Date:

Inflammation:-

is a Reprotective Response involving host cells, blood vesseles, Protein and other mediators. to eliminate the initial caure of cell injury as wellas necrotic cells and tissues and initiate the process of Repair.

Characteristics:-

① - Inflammation accomplish its mission by first diluting destorying or neutralize harmful agent

② - Healing and Repair of cell at site of injury

③ - Inflammation helps clear infection but also capable of normal tissue

④ - Inflammation is normal and beneficial

⑤ - but Rx very strong, Prolonge or inappropiate lead to chronic Inflammation

⑥ - Inflammation act as innate immunity - Protectic Response

Function:-

- Main Goal is to bring
- Cell - and mole cule of host
- defenre - leukocytes + plasma Protein ↓ to site of infection.

Date:

Cardinal Sign of Inflammation:-

Heat - Calor
Redness - Rubor
Swelling - Tumor
Pain dolor
Loss of fun- function laesa.

Main component of Inflammation:-

①- Vascular Event] activated by mediator derived
②- Cellular Response] from Plasma Protein and cells

Inflammation Induced by Mediators:-

①- Mediators → Produced by Plasma Protein
↓
Induce Inflammation - Response to injury stimuli
Mechanism:-

②- Microbes
↓
enter the cell, does infection and damage
↓
sensed by ①- Macrophages
②- Dendritic cells
③- Mast cells
↳ to secrete ①- Cytokines
②- Mediators
↳ induced Inflammation
①- vascular change
②- cellular change
↓
①- healing → Repair
②- sometime lead → chronic inflammation

Date: _____

Type of inflammation:-

02 type of inflammation.

① - Acute Inflammation

② - Chronic Inflammation

	Acute Inflammation	Chronic Inflammation.
① - Onset	Rapid onset	Slow onset.
② - Duration	Short duration few minutes	Long duration days - Years
③ - Component	contain fluid + Plasma Protein	Contain lymphocytes, Macrophages
④ - "	Predominantly neutrophills leukocyte	Predominantly macrophages
⑤ - Systemic Menifistation	Fever often high	low grade fever
⑥ - out come	Healing Abscess formation	Fibrosis Tissue destruction
⑦ - Cardinal sign	Present	Slightly / Absent

Date: _____

⑧- Blood vesseles	Vasodilation	Proliferation
⑨- Mediators	Histamine Brady Kinin	T-lymphocytes. GFs.
⑩- →	Tissue + fibrosis. Mild and self limited	Tissue - fibrosis Severe + progressive

Acute Inflammation:-

Two component.

① - Vascular changes
② - Cellular changes

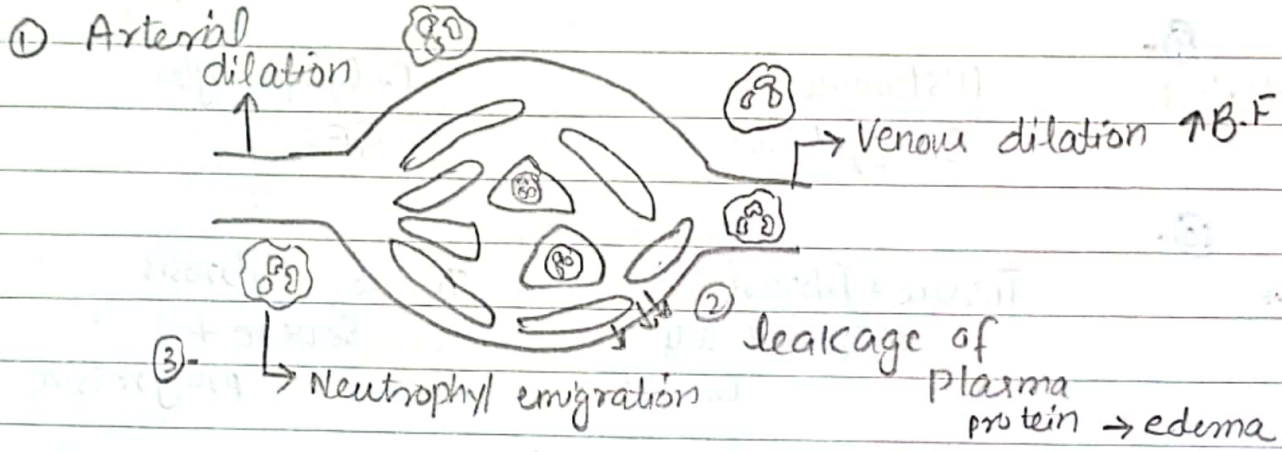
Vascular changes:-

① - Alteration in vessel calibar
↓
↑ Blood flow
↓
Vasodilation

② - ↑ vascular Permeability
↓
allow plasma Protein to leave circulation.

③ - Activation of endothelial cells
↓
↑ adhesion of leukocytes
↓
Migration of leukocytes

Date: _____



Cellular Events:-

①- Emigration of leukocytes from Circulation

↓

②- Accumulation at site of injury

↓

③- Activation of leukocytes

↓

④- Mainly Neutrophills (polymorphonuclear leukocytes)

↓

⑤- eliminate offending agents.

Date:

Stimuli of Acute Inflammation:-

Foreign Bodies	Tissue Necrosis	Trauma	Infection	Immune Rx
↓	↓	↓	↓	↓
Splinters	Ischemia	Thermal injury	Bacterial	Autoimmune Rx
Dirts	Physical injury	Burns	Viral	
Sutures	Chemical Inj	Irradiation	Fungal	
Crystal deposites		Toxicity	Parasitic	

Recognition of Microbes:-

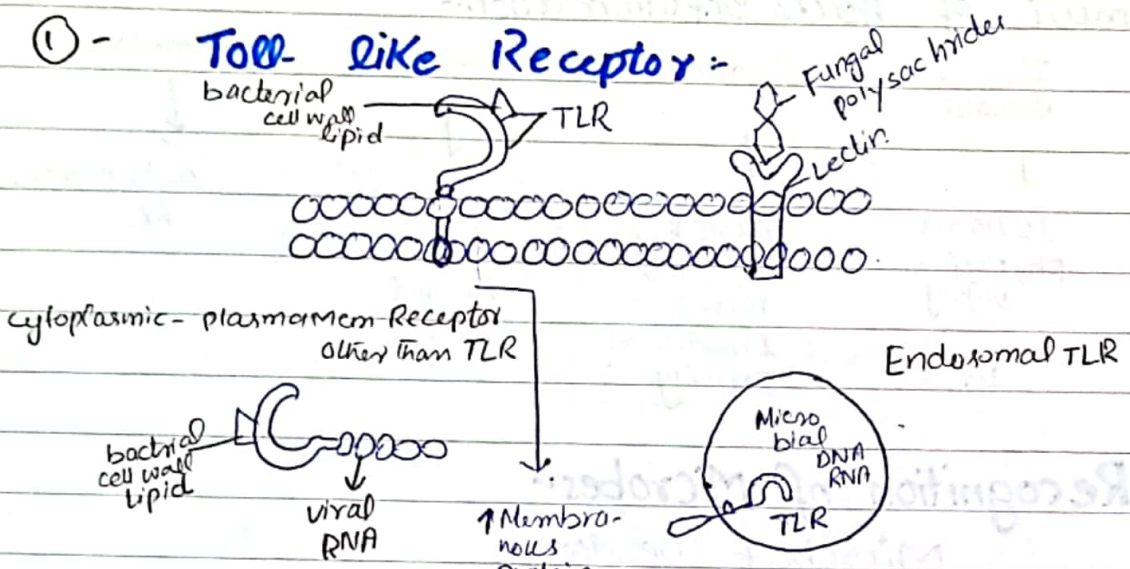
Microbe + Deadcells
↓ send
Danger Signals to
↓
Phagocytes
Dendritic cells
Epithelial cells
↓
Express Receptor
↓
Serve the prerence of infections pathogens + sub-related to dead cells

also called Pattern recognizing Receptor →

Date:

02 Type of Recognizing Receptor.

① - Toll- like Receptor :-



↓

more cytokines.

TNF + Mediator

Releare at extracellular

↓

Blood vessel

↓

↑ adhesion of leukocytes

↓

Site of inflammation

↓

Activation

↓

Acute Inflammation

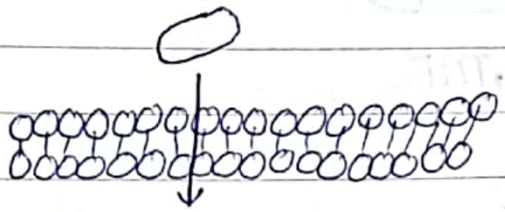
Date: _____

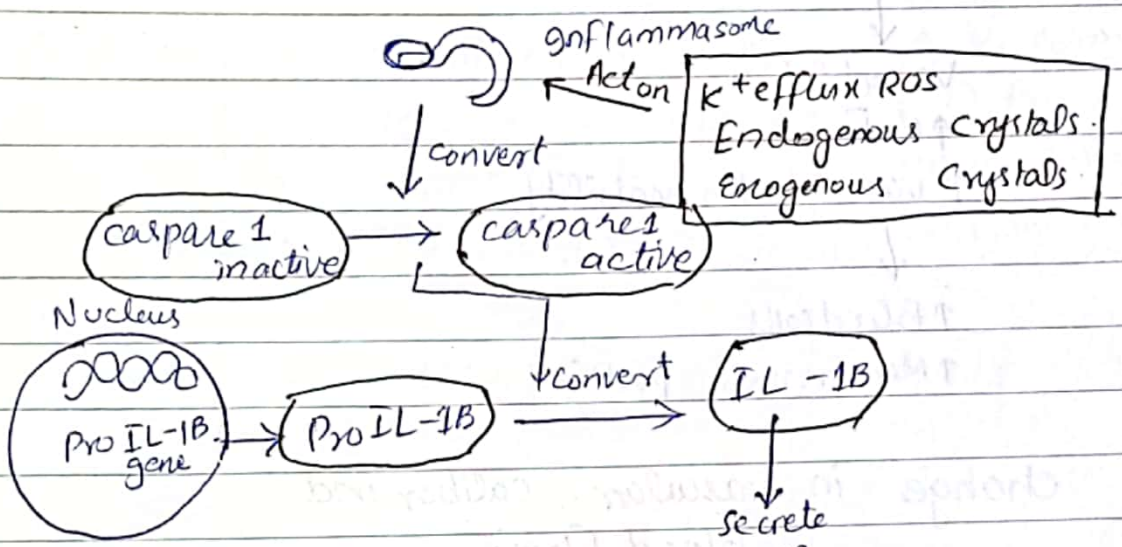
② - Inflammasome :-

Multi protein cytoplasmic complex.

