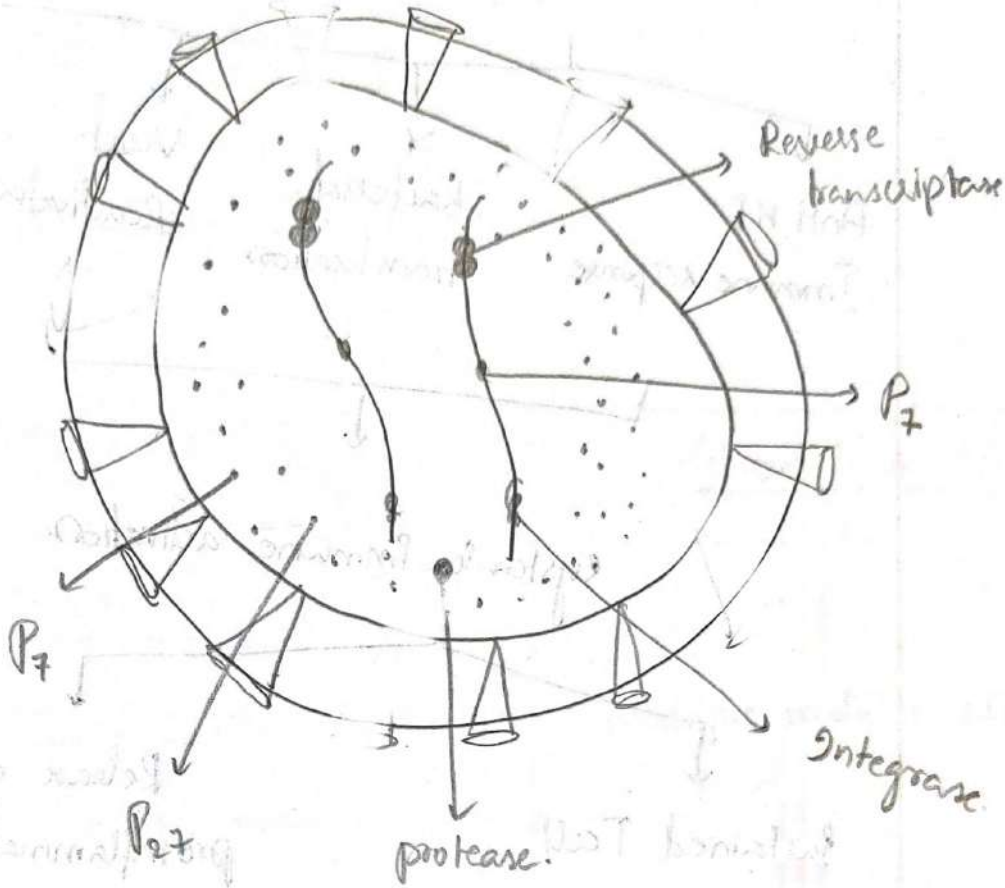
class IV - Most severe & most common.
- proliferation of mesangial cells.
- scarring of the cells.
- hematuria, proteinuria are present in patients suffering this class

class V - sub epithelial deposition of immune complexes.

class VI. - ~~Sector~~ sclerosis of >90% of glomeruli

~~class~~ VII -

2) Structure of Human Immodeficiency virus.

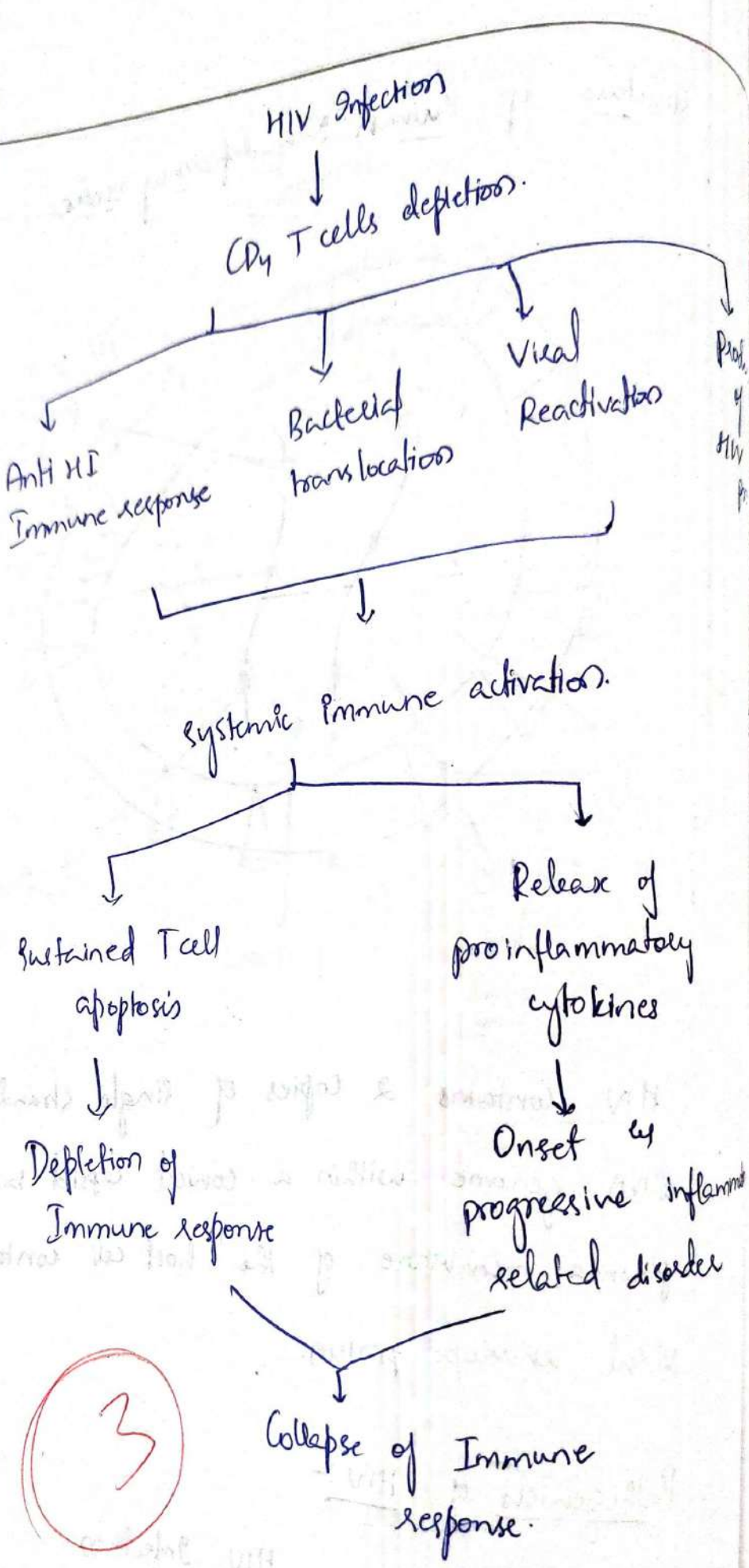


HIV contains 2 copies of single stranded RNA genome within a conical capsid by plasma membrane of the host cell containing viral envelope protein.

Pathogenisis of HIV :-

HIV Infection

↓


HIV Infection
CD4 T cells depletion.
Anti HI Immune response
Bacterial translocation
Viral Reactivation
Systemic Immune activation.
Sustained T cell apoptosis
Release of proinflammatory cytokines
Depletion of Immune response
Collapse of Immune response.

3

3) According to Ridley - Joplin classification, leprosy is classified into.

Tuberculoid leprosy
Borderline leprosy
Lepromatous leprosy.

