Amrutha K
Roll No: (5)

15/7/22. Department of Ophthalmology – Assignment

15/20

## Anatomy & Development of Eye

- The development of the eye is a marvel of complexity, involving a series of intricate cascading cellular events, precisely choreographed by a set of molecular signals that can switch on and off genetic programs

### Development of Eye:-

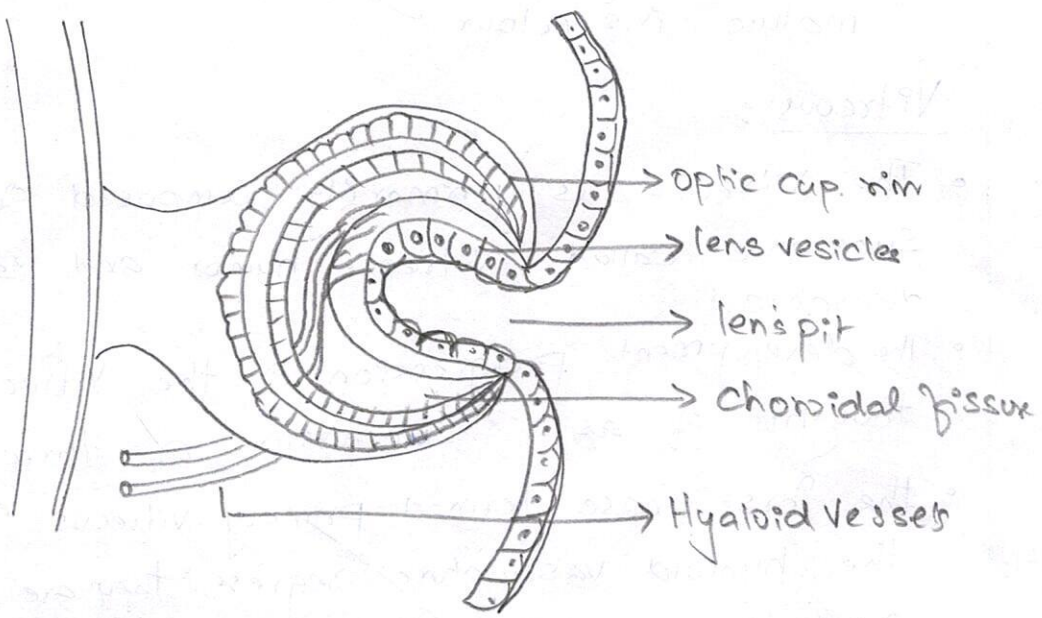
- During the fifth week of gestation, the eyelids begins to form from contributions of both neural crest infiltrated mesenchyme and nearby surface ectoderm
- By the Seventh week, two distinct eyelid folds are apparent and epithelial cells begin to invaginate in the area of the medial lid margins, forming the precursors to the future lacrimal puncta and canaliculi.
- During the 8th week, the upper and lower eyelid proceed to refuse via epithelial cell migration and proliferation
- Later mesenchyme begins to infiltrate the lids and begins to form the palpebral musculature.
- In the mid second trimester, a gradual re-seperation of the lids commences forming the mature palpebreal fissure

1) Lacrimal gland:-

- The lacrimal gland begins its development in the 7th week of gestation.

derived from Successive waves of invading neural crest cells.

- The mature transparent cornea is ultimately formed when the mesenchymal derived stromal keratocytes produces a highly organised stromal collagen matrix.




## Iris and ciliary body

- The iris and ciliary body are a product of the anterior rim of optic cup.
- The inner and outer layers of the anterior rim induced by surrounding mesenchymal derelopment, give rise to the posterior and anterior portion of iris epithelium respectively
- The intrinsic muscle of the iris (Sphincter pupillae/ dilator pupillae)

Krishnan. C.T
Roll. no: 66

29/9/21

# ENT TEST

18/20

## I. Short notes

### 1. Lateral wall of nose:-

⇒ The lateral wall of nose is formed by bone, cartilage, soft tissues.

⇒ Bony support is provided by:-

1) Ethmoidal labyrinth & uncinate process.
2) Perpendicular plate of palatine bone.
3) Pterygoid process of sphenoidal bone.
4) Medial surface of lacrimal bone.
5) Medial surface of maxillary bone.
6) Inferior concha.

* Contents:-

1) Naso lacrimal duct - under anterior lip of inferior concha.
2) Frontal sinus:- frontonasal duct & ethmoidal infundibulum into anterior end of semilunar hiatus.
3) Anterior ethmoidal cells:- drain into frontonasal duct or ethmoidal infundibulum.
4) Middle ethmoidal cells:- Open into ethmoidal bulla.
5) Posterior ethmoidal cells:- open into lateral wall of superior nasal meatus.

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6) Maxillary sinus :- open into semilunar hiatus.

* It is subdivided into 3 areas

1) vestibule.

2) Nasal concha & meatus.

- conchae — Superior, Middle, Inferior.
- Meatus — Superior, Middle, Inferior

- Blood supply :-

1) External Carotid artery → sphenopalatine artery.
→ Greater palatine artery
→ Superior labial artery
→ Lateral nasal artery.

2) Internal carotid artery — Anterior & posterior ethmoidal arteries.

2) BLOOD SUPPLY OF TONSIL :-

⇒



* Arterial supply :-

i) Tonsillar branch of facial artery.

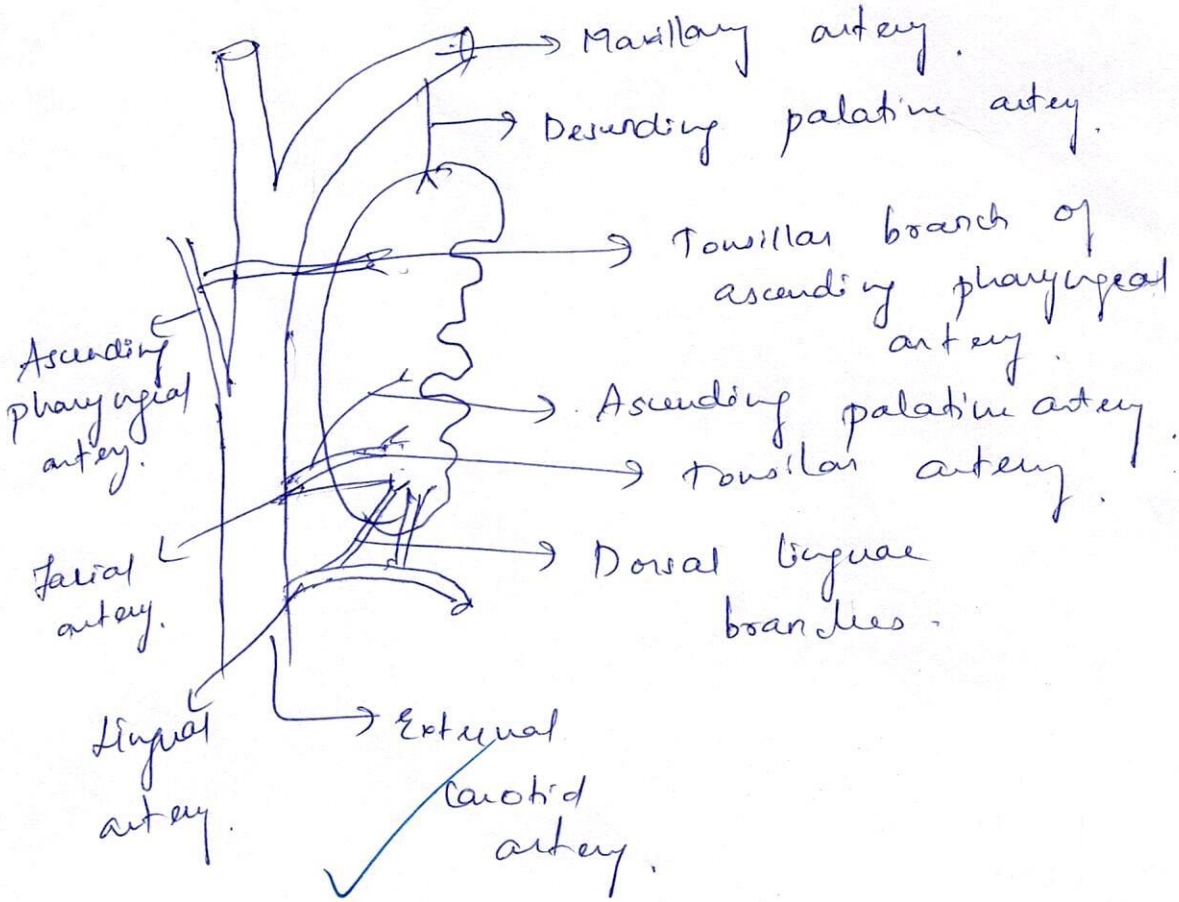
ii) Twigs from dorsalis linguae artery.

iii) Twigs from ascending pharyngeal artery.

iv) Twigs from ascending palatine artery.

* V enous drainage

→ Paratonsillar vein to pharyngeal plexus.

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28/10/20

# General Surgery - Test

INDUMATHY S
Regno: 521621566

16/20

## ① CA - esophagus:

Esophageal cancer is of 2 types
- Squamous cell carcinoma.
- Adenocarcinoma.

### Squamous cell ca:

most common overall & is located in middle one third

Risk factors: smoking, alcohol, preservative rich food, tylosis, achalasia cardia, vit E & selenium deficiency, plummer vinson syndrome.

### Adenocarcinoma:

Located in lower one-third

Risk factors: smoking, alcohol, GERD, CREST syndrome, Barrett's esophagus, Obesity

### Clinical Features:

① Dysphagia: Earliest & m/c symptom
Progressive
solids > liquid

② weight loss

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Investigation of choice to confirm the diagnosis — endoscopic biopsy.

IOC for staging - PET CT.

IOC for T & N staging - Endoscopic ultrasound.

Barium swallow:

- Rat tail appearance or
- Apple core deformity
- Shouldering effect
- Irregular strictures.

TNM staging:

Tis - High grade dysplasia
T1 - Invasion into lamina propria, submucosa.
T2 - Invasion into muscularis propria
T3 - Invasion into adventitia
T4a - Invades resectable adjacent structures.
T4b - Invades unresectable adjacent structures

N0 - No regional lymph node metastasis
N1 - 1-2 positive regional lymph node.
N2 - 3-6 positive regional lymph node.
N3 - 7 or more positive regional lymph node.

M0 - No distant metastasis
M1 - Distant metastasis.

## Surgical management:

### Esophagectomy.

Proximal margin Resection – 10cm.
Distal margin resection – 5cm.
wide margins are required to resect skip metastasis in lymphatics running longitudinally along submucosal layer.

* Transhiatal / Orringer's esophagectomy : Lower 1/3rd
mid 1/3rd
2 incision
Neck – site of anastamosis

* Ivor Lewis esophagectomy : mid 1/3rd
Lower 1/3rd.
2 incision
site of anastamosis – thorax

* Mckeown / Three field esophagectomy : upper 1/3rd
mid 1/3rd.
3 incisions
site of anastamosis – neck

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* chemotherapy :

- 5 fluorouracil (5 Fu)
- cisplatin
- taxanes (paclitaxel).

chemo & RT have been proved to improve the survival rate.

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(2) CA stomach:

Risk factors of gastric cancer:

- smoking
- Alcohol consumption
- consumption of smoked food / fish
- Menetrie's disease.
- Gastric resections & reconstruction
- Gastritis
- polyps

Classification of gastric cancer:

→ Lauren's classification → Pathological classification
→ Japanese classification
→ Bormann's classification
→ molecular classification

Clinical presentation of gastric cancer:

→ Lump
→ Gastric outlet obstruction
→ Anemia, Anorexia
→ Acedyspepsia
→ Late presentation.

Atypical presentation of gastric & GJ cancer:

1. Sister mary Joseph's nodule.
2. Krukenberg Tumor
3. Irish nodule – Left axillary lymph node in gastric cancer
4. Blumer shelf.
5. Left supraclavicular lymph nod.
   ↳ Virchow's node / Troisie sign.
6. Migratory Thrombophelebitis.
7. Leser Trelat sign.
8. Triple palms.

## Investigations for gastric carcinoma:

confirmation of diagnosis – Endoscopic biopsy

IOC for overall staging : PET-CT – Done T 18-FDG

IOC for T & N stage of cancer → Endoscopic USG

## TNM classification:

$T_x$ – 1° Tumour cannot be assessed.

$T_0$ – No evidence of 1° tumor

$T_{is}$ – carcinoma in situ

$T_{1a}$ – Tumour invades lamina propria, muscularis mucosa

$T_{1b}$ – Tumour invades the submucosa

$T_2$ - Tumor invades muscularis propria.

$T_3$ - Tumor penetrates subserosal connective tissue without invasion of the visceral peritoneum or adjacent structures.

$T_4$ - Tumor invades the serosa or adjacent structures.

$T_{4a}$ - Tumor invades the serosa.

$T_{4b}$ - Tumour invades adjacent structures.

$N_x$ - Regional lymph nodes cannot be assessed.

$N_0$ - No regional lymph node metastasis.

$N_1$ - Metastasis in 1-2 regional lymph nodes.

$N_2$ - metastasis in 3-6 regional lymph nodes.

$N_3$ - metastasis in 7 or more regional lymph nodes.

$N_{3a}$ - metastasis in 7-15 regional lymph nodes.

$N_{3b}$ - Metastasis in 16 or more regional lymph nodes

$M_0$ - No distant metastasis.

$M_1$ - Distant metastasis or positive peritoneal cytology.

most common site for distant metastasis → Liver.

## management:

- Management of gastric cancer
  - Surgical management
    - 1° tumor resection
    - lymph node resection
  - chemotherapy.
  - Radiotherapy.

## Surgery of 1° tumor:

Proximal margin → 5 cm.
Distal Margin → pylorus, irrespective of site of cancer.
Distal gastrectomy → Tumor at pylorus or antrum.

## Subtotal / Partial gastrectomy:

Tumor in the body of stomach.
→ Proximal margin → 5cm proximal to tumor.
Distal margin → pylorus.
60 - 75% stomach is removed.

## Total Gastrectomy:

Tumor at the fundus or Siewert type III.
→ Entire stomach removed till pylorus.
→ Reconstruction by esophago-jejunal anastomosis.

23/8/21

# General Medicine - Test.

MANIMOZHI.]
REGNO: 521721578

## ① AML:

common progenitor cells.

- B lymphocytes
- T lymphocytes maturing in thymus
- NK cells.

25/30

most common leukemia in adults → AML.

AML is a neoplastic disease characterized by
- infiltration of blood,
- bone marrow
- proliferative, clonal undifferentiated cells of the hematopoietic system.

### Etiology:

* Heredity:
  - aneuploidy
  - Inherited disease + defective DNA repair.
  - congenital neutropenia.
* Radiation
* exposure to benzene.
* Anticancer drugs.

## Classification:

$M_0$ : AML T minimal differentiation
$M_1$ : AML without maturation
$M_2$ : AML with maturation CM.
$M_4$ : Acute myelomonocytic leukemia
$M_5$ : Acute monocytic leukemia
$M_6$ : Acute erythroid leukemia
$M_7$ : Acute megakaryoblastic leukemia
Acute basophilic leukemia
Acute panmyelosis T fibrosis

## C/F:

* Fatigue
* Fever
* Night sweats
* Loss of appetite
* weight loss.
* Signs of abnormal hemostasis
* Findings → splenomegaly.
  Hepatomegaly.
  Lymphadenopathy.
  Sternal tenderness.

## Diagnosis:

CBC
PT, PTT
Bone marrow aspiration

## Treatment:

* Induction chemotherapy.
* Postremission therapy.

## ② Hodgkin's Disease:

Classification: Classical HD.
Nodular lymphocyte.
- → Lymphocyte rich
- → Lymphocyte depleted
- → Nodular sclerosis
- → mixed cellularity.

C/F: unexplained painless, rubbery, firm cervical lymphadenopathy.

lymph node: cervical > supra clavicular > axillary

Alcohol → Induce pain in lymph node

Nodular sclerosis → mediastinal lymphadenopathy
↓
Dyspnea, SVC obstruction, pleural effusion; pediatric & elderly → mixed cellularity

Reed Sternberg cell is seen.

Treatment:

~~Early disease~~

Adriamycin [cardiac toxicity]
Bleomycin [Lung toxicity]
Vincristine [Neurotoxic]
Dacarbazine

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## ③ Amyloidosis

Precursor soluble protein ⟶ extracellular insoluble fibrillar proteinaceous deposit.

Fibrils are due to :
- β configuration of precursor protein
- Proteolytic processing.
- Amyloid P component.
- Glycosaminoglycans.

### AL amyloidosis:

- → Amyloid proliferation
- → Lambda more common.
- → cardiac cause : non ischemic cardiomyopathy
- → Renal cause → proteinuria, non diabetic nephrotic syndrome
- → Hepatomegaly.
- → ors finding ++, CIDP & a monoclonal protein
- → Anti factor Xa antibodies
- → carpel tunnel syndrome.
- → macroglossia.
- → shoulder droopy.



### Treatment:

- High dose IV melphalan & dexamethasone
- Autologous transplant

(4). Antiplatelet Drugs:

MOA

Aspirin – COX inhibitor.
Dipyridamole – Phosphodiesterase inhibitors.
Treprostinil – Analogue of prostacyclin.

Clopidogrel
Prasugrel
Ticagrelor
Ticlopinide
} ADP antagonist.

Abciximab
Eptifibatide
Tirofiban
} GP IIb/IIIa inhibitors

⑤ Hyperproliferative Anemia :

→ Reticulocyte index ≥ 2.5.

* Hemolysis : Immune destruction
  Genetic conditions of hemoglobin
  Red cell enzymopathy or membranopathy
  Thrombotic microangiopathy.
  Drugs, toxins, trauma, mechanic heart valves

* Acute blood loss

⑥ Essential Thrombocytosis :

* Sustained increase in platelet count ≥ 4.5 L/cum
* Exclusion of WHO diagnostic criteria for CML.
* megakaryocytic hyperplasia / proliferation on bone marrow biopsy.



megakaryocytes → staghorn appearance on bone marrow biopsy.

* Presence of JAK 2 V617F, MPL or calreticulin mutation

## Clinical features:

* Increased platelets can clog small arteries of hand & feet causing throbbing pain
* Paraesthesias
* Transient ischemic attack.

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JAVISHI KRISHNA
Roll no: 521721634

Department of Pediatrics

31/40

① Pneumonia.

- Pediatric pneumonia is responsible for the death of >800,000 young children world wide
- Newborns with pneumonia commonly present with poor feeding and irritability, as well as tachypnea, retractions, grunting and hypoxemia.
- Infections with group-B streptococcus, listeria monocytogenes (or) gram-negative rods (eg: Escherichia coli / klebsiella Pneumoniae) are common causes of bacterial Pneumonia.
- Group B streptococci infections are most often transmitted to the fetus in utero. the most commonly isolated virus is respiratory syncytial virus (RSV)

## Pathophysiology

- An inhaled infectious organism must bypass the host's normal non-immune defense mechanisms in order to cause pneumonia.
- Respiratory tract host defenses
  - To prevent and minimise injury and invasion by microorganism and foreign substances. Various defense mechanisms have evolved, both systemically and with in the respiratory tract.
- Some mechanisms are non specific are directed against only microbes or substances with specific antigenic determinants.

- Non specific defenses include the glottis and vocal cords, ciliary escalator, airway secretions, migratory and fixed phagocytes, non specific antimicrobial Proteins and opsonins.
- Mucoid airway secretion provides a physical barrier that minimizes epithelial adhesion and subsequent invasion by microorganisms.
- These secretions contains complement components, fibronectin and other proteins that binds to microbes and renders them more susceptile to ingestion by phagocytes.
- Alveolar and distal airway secretions also include whole surfactant, while facelitates opsonisation and phagocytosis of pathogen, as well as surfactant-associated proteins A (sp-A) and D (sp-D).
- Both of these latter protein can modulate phagocytosis, phagocytes production of oxyradicals and cytokine elaboration.

## Systemic host defenses



- Immunologic defense mechanism targeted against particular pathogen typically emanate from specifically Primed lymphocytes following the presentation of Processed antigen by macrophages
- These mechanism include cytotoxic / killer / Suppressor and memory functions.

Systemic and Secretory antibiotics and Consequent Cascade of Cytokines

- Secretory antibodies and mucosal lymphoid tissues are absent or minimally functional for the first month of Postnatal life
- The neonatal granulocyte number frequently decreases in response to early infection. On the other hand the phagocytes that are present often move much more sluggishly to the inflammatory focus where
- Viral infections are characterized by the accumulation of mononuclear cells in the Submucosa and Perivascular Space, resulting in Partial obstruction of the airway
- Patients with these infections present with wheezing and Crackles.
- In bacterial infections, the alveoli fill with Proteinaceous fluid, which triggers a brisk influx of RBC and Polymorphonuclear (PMN) cells followed by the deposition of fibrin and the degradation of inflammatory cells (gray hepatization)
- During resolution, intra alveolar debris is ingested and removed by alveolar macrophages

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## Workup Management of CAP

- CURB 65
  - uses 5 Variables to assess overall death risk in CAP patient
    - a) Confusion
    - b) urea > 7 mmol (> 42 mg/dl)
    - c) respiratory rate > 30/min
    - d) Blood pressure: Systolic < 90 mm Hg
      Diastolic ≤ 60 mm Hg.
    - e) Age ≥ 65 years

## Treatment of Community aquired pneumonia

### Outpatient therapy regimens

1. Amoxicillin 1gm TDS + Azithromycin 500mg OD.
2. Amoxyclav 625mg + Doxycycline 100mg BD
3. Betalactum (Ampicillin + Sulbactum 1.5 - 3g q6th hrly + macrolide (azithromycin 500mg / Levofloxacin 750mg/day)

### Severe manifestation



1. Betalactum + Macrolide
2. Betalactum + Levofloxacin
3. MRSA / Gram negative bacilli (GNB - Pseudomonas)
   1. Vancomycin
   2. Piperacillin + Tazobactum

## treatment approach to CAP

CRUB 65 Score

- Inpatient
  - ① Sputum Gramstain / AFB / Gene expert Culture & Sensitivity
  - ② CXR PA view
  - ③ HRCT Chest.
    - Non Severe pneumonia → Ampicillin + Sulbactam + Azithro / Levofloxacin
    - Severe pneumonia → Ampicillin + Sulbactam + azithro / Levofloxacin

GNB / MRSA → Vancomycin / Piperacillin + Tazobactam.

- Outpatient
  - 1ed risk factors → Amoxyclav 625mg + Azithromycin 500mg
  - No risk factors → Amoxicillin 500mg + azithromycin

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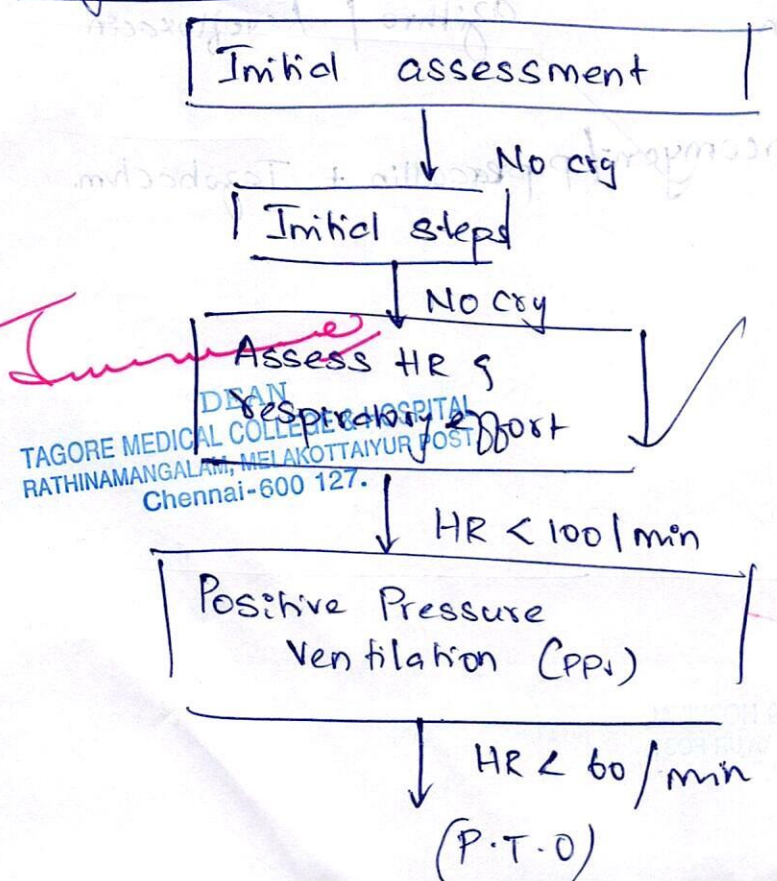
Short notes

① Neonatal resuscitation :-

- The guideline for neonatal resuscitation are based on NRP ( neonatal resuscitation programme) 2021
- In newborn priority is respiration >> temperature > Circulation
- Resuscitation in newborns is not done under room air, done under warmer air
- Initial assessment : 4 pre birth questions before delivery
  1. Term gestation or not.
  2. Amniotic fluid clear or not
  3. Additional risk factors.
  4. Umblical cord mx plan → delayed cord clamping by 30-60 seconds.

Algorithm



Chest Compression (with Ventilation)

↓ HR < 60/min

Adrenaline injection

↓

Volume expansion

## Chest Compressions

- a 2 thumb technique
- Site: Lower 1/3rd of sternum
- Avoid xiphisternum (easily injured).
- 1/3rd of the antero posterior (AP) diameter.
- Chest Compression + Ventilation
  (Ratio = 3:1 → 90 Compression + 30 Ventilation in 1min)
- Ventilation is given alternate airway (ET)
- monitor HR with Cardiac monitor

## ② Posterior Urethral Valve

- Posterior urethral valve (PUV) disorder is an obstructive developmental anomaly in urethra and genitourinary system of male newborn
- A Posterior urethral Valve is an obstructive membrane in posterior male urethra as a result of abnormal in utero development

- It is the most common cause of BOO in male new born
- The disorder varies in degree ē mild case presenting due to milder symptoms.
- More severe cases can have renal & respiratory failure from lung underdevelopment as a result of low amniotic fluid volumes

## Presentation

- PUV can be diagnosed before birth or even at birth when USG shows that a male baby has HUN.
- Some might also have oligohydramnios due to urinary obstruction
- the later presentation can be UTI / or voiding pain

## ③ Febrile Convulsion

**Definition** Condition ē fever and seizure in age group 6month - 6years

- Fever Temp > 100.4°F without the evidence of CNS infection / electrolyte or metabolic abnormalities
- No h/o preceding trauma or afebrile seizures



types of febrile seizures

| | Simple seizures | Complex FS |
|---|---|---|
| ① | GTCS | Focal |
| ② | lasting < 15 mins | lasts > 15 min |
| ③ | does not recur within 24 hrs | recurs with in 24h |

## Investigations:

- In Simple febrile seizure investigation is not needed.
- CBC → Complex FS or FSE
- Blood Sugar or Calcium estimation → CFS or FSE.
- Serum electrolyte → FSE
- Urine analysis only if < 18 month without focus of infection
- MRI
- EEG
- Lumbar puncture →

## 3 Treatment ✓

- Control fever → antipyretics / para — 10-15 mg/kg/dose q6th hrly
- tepid sponging
- Control seizures → IV benzodiazepine → Inj. Lorazepam / Inj. midaz (0.1 mg/kg)

## (4) Bone Marrow aspiration

- Bone marrow examination refers to pathologic analysis of samples of bone obtained by bone marrow biopsy and bone marrow aspiration, used for diagnosis of Leukemia / multiple myeloma / anaemia and pancytopenia

## Types of Bone marrow needles

1. Sternal needle → Klima
2. Salah bone marrow aspiration needle
3. Modern Jamshidi needle

## Technique of Bone marrow aspiration

- The best site for Bone marrow aspiration is Posterior Superior iliac spine
- The biopsy is usually performed ~~c~~ Jamsidi (Size 8–11) needle
- Administer local anaesthetic at site of biopsy when desired depth is reached the stylet is with drawn and needle is advanced ~~c~~ Same rotating movement
- The depth of the needle is measured the ~~stylet~~ Styler
- A Specimen measuring approx 2cm in length should be collected
- When the correct depth has been ascertained ~~the~~ needle is rotated in opposite direction to help loosen the Specimen
- The needle ~~c~~ the Specimen is withdrawn using a rotational movement

⑤ Scorpion sting

- ~~Asce~~ Indian red scorpion and black scorpion are more common
- Venom
  nature of venom is similar to cobra but fatality is less (as amount ingested is less)
- It is both neuro and haemo toxic
- It stimulates the sodium channels and inhibits $k^+$ channels → Produces autonomic storms

Clinical features

① Severe local pain

treatment

- local lignocaine / Icepacks / NSAIDs /

② local pain + no other symptoms → dry bite

③ cholinergic ~~cbs~~ crisis (early) → Salivation / Sweating / Paraplism / Bradycardia / hypotension

④ Adrenergic crisis → tachycardia / hypertension / (later) myocardial dysfunction / cold extremities / Cardiac failure / Shock & Pulmonary edema leading to Multi organ failure

⑤ CNS manifestations

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Asx Pragosen

- Symptomatic treatment

Bala Shankar. M
Roll : 17

18/20

1) Mammary gland

situation

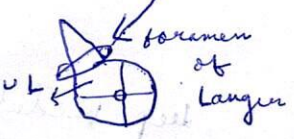
- lies in superficial fascia of pectoral region.

Extent

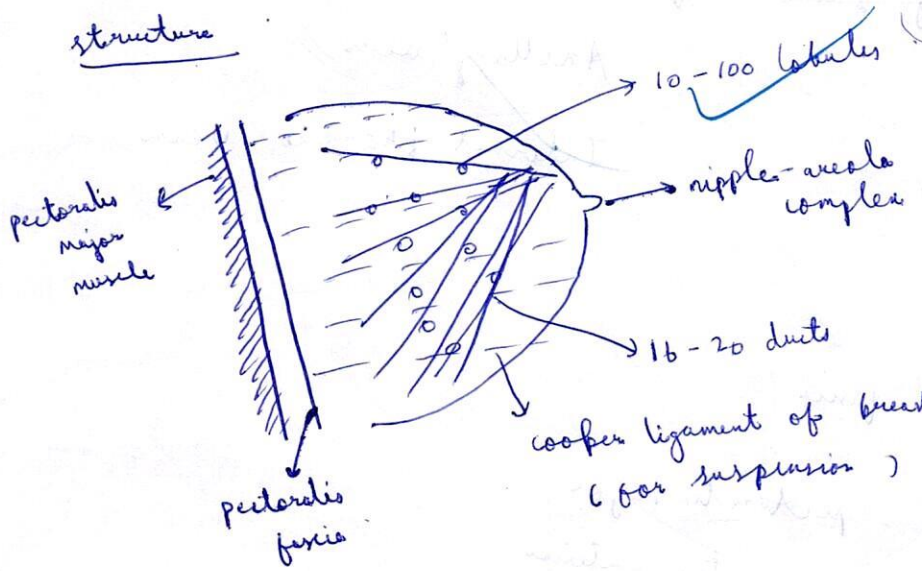
Vertically - from 2nd - 6th ribs

Horizontally - Lat. border of sternum to maxillary line.

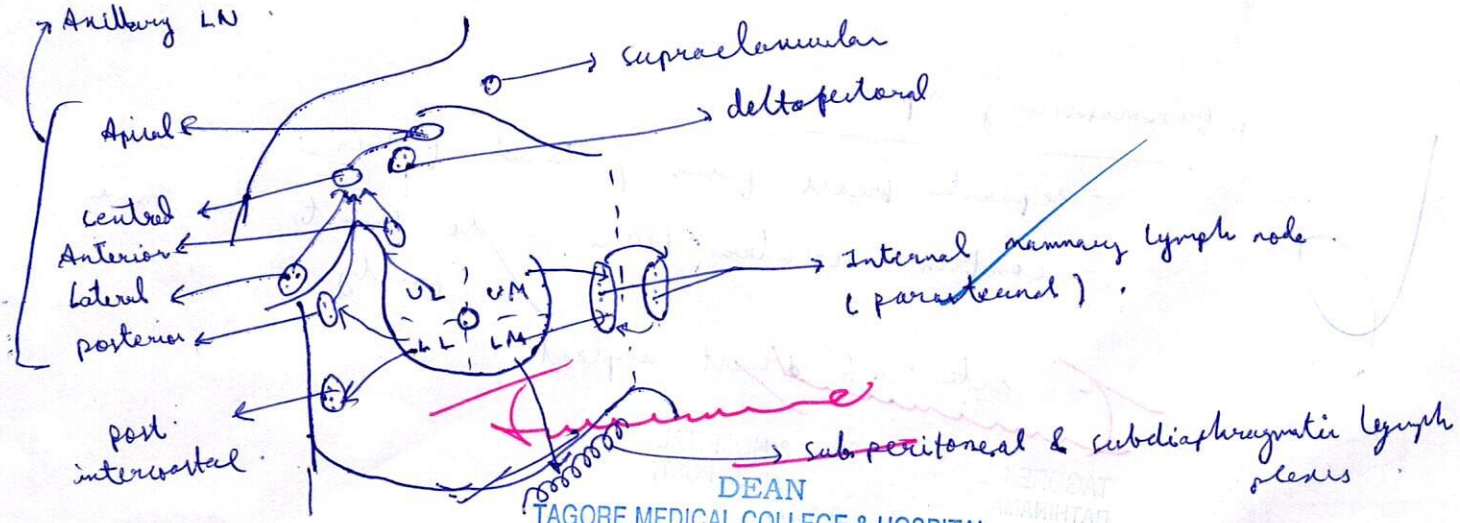
- Lies on deep fascia & separated from fascia by retromammary space → superolateral part called axillary tail of spence pierce the deep fascia & extend in the axilla




structure




Lymphatic drainage



## Blood supply

### Arterial supply

- Axillary artery via several branches
  - Superior thoracic
  - thoracoacromial
  - lat. thoracic
  - subscapular
- Internal thoracic artery via medial mammary arteries
- perforating branches of 2nd, 3rd & 4th intercostal arteries

### Venous drainage

~~perforating branches of~~ 2nd, 3rd & 4th Intercostal veins

~~1~~

Axillary vein

Internal thoracic vein

### Deep Relations

- lies on deep fascia
- 3 muscles
  - pectoralis major
  - Serratus anterior
  - External oblique
- Retromammary space
  - separate breast from pectoral fascia
  - contain areolar tissue
  - so breast can move freely
- site of breast implant

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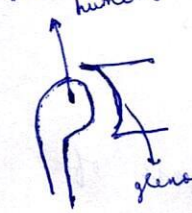
2) Shoulder joint

Type & articular surface

- glenohumeral joint
- synovial ball & socket articulation between head of humerus & glenoid cavity of scapula




↓ ball ↓ socket

- A dense fibrocartilagenous lip called labrum glenoidale is attached to the rim of glenoid cavity & makes it wider & deeper.

Capsule & ligaments

a) capsular ligament (ie capsule)

b) glenohumeral ligament

3 thickenings in ant. part of fibrous capsule

↓ to strengthen the capsule

- sup.
- middle
- inf.

c) coracohumeral ligament

- strong band of fibrous tissue
- base of coracoid process to ant. part of greater tubercle of humerus

d) Transverse humeral ligament

- broad fibrous band
- spans bicipital groove b/w greater & lesser tubercle changing it to canal

↓ provides passage to tendon of long head of biceps surrounded by synovial sheath

Accessory ligament

- coracoacromial ligament
  extends b/w coracoid & acromion process.

- coraco-acromial arch
  formed by coracoid process, acromion process & coracoacromial ligament.

joint capsule
- fibrous sheath which encloses the structures of joint.
- extends from anatomical neck of the humerus to the border of glenoid fossa.
- The synovial membrane lines the inner surface of jt capsule, & produces synovial fluid
  ↓
  to ↓ friction b/w articular surfaces

Relations

Ant
- subscapularis muscle.
- Deltoid
- The axillary vessel & brachial plexus.

post
- infraspinatus.
- Teres minor muscle.

Sup
- supraspinatus muscle.
- subacromial bursa
- coracoacromial ligament.
- Deltoid muscle

Inf
- Long head of triceps muscle
- axillary nerve.
- post. circumflex humeral vessels.

tendon of long head of biceps muscle passes through the joint & emerges beneath the transverse ligament

## Movements of shoulder jt

**flexion**
- pectoralis major (clavicular head)
- Anterior fibers of deltoid
- coracobrachialis
- biceps

**Extension**
- pectoralis major (sternocostal head)
- post fibers of deltoid
- Teres major
- Lattismus dorsi

**Abduction**
- supraspinatus (upto 15°)
- Deltoid middle fibres (up to 90°)
- Trapezius & serratus anterior (above head)

**Adduction**
- pectoralis major
- Teres major
- Lattismus dorsi
- Coracobrachialis
- subscapularis

**Medial rotation** - Pectoralis major
- Teres major
- Latissimus dorsi
- Anterior fibres of deltoid
- subscapularis

**Lateral rotation** - Infraspinatus
- teres minor
- post. fibers of deltoid

circumduction

combination of flexion, abduction
extension, abduction

3) great saphenous vein

origin, course, termination

formed by union of dorsal venous arch with medial margin vein

↓

passes upward in front of medial malleolus

↓

crosses the lower 1/3rd of medial surface of tibia obliquely

↓

Runs along medial border of tibia to reach the back of knee

↓

In the thigh, inclines forward to reach the saphenous opening

↓

Pierces the cribriform fascia & opens into the femoral vein

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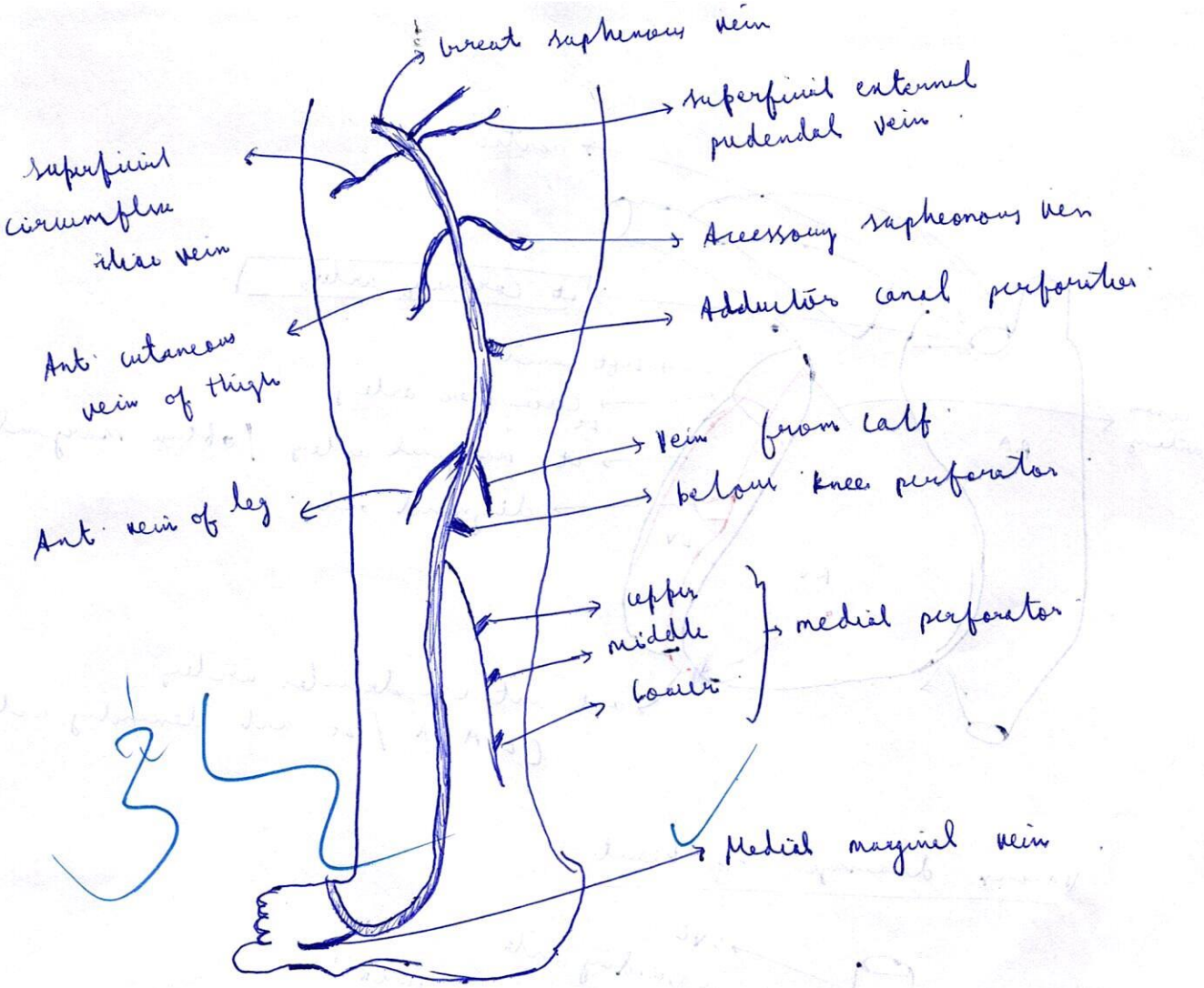
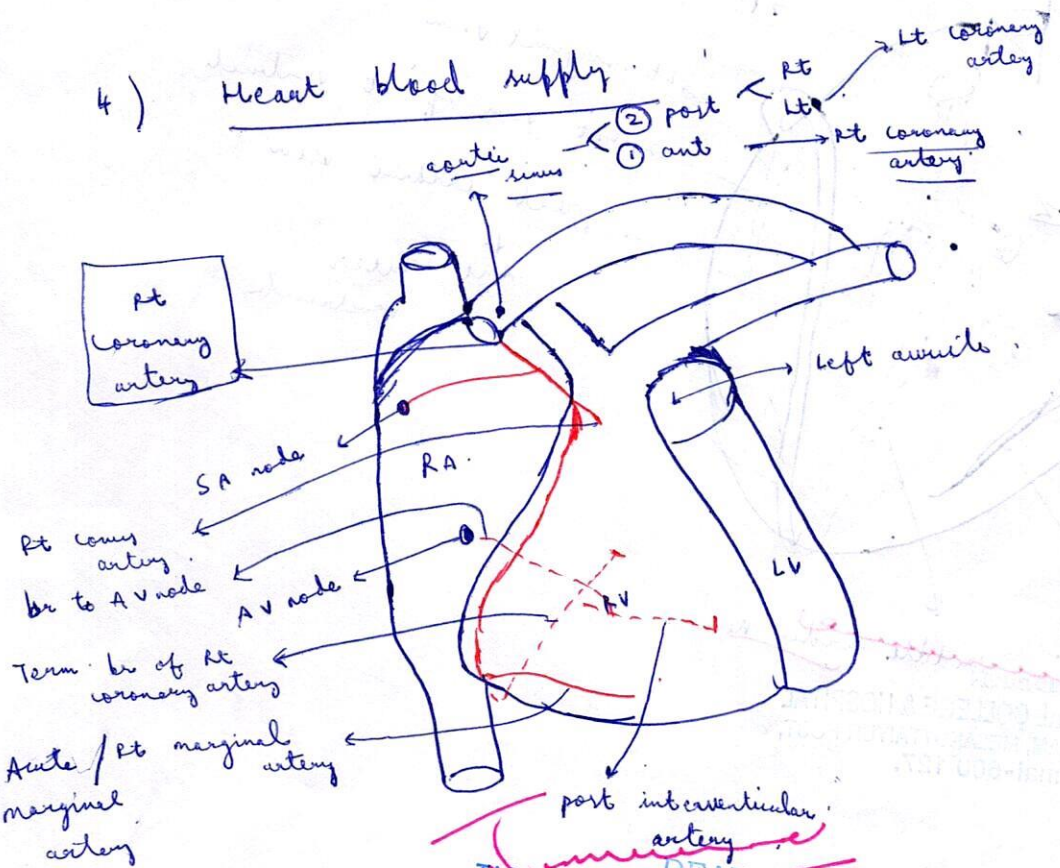
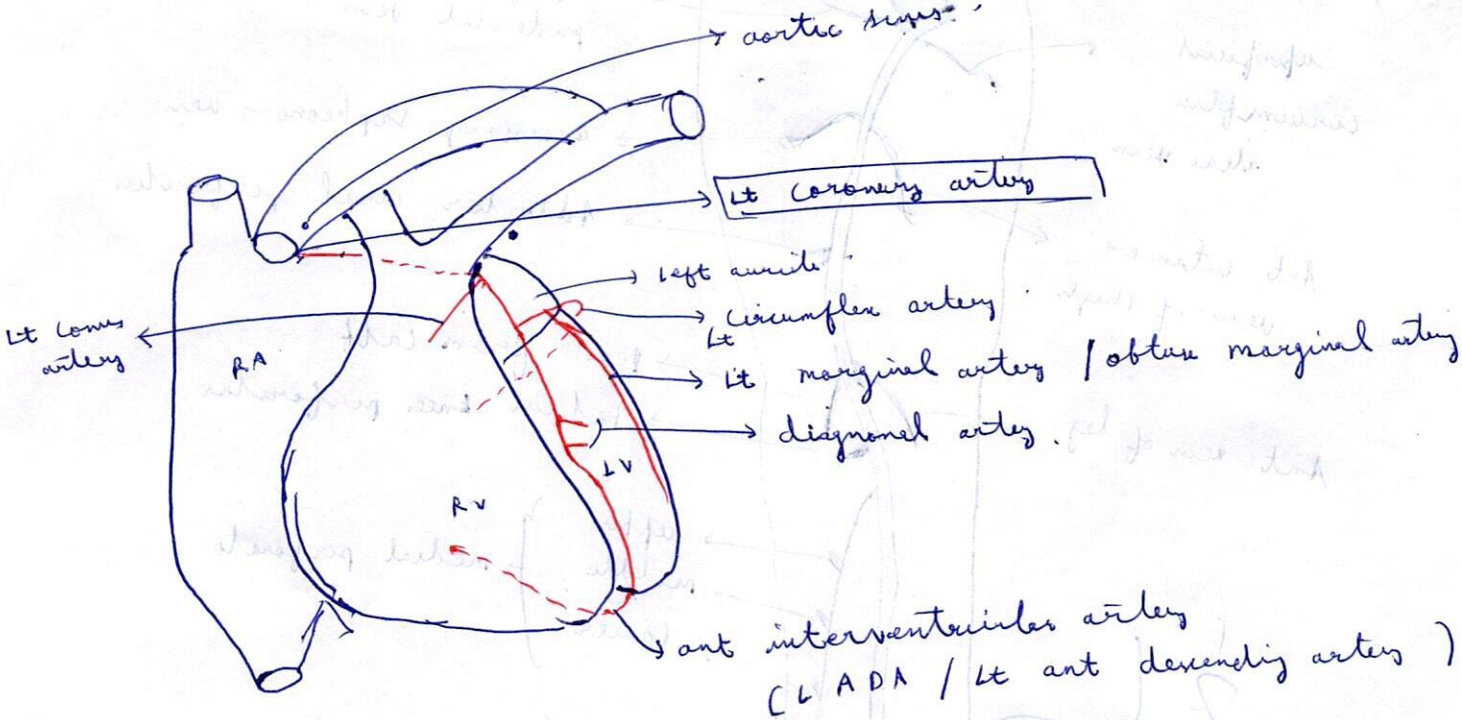
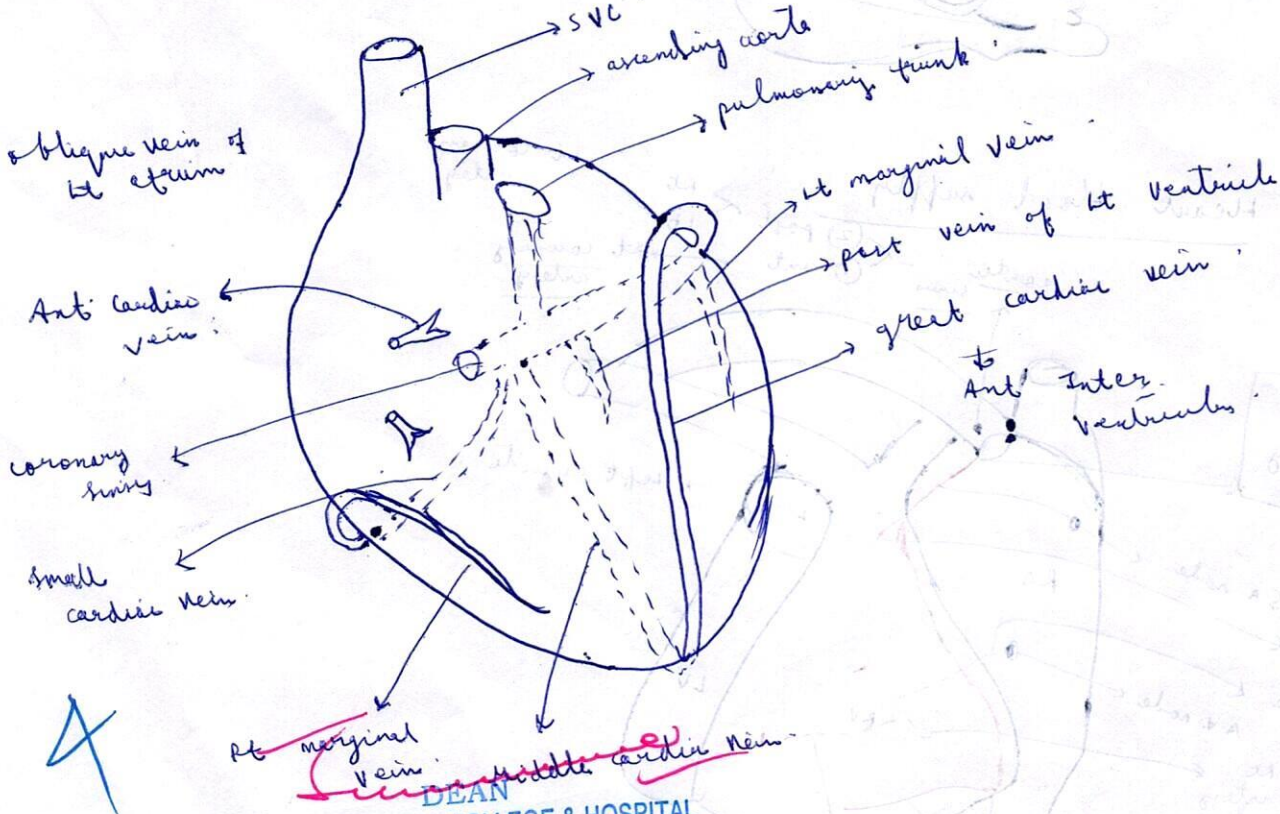



4) Heart blood supply






## Venous drainage of heart



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Apson Ranjith. B
Roll. no. 13

# Physiology Test.

1. **Erythropoiesis :-**

⇒ Erythropoiesis is the process of origin, development or maturation of erythrocytes.

**Site :-**

→ Fetal life - yolk sac.
→ Adult life - bone marrow.

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***Stages :-**

→ Proerythroblast.
→ Early normoblast.
→ Intermediate normoblast.
→ Late normoblast.
→ Reticulocyte.
→ matured erythrocyte.

*** Factors regulating erythropoiesis**

- **General factors :-**
  - i) Hypoxia → erythropoietin.
  - ii) Growth inducers.
  - iii) Vitamins.
- **Maturation factors :-**
  - i) Vitamin $B_{12}$
  - ii) Folic acid.
- **Factors necessary for hemoglobin production :-**
  - i) Vitamin c → helps in absorption of iron.
  - ii) Proteins → amino acids for globin synthesis.

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iii) Iron & Copper → heme synthesis

iv) Calcium, bile salts, Cobalt & nickel

## Regulation of erythropoiesis

### General factor

↓ Hypoxia

↓ Stimulate JG apparatus

↓ Bone marrow

↓ Erythropoietin

↓ Accelerate erythropoiesis

1) Thyroxin
2) Hemopoietin
   Growth factor.
3) Colony promoting factor
4) Vitamin.

### Special factors

Castle Hematic Principal

a) Intrinsic factor :- Secreted in gastric juice.
   - It helps in absorption of vit. $B_{12}$

b) Extrinsic factor
   - Vit. $B_{12}$ taken in diet for development of RBC.

### Dietary factors

→ All factors which are required for synthesis.

## Functions of erythropoietin

→ ↑ RBC production by
   → promoting erythroblast production
   → shortening maturation time through normoblast

→ Promotes early release of reticulocytes.

2. Resting Membrane Potential (RMP) :-

→ the electrical potential difference between the inside of the cell and the surrounding extracellular fluid is called RMP.

- Factors determining RMP

⇒ Pump potential

→ $3 Na^+$ are pumped out and $2 K^+$ are pumped in against the concentration gradient.

→ Requires energy.

⇒ Donnan Potential

→ Large impermeable negatively charged intracellular molecules (proteins) attracting positively charged ions ($Na^+$ and $K^+$) and repelling negative ions (eg:- $Cl^-$)

* Origin of normal RMP

→ Contribution of potassium diffusion potential

→ Contribution of sodium diffusion through the nerve membrane.

→ Contribution of sodium potassium pump.

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SHIFA AZEEZ .K.P
Roll no: 116

3/7/21

Biochemistry assignment test

14/20

Diabetes

Classification

a) Type I IDDM
- Characterised by a severe insulin dificiency.
- Patient requires insulin to live.
- the onset of the diabetes is in youth (age before 20)
- this type of diabetes is not related to obesity.

b) Type II NIDDM
- Constitute 90% or more of all diabetic pt.
- Insulin levels are normal or high but may diminish over years of having diabetes.
- Insulin resistance plays a major role in Pathogenesis.
- It is often diagnosed above age of 30.

Pathogenesis of type I diabetes.

- It is an autoimmune disease, the immune system mediates the destruction of β cells in the pancreas
- It develops in genetically susceptible individuals who are exposed to environmental factors which the autoimmune response and β cell destruction ensues.
- Overt IDDM does not appear until above 90% of the β cells in the pancreas are destroyed.

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Pathogenesis of type II diabetes.

a) Risk factors

- Obesity
- Genetics
- Age (Insulin Production decreases ↑ age
  Insulin resistance increases ↑ age)

Obesity:

- Obesity is associated with increased level of free fatty acid, which makes the muscle more insulin resistant and reduces glucose uptake by muscles. Therefore obesity exacerbates.
- In the liver free fatty acid increases the production of glucose contributing to high glycemic levels.
- In normal individuals, the pancreas secrete more insulin in response to free fatty acid, therefore
- Compensation does not occur and hyperglycemia develops → β cell becomes desensitised to glucose leading to decreased insulin secretion

Biochemical test for diagnosis of Diabetis mellitus

- Screening of all adults > 45 years every 3 year
- The following test are on two seperate days

a) Fasting plasma glucose ≥ 100 mg/dl
If between 100 - 150 mg/dl perform a 75g oral glucose tolerence test or recheck fasting plasma glucose

b) Random plasma glucose :. < 200 mg/dl in patient with symptoms of Diabetes

c) Two-hours postprandial plasma glucose level < 200 mg/dl

d) Hemoglobin $A_1C$ (%) – 5.7 - 6.4 %. (good glycemic control)
6.5 - 7.5 %. moderate glycemic control
7.5 – 8 %. Poor glycemic control
> 10 %. Poor glycemic control

| Diagnostic Criteria for Diabetes mellitus | | |
|---|---|---|
| Plasma glucose test | Impaired glucose tolerance | Diabetes mellitus |
| Random plasma blood glucose | | > 200 mg/dl c diabetic symptoms. |
| Fasting blood glucose | 100 - 120 mg/dl | > 120 mg/dl |
| 2hrs post Prandial blood glucose | 140 - 200 mg/dl | > 200 mg/dl |
| Haemoglobin $A_1C$ | 5.7 - 6.6 | > 6.5 |

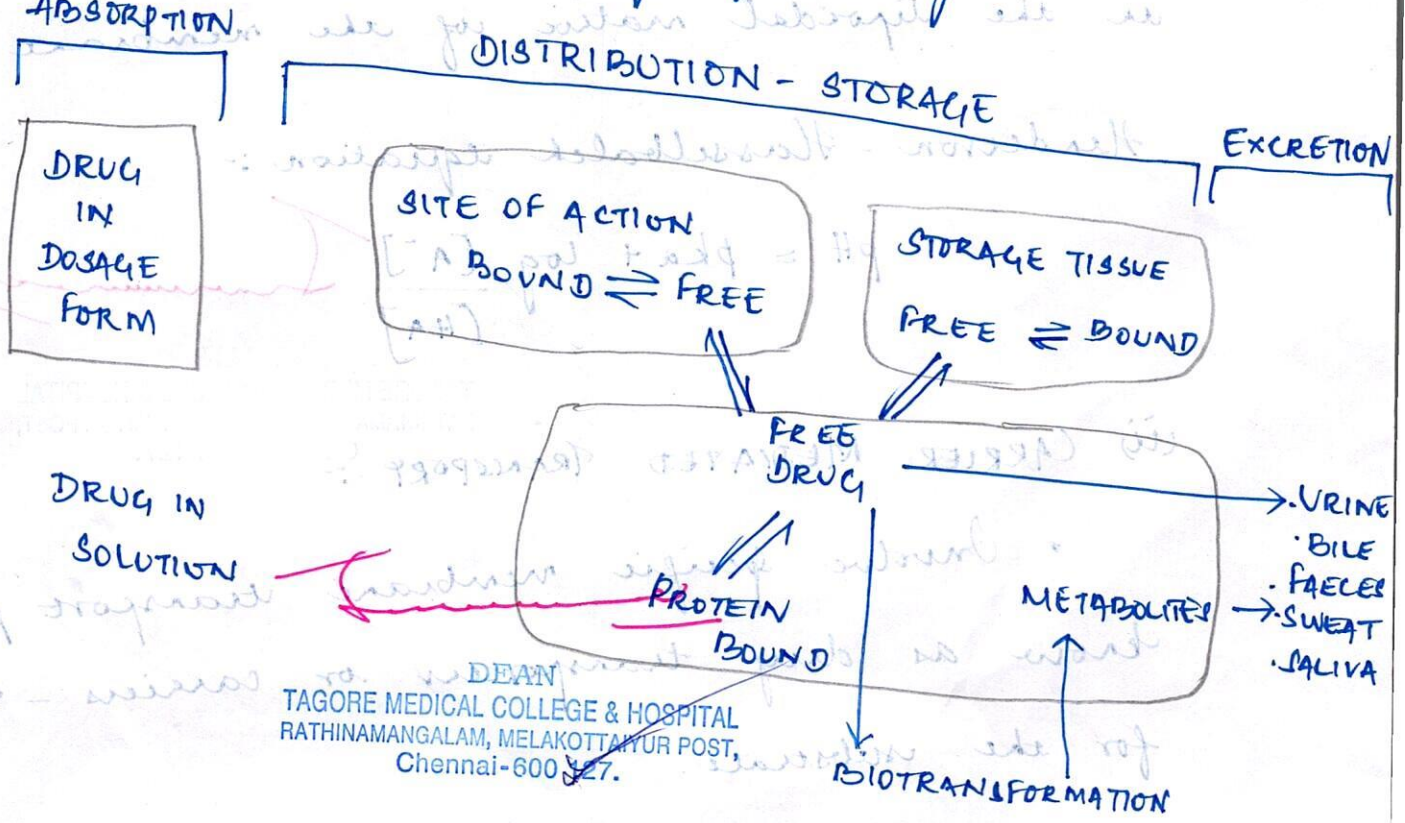
Dinesh. M
Roll No : 24

14/20

1) PHARMACOKINETICS :-

Definition :-

- Pharmacokinetics is the quantitative study of movement in, through and out of the body.
- Intensity of effect is related to concentration of the drug at the site of action, which depends on its pharmacokinetic properties.
- Pharmacokinetic properties of particular drug is important to determine the route of administration, dose, onset of action, peak action time, duration of action and frequency of dosing.



## DRUG TRANSPORTATION :-

- Drug molecules can cross cell membrane by.
  - (i) Passive diffusion
  - (ii) Protein - mediated transport (Carrier Mediated)
    - Facilitated Transport
    - Active transport.
      - Primary
      - Secondary.

### (i) PASSIVE TRANSPORT :

- Majority of drugs diffuses across the membrane in the direction of concentration gradient.
- Propotional to lipid : water partition coefficient.
- lipid soluble drugs diffuse by dissolving in the lipoidal matrix of the membrane.

Henderson - Hasselbalch equation :-

$$pH = pka + \log \frac{[A^-]}{[HA]}$$

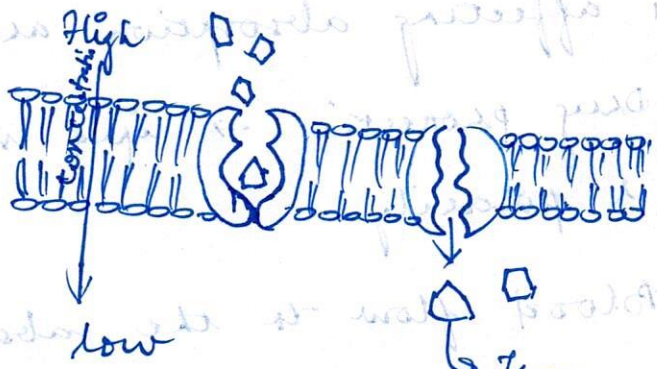


### (ii) CARRIER MEDIATED TRANSPORT :-

- Involve specific membrane transport proteins know as drug transporters or carriers - specific for the substrate.

• Drug molecules bind to the transporter, translocated across the membrane & then released on the other side of the membrane.

FACILITATED DIFFUSION :-




• Moves substrate of a single class (Uniporters) down a concentration gradient.

• No energy dependant.

• Similar to entry of glucose into muscle (GLUT-4).

ACTIVE ~~TRANSPORT~~ ENERGY DEPENDANT :-

• Can move molecules against a concentration

gradient and energy dependant.

## Absorption of drugs :-

- Absorption is the transfer of a drug from its site of administration to the blood stream.
- Most of the drugs are absorbed by the way of passive transport.

factors affecting absorption are :-

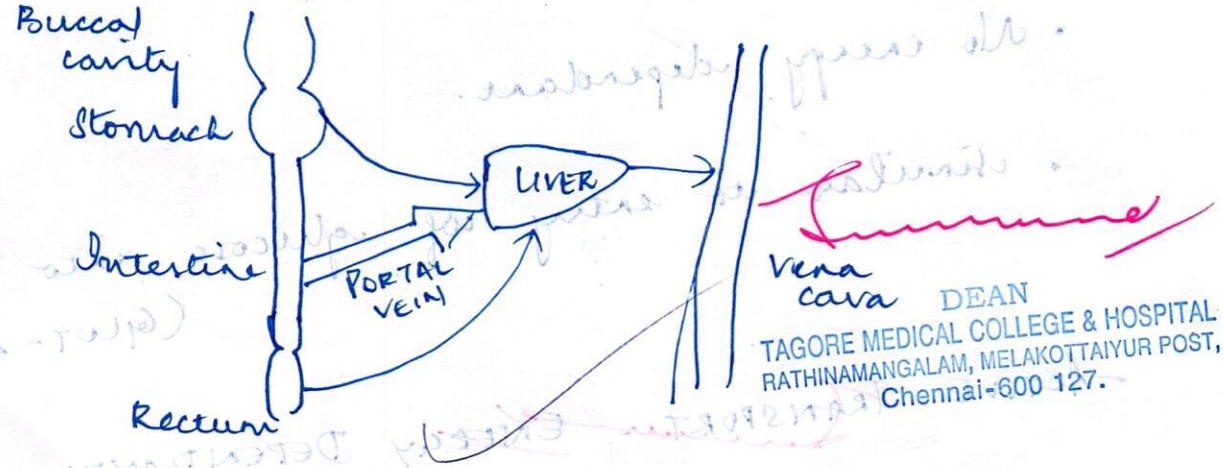
→ Drug properties → lipid solubility; molecular weight & polarity.

→ Blood flow to the absorption site

→ Total surface area available for absorption.

Routes :- Topical; Intravenous; Intramuscular; subcutaneous; Intraosseous.

## 1st pass metabolism :-

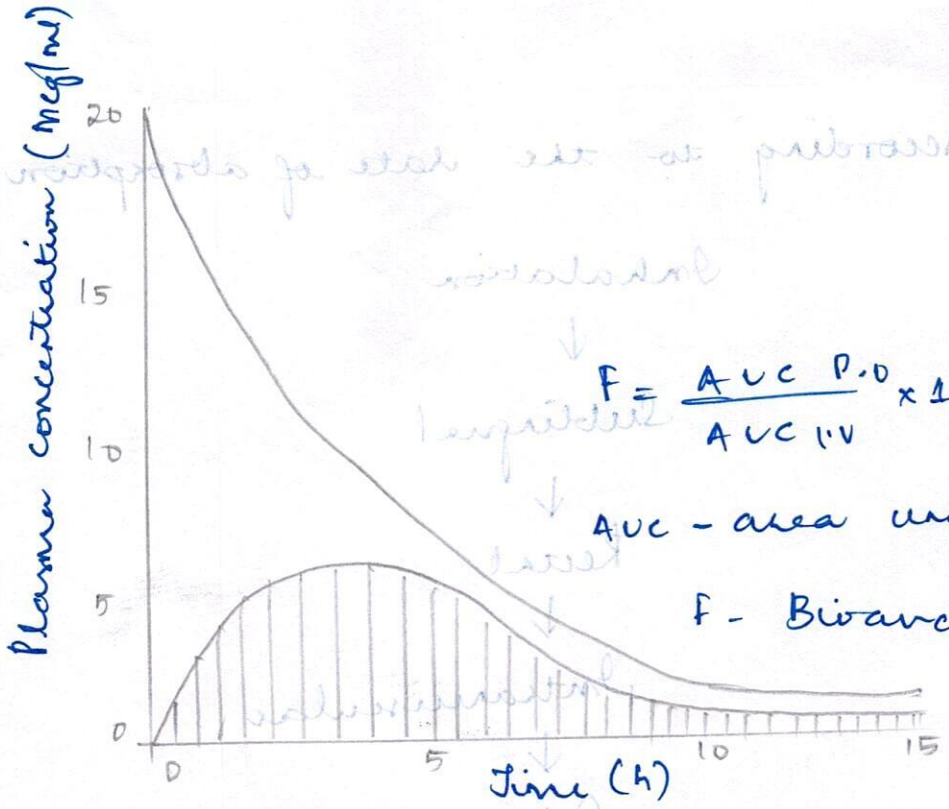


According to the rate of absorption :-

Inhalation
↓
Sublingual
↓
Rectal
↓
Intramuscular
↓
Subcutaneous
↓
Oral
↓
transdermal

BIOAVAILABILITY :-

- Bioavailability refers to the rate and extent of absorption of drug from dosage form as determined by its concentration-time curve in blood or by its excretion in urine.
- It is a measure of fraction (F) of administered dose of a drug that reaches the systemic circulation i.e. the unchanged form.




$$F = \frac{AUC\ P.O}{AUC\ I.V} \times 100\%$$

AUC - area under the curve

F - Bioavailability.

Volume of distribution :-

$$V_d = \frac{\text{Dose administered IV}}{\text{Plasma concentration}}$$

factors affecting $V_d$ :-

i) lipid solubility

ii) Pka of drug

iii) Affinity for different tissues

iv) Blood flow

v) Disease states

vi) Plasma protein binding.

29/6/21

Dakshana Rajendran
Roll: 22

# MICROBIOLOGY TEST

1. **Antigen Antibody Reactions :-**

→ The types of antigen antibody reactions are :-

- Precipitation reaction
- Agglutination reaction
- Complement fixation.
- ELISA
- Immunofluorescence.

19/20

## * AGGLUTINATION REACTIONS :-

→ The interaction between antibody & a particulate (insoluble) antigen results in visible clumping is called agglutination.

→ The various particulate antigens are :-

- WBCs
- RBCs
- Latex particles.

- **Principle :-**

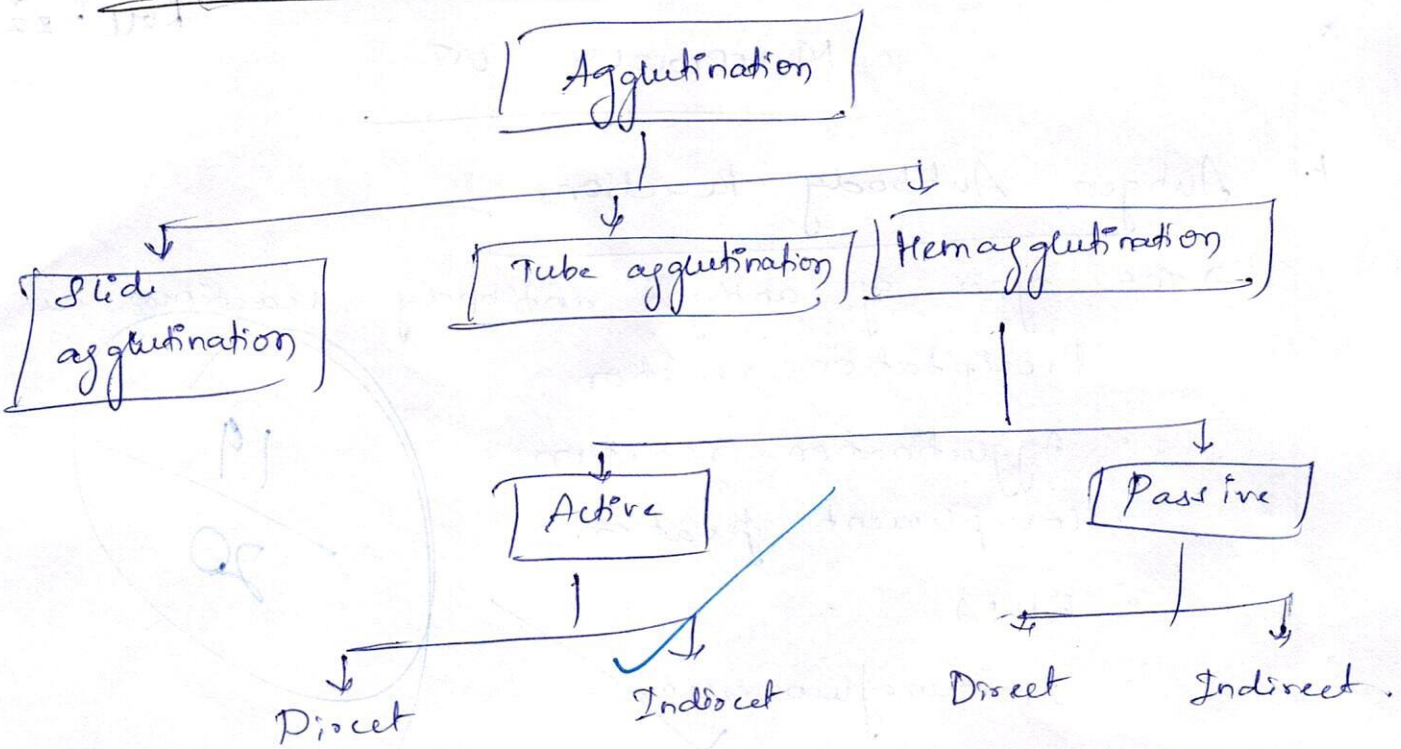
→ It is the visible aggregation of particles caused by combination of with specific antibody.

→ Antibodies that produce such reactions are called agglutinins.



→ The antigen must be exposed & able to bind with antibody.

• Types of agglutination reactions :-




• steps in agglutination :-

1) sensitization

↓

2) lattice formation

→ formation of cross links

↓

visible aggregates.



• Phases :-

1) Primary phase

2) Secondary phase.

* Applications :-

→ ABO Blood grouping.

→ Rh blood grouping.

→ Widal test for typhoid.

→ Weil Felix test for typhus.

→ Coomb's test for identification of anti Rh antibodies

→ Brucella agglutination test for brucellosis

→ Leptospira agglutination for leptospira

→ Cold agglutination test for pneumonia, malaria, trypanosomiasis.

→ Hemagglutination inhibition test.

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28/8/24

Arun kumar. A
Roll. no:- 17

# PATHOLOGY TEST

1. Define shock. Write about pathology, morphology, lab diagnosis of shock.

19/20

## *Shock:-

→ shock is defined as a life threatening condition due to poor tissue perfusion with impaired cellular metabolism, manifested by serious pathophysiological abnormalities.

### • Classification:-

→

- Shock
  - Hypovolemic shock
  - Cardiogenic shock.
  - Extracardiac obstructive shock
  - Distributive shock
    - → Septic shock
    - → Anaphylactic shock
    - → Neurogenic shock.

### • Pathophysiology:-

Dilatation of systemic vasculature

↓

Decrease systemic arterial pressure

↓

Pooling of blood in systemic venules & small veins

↓

The right heart filling of stroke volume decreases

↓

Decreased pulmonary blood volume & filling

Discharge of angiotensin & vasopressin though they fail to restore the cardiac output to normal,

- Hypovolemic shock → ↓ Effective circulating blood volume
  - → Hemorrhage
  - Trauma
  - Surgery
  - Burns
  - Dehydration
- Septic shock
  - → Gram -ve Septicemia
  - → Gram +ve Septicemia
- Cardiogenic shock

↓ Venous return to heart
↓ Cardiac output
↓ Blood flow
↓ Supply of $O_2$
↓ Anoxia
↓ Shock

## * Morphology of shock :-

- Brain – Ischemic encephalopathy.
- Heart – Coagulative necrosis / subendocardial hemorrhage / contraction band necrosis
- Kidney – Acute tubular necrosis
- Lungs – Diffuse alveolar damage develop shock lung.
- GIT – Hemorrhagic enteropathy.
- Adrenal gland – Cortical cell lipid depletion may occur.
- Liver – Fatty change, central hemorrhagic necrosis.

* Lab diagnosis of shock :-

* Arterial blood gas :- most useful laboratory investigation.

↳ shows Metabolic acidosis — an elevated lactate level indicate inadequate tissue perfusion.

* Hemoglobin & hematocrit :-

→ Not useful in diagnosis.

→ May remain normal.

→ Serial estimation of Hb is useful.

* Coagulation studies

* Electrolytes

* Urinalysis.

* Blood grouping & cross matching.

*Treatment :-

→ IV fluids.

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