↓ Recognize

Products of dead cells
① Uric Acid
② ATP extracellular





Secrete IL-1B.

↓

Inc adhesion of leukocyte

↓

Acute inflammation

Autoimmunity / Autoinflammatory syndrom:-

↓

Autoactivation of inflammosome.

↓

Releare IL-1B

Gout (urate crystal), cholesbol crystal (atherosaclarosis.) amyloid in brain (Alzahmir disease) Type 2 diabetes obesity

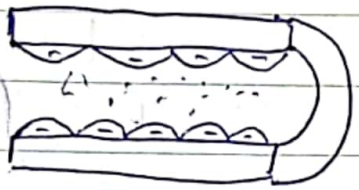
IL-1B antagonists for the treatment of them

Date:

Vascular changes:-

Normally:-

↑ hydro static pres.



↓ colloid Osmotic Pressure

Secreted IL1B., TNF.

↓

Blood Vessel

↓

Vasodilation

↑B.F

↑ vascular Permeability.

↓

↑ Blood cells

↑ Membrenous protein.

① change in vascular caliber and Blood flow:-

Inflamatory Stimulus

↓

Transate Vasoconstriction.

↓

vasodilation

↓

↑ B.Flow

↓

engorgment of down stream capillary bed.

↓

Cawres - Redness. erthyma.
stasis, Margination
Heat
Swelling

① - Stasis :-
small dilated vessel
e- RBC

② - Margination:-
Leukocyte accumulate vascular endothelial surface

Date: _____

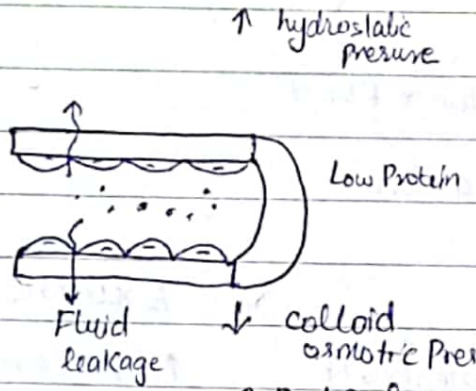
②- Inc Vascular Permeability:-

↑ vascular Permeability
↓
movement of protein + fluid
↓
Two Type of action

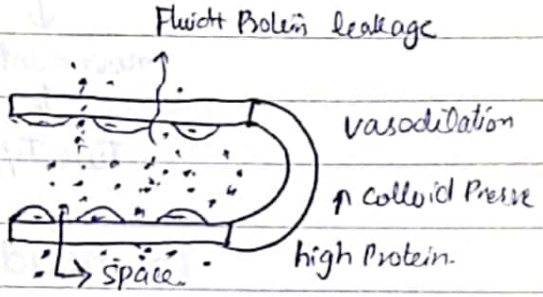
	Transudate	Exudate
Defination:-	Inc vascular Permeability leads to ↓ osmotic Pressure ↑ Protein loss. ↑ hydrostatic Pressure	↑ V. Permeability ↑ O.P. Damage serous membrane out flow of water + Protein
Edema	No inflamatory edema	Inflammatory Edema.
Protein	Low protein Content	High protein Content.
Coagulant	No tendency to Coagulate	Coagulate.
fibrinogen	No fibrinogen	Fibrinogen ↑ content.
LDH	Low LDH	high LDH
spaces:	Absent	Interendothelal space Present
leak mol.	Fluid leakage	Protein + fluid leakage

Date: _____

Nephrotic Syndrom
Pulmonary embolism



Malignancy
TB.



Cause of ↑ Vascular Permeability:-

① - Endothelial Cell Contraction:-

histamine
bradykinin
mediator
↓
endothelial cell contraction
↓
Short lived (15-30min)
Gap form

TNF & Interleukin
↓
cell contraction
↓
long liver
(4-6 hour)
↓
24 hour.

② - ↑ Transcytosis

③ - Leakage of New B.V.

Date: _____

② - endothelial cell injury:-

Cell Necrosis

direct indirect

↓

Begin after 2-12 hours

cause ↑ vascular Permeability

Cellular Event:-

02 Steps.

① - Leukocytes Recruitment → in ECF

② - leukocytes Activation.

cause acute inflammation

but it may prolong to damage other normal cells.

① - Leukocytes Recruitment:-

Four steps.

① - Margination and Rolling along vessel wall.

② - adhesion to endothelium.

③ - Transmigration b/w endothelial cell.

④ - Migration towards chemotaxis.

(a) Migration and Rolling:-

02 sub steps

ⓐ Migration

ⓑ - Rolling.

Date: _____

(a) **Migration:- / Margination:-**

Blood from capillaries

↓ entering

post capillary venule

↓

Cause swept process of circulating cells

↓

(a) RBCs are small.
They move faster
They are pushed away from endothelium

(b) Leukocytes large in size
More less faster
Pushed away towards endothelium
so that interaction is easily. → Margination

(b) **Rolling:-**

Cytokines and other mediators activates the endothelial cells which express adhesion molecules or Receptor or ligand to which leukocyte is loosly attach and detects again & again
Rolling of leukocyte starts.

Date:

Rolling is mediated by selectin

Receptors on endothelium and leukocytes contain EC domain for binding sugar.

Types

E selectin :- Receptor on endothelial cell
CD62E

P selectin Receptor on platelet endothelium.
CD62P

L-selectin Receptor on surface of mast leukocytes
CD62L

They are in low amount - unactivated form
but when stimuli come -> activate in large amount

Histamin
Prothrombin
↓
P selectin
↓
Facilitate leukocyte binding due to Rolling phenomena

Cytokine TNF
IL-1
↓
E-L selectin
↓
Facilitate leukocytes Binding due to rolling phenomena.

Date:

② Adhesion:-

Rolling leukocyte
↓
Initiate
↓
Adhesive
↓
mediated by integrins

① - Transmembrane Glycoprotein

② - Types
- ICAM
- VCAM

binds to leukocyte function associated Antigen 1
Macrophage 1 antigen.

Binds to VLA-4.
very Late antigen 4

③ - mediate adhesion

④ - Show less affinity when inactivated.

⑤ - high affinity when activation of lymphocytes

chemokines
↓
Leukocytes
↓ activated
Conformational change in affinity of integrins on leukocytes.
↓
Recieve signal for attachment

TNF-IL1
↓
endothelial integrin ligands
↓
↑ expression of ligands for integrins.
↓
send signal for attachment

firm adhesion.

Date: _____

③- **Transmigration :-**

Leukocyte migrate through vessele wall
↓
By sequeezing b/w cells at I/C Junction
↓
called ~~diapup~~ di'pedes.
↓
after dipedes leukocytes Release
↓
collagenases.
↓
help them to pass through vasculer membrane
↓
extra vasation of leukocytes
↓
move toward site of infection along. e- chemical gradient called chemotaxis.

④- **Migration towards chemotaxis:-**

Exogenous ← Bacterial Product
N-formyl meth

endogenous
Cytokines.
(chemokines family)
Products AA.
LTB4

Chemotactic Molecules
↓
Bind + Receptor.
↓
Release cytoskeleton contractile element

Leukocytes move by extending pseudopods
↓
anchor ECM
↓
pull leukocytes
↓
direction depends on chemokines Receptor

Date:

In acute inflammation
6-24 hrs. Neutrophil - Short lived
Replace by Monocytes - longer

Leukocyte Activation:-

Leukocytes at reached
at site of microbe
or pathogen.
↓
Activated
↓
Perform following function.

Phagocytosis of particals
Intracellular Destruction of phagocytosed microbes
Liberation of substance that destroy extracellular microbes and dead cells.
Production of mediators.

Phagocytosis of Particals:-

① - Leukocyte bind to Micro-organism throug Receptor
↓
Recognize
↓
component of Microbes / dead cells
opsonines

Date: _____

② - Microbe also have coating by phenomena of opsonization
↓
③ - Recognize by Receptor and engulf by leukocytes

④ - Most imp opsonins.
↓
Antibodies of immunoglobins
q IgG.
↓
Bind to mitrobial surface antigen
↓
Breakdown the product of complement protein C_3

Plasma Carbohydrate - binding Lectin.

⑤ - Fc receptor for IgG (FcγRI)

Complement Receptor 1, 3 } for compliment fragments

$C1q$ → collectins.

Binding of opsonized particals → Receptor → engulfment
↓
↑ cellular activation
degradation of Microbes
formation of phagocytic vacuole
attach to lysosome → Phagolysorome

Date:

Morphological Changes:-

There are Four morphological changes.

1. Serous Inflammation
2. Fibrinous Inflammation
3. Suppuralive Inflammation.
4. Ulcer.

①- Serous Inflammation:-

①- Outpouring of watery fluid with less protein.] depend on site of injury

②- Secretion from.
- ①- Plasma
- ②- Mesothelial cell lining — Pleura, Pericardiam, Peritonium.

Fluid in serous ~~sac~~ cavity → effusion

③- Accumulation of serous effusion either within epidermis. beneath the epidermis of skin

Example:-

①- Viral Infection
②- Skin blister.

Date:

Fibrinous Inflammation:-

→ Severe Injury
↓
↑ Vascular Permeability
↓
allow large molecule to pass through the endothelial Barrier (Fibrinogen)

→ Inflammation of Body cavity such as meninges, pleura and Pericardiam

→ Exudate can Remove by Macrophages
↓ Fibrinolytic.
lead to Resolution

→ If more fibrin occure
↓
No Resolution
↓
Organization occure
newly B-Formation
Fibroblast
Scar formation

Example:-

Fibrinous pericardium Inflammation.