	Tuberculoid leprosy	Lepromatous leprosy
1.	Skin lesions.	
	Hypopigmented, annular macules	multiple nodules are seen.
2)	Nerve lesions	
	Enlarged thickened nerves Nerve abcess is seen	- Nerve lesion appears late. - Symmetric involvement of nerves - Trophic changes of nerve lesions are seen in hands or feet.

3

3)	Inflammation: Granulomatous inflammation with few langhan cells	No inflammation, Virchow's lepra cells, lymph nodes have filled with bacteria
4)	Dry, scaly	mostly skin lesions are Anaesthetic or hypoesthetic
	Nerve degeneration, skin anaesthesia, leaving chronic skin ulcers	Mostly ulnar and peroneal nerves are involved

20

Short Essays:

(2) Kwashiorkor:

- protein deficiency with no protein intake in the diet but with sufficient calorie intake.

- In kwashiorkar disease, protein intake is less but with sufficient calorie intake.

Clinical features:

- Wasting of muscles
- It is seen in children b/w 6 months to 3 yrs of age.
- Adipose tissue is preserved.
- Oedema is present in both generalized & localized
- failure in growth.
- Serum protein is low
- Hepatomegaly

- There are altered light & ~~band~~. dark bands in hair

Morphology :

- Atrophy of different tissues but subcutaneous fat is preserved

Kwashiorkar	Marasmus
- protein deficiency with sufficient calorie intake	- Deficiency of both proteins & calories.
- seen in 6 months to 3 yrs children.	- common in infants of 1 yr of age.
- Growth failure of muscle is seen but preservation of adipose tissue.	- wasting is seen along with loss of adipose tissue.
- Hepatic enlargement is seen	- Growth in the failure of muscle.
- Oedema is present	- Hepatic enlargement is not seen.
	- oedema is absent.

No protusion of abdomen	protrusion of abdomen

(3) Anemia:

Anemia is defined as decreased haemoglobin level in the blood less than normal age range with regard to age & sex of the person.

Adult male - 16 mg/dl.

Adult female (menstruating) - 12-14 mg/dl

It is also defined as reduced oxygen carrying capacity in the blood.

Etiopathogenesis:

- Iron deficiency occurs due to decrease absorption of iron.
- Due to defective heme synthesis.

It is divided into 3 stages:

1) Iron depletion:

Iron is adequate to maintain the normal haemoglobin levels & only serum ferritin is decreased.

2) Iron deficiency erythropoiesis:

Lowering of serum iron & transferrin saturation level without anemia.

- MCV, MCH, MCHC are normal in this stage.

3) Iron deficiency anemia:-

- Low serum iron, serum ferritin, transferrin saturation impaired Hb production.

- Morphologically, first there is reduction in size, microcytic anemia & central pallor is seen.

Laboratory findings:

- peripheral blood smear.
- Hb & PCV values are decreased
- Blood cell indices is decreased
- MCV, MCH, MCHC all are decreased.
- Bone marrow shows microcytic erythroid. normoblastic hyperplasia.
- Reticulocyte count decreases.
- Serum ferritin level decreases.

These are the laboratory findings which show positive for Iron deficiency anemia.

Long Essay

(1) Clinical impression with the given symptoms is the patient has megaloblastic anemia.

22

because

- This anemia occurs due to vitamin B_{12} and folic acid deficiency
- This leads to impaired DNA synthesis & a distinct megaloblast is found in Bone-marrow.
- Pallor of ~~cont~~ conjunctiva & the palmar creases are seen in megaloblastic anemia.
- Weakness, fatigue, palpitations, beefy red tongue are the cardinal symptoms of anemia.

Laboratory findings:-

Hematological

- Haemoglobin & Hematocrit (~~PCV~~ PCH) is decreased.

Red Blood cell indices:

MCV, MCH, MCHC → normal.

↓
increases

- Dimorphic anemia - in peripheral smear shows the true population of RBC's.
- Reticulocyte count is normal (or) slightly low.
- ~~Macrocytes ovulole~~ cy.
- Macro ovulocytes cy Microcytic hypochromic.

Biochemical findings:

- Serum homocysteine levels increases
- Serum bilirubin. mild increase can cause jaundice.
- Increase in plasma lactate dehydrogenase.
- Increase in serum iron ferritin.
- Vitamin B_{12} decreased.

- deoxy uridine supression is abnormal even before the morphological changes.

Bone marrow findings:

cellularity - hypercellular.

- M:E ratio - marked erythroid ratio hyperplasia

From 1:1 to 1:6

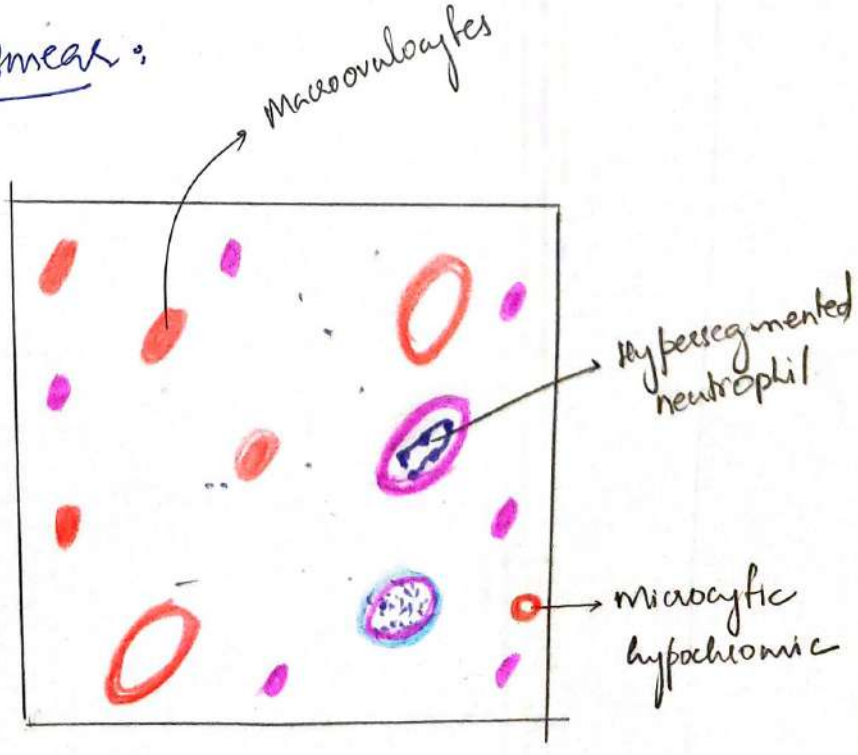
where as normal is 2:1 to 4:1

- M:E reverser ranging.

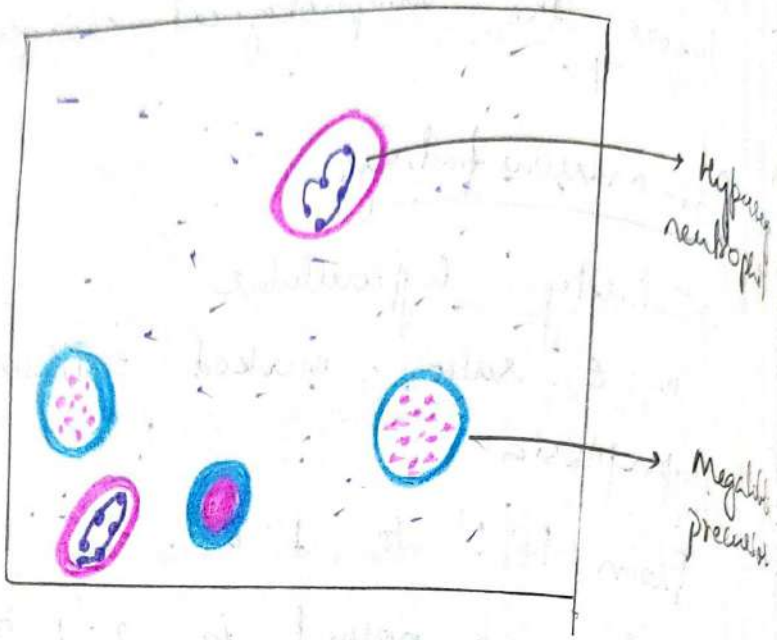
- most macrocytes lack the central pallor

- marked variation is seen in size & shape.

Peripheral smear:



Bone Marrow findings



MONTHLY TEST - 1 25/03/24

NECROSIS :-

Necrosis is form of localized cell death which leads to degradation of cell by hydrolytic enzyme later accompanied by inflammation.

There are five types of Necrosis

- Coagulative Necrosis
- Liquefactive
- Caseous
- fat
- fibrinoid

Coagulative Necrosis :-

- Necrosis caused due to Ischaemia i.e due to less blood supply.

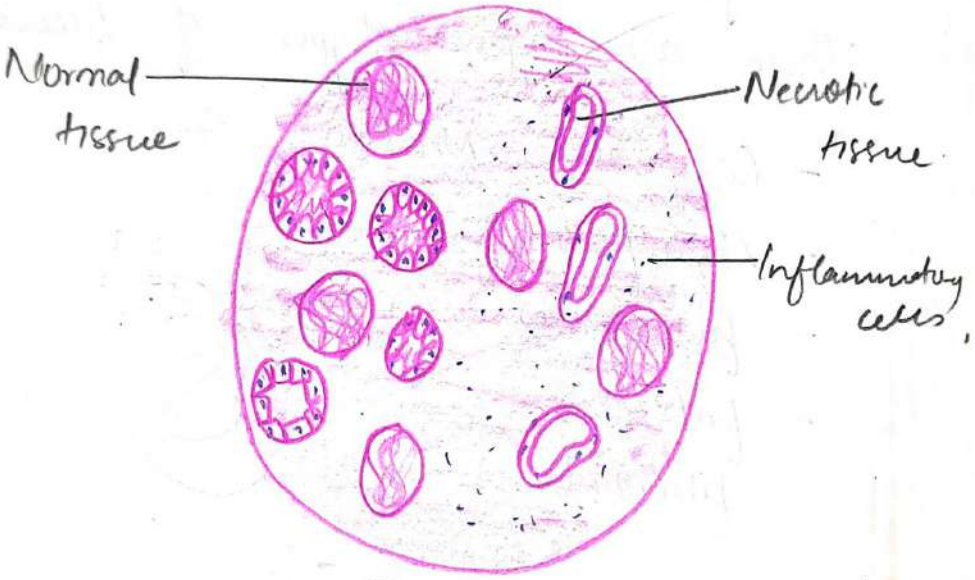
Eg :- Infarcted kidney spleen, except brain

- Gross appearance - dry, shrunken & dark.

Pathogenesis :- nuclear acidosis
- cellular disruption.

Macroscopic - Cell wall is intact
Cell membrane is disturbed
Nucleus is disintegrated

Histological appearance -



KIDNEY

LIQUEFACTIVE NECROSIS :-

- Necrosis caused due to pus accumulation & disintegration of cell by putrifaction

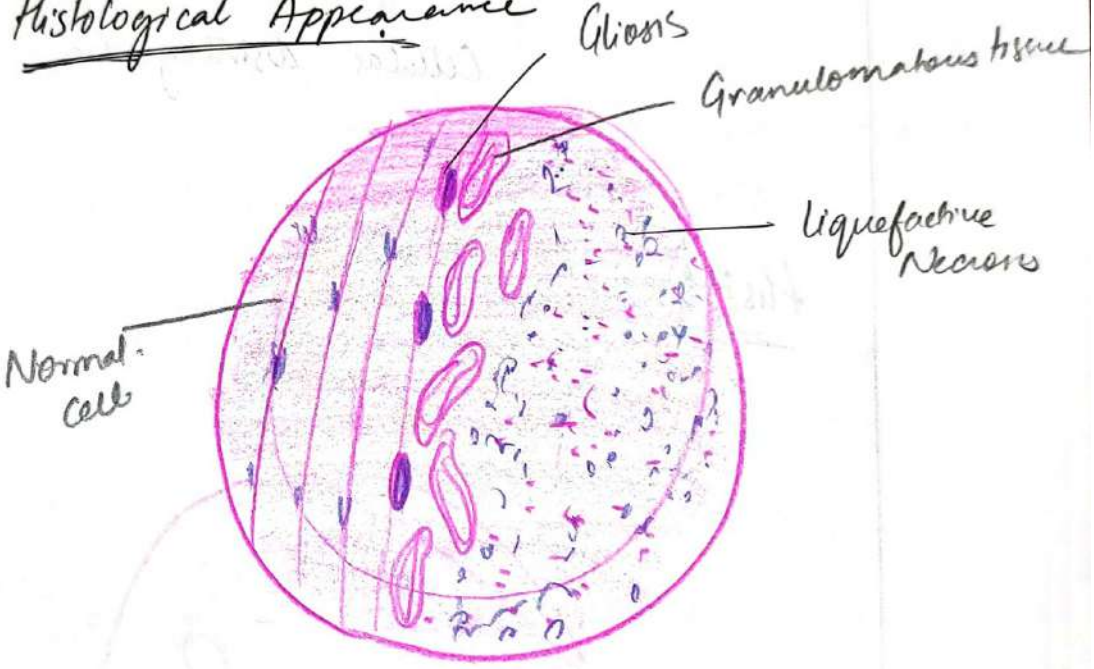
Eg:- Infarcted brain

Pathogenesis :- ↑ in hydrolytic enzymes
↓
formation of pus

GA: pale, swollen, putrified.

Microscopic - cell wall is disturbed
Nucleus is disintegrated - pyknosis
Cellular debris is formed

Histological Appearance



BRAIN:-

~~Path~~

CASEOUS NECROSIS:- Combination of coagulative & liquefactive Necrosis
- Necrosis having cheese like appearance

Eg- Tuberculosis of organs

Pathogenesis:- Phagocytosis of cellular debris by macrophages
~~Modified~~ ↓
Modified macrophages accumulate

Gross Appearance :-

Cheese like yellowish

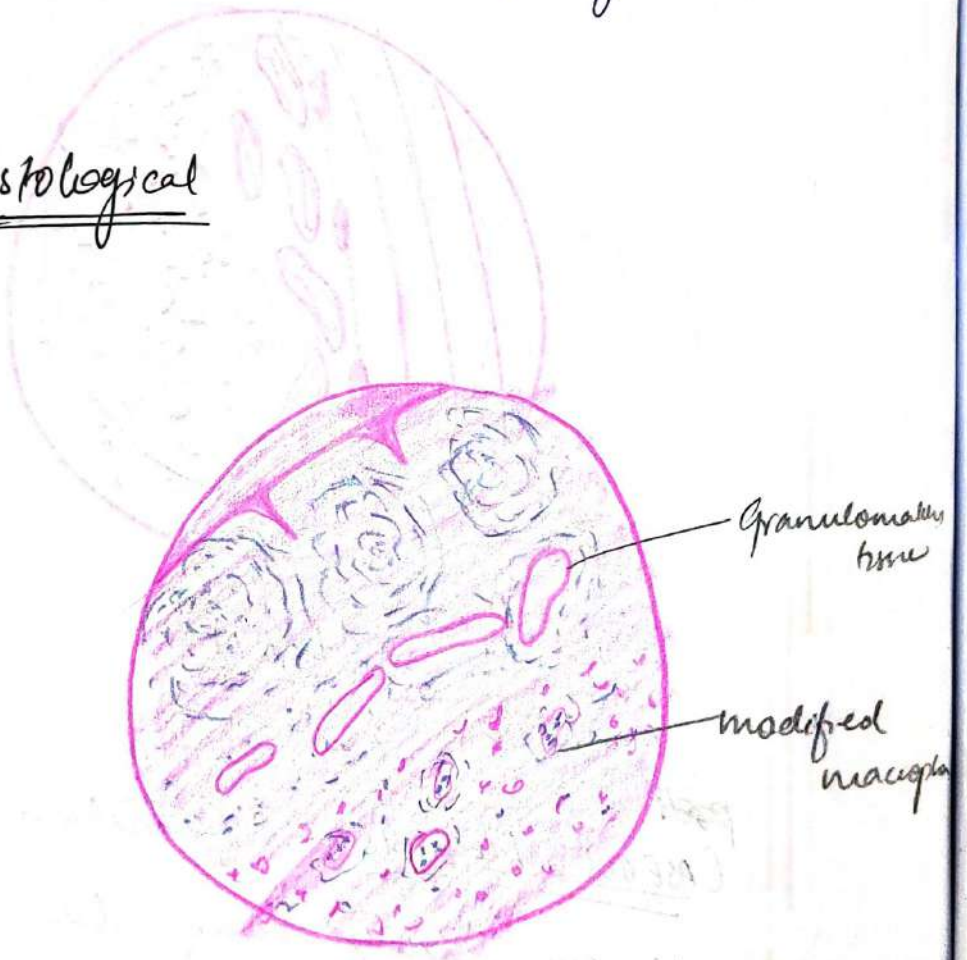
Matual is seen

Microscopic :- granulomatous tissue is seen

Modified macrophages

Cellular disintegrata

Histological



Tuberculosis of Lymph node

FAT NECROSIS :-

Caused by excessive accumulation of fat.

Eg:- liver, adipose tissue

Pathogenesis :-

fat from adipose tissue

↓

combines with Ca^{+2} present in the cell

↓

Saponification

GA: white, amorphous looking

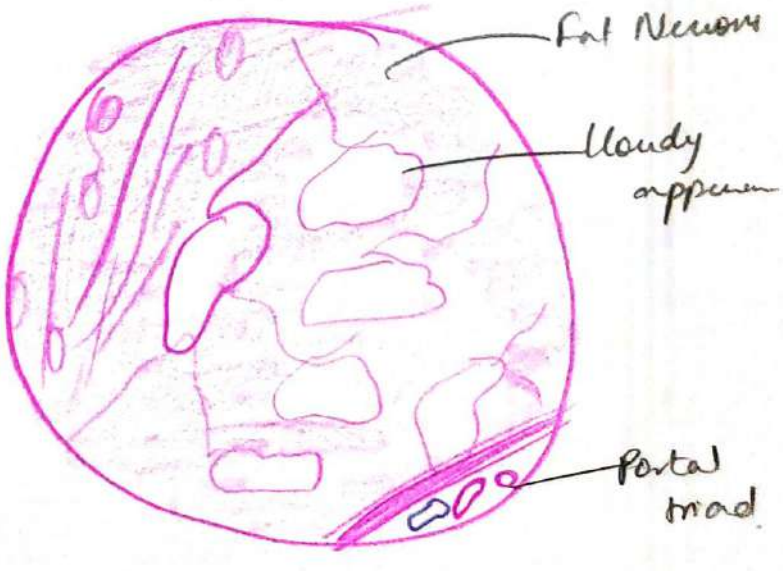
Microscopic :- colloidal white substance

white is due to Ca^{+2} salts

Inflammatory cells are present

Cloudy appearance

Histological



Fibrous Necrosis –

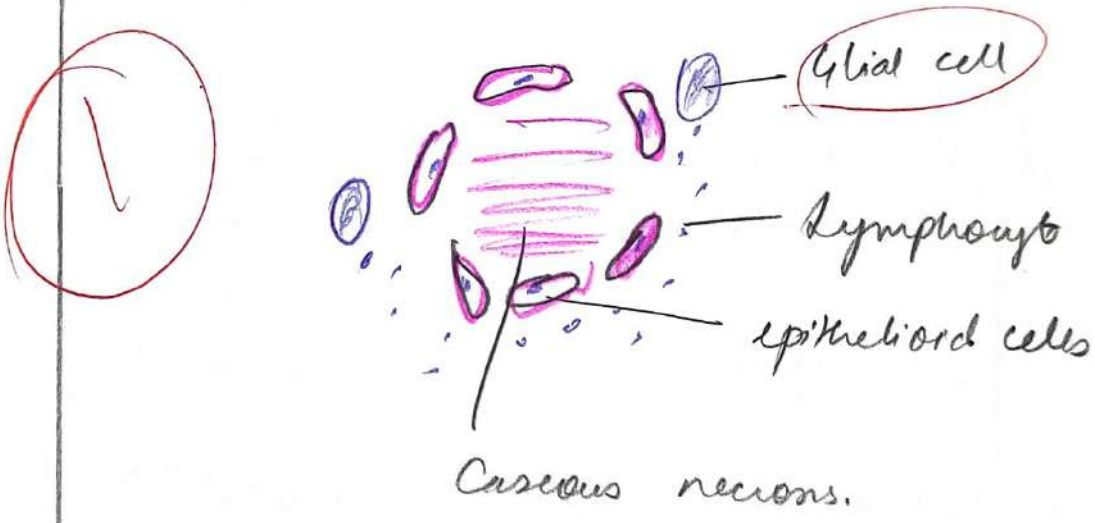
caused in fibrillar tissue

2. PATHOGENESIS OF GRANULOMA

Collection of modified macrophages
- Epithelioid cells.

These macrophages are surrounded by ~~xx~~ lymphocytes

Eg:- Caseous necrosis.



3. Calcification :- abnormal accumulation of calcium in the organ or tissue except at other than bone or enamel.

	DYSTROPHIC	METASTATIC
→	Occurs in dead or degenerated tissue	Occurs in living tissue
→	Serum calcium levels are normal	Serum calcium levels are increased
→	Irreversible	Reversible
→	Calcium metabolism is normal	Calcium metabolism is deranged
	Causes -	
	→ Necrosis	→ Hyperparathyroidism
	→ Thrombi	→ Milk-alkali syndrome
	→ Atheromas	→ Hypervitaminosis D
	→ Old scar	
	→ Tumours	

DYSTROPHIC

Pathogenesis -

cellular injury

↓

destruction of cell membrane

↓

release of phospholipid

↓

activation of phosphatase

↓

release phosphate ion

Ca^{+2} ions

Calcium phosphate

Sites :-

- psammoma bodies in papillary carcinoma
- Gamma Gandy bodies in spleen

METASTATIC

↑ Ca^{+2} levels in blood

inorganic phosphate ions

formation of Calcium phosphate

Sites :-

- alveoli of lungs
- fundus of stomach

MONTHLY TEST - 2.

LONG ESSAY

27-05-24.

1. a) Systemic Lupus Erythematous (SLE) is the probable diagnosis.

b) Etio Pathogenesis :-

Genetic factors :-

HLA DR_2 HLA DR_3

~~Hoo~~

due to the mutation of these two genes

Environmental factors :-

- UV radiations
- Hormones
- Drugs - phenylamine

Immunological factors :-

- presence of Anti Nuclear Antibodies
- presence of Anti smith Antibodies
- presence of Anti ds DNA antibodies
- presence of excess of Interferon 1.

↓

Systemic Lupus Erythematous

C. Morphological pattens of renal lesions.

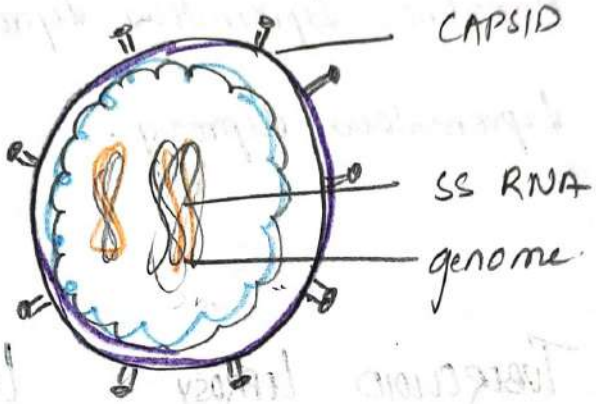
1. minimal lupus nephritis.
2. Systemic lupus proliferative nephritis.
3. Focal lupus Nephritis
4. Systemic Fibrinoid Lupus Nephritis
5. ? Systemic Abiers Lupus nephritis
6. Systemic sclerotic nephritis.

1

b. CONTI-

2. Human Immuno Deficiency Virus (HIV)

Two copies of single stranded RNA genome within capsid surrounded by plasma membrane of host cell containing Viral envelope of protein



3. LEPROSY :-

Classification according to modified Ridley Dopler classification.

1. Tuberculoid Leprosy
2. Borderline Tuberculoid
3. Borderline
4. Borderline Lepromatous Leprosy
5. Lepromatous Leprosy.

TUBERCULOID LEPROSY	LEPROMATOUS LEPROSY.
• Involves skin only ~~pheripheral~~	• Involves Skin peripheral Nerve Anterior chamber
• (Macrophages), Epitheloid cells are present. Langerhan's cells absent. • Granuloma is absent. Epitheloid cells are present. • Immunity is present	• Macrophages. NK cell • Granuloma is present • Immunity is absent • Lepromin test positive. -only to differentiate b/w Tuberculoid & lepromatous leprosy

Tuberculoid Leprosy	Lepromatous Leprosy
Immune response is not supressed.	Cell mediated immunity is supressed.
Microscopically :- Caseous necrosis observed.	Granulomatous tissue observed.

16

LONG ESSAY. MONTHLY - III 24/06/24

1. A) Megaloblastic Anaemia or Vit B_{12} deficiency Anaemia

2

B) LABORATORY FINDINGS :-

1. Blood Picture and Red cell Indices :-

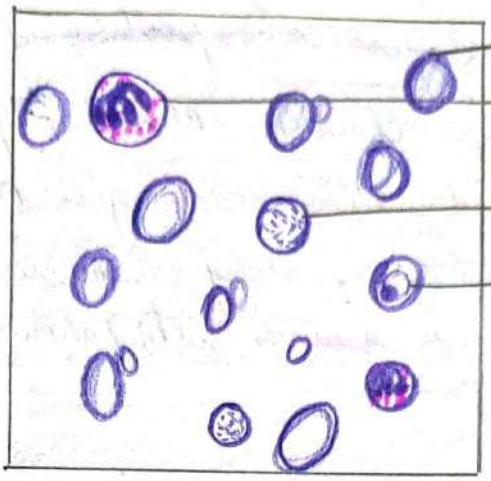
- Haemoglobin - Values below the normal range.
- Red cells :- picture shows macrocytosis. along with anisocytosis, Poikilocytosis, macroovalocytosis, Basophilic Stippling. Occassionally Normoblast is also Seen.
- Reticulocyte Count - Low to normal.
- Absolute Values - ↑ MCV
  ↑ MCH.
  Normal or Reduced MCHC
- Leucocytes :- Reduced, presence of hypersegmented Neutrophils
- platelets :- Reduced or normal

2. Bone marrow findings:-

- Marrow cellularity - hypercellular with decreased myeloid & erythroid ratio
- Erythropoiesis:- Erythroid hyperplasia due to megaloblastic erythropoiesis

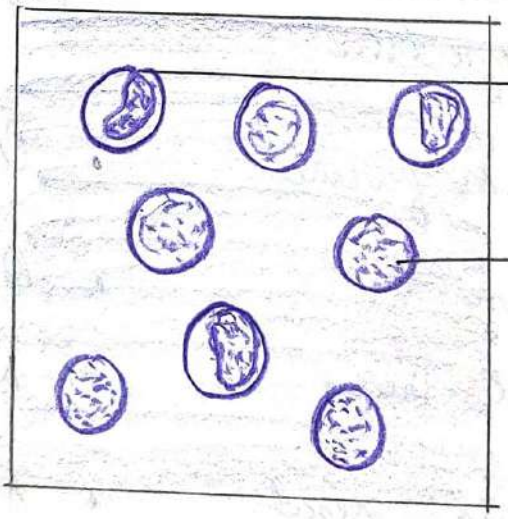
3. Biochemical findings:-

C. PERIPHERAL SMEAR.



6

BONE MARROW PICTURE



2. KWASHIORKOR :-

Kwashiorkor is a protein malnutrition seen in children above 1 year of age. associated with wasting of muscle but not adipose tissue, along with edema (localized) leading to growth retardation.

	KWASHIORKOR ~~(6 months - 4yr)~~	MARASMUS
•	Protein deficiency (above 1yr)	• Protein & calorie deficiency (below 1 year)
•	Wasting of muscles	• wasting of muscles along with adipose tissue
•	but preservation of adipose tissue	
•	Growth failure	• Growth failure present
•	Oedema.	• Oedema is absent
•	Enlarged liver	• Not observed
•	Anaemia present	• Anaemia absent
•	Atrophy of Organs	• Not observed.
•	presence of fatty liver	• absence of fatty liver

8. ANEMIA :- Is defined as decrease in haemoglobin levels below normal range males - < 13 gm/dl
females - < 10 gm/dl
Newborns - < 15 gm/dl
pregnancy - < 10.5 gm/dl.

(2)

ETIO PATHOGENESIS.

Iron deficiency anaemia is caused due to ↓ in iron in bone marrow for the synthesis of haemoglobin.

↓

uptake from peripheral iron stores

↓

depletion of iron stores

↓

decrease in Overall iron

↓

Causing iron deficiency anaemia

- decrease intake of iron in diet
- decrease absorption of iron from gut
- Increase in excretion of iron.
- Increased blood loss.

Etiology:-

1. Female reproductive life - menstruation
   - due to IUCD's
   - ~~post trauma~~
   - post partum haemorrhage

2. post menstruation.

3. Adult men. - dentition dance

Laboratory findings :- Microcytic, hypochromic anemia

Blood picture & Red cell indices

i) Haemoglobin - decrease in hb concentration

ii) Red cells - hypochromic, microcytic, (central pallor)
   anisocytosis
   poikilocytosis.

   Other cells - Target cells, elliptical forms
   Polychromatic cells.

iii) Reticulocyte count - normal or reduced.

iv) Absolute values - MCV ↓
   MCH ↓
   MCHC ↓

v) Leucocytes - Normal.

Necrosis

15)

Defination :- Type of cell death in living organisms which is localised in tissues by degrading hydrolytic Enzymes accompained by Inflammatory reactions.

Causes of Necrosis :-

(i) Degradation of hydrolytic Enzymes due to ischemia and hypoxia
(ii) Denaturation of proteins

Types of Necrosis :-

(i) Coagulative Necrosis
(ii) Liquificative Necrosis
(iii) Caseous Necrosis
(iv) Fat Necrosis
(v) fibrinoid Necrosis

Coagulative Necrosis :- Necrosis of tissues due to (i) Caesation of blood
(ii) Reduced blood supply due to hypoxia (Ischmia)

Mainly Seen in kidney, Heart, Lungs but not the brain.

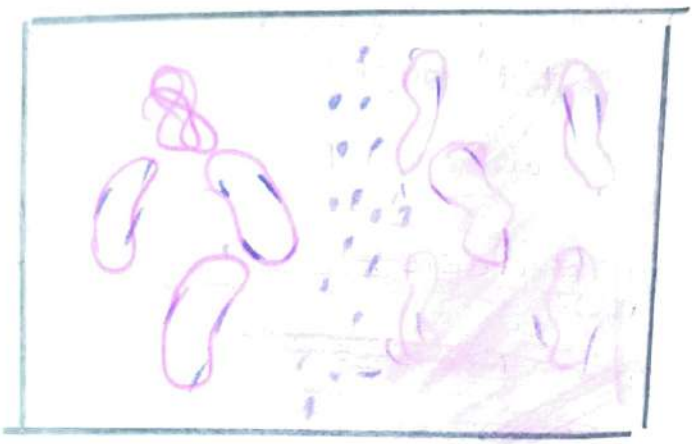
Morphological features :-

Cell become slightly swollen → Cell becomes Pale, Yellow Mor swalloing

Microscopic features :-

⇒ Marked feature of coagulative Necrosis is formation of Thrombstones

→ Highly eosinophilic stain, Nucleus and cytoplasm cannot be differentiated.



Liquificative Necrosis :-

Cell death occur due to ischemia, hypoxic conditions. ⊗ It is type of Metastatic calcification

Mainly seen in Brain infracts

Morphologically :- Cells are swollen & Pale yellow Bebs are seen.

Microscopically :-

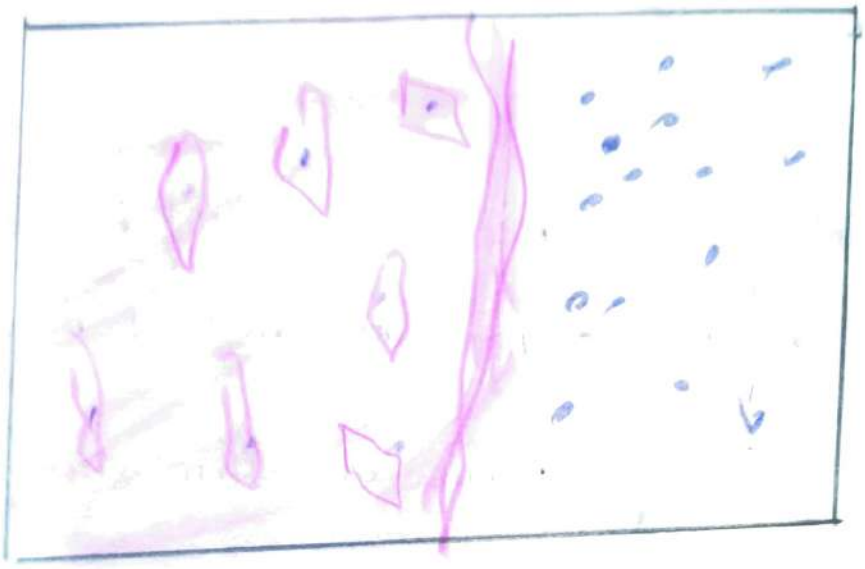
→ Highly eosinophillic stain, due to liquication of cells

→ Karyorrhexis, pkynosis, karyolysis of cells is seen.

Pyknosis :- Condensation of Nucleus

karyorrhexis :- fragmentation of Nucleus

∴ karyolysis :- Dissolution of Nucleus.



Caseasous Necrosis :-

→ caseosous means chessy like appearence

→ This type of cell death occur due to Tuberculosis condition

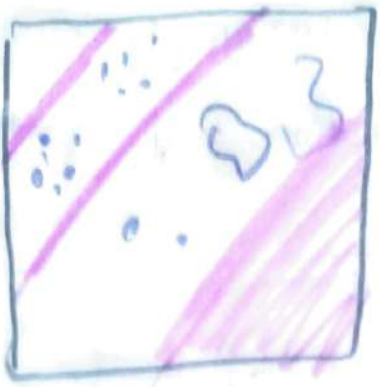
Morphologically :- Cheesy & soft, Swollen and Organ become yellow.

Cheesy appearence due to lipopolysaccharide capsule of Mycobacterium.

Microscopically :-

Highly eisnophills stain

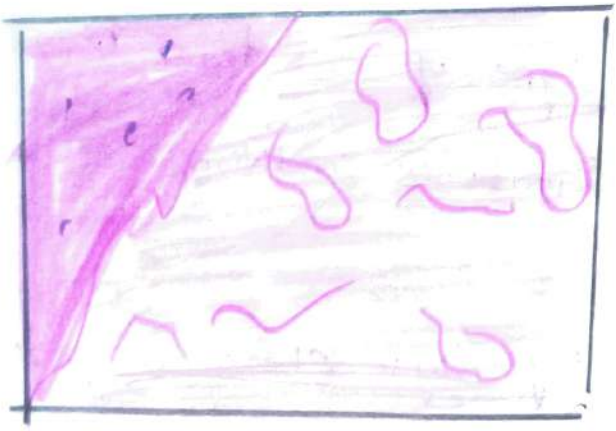
→ Nucleus is condensed and fragmented



Fat Necrosis :- Accumalation of fat substance

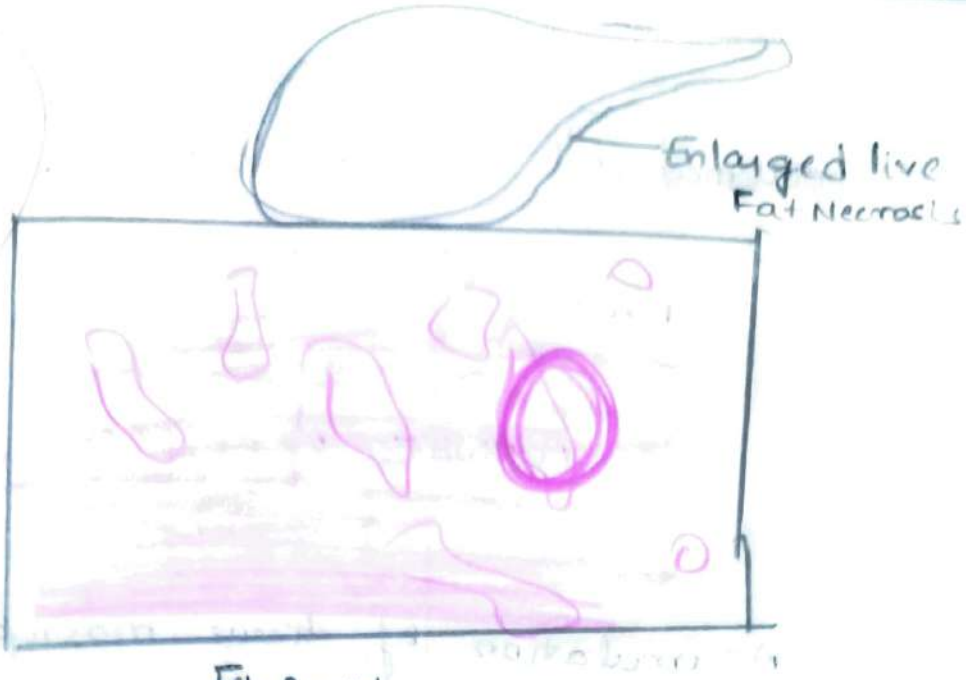
~~be~~ in breast pendulus, Acute Pancreatis

Microscopically :- Yellowish firm deposits



Fibrinoid Necrosis :- Depositing of fluid like Material ; Autoimmune diseases, Arteriales in hypertension, Peptic ulcer, .

Microscopically :- Highly eisonophically, hyaline deposit



2) Pathogenesis of granuloma formation

Granuloma means gaint cells surrounded by lympocytes occur in inflammatory reaction.

Cell injury

↓

Failure to digest agent

↓

Weak Acute Anflammatory response

↓

Engulfment by Macrophages

↓

Persistance of injurious Agent

T cell mediated immune response

Poorly digest agent

Activation of CD4+ T cell

Accumulation of tissue macrophages

Proliferation of T cells

Macrophage activation

Transformation to epitheloid cells

giant cells

Secreting of fibroblastic Proliferating cytokines

③

Metastatic calcifications	Dystropic calcification
* Deposition of calcium in Normal tissues	Deposition of calcium salts in degenerated tissues
* Calcium Metabolism is deranged	Calcium Metabolism is Normal
* Serum calcium level is increased due to Hypercalcemia.	Serum calcium level is Normal
* Reversible if we correct Metabolic disorder	Generally Irreversible if infective agent.
Causes of Metastatic calcification.	Causes of Dystropic calcification.
→ Hyperparathyroidism (due to adenoma)	→ Increased binding of phosphates due to Necrosis i- (i) caseosous (i) liquificative
→ Hypervitaminosis D,	→ Hematoma
→ Bony destructive lesions (Mylenoma)	

Metastatic	Dystropic
Pathogenesis :-	Pathogenesis :-
Increased accumulation Calicum phosphates due Hypercalcaemia at sites of lung, stomach blood vessels	Increased binding of phosphates with Necrotic and degenerative tissue, turn bind to calicum forming Calicum phosphates Precipitates

1A) Sys: My probable diagnosis is Systemic Lupus erythramatous because buttafly rashove face, episodes of hamoptysis from past 1 week and positive anti-neutrophilic antibodies and anti-double stranded DNA antibodies.

b) Etio-pathogenesis of Systemic Lupus erythrematous:-

~~Etiology~~:- Etiology:-

Exact cause is not known

→ ~~Antinuclear~~ Ant antibodies are detected against nuclear and cytoplasmic components of cells are demonstrable in ~~ee~~ plasma. Discoid form.

(i) Antinucleas Antibodies (ANA):- Antibodies against nucleas Antigens. Which destroy neutrophilic antibodies.

(ii) Antibodies to Double stranded DNA antibodia Antibodies against nucleas Antigens which are Double stranded

(iii) Anti Smith Antibody

(iv) Other non specific antibodies :-

Anti ribonucleoproteins

Anti phospholipid Antibodies

Pathogenesis of Systemic Lupus erythramatous.

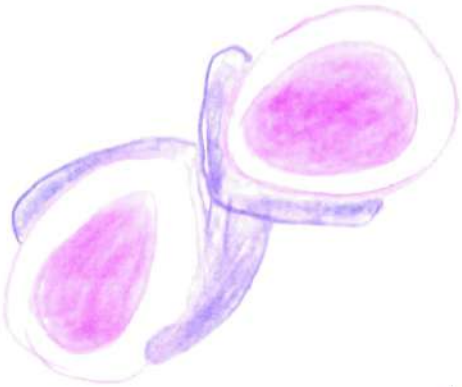
Autoantibodies formed by any of mechanism cause tissue injury

Two types of Tissue injury :-

Type II hypersensitivity characterized by Mast cell degranulation

Type III hypersensitivity Characterized by cytocelltoxicity and ADCC mechanism.

Le cell phenomenon



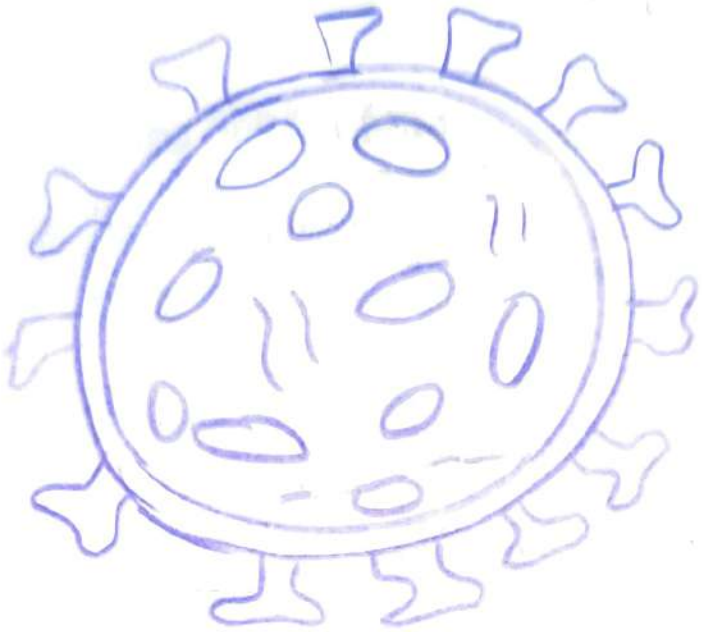
Typical LE cells

Morphological patterns of renal lesions :-

Small blood

28

· Structure of human Immunodeficiency virus (HIV)



· Pathogenesis.

Entering of HIV virus

↓

Incorporate into Host DNA

↓

Replicate in Host DNA

↓ by Reverse transcriptase

Viral proteins are produced

↓

Invade the other cells

↓

Immunodeficiency in Host

Cause

Cause HIV

3A) classification of Leprosy according to Modified Ridley - Jopling classification:

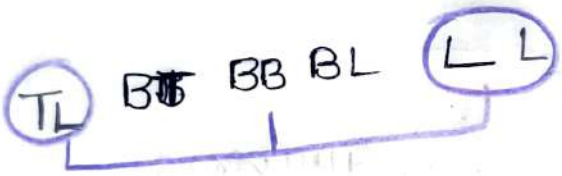
(1) Tuberculous Leprosy (TL) polar end

2) To Lepramatous Leprosy (LL) Polar end

3) Border Tuberculoid Leprosy (BT)

4) Border Lepramatous Leprosy (BL)

(5) Between Border line Leprosy (BB)



Tuberculoid Leprosy	Lepramatous Leprosy
Skin lesions Asymmetrical, Homogenous erythramatous	Skin Lesions Symmetrical, Multiple, homogenous erythramatous
Neve Lesions:- • Presents with More Sensory Associated	Neve Lesions:- • Present with Less Sensory Association disturbances
Histopathology:- Hard turbeculi like granulomatous formation eroding the basal layer	Histopathology Collection of foamy Macrophay & Lepraecells clear zone Lympocyte

Backpiology :

Leprae cells few, and destroyed

Complications :-

Type 2 reactional Leprosy

Neurological damage

~~Lep~~ Backpiology :-

Lepra ~~bacili~~ cells are highly positive to Leprae bacili

Type 2 reactional Leprosy may occur

04/6/24 Monthly test - 3

1A) My diagnosis is 'Pernicious Anaemia' [one type of "Megaloblastic Anaemia"]. It is because :-

1) Patient has Past history of Hashimoto's thyroiditis [Autoimmune disorder]

2) Patients presented with 'fatigue' & sore throat 'Pallor' of conjuctiva, tongue was "beefy red" & smooth

3) Pernicious Anaemia occur in old age '>60yrs'.

B)

. Laboratory findings :

1) Hemoglobin Estimation :-
Hemoglobin level is "decreases" than normal limit causing pallar

2) pherepheral blood smear :-
Megablastic, Macrocytic Anaemia.
Red blood size increased. (Normochromacia)

3) Red cell indices :-
'MCV' level increased than normal
MHC level increased than normal
'MCHC' level may normal & decreased

Reticulocyte count :- May be low & Normal level

Platelet count :- slightly reduced than normal

Neutrophilis ~~count~~ :- "Hyper segmented Neutrophilis" on blood smear Multilobed.

Leucocyte count :- slightly reduced.

Biochemical findings

FIGLU Test for folic acid deficiency :- on giving Homocysteine orally : folic acid level will be increased in urine

B_{12} findinding :- Microbiological B_{12} assy
Radioassy test

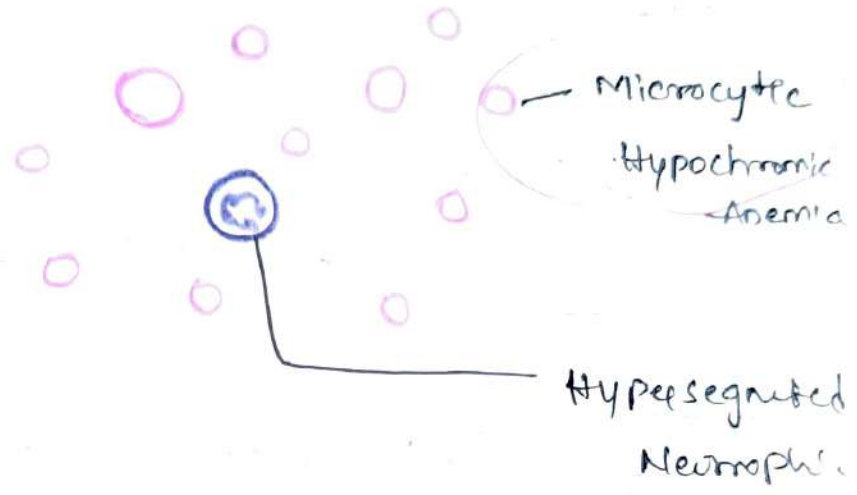
Sqphilling test :- Whether it differentiate Vit B_{12} & folic acid deficiency.

Bone Marrow findings

Marrow cellularity :- Cellusity of Marrow

"Increased" due to over production of cell from Bone Marrow

Erthryopoiesis :- erythroid Hyperplasia
Seen in Megaloblastic Anaemia.



Difference between	
Kwashiorkar	Marasmus
• Occur due "protein" deficiency with adequate calorie level	• occur due to "calorie" deficiency
• Seen in children between 2-4 years	• Seen in "infancy" <1 year
• Edema Present	Edema Absent
• Fatty liver enlarged	No fatty liver
• Lethargic	Alert and irritable

Kwashiorkor :- "Malnutrition" caused by "Protein" deficiency with Sufficiency calorie intake.

Commonly seen children between "2-4 years" more than 1 yr.

due to Breast feeding decreased for "1st child" if "Second pregnancy" occur within 2years after 1st child..

Clinical features:- Edema, Emaciation, Bloated body,

34) Anemia :-

Defined as "decreased Haemoglobin Concentration" lower than Normal limit of Age, Gender of an individual.

Red blood cells (RBC) and oxygen carrying capacity reduced.

Iron deficiency Anemia :- Nutritional deficiency Anemia.

Etiology :-

(i) .Women in Reproductive Age :-

Anemia coomonly seen due to menstruation causes Excess Blood loss

(2) Decreased Dietary intake of "iron"

(3) Decreased "Iron absorption" due to

. Gastric ulcers, ↓ Brush Borders in Anorexia

(ii) Adult Males :- Rarely seen due to chronic liver disorders, Renal disorders Haemolytic disorders

(iii) Pregnancy :- Hemodilution cause Anemia

Infancy poor feeding of iron and Milk is poor source of iron

Pathogenesis :-

(i) Decreased iron intake :- due to Malabsorption, Anorexia etc.

(ii) Decreased iron absorption :-

Due to GIT disorders like Achlocardia.

decrease in vitamin C.

(iii) Decreased synthesis of erythropoietin :-

Renal disorders

Laboratory findings :-

Hb concentration :- Hb con^c decreased

Red cell indices :-

MCV, MHC, MCHC will be decreased

Phepipheral blood smear :-

Microcytic, Hypochromic Anaemia

RE Count :- Normal leve

Marrow cellulaplity :- decreased Marrow Cellulaplity

Serum ferritin :- Low ferritin level

TIBC capacity :- TIBC level increased

PES INSTITUTE OF MEDICAL SCIENCES & RESEARCH
KUPPAM-517425
DEPARTMENT OF PATHOLOGY

II MBBS TERM MONTHLY TEST- MARCH 2024

Date: 25.03.2024
TIME: 1 hour

(Write neat diagrams using color pencils wherever relevant)
MAX. MARKS:20

LONG ESSAY 10X1=10 MARKS

Define necrosis and enumerate the different types with examples.

SHORT ESSAY
2X5=10 MARKS

2. Describe the pathogenesis of granuloma formation.
3. Differentiate between metastatic and dystrophic calcifications

PES INSTITUTE OF MEDICAL SCIENCES & RESEARCH
KUPPAM-517425
DEPARTMENT OF PATHOLOGY
II MBBS SECOND MONTHLY TEST FOR MAY 2024

Date: 27-05-2024
TIME DURATION: 1 Hour
MAX. MARKS: 20

(Write neat diagrams using color pencils wherever necessary)

LONG ESSAY **10X1=10 MARKS**

1. A 24-year-old female presented with history of recurrent episodes of fever, pleuritic chest pain, and arthralgia of two-year duration. The patient also had episodes of haematuria from past 1 week. Physical examination revealed a butterfly rash over the face and similar rashes over the extremities. Laboratory investigations revealed anaemia and thrombocytopenia. Further laboratory work-up showed positive anti-neutrophilic antibodies (ANA titre: 1:600) and strongly positive anti-double stranded DNA antibodies (1400 IU/ml). Urine examination showed few RBC casts and mild proteinuria.

a. What is the probable diagnosis. [2 marks]

b. Describe etio-pathogenesis of the condition with neat labelled schematic diagram. [5 marks]

c. Enlist six morphological patterns of renal lesions in the above mention clinical condition. [3 marks]

. **(2+5+3 marks)**

SHORT ESSAY **5X2=10 MARKS**

2. Describe the structure of Human Immunodeficiency Virus (HIV) and pathogenesis of HIV with schematic diagram. **(2+3 marks)**

3. Classify leprosy according to modified Ridley-Joplin classification. Tabulate the differences between tuberculoid leprosy and lepromatous leprosy. **(2+3 marks)**

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DEPARTMENT OF PATHOLOGY

Date:24/6/2024

Long Essay **1x15= 15 marks**

1. A 65-years –old woman presented with fatigue and a sore tongue. She has a past history of hashimoto's thyroiditis for which she has had a thyroidectomy done. Physical examination revealed pallor of the conjunctiva and the palmar creases . Her tongue was beefy red and smooth. Vibratory sensation was absent in the lower extremities.

A) What is your clinical impression and why? 3marks

B) Describe the laboratory (hematological and biochemical) and the bone marrow findings . 8marks

C) Draw a neat labelled diagram of the Peripheral smear and Bone marrow picture. 4marks

Short Essays **2 x 5=10marks**

2.Describe Kwashiorkor. Tabulate the differences between Kwashiorkar and Marasmus.(3+2)

3.Define Anemia. Describe the aetiopathogenesis and Laboratory findings in Iron Defeciency anemia (1+4)