

INFLAMMATION

TYBSc PAPER -2, UNIT-4

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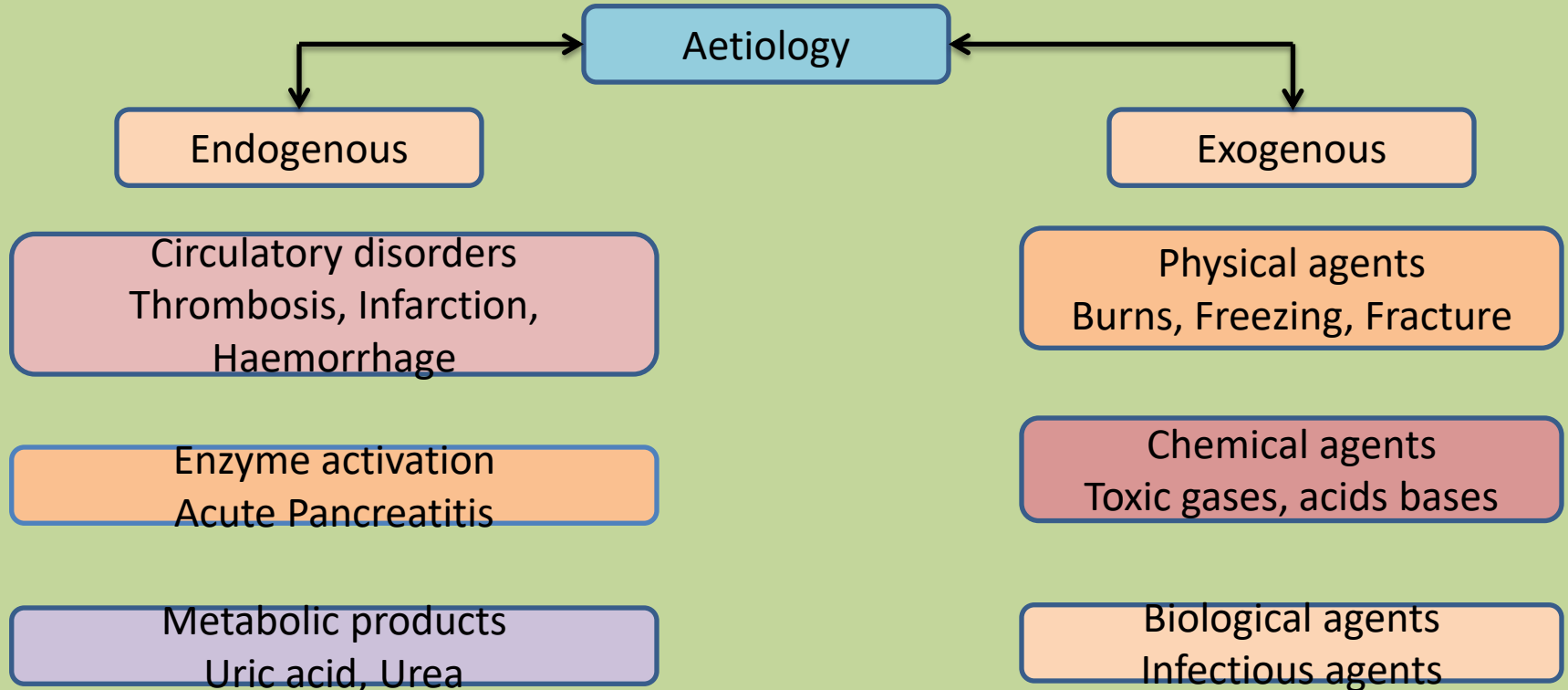
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Inflammation is local response of living tissue to any agent.

Process by which body's immune system malfunctions

Examples: Diabetes, Arthritis, Myasthenia gravis

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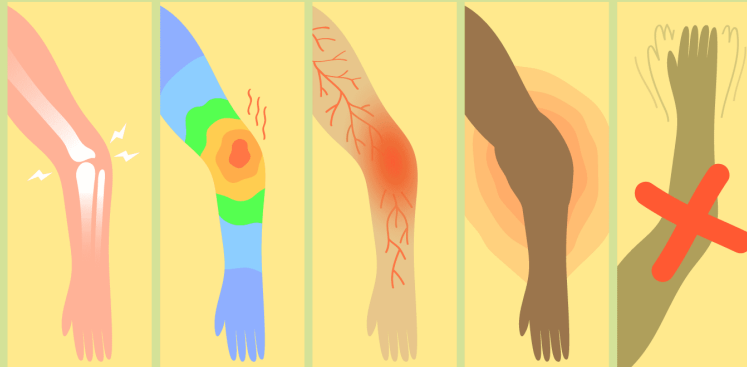
Signs of inflammation

Pain
Dolor

Heat
Calor

Redness
Rubor

5 Cardinal Signs of Inflammation



Pain

Heat

Redness

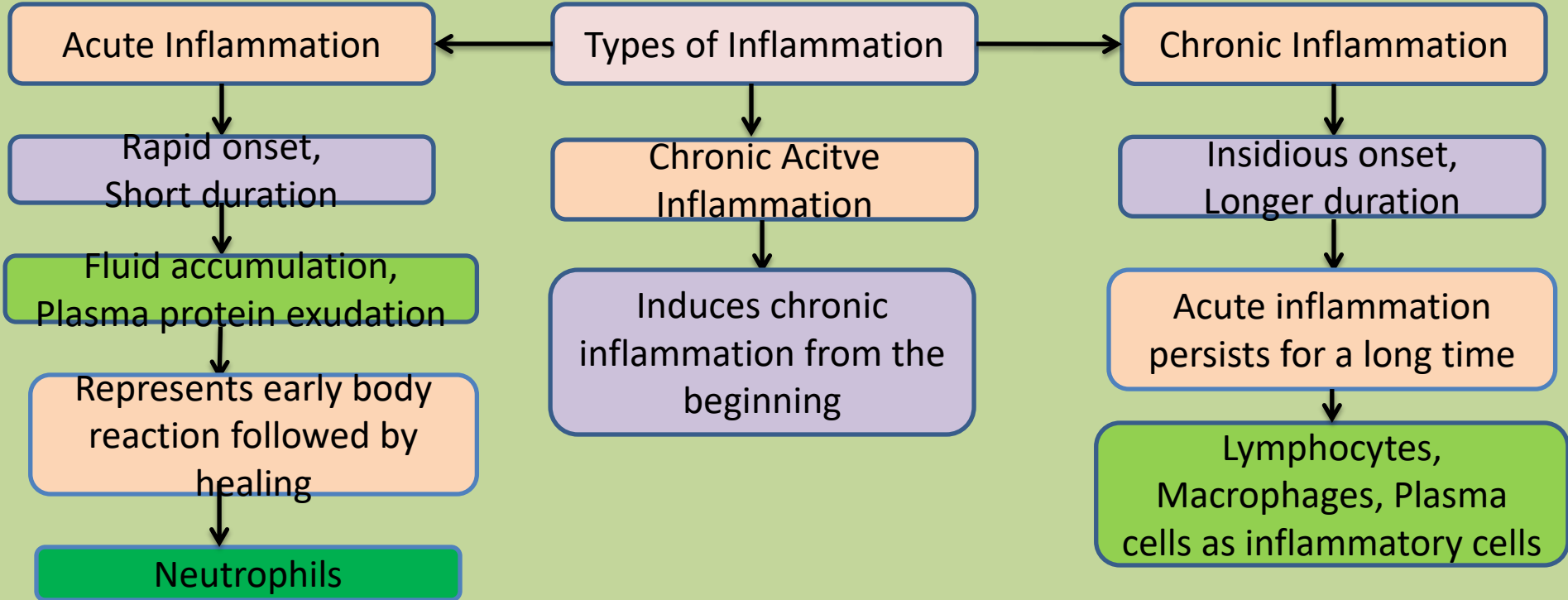
Swelling

Loss of
Function

Swelling
Tumor

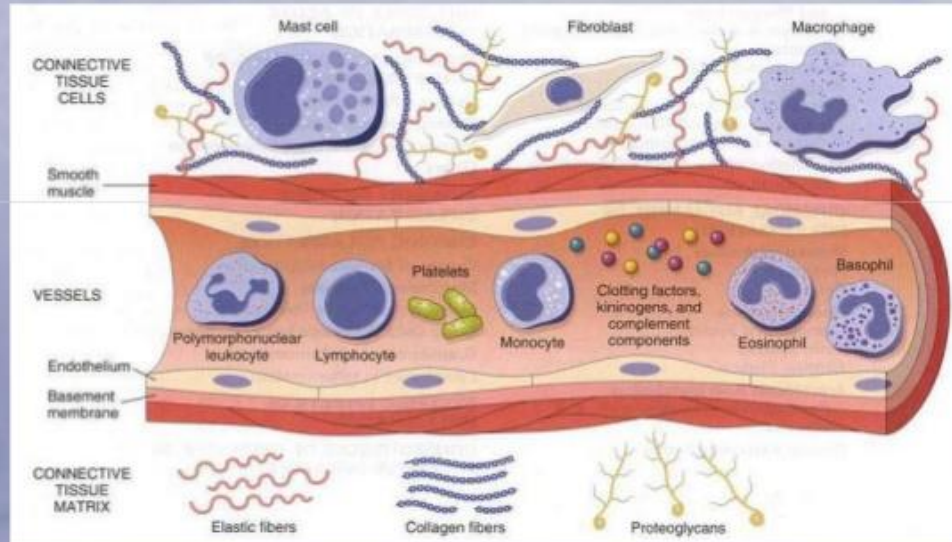
Loss of function
Functio laesa

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Components of Acute and Chronic Inflammatory responses



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Pathogenesis

Three main processes occur at the site of inflammation, due to the release of chemical mediators

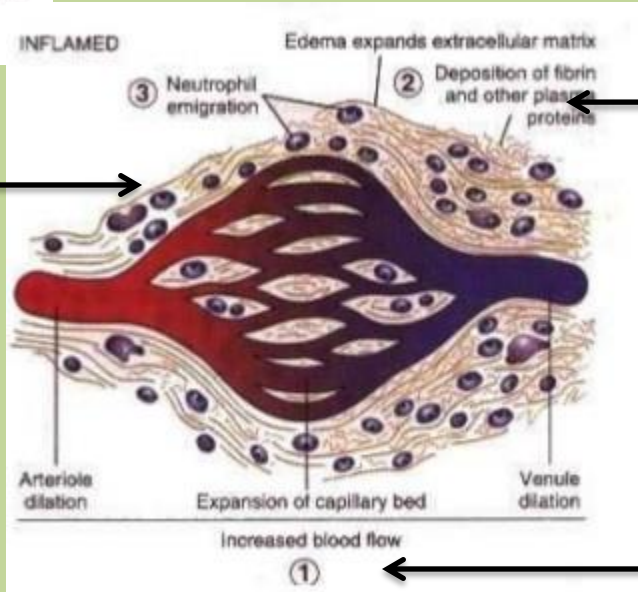
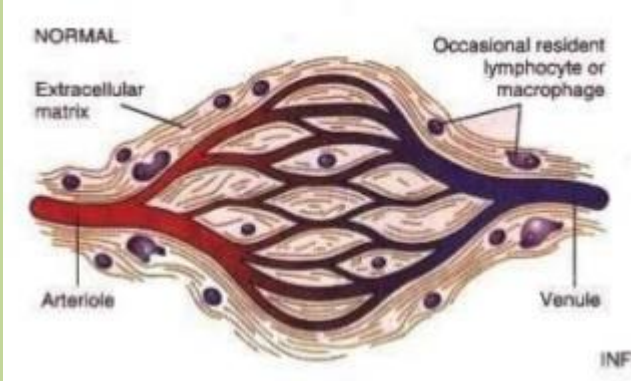
Increase in the blood flow (redness and warmth)

Increased vascular permeability (swelling, pain and loss of function)

Leukocytic infiltration

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Mechanism of inflammation



Exudation
Edema

Vasodilation

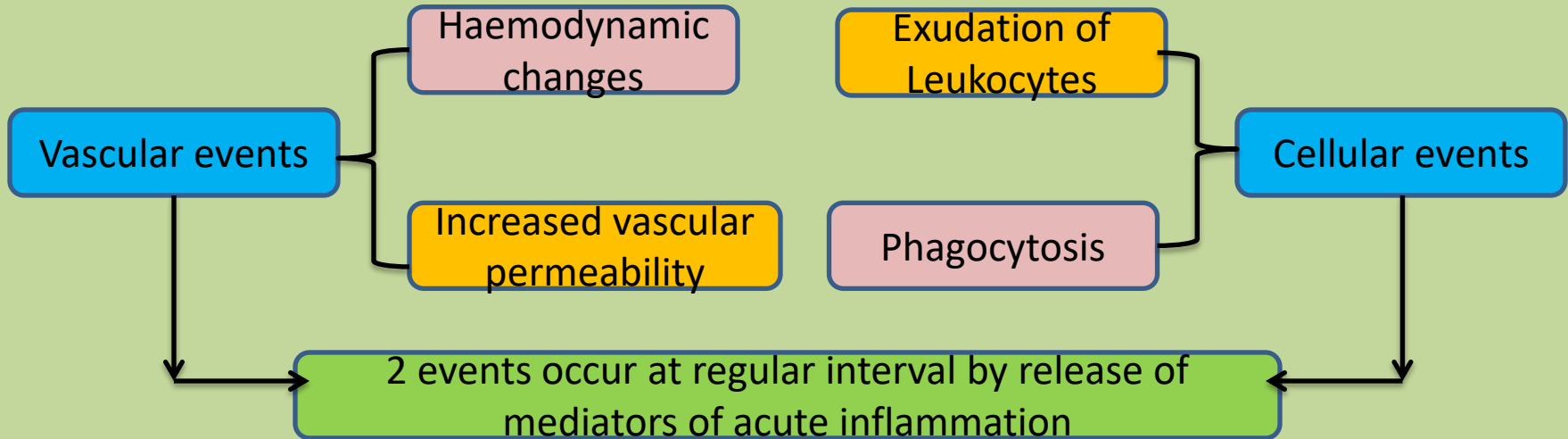
Emigration of
cells

Chemotaxis



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Acute inflammation



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Haemodynamic changes

1. Transient Vasoconstriction

2. Vasodilation
(within half an hour of injury)

Increased blood flow in the micro-vascular bed

Redness and warmth

3. Elevation of hydrostatic pressure

Transudation of fluids in extracellular space

Swelling

4. Slowing or Stasis

Increased vascular permeability

Increased concentration of RBCs

Slower blood flow

Raised blood viscosity

5. Leukocyte migration

Neutrophils

Extravascular spaces

Emigration

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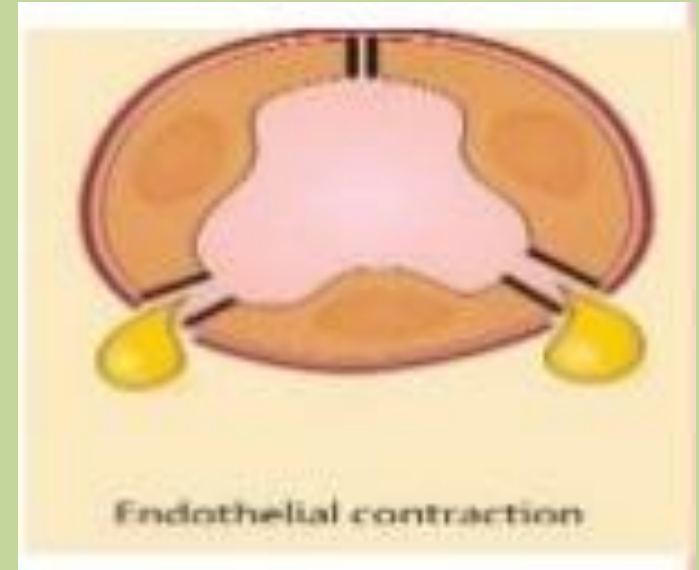
Mechanism Increased vascular permeability

1. Gaps due to endothelial cell contraction

Reversible process

Mediated by Histamine, Bradykinin, Leukotrienes

Short duration
15-30 minutes



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Mechanism Increased vascular permeability

2. Structural reorganization of cytoskeleton of endothelial cells

Mediated by cytokines such as interleukin1, Tumor Necrosis Factor alpha (TNF)



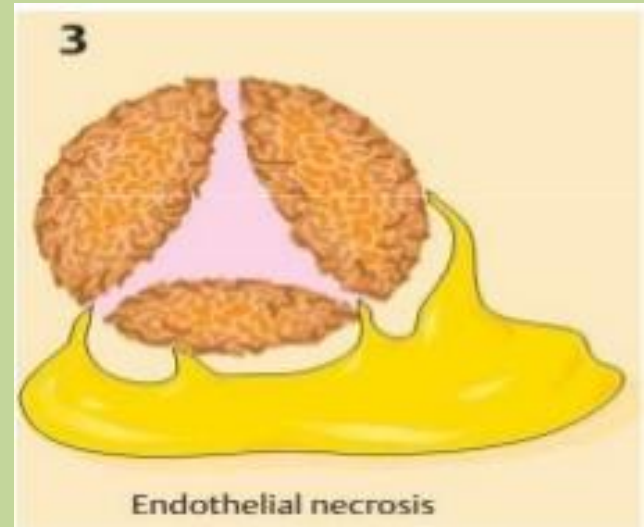
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Mechanism Increased vascular permeability

3. Direct injury to endothelial cells

Cellular necrosis, Thrombosis, appearance of physical gaps

Lasts for several hours or days



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Mechanism Increased vascular permeability

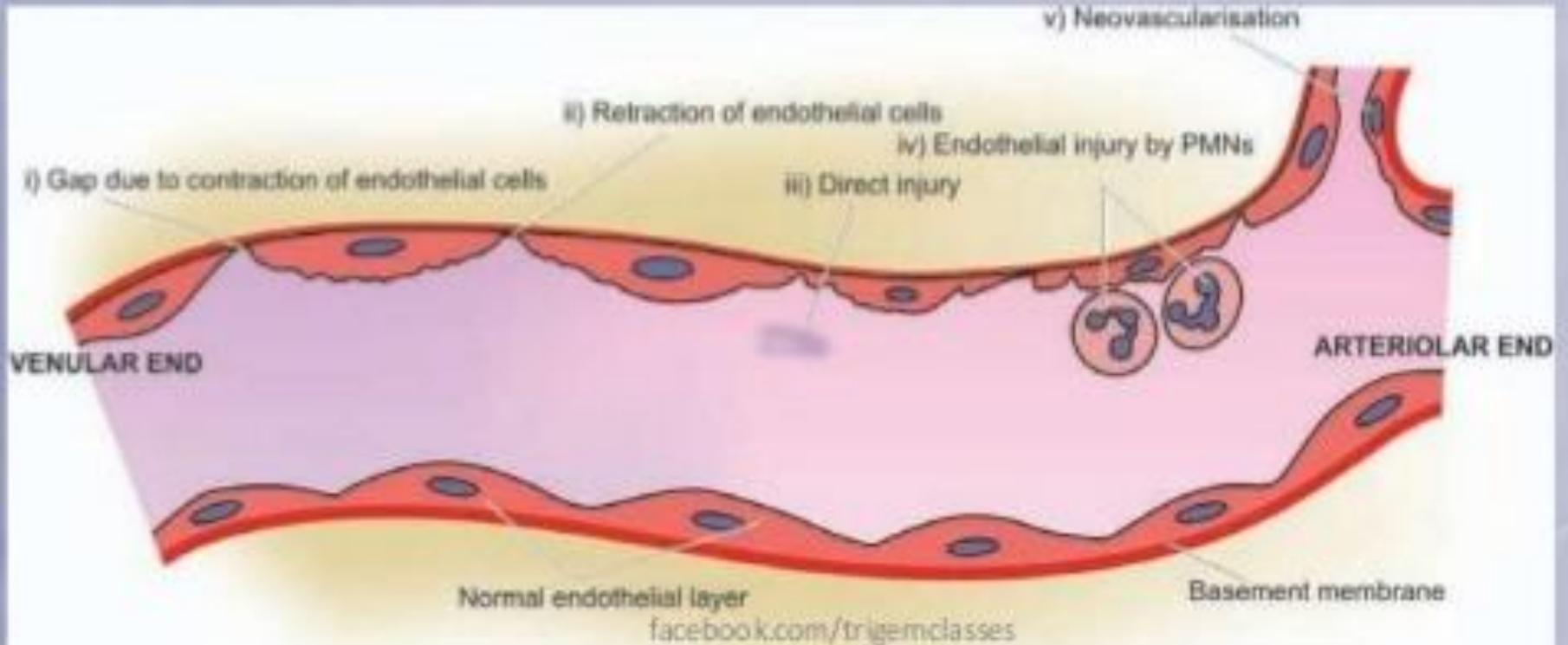
4. Leukocyte mediated cellular injury

Adherence of leukocytes to endothelium at the site of inflammation

Activation of leukocytes – proteolytic enzymes

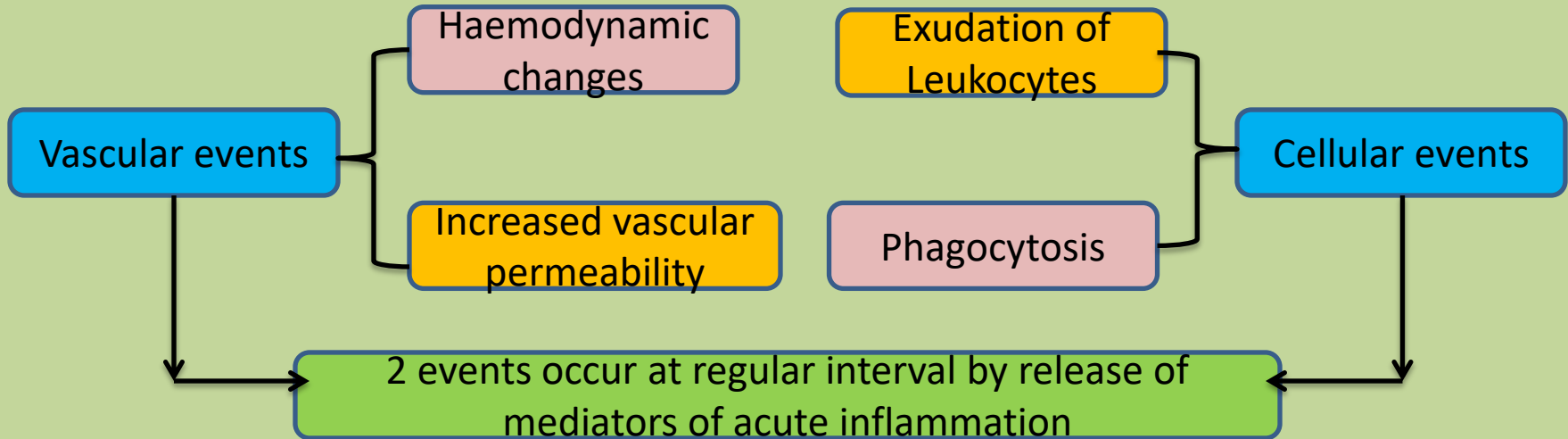
5. Neo Vascularization
Vascular Endothelial Growth Factor (VEGF)

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Acute inflammation



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Cellular events

Exudation of Leukocytes

Leukocytes escape from the lumen of microvasculature to the interstitial tissue space

In acute inflammation → Polymorphonuclear neutrophils, monocytes, macrophages

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Changes leading to migration

Changes in the formed elements of the blood

Vasodilation and subsequent slowing of blood stream

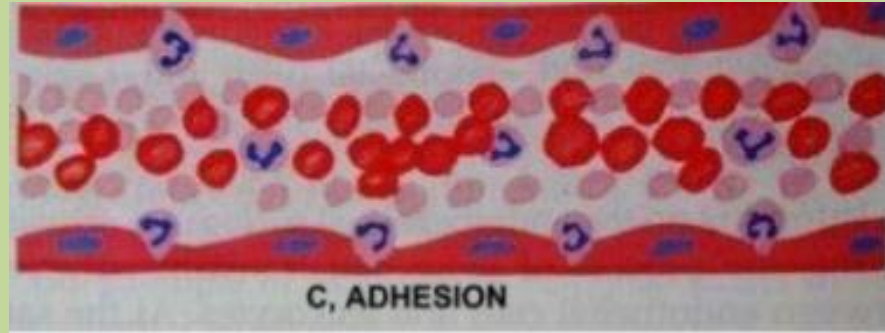
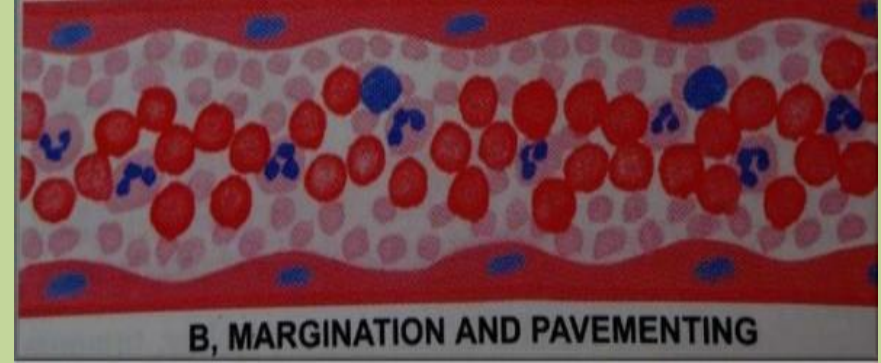
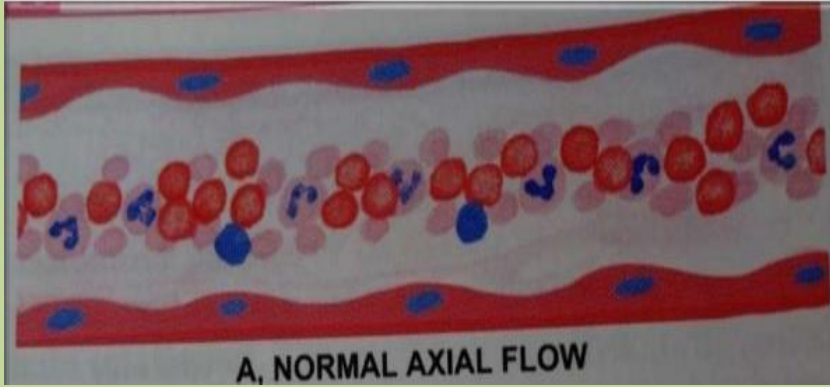
Margination: The central stream of cells widens and peripheral plasma zone becomes narrower because of loss of plasma by exudation

Pavementing: The neutrophils of the central column come close to the vessel wall

Rolling: Peripherally marginated and pavemented neutrophils slowly roll over endothelial cells lining the cell wall

Adhesion: Transient bond between the leukocytes and endothelial cells becoming firmer

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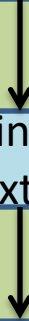


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Neutrophils move till suitable site is reached

Cross the basement membrane by damaging it locally with secreted collagenase and escape out into extravascular space

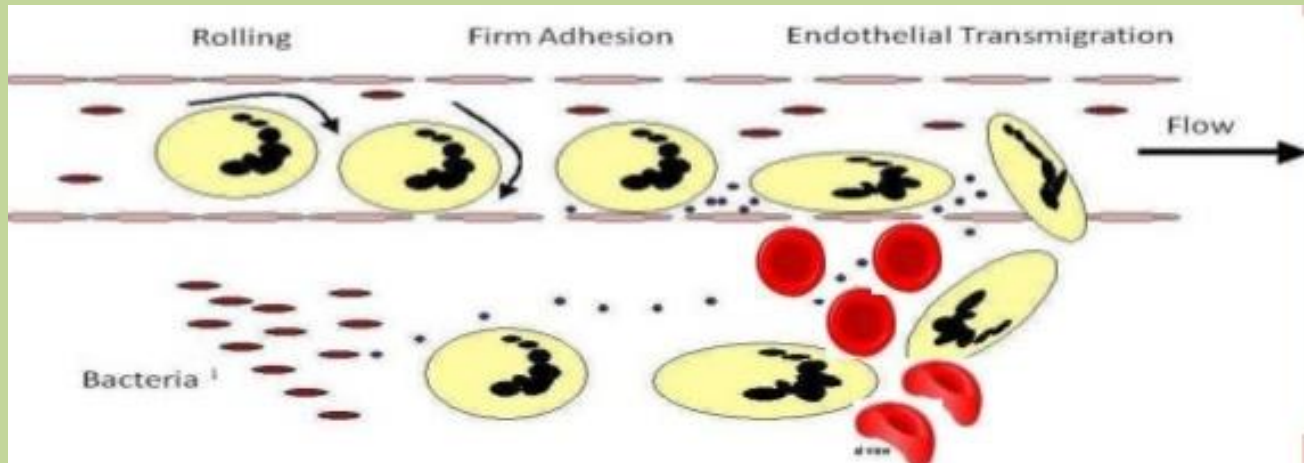
Emigration



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Diapedesis: Simultaneously escape of RBCs takes place through the gaps between the endothelial cells

Diapedesis gives **haemorrhagic appearance** to the inflammatory exudate



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Chemotaxis

After moving from blood, leukocytes migrate towards site of injury along the chemical gradient by a process called Chemotaxis

Potent chemotactic substances or chemokines for neutrophils are:

↓
Leukotriene B4 (LT-B4) Arachidonic acid metabolites
Components of Complement systems (C5a and C3a in particular)
Cytokines
Interleukins (IL8)

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Phagocytosis

Process of engulfment of solid particulate matter by the cell

Polymorhonuclear
Neutrophils

Macrophages

Microphages

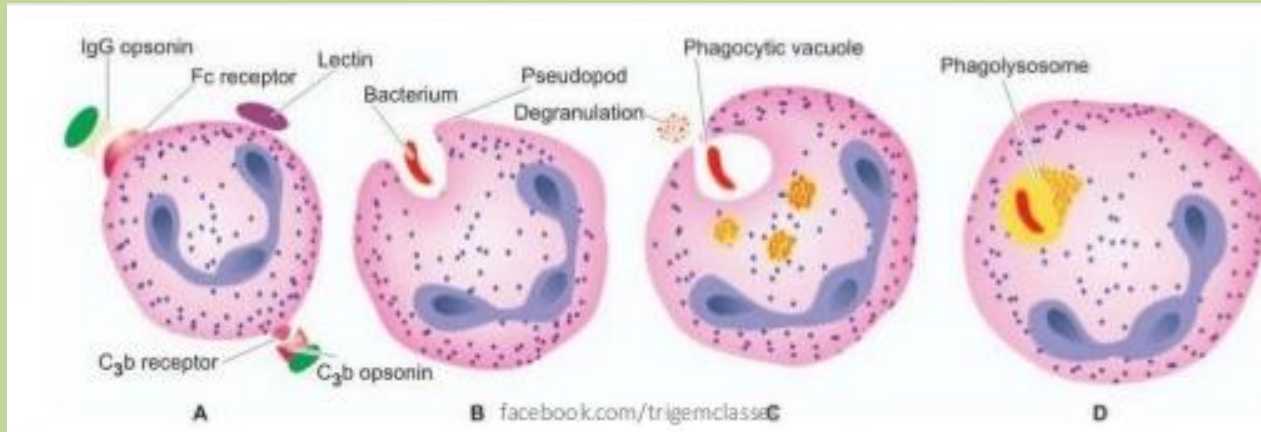
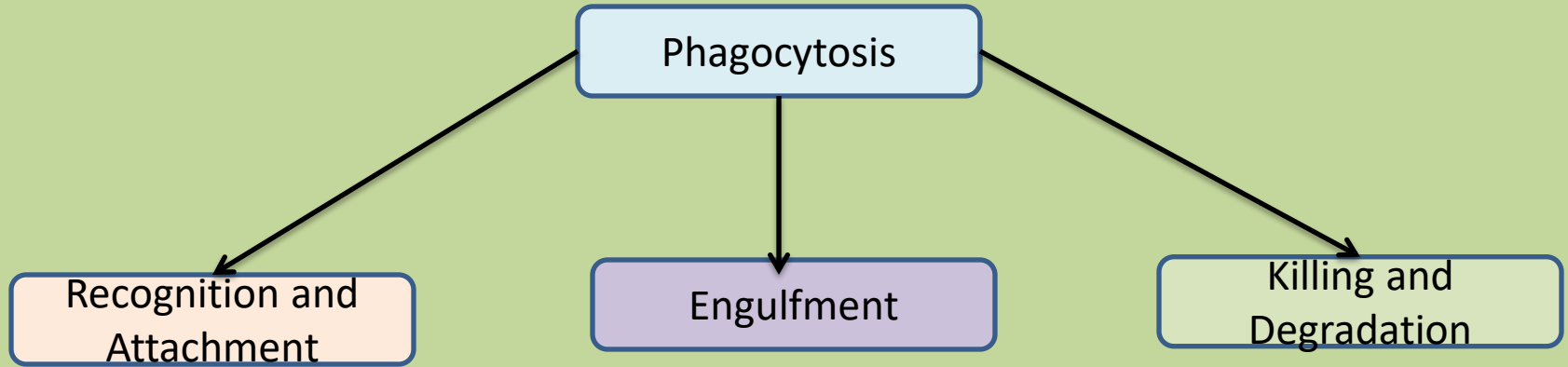
Circulating
monocytes

Fixed Tissue Mononuclear
phagocytes

Early in acute inflammatory
responses

Phagocytic cells release proteolytic enzymes – lysozyme, protease, collagenase, elastase, lipase, proteinase, gelatinase, acid hydrolases

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Outcomes of Acute Inflammation