Date: _____

Suppurative Inflammation:- purulent..

Pus collection (Neutrophills, necrotic cells, and edemaf fluid)

Caure by pyogenic (pus forming) such as staphylococci.

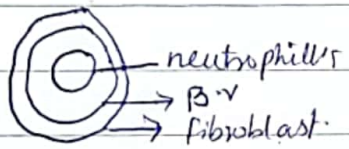
Abscesses formation

↓

Local collection of pus.

have central large necrotic Region

↓



Rimmed by preserved neutrophills

↓

surrounding zone of Blood vessels

↓

surrounding by fibroblast prolifration

↓

Scar formation.

Ulcer:-

Local defect or extravation of the surface of organ.

↓

occur due to necrosis of tissue

" Inflammation near a surface

Commly occur in GIT
Urinary Tract
Subcutaneous tissues of low extremities of old people

Scar-formation + accumulation of Lymphocytes Macrophages.

Peptic gastri ulcer.

Date: ✓

Q:-

Define Inflammation?

"Inflammation is protective Response involving host cells, blood vessels, Protein, and madiator. to eliminate the cause of injury as well as necrotic cell. and process of repair start."

① → In this process dilution of harmful agent destorying

② - Repair and healing occure

③ - It is beneficial Rx and normal

④ - act as innate immunity.

⑤ - If prolong cause chronic inflammation

Q:-

Briefy mention of morphological Pattern of acute inflammation?

There are four pattern of acute inflammation

① - Serous Inflammation

② - Fibrinoid Inflammation

③ - Suppurative Inflammation

④ - Ulcer.

Date: ✓

①- Serous Inflammation:-

①- Outpouring of watery fluid
e- less protein

②- Secretion from ·) Plasma
·) Mesothelial cells linning

Fluid- effusion
①- · pleura
②- · pericardiam
③- · peritonium

③- Accumulation of fluid within epidermis

Example.
Viral Infection
Skin blister ✓

②- Fibrinous Inflammation:-

①- Severe Injury — large protein leakage
②- ↑ Vascular Permeability
③- Large molecule fibrinogen pass the membrane ↳ into tissue.
④- Fibrin is form
⑤- Deposite in EC space
⑥- If fibrin is not removed Then
Scar formation ocure

Example:-
Ocure in body cavity of · Meninges
· Pericardium

Date:

③- Suppurative Inflammation:-

①- Pus collection (Neutrophils, debris of edema, necrotic cell)

②- Infection due to bacteria (staphylococci) cause liquefective tissue Necrosis.

③- Infection due to pyogenic bacteria

④- Abscesses formation

⑤- Center → neutrophyll surrounded by blood vessele + fibroblast

Example

Acute appendicitis.

④- Ulcer:-

①- Local defect + extravasation of surface of organ and tissue produced by shedding of necrotic tissue

②- Prerent in mucosa of mouth, stomach, intestine or GIT

③- and also in skin or sabaceous tissue.

④- Scar formation

⑤- Accumulation of Macrophages and lymphocytes

Example :-

Peptic gastric ulcer.

Date:

Q:- Chemical Mediators of Inflammation :-

Date:

Liver

Plasma

Protein Plasma Activation

Complement activation → Leukocyte activation, direct killing (MAC)
$C3a, C5a, C3b$ (Anaphylatoxin) target
$C5b-9$ Membrane attack complex
Vasodilation

Plasma

Factor XII activation → (Bradykinin) Kinin → Vascular Permeability, Smooth muscle contraction. Vasodilation, Pain

Plasma

Protein activation during coagulation → Endothelial activation Leukocyte recruitment

niceday

Date:

Q:- Describe vascular changes:-

Vascular Changes:-

Two changes

① - Vasodilation

② - Inc Vascular Permeability

① - Vasodilation:-

① - Vasodilation (TNF, IL)

↓

② - ↑ Blood flow

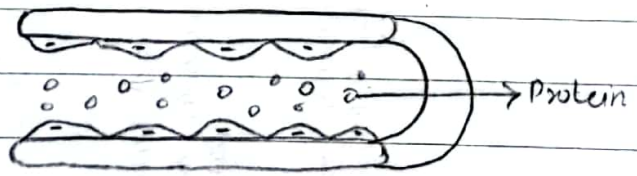
↓

③ - engorgment of down stream capillary bed

↓

④ - Cause Redness
Stasis
Heat
swallowing
loss of function

② - Inc Vascular Permeability:-



① - Increare Hydrostatic prerrure

② - Decreare Golloid osmotic Prerrure

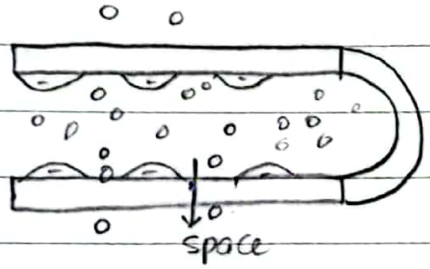
③ - Retraction of endothelial cells by histamine

④ - and injury cause by burn

niceday

Date:

(i) Exudate:-



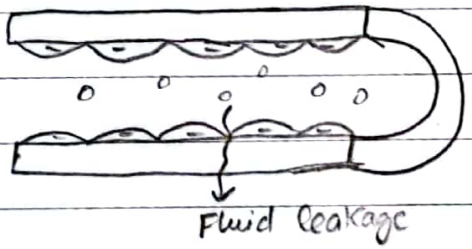
Example

① Malignancy
② Tb

① - Fluid - Protein leakage ✓ ✓
② - Vasodilation
③ - Stasis
④ - Increare endothelial Space ✓
⑤ - high Protein Content ✓
⑥ - ↑ Fibrinogen content
⑦ high LDH ✓
⑧ Damage serous membrane

coagulation inflam. Adema

(ii) Transudate:-



Example:-

① - Nephrotic Syndrome
② - Pulmonary embolism

① - Fluid leakage
② - ↓ osmotic colloid Pre.
③ - dec Protein synthesis
④ - Inc Protein loss
⑤ low Protein content
⑥ - ↓ Fibrinogen
⑦ - ↓ LDH low
⑧ - ↑ hydrostatic Pressure

Q:-

Date:

Cellular Changes:-

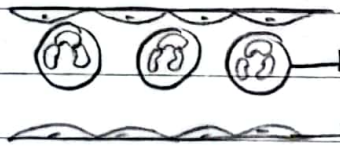
Steps of cellular changes

① - Margination
② - Rolling
③ - Adhesion
④ - Trans migration

Margination:-

① - Blood enter in vanules.
↓
② - Leukocyte large in size that's why ot pushed towards endothelium.
↓
③ - leukocytes align in a straight pathway.

RBC small size away from endothelium



Rolling:-

① - Rolling is mediated by Slectin here integrin in low affenity state.

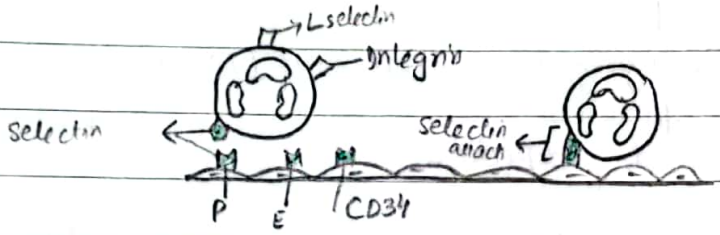
② - Selectin Receptor on endothelial
Ligand on leukocytes.

③ Three type of selectin

E-selectin L-selectin

Pselectin.

Date:



E - selectin- Receptor on endothelial cell CD62E

P- selectin Receptor on platelets endothelium CD62P

L- selectin surface of Mast leukocytes. CD62L

③- Adhesion:-

①- Adhesion is mediated by integrin here integrins in high affinity state.

②- Integrin Receptor on leukocyte but ligand on endothelium.

③- Two type of ligand of integrins

①- (ICAM - 1) - bind to leukocytes associated e- Antigen MAC1

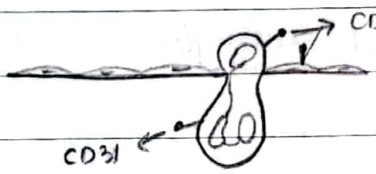
②- (VCAM) bind to VLA - 4 - Antigen 4..

Date:

④- Transmigration:-

①- Leukocyte migrate through vessele wall
↓
by squeezing b/w cell-and I/C junction
↓
called dipediesis.

②- This is occure due to the CD31 dimmer



dipedesis.

③- CD31 hom type interaction

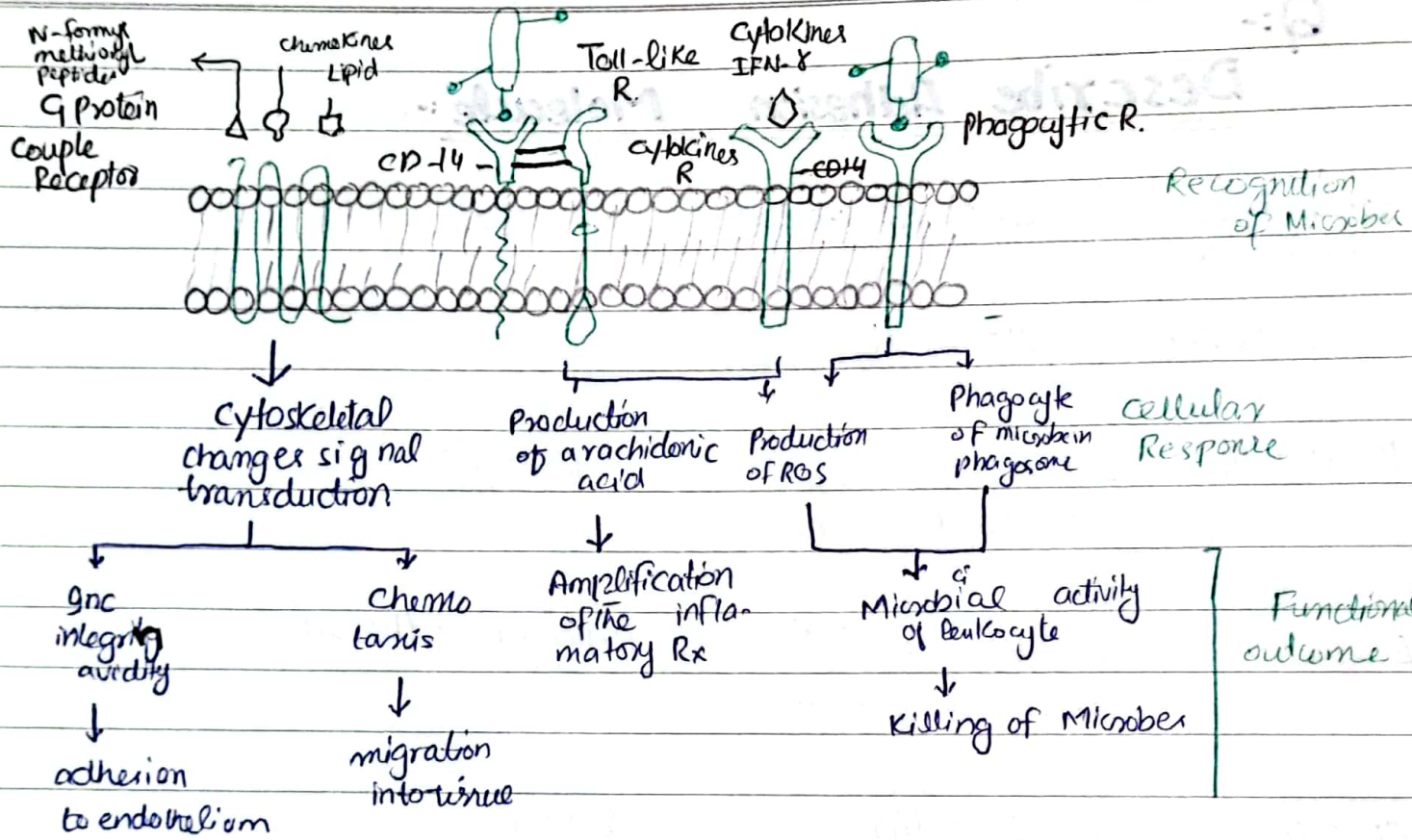
Date: _____

Q:-

Describe Adhesion Molecule:-

Endothelial Molecules	Leukocytes Molecule	Role.
Selectins	Selectin Ligands.	
P-selectin	Sialyl-Lewis X modified proteins	Rolling
E selectin	Sialyl-Lewis X modified proteins	Rolling + adhesion
CD 34	L-selectin	Rolling
Integrins	Integrin Ligands	
ICAM-1	CD11/CD18 MAC 1, LFA-1	Adhesion
VCAM-1	VLA-4	Adhesion
Others		
CD31	CD31 homotypic	Tran diapedesis Migration of leukocytes

Date: _____



Q:- **Leukocyte Activation:-**

Date: .

Q:- Chemotaxis:-

Define:-

"Movement of leukocytes from vessele lumen into a damage area is called chemotaxis."

Process:-

Extravasation of leukocytes.

↓

move towards site of infection along e- chimical gradient called chenotaxis.

↓

can be exogenous

① - Bacterial product
(N-formyl methionine Termini)

② - Cytokines
(chemokines)

③ - Product of AA
(LTB4)

endogenous.

Date: _____

Chemotaxic molecule

↓

Bind Receptor

↓

Release cytoskeleton contractile elements

↓

Leukocyte move by the formation of pseudopods

↓

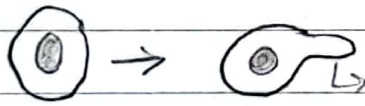
anchor ECM

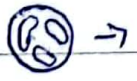
↓

Pull the leukocytes

↓

direction depend on chemotaxis Receptor



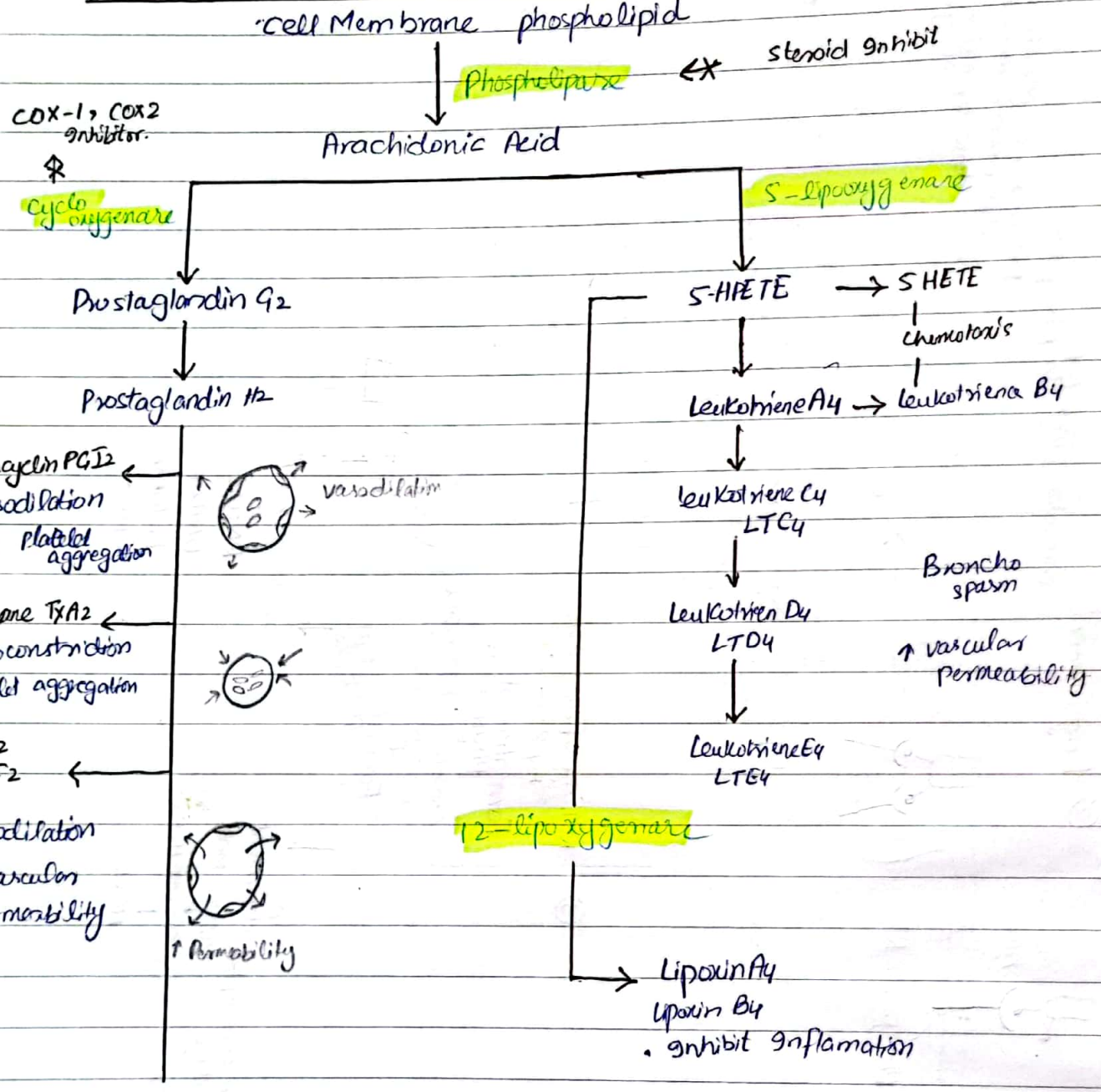


Q:- Phagocytosis:-

Date: _____

Date:

Q:- Production of Arachidonic Acid Metabolites:-



Date:

Q:- Outcome of Acute Inflammation:

Date:

Q:- Define CytoKines:-

Define:-

"Group of low MW - polypeptide / protein which are secreted by activated immunocytes or some matrix cell and possess high activity and function:"

→ Regulate Immune Response

Cytokines. **Functions:-**

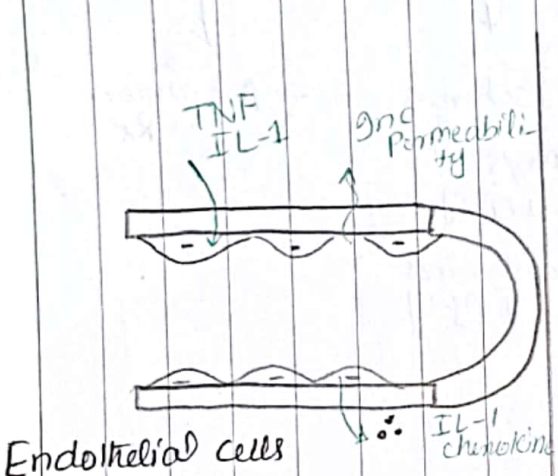
In acute Inflammation:-

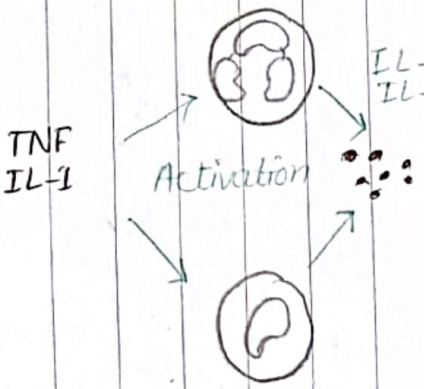
TNF	(Macrophage/Mastcell) stimulate expression of endothelial adhesion and secretion of other cytokines.
IL-1	(Macrophage) stimulate TNF (Role in Fever)
IL-6	(Macrophages) acute phase Response
ChemoKines	(Macrophages, Tlymphocyte, Mastcell) Recurritment of leukocytes. migration of cells in normal tissue
IL-17	(T-lymphocytes) Recruitment of neutrophills & Monocytes

IN CHRONIC INFLAMMATION:-

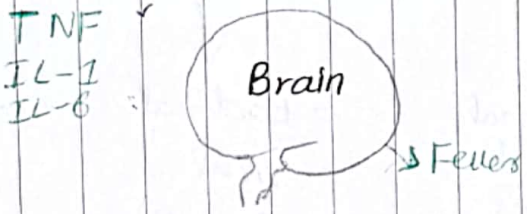
IL-12	(Dendritic cell, Macrophage) Inc Production of IFN-δ
IFN-δ	(T-lymphocytes NK cell) Activation of macrophages Kill microbes - tumor cells
IL-17	(T-lymphocytes) Recruitment of neutrophill - monocytes

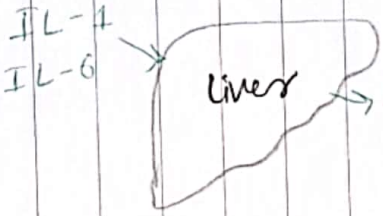
Local Inflammation

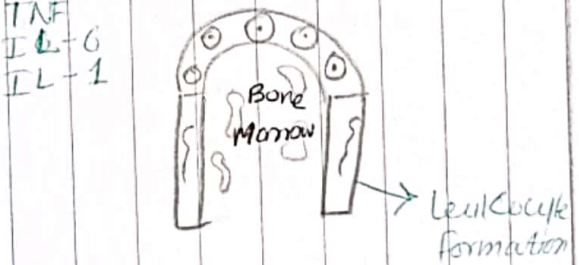




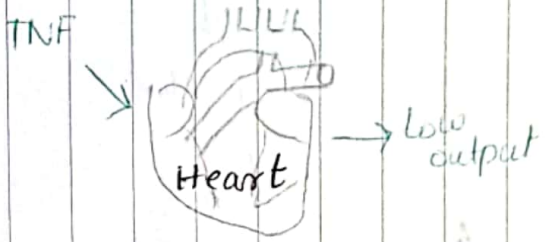
Systemic protective effects

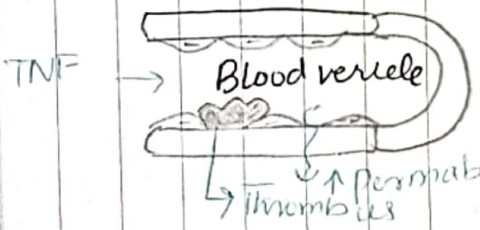


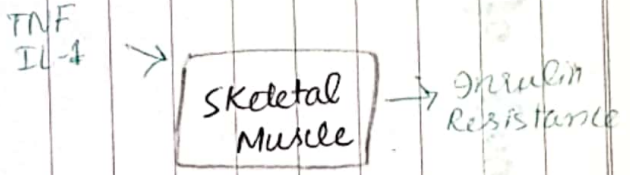




Systemic Pathological effect





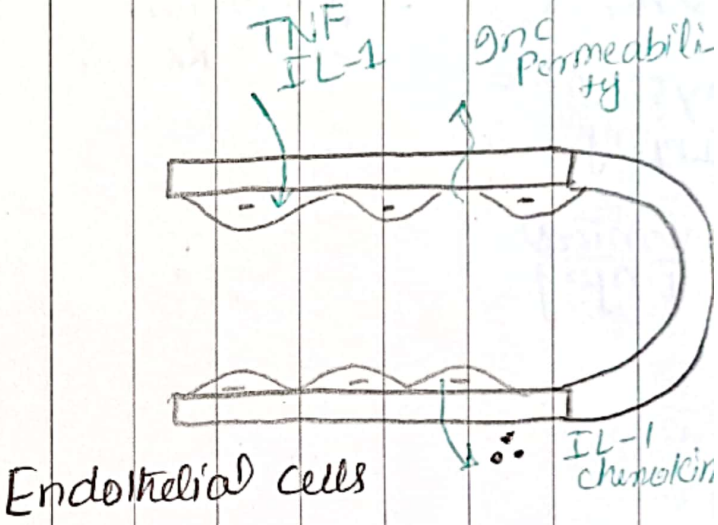


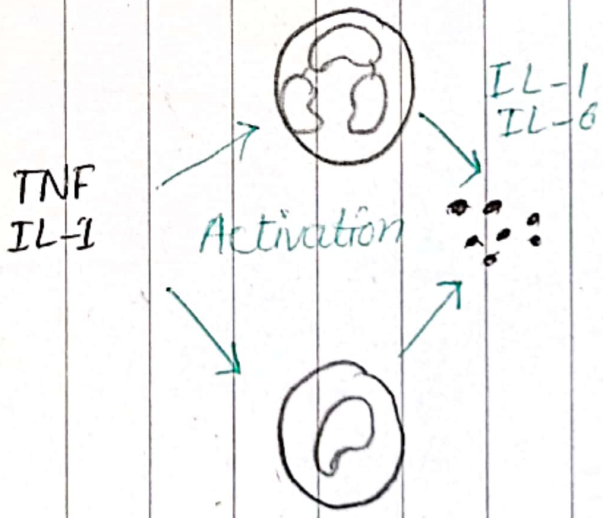
Role of cytokines in acute inflamation:-

niceday

Date:

Local Inflammation





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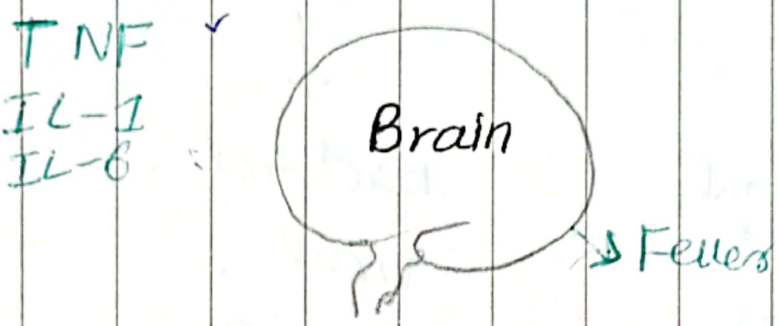
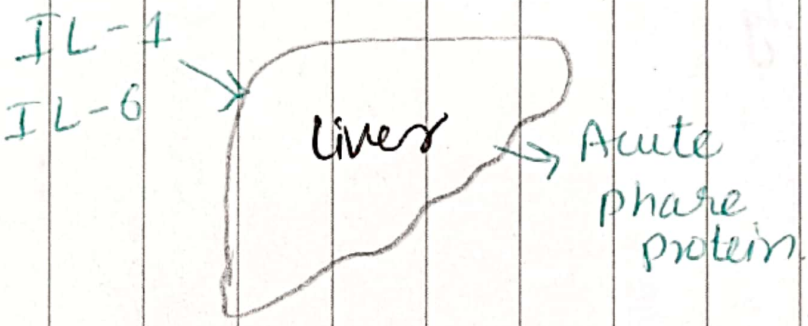
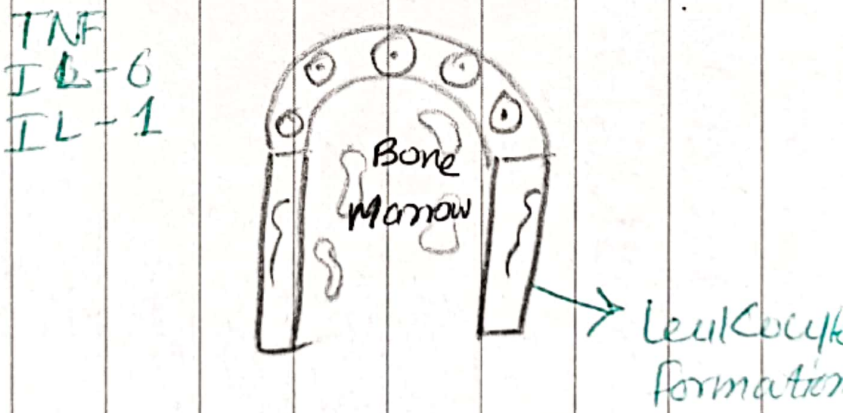
Systemic protective effects

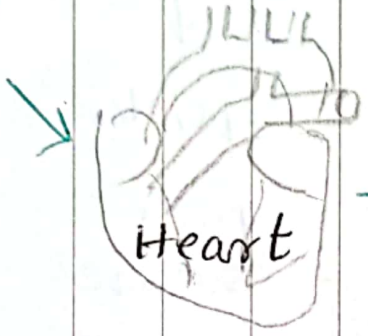




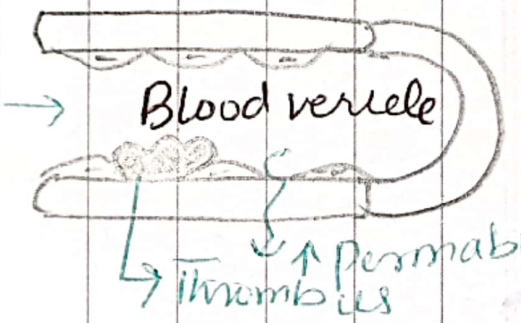


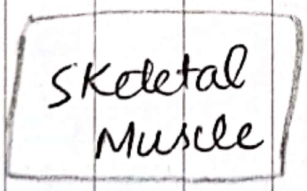
Role of cytokines in acute inflamation:-

Systemic Pathological effect



→ Low output





→ Insulin Resistance

Date: _____

Date:

Stimuli of inflammation:-

- Foreign Body
  - Splinters
  - Dirts
  - Sutures
  - Crystal deposits
- Trauma
  - Thermal injury
  - Burns
  - Irradiation
  - Toxicity
- Tissue Necrosis
  - Bacterial
  - Viral
  - Fungal
  - Parasite
- Infection
- Immune Rx
  - Autoimmune Rx
- Ischemia
- Physical Injury
- Chemical Injury

Date:

Ques 1:-

① - Pain in Right lower quadrant 12 hr
Appendicetomy. Performed
Neutrophill in wall of appendix

Ans:-

~~Pla~~ Pathology:-
(Acute Appendicitis) acute suppurative inflammation

sign- symptoms:-

① - Loss of appetite
② - Dull pain near naval
③ - Naurea
④ - vomting
⑤ - Abdominal Swelling
⑥ - feuer. - 99-102
⑦ - ↑ WBC
⑧ - Anorexia
⑨ - Tendrness at MucBurny points.

→ Pus formation in center
Then neutrophill
and Then surounded by
→ dilated Blood vessele and
fibro blast

Ques 2:-

12 year old boy injury a tender, Red. hot swelling developed at local site.
Trace sequence of event of this Picture.

Ans:-

Redness . tumor
loss of function pain
heat

} symptom of acute inflammation

Date:

→ Sequence of event of acute inflamation

Notes

Ques 3:-

cellular exudate form in acute inflammation

Ans:- (a) What factor attract WBC out of Blood Vesseles

Chemokines.

act on WBC and stimulate cell to migrate through spaces towards chemical conc-gradient

Chemokines IL-8

CD 31.

(b) What are the object of their exudation out of vessele-? how do they achieve this objectives

Phagocytosis process. — Notes

Q No4:-

CBC Test of 22 yearold student abnormal Leukocyte count

Weeks complain of sneezing - and watering eyes in spring- summer.

Which is cell type increase

Ans:-

Inc Releare of histamine in Responce of allergic substance from Mast cell- and basophills which caure ↑ eosinophills

Date:

Q NO 5:-

25 old man Red hot fluctuant swelling in arm after recieving IM-injection.

local fluid exudate formation

Describe mechanism of fluid exudate formation

Ans:-

Major Three Component

① - Vasodilation

② - ↑ Vasular Permeablity

③ - Emigration

Explain Vasular Permeability Notes

Q NO6:-

30 old house wife , skin blisler e- serous fluid Resulting from burn.

Name the morphological Pattern.

Ans:-

Serous Inflammation

Q NO7:-

3 Major Component of acute inflammation

Ans:-

Vasular

Cellular

Humoral - Plasma proteare

Platelets activate factor

Arachidonic Acid

Date:

Que. 8:-

20 year old patient
Appendicitis.
Pain in R iliac fossa.
Appendex Removed (Red. swell)
show edema.
Enlist mechanism cause edema

Ans:-

Change in Vasular flow.
Vasodilation by histamine
" " NO.
Vascular Permeability
Space formation
Endothelial injury - Leakage of fluid

C5a, C3a inflammation
C3b Phagocytes
MAC Microbe lysis
Fever IL-1 - TNF
Pain PG, Bradykinin

Date:

Difference b/w

Acute Inflammation.	Chronic Inflammation.
① - Defination	
Rapidly reponding Protective mechanism delivering of neutrophills to site of infection for clearing the invaders and initiating the process of necrotic tissue	Inflammation of prolong duration in which tissue injured. and attempts to repair.
② - Major cell involve	
Neutrophills Macrophages	Mononuclear cells Monocyte Fibroblast
③ - Onset	
Immediate Onset	Delayed
④ - Duration	
Few days	up to months.
⑤ - Systemic Manifestation	
High grade fever	Low grade fever
⑥ - Cardinal Sign	
Present	Absent

niceday

Date:

⑦- outcomes

Healing
Pus formation

Fibrosis
Tissue destruction

⑧- Blood Vesseles

Vasodilation

Proliferation

⑨- Mediators

Histamine
Brady Kinin

T-lymphocyte
GFs

⑩- Vascular change

Vasodilation
↑ vascular Permeability

New Vessel formation

Differentiate b/w

Date:

Transudate	Exudate
① O.P	
↓ osmotic Pressure	↑ osmotic Pressure
② Feature	
↑ Protein loss ↑ hydrostatic Pressure	Damage serous membrane
③ Edema ✓	
No inflammatory edema	Inflammatory edema
④ Protein	
↓ Protein Content	↑ Protein content
⑤ Coagulation ✓	
No tendency to coagulate	Coagulation
⑥ Fibrinogen	
No fibrinogen	Fibrinogen content
⑦ LDH	
low LDH	high LDH
⑧ Spaces	
Absent	Intercendothelial space present

Date:

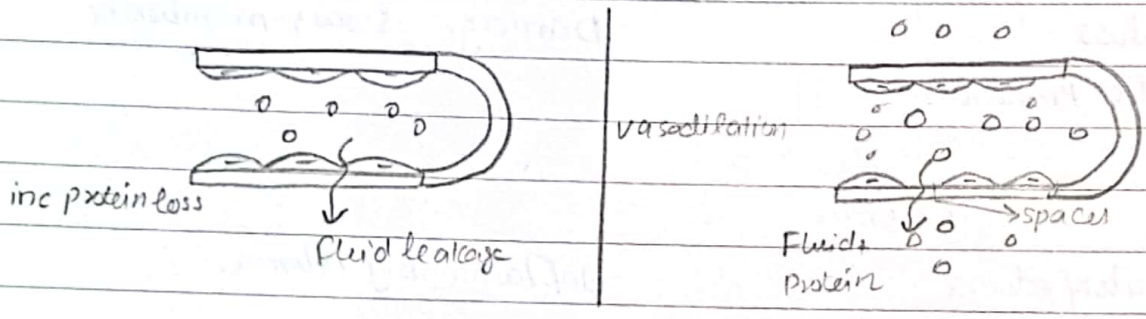
@- Leakage

Fluid leakage	Protein + fluid leakage

⑩- caure

Nephrotic Syndrome Pulmonary embolism	Malignancy Tb

⑪- Diagram



Date:

Chronic Inflammation:-

Q:-

Define:-

It is a Response of prolonge duration (months) in which inflamation, tissue injury. and then Repairing ocurse

Prolong duration. delay onset. fibrinous form Mono nuclear cell infiltration Tissue damage Attempt at healing.

Cause:-

1- Persistant Infections.
2- Hypersenstivity disease
3- Prolonged exposure to potentially toxic agents
4- either exogenous- endogenous
5- Immune- Mediate Inflammatory diseases

★ 1- **Persistant Infections:-**

i- by microorganism that are diffiault to eradiccate
such as mycobacteria, fungi, Virus, Parasites] They may evolve immune Rx → delayed Type hypersenstivity

2- Sometime acute bacterial Inf of lung ↓ progressive to chronic lung disease

★ 2- **Hyper Senstivity Disease**

1- Chronic Inflammation play imp role in disease which is cause by excessive abnormal activation of immune system

Date: _____

② - Auto immune diseare Ocurse
On this auto antigen evoke
a self-perpelvating immune
Rx that result chronic
tisrue damage

Example :-

- Rheumatoid arthritis
- Multiple sclerosis.
- Microbes - Inflammatory bowl Syndrome/diseare
- Allergens - bronchial asthma

★ ③ - **Prolonged exporure to potentially toxic agents either exogenous- endogenous :-**

① - Exogenous agent → silica non degradaable
Inhale for long time caure lung Diseare Silicosis

② - Deposition of cholestrol +lipid in the wall of arteries
caure Atherosclerosis.

★ ④ - **Some other disease :-**

Neurodegenrative diseare
↳ Alzheimes Diseare
Metabolic Syndrom
Type 2 diabetes
Cancer

Date:

Morphological Feature :-

There are three feature

(1)- Infilteration e- mononuclear cells
include Macrophage
Lymphocytes
Plasma cells

(2)- Tissue destruction.
induced by inflammatory cells.

(3)- Attempt at healing.
by Connective Tissue replacment
Damage Tissue
Angeogenesis.
Fibrosis

Date:

Role of Macrophages:-

① - Dominent cell.
② - secrete cytokines and Growth factors.
③ - act on various cell
④ - destory foreign invader. and tissue
⑤ - by activating other cells - like T-lymphocytes

Function:-

① - Macrophages is phagocytes that act as filter for participlate matter, microbs.
② - Also eliminate microbes in cellular and humoral immune Response.
③ - also role in inflammation and Repair

Macrophage feature.

① - Macrophages derived from hematopoietic stem cells in bone Marrow and progenitors in embryonic yolk sac and
② - fetal liver during early development
③ - Circulating cells → Monocyt

Q:-

Reticuloendothelial System :-
also called Mononuclear phagocyte system.

① - In liver - Kuffer cells
② - Spleen- lymph node - Sinus histiocyte
③ - CNS. - Microglial cells.
④ - Lungs - alveolar Macrophages

Date:

Two Path way

① - Classical Macrophage activation

② - Alternative macrophage activation

nice

Phagocytes
Kill microb
Inflamation
Repair
Scar form
fibrosis

Date: 6

secrete mediator

Function of Macrophages:-

① - Macrophage is a phagocytes to eliminate and ingest Microbes and dead cells.

② - Macrophages Initiate the process of tissue Repair

③ - Macrophage involve in formation of scar and fibrosis.

④ - Macrophages secrete mediator of inflammation such as cytokines (TNF) ecosanoid

⑤ - Macrophage display antigen to T-lymphocytes and respond to signals from T cells help in (cell mediate immune Responses).

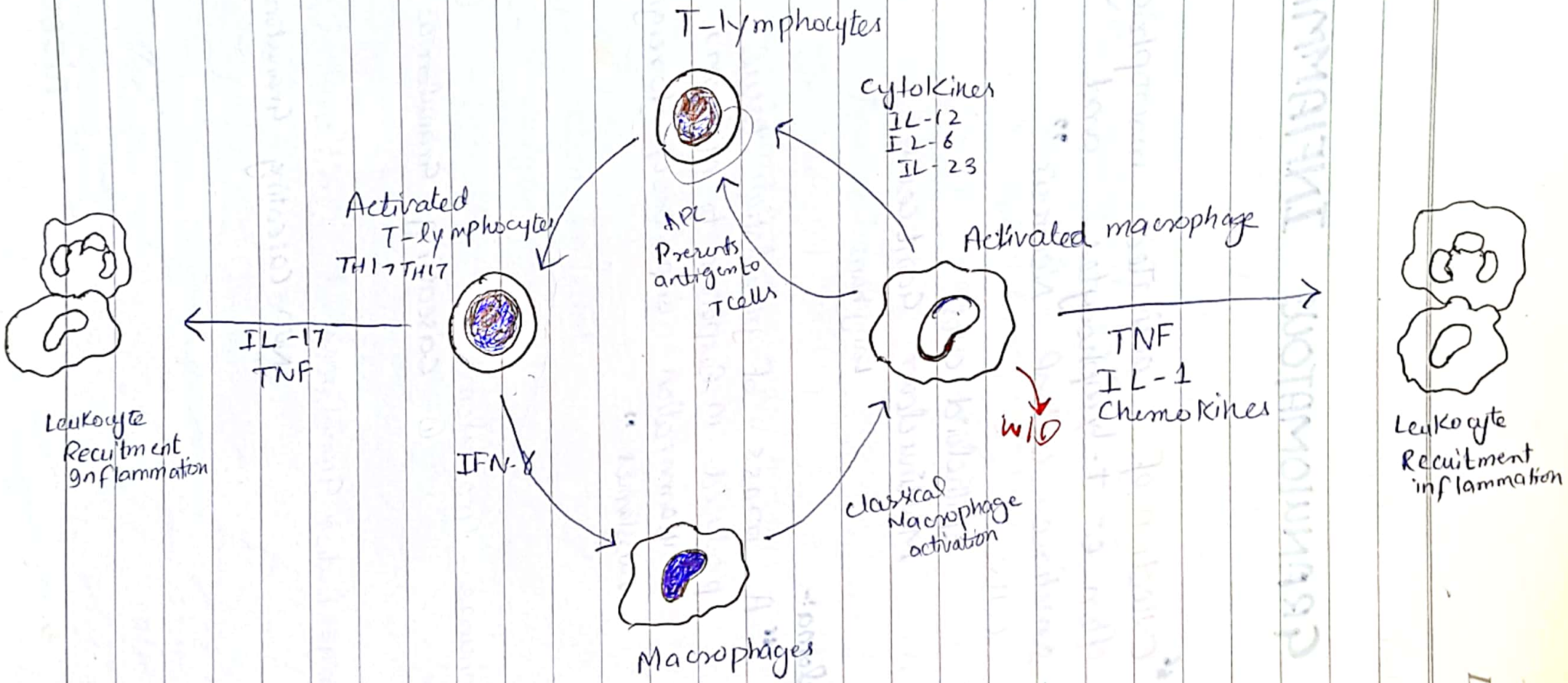
Role of Lymphocytes :-

Microbes activate T-Lymphocytes - B-Lymphocytes. which amplify and propagate chronic inflammation defense against infectious Pathogens.
Chronic inflammation → granulomatous inflammation.
Seen in autoimmune direare. and hypersensitivity direare

TH1 Cell produced cytokines IFN-γ which activate macrophage by classical Method

TH2 cell secrete IL-4, IL-5 and IL13 activate eiosenophills alternate Pathway for Macrophage activation

TH17 cell secrete IL-17 and cytokines induce secretion of chemokines for reciting neutrophills



Macrophage - lymphocyte interaction in chronic inflammation

Date: / /

niceday

Date:

MCells of lymphocytes.

B-lymphocytes, Mast cell, Neutrophills

GRANULOMATOUS INFIAMMATION:-

Define:-

"Collection of activated macrophages often e- T-lymphocytes and sometime central Necrosis"

Cells

a) Epitheloid cells

b) Multinuclear Giant cells.

Lang-hans

Granuloma:-

"A mass of granulation tissue produced in Response to infection, inflammation or prerence of foreign substances"

Type:-

(•) Immune Granuloma

① - Caseating Granuloma

(•) Forign Bodies Granulomas

Non Caseating Granuloma

Date:

Types of Repairing:-

Two types
- ✓ Healing of skin wound
- ✓ Fibrosis in the injured parachymal cells

①- Healing of skin Wound:-

Two step.
- ⊙- Epithelial regenration
- ⊙- Formation of C.T scan

Type of healing
①- Primary healing
②- 2nd intention, 2ndary healing

First intention:-

①- clean - uninfected
②- surgically incised
③- With less cell and tissue loss
④- Edges of wound are approximated by surgical sutures
Scanty granulation, Neat linear scar

①- Initial hemorrhage + Hemostatic Function:-

Incison
↓
destruption of epithelial membrane
Death of epithelial cell
Death of connective tissue

Date: _____

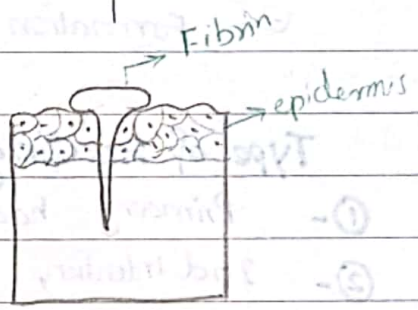
Scar formation
↓
Minimal Wound Contraction

space fill e- blood
↓
Blood clot
↓
Hemostatic function

② Acute inflammatory Responre:-

9n 24 hrs.

Neutrophils arrived at injury site
↓
Migration towards clots



fibrin plug joined the edges

9n 3rd day

Neuthophil Replace by Macrophages

③ Proliferation:-

① - Epithelial changes:-

24 hrs	Mitotic activity of basal cells is inc.
24-48 hrs.	epithelial cell migrate.
	Dermis proliFrate
	epidermal layer forming.
03 days.	Thick epidermal form
05 days	Malure epidurmal form e- Kretinization.
5day - 2week	Angiogenesis.

Date: _____

② - **Fibroblasts cells:-**

Prolifration

↓

Migrate to the sile of injury.-

↓

Collagen fiber deposition

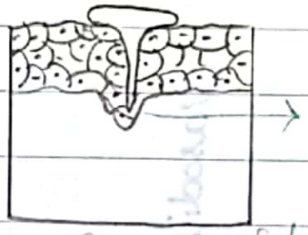
↓

scar formation

3 days collagen at margins.

5 day Aburdant collagen

2nd week Collagen accumulation and Blanching process



Regrowth of basal epidermis

③ - **Granulation Tissue**

Fibroblast + loore CN + New B.V.

combined

called granulation tissue

3 days- granulation invade in space of wound

④ - **Organization:-**

3 day- fibroblast invade in wound

5 day collagen bridges. The wound

→ fibrous union

Restortion of intact skin

⑤ **Remodeling:-**

after 2-3week Capillary remodling

↓

3- Months. Recruitment of preendothelial cells

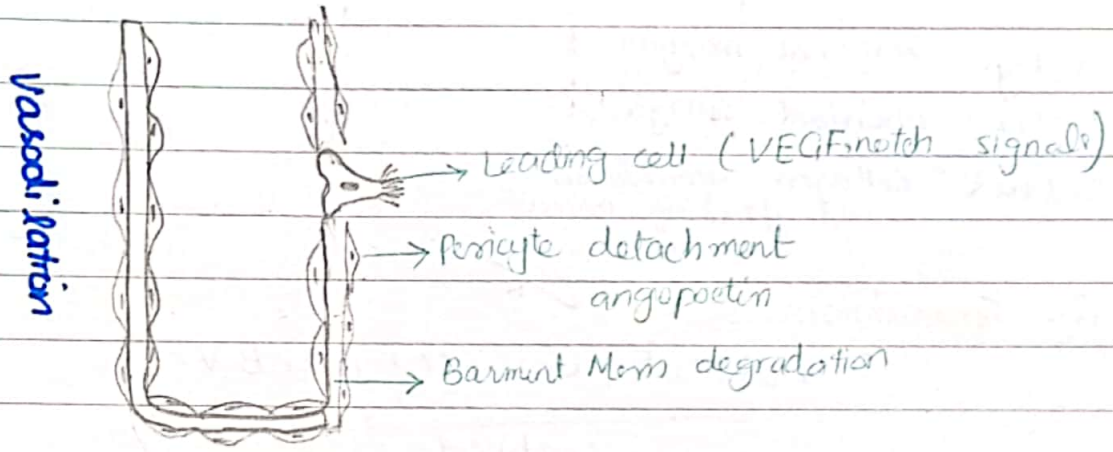
Suppressor of entothelial prolifration - migration

Date: _____

Q:- Angiogenesis? Formation of new blood vessels from pre exsisting vesseles.

Steps :-

① - Vasodilation in response of NO and inc permeability induced by VEGF



② - Separation of pericytes from adluminal surface

③ - Migration of endothelial cells toward area of injury.

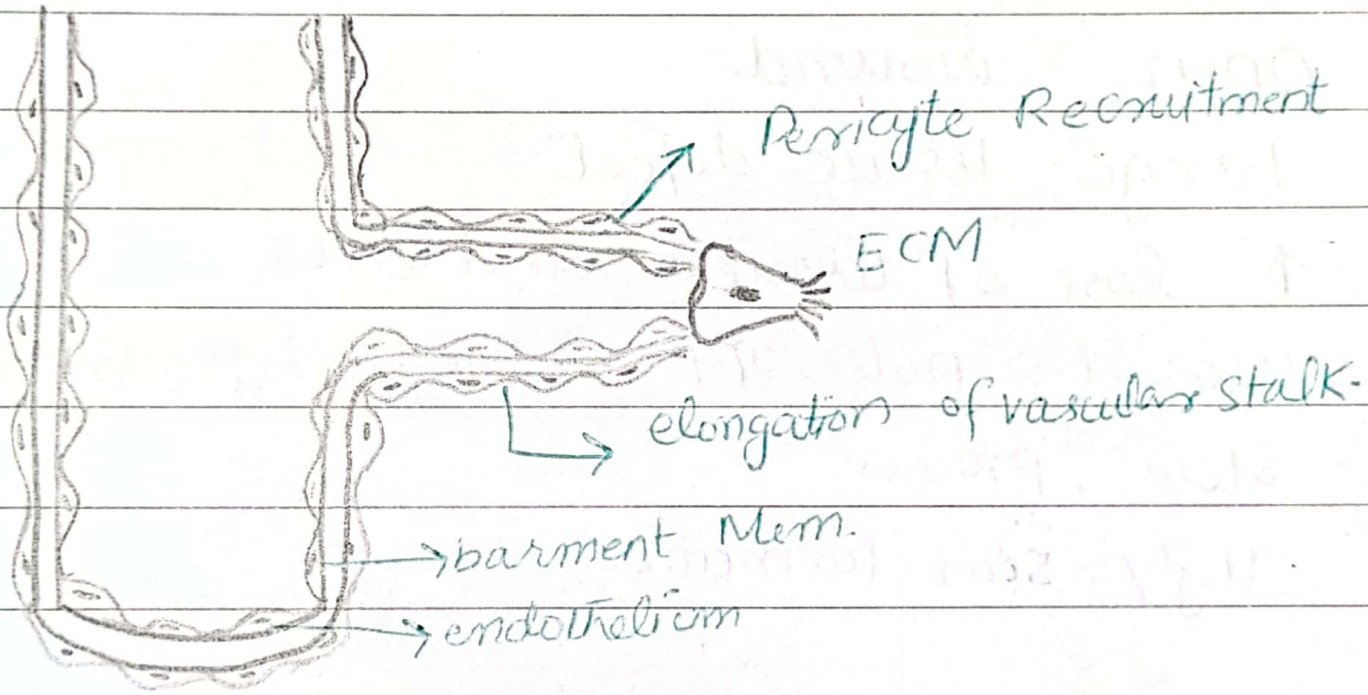
④ - Prolifration of endothelial cells

⑤ - Remodelling into capillary tubes.

⑥ - Suppression of endothelial prolifration + Migration

⑦ - Deposition of B. Membrane

Date: _____



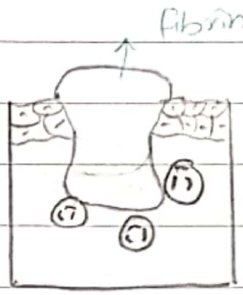
Date: _____

Secondary healing :-

Open wound.
Large tissue defect
↑ loss of tissue and cells
Wound not approximated by surgical suture.
Slow process
Ugly scar formation

①- Initial haemorrhage :-

Wound space is filled ∵ blood and fibrin clot

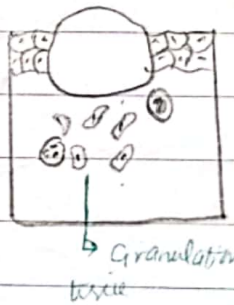


②- Inflammatory Phase :-

acute inflammation
Appearence of macrophages

③- epithelial changes :-

Proliferation of epidermal cells
and migration of epidermal cells



④- Granulation Tissue :-

Prolifration of fibroblast
and neovascularization
→ deep red
very fragile

Close Wound.

Date:

④- Wound Contraction:-

Myofibroblast

α actin work

↓

Wound contraction

80% contraction

about 1/3rd, 1/4th of orignal size

Pallatet derive GF
Fibroblast GF

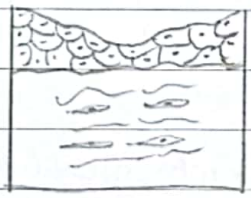
Wound contraction → active granulation tissue is formed

⑤- Prerence of infection:-

Bacterial contamination delays healing.

due to releare of bacterial Toxins

and provoke Necrosis.



Wound contraction

Suppuration

Thrombosis

Date: 14

Feature:-	Primary Union	Secondary Union
Cleanliness of wound.	clean	Unclean
Infection	Uninfected	Infected
Margins	Surgical clean	Irregular
Sutures	Used	Not used
Healing	Scanty granulation tissue at incised Gap ↓ tissue	Exuberant granulation Tissue to fill gap More tissue
Outcome/ scar	Neat linear Scar	Contracted Irregular Scar Wound contraction
	No wound contraction	
Complication	epidermal cyst formation	May require debridment
	Small damage	Large tissue damage
	Rapid P	Slow Process

Date:

Complication of Wound healing:-

① - Infection :- of wound due to bacteria. delay healing

② - Implantation :- (epidermal cyst formation.)

③ - Pigmentation :- Rust like colour due to staining e⁻ haemosidrin

④ - Deficient scar formation :- Inadequate formation of granulation tissue.

⑤ - Incisional hernia :- Weak scar formation
↳ may be site of bursting open of a wound

⑥ - Hypertrophied scars :- ↑ scar, ugly and painful

⑦ - Keloid formation

↑ collagen formation (Claw like) → Kleoid.
scar tissue grow beyond the boundaries dnt regress seen in blacks

⑧ - Excessive Contraction :-

~~contraction~~ ↑ wound contraction may result in formation of cicatrisation
- Peyronie's disease
- Dupuytren's contractures.

⑨ Desmoids.

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⑨- Neoplasia :-

carcinoma formation

Factor Influencing Healing :-

Local Factors :-

① - Infection
② - Poor blood supply — Hematoma
③ - Foreign bodies
④ - Movement delays wound healing
⑤ - Ionising Radiation → delays granulation tissue form
⑥ - Exposure to UV light.

Systemic Factors :-

① - Age
② - Nutrition Deficency — Anemia, drugs, hormones, Malignant disease, obesity
Vit C and Zinc delay healing
③ - Infection
④ - uncontrolled DM
⑤ - Administered of Glucocorticoids
⑥ - Haemologic Abnormality.
defect of neutrophill function and Neutropenia.

Delayed. Wound :-

Date: _____

Factor from Blood :-

① - Infection :

due to bacteria → Inflammation

② - Nutrition :-

Vit C deficency inhibit collagen synthesis

Retard healing

③ - Glucocorticoids :- (Steroid)

weak the scar

Inhibit TGF-β

diminsh fibrosis

Corneal infection

④ - Machanical variable :-

↑ local Prerrure / Torsion

cause wound pull apart.

⑤ - Poor purfusion :-

Obstruction of veins. (diabetes → Atherosclerosis, varicose vein)

Impair healing

⑥ - Foreign Bodies

steel, glass. → Impair healing

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⑦- Keloids and Hypertrophic scar

↳ Accumulation of collagen
↑ scar

↳ Scar tissue grow beyond boundaries of orignal Wound, dnt Regress

⑧- Exuberant granulation (Proud Flesh)

↑ granulation tissue
Protude above level of surrounding skin

↑ contraction of wound.
cause contractures
deformity occur

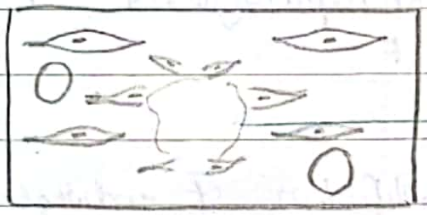
⑨- ✓ Desmoid.

Incisional scar and injuries may form by proliferation of fibroblast
Recur after excision ⇒ desmoid

Scar Formation :-

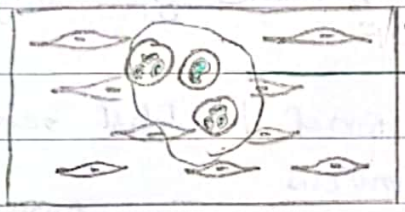
① - Formation of new blood vessels (Angeogenesis)
② Migration and proliferation of fibroblasts
③ - Deposition of C.T
④ - Maturation and Recognization of fibrous tissue to produced stable scar

① -



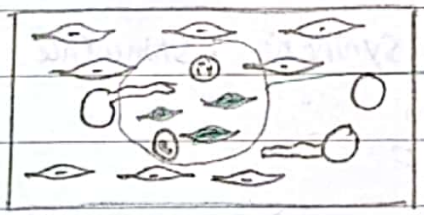
→ Area of injury.

② -



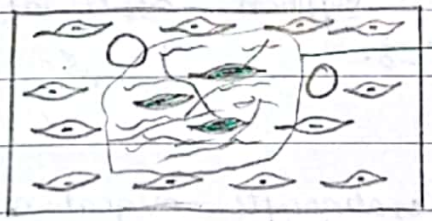
→ Inflammation

③ -



→ Formation of Granulation Tissue

④ -



→ Scar formation
C.T deposition

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Growth Factors :-

	Functions
Epidermal Growth Factor (EGF) Macrophages - salivary G ; Keratinocytes	Mitogenic for Keratinocytes and fibroblast stimulates formation of granular tissue EGF
Transforming (GF) Macrophage - Keratinocyte	Proliferation of hepatocytes TGF
Hepatocyte (GF) (Scatter) Fibroblast , stromal cell	↑ Prolifration of hepatocytes. , ↑ cell motility HGF
* Vascular endothelial (GF) Mesenchymal cell	Stimulate Prolifration of endothelial cell ↑ vascular permeability Angio g* VEGF
Platelets derived (GF) ✓ Macrophages, Smooth M, Keratinocytes	activates Prolifertion of fibroblast , endothelial cells ECM protein Synthesis PDGF W. C. Angio.
* Fibroblast GF. (1,2) ✓ Macrophages, Mast cell	ECM protein Synthesis , Stimulate Angeogenesis Wound con FGF
Tranforming (GFB). T-lymphocytes , Macrophages smooth muscle	suppress acute inflammation TGF-B.
Keratinocyte (GF) Fibroblast	Stimulate Keratinocyte migration , Proliferation KGF

Date:

Granulomatous Inflammation

Disease	Cause	Tissue Rx
Caseating		
Tb	Mycobactrium tuberdosis	✓ Caseating Granuloma activated macrophages rimmed by fibroblast. Central Necrosis. cell. Lang·hans Gaint cell
Syphills	Trepanema Pallidum	✓ Plasma cell infiltrates ✓ Central cell necrosis
Cat-Scratch direare.	Gram (-)ve bacillus	✓ stallate granuloma ✓ Central Granular debris. ✓ Gaints cells
Non Caseating:		
Leprosy	Mycobactrium Leprae	✓ Non caseating Granuloma ✓ Acid fat bacilli in macrophag
Sacrcoidosis	Unknown	✓ Non caseating Granuloma. ✓ activated macrophages
Crohn direare	Immune Rx against intestinal bactria self antigen	" " ✓ chronic inflammatory ✓ infiltration

Date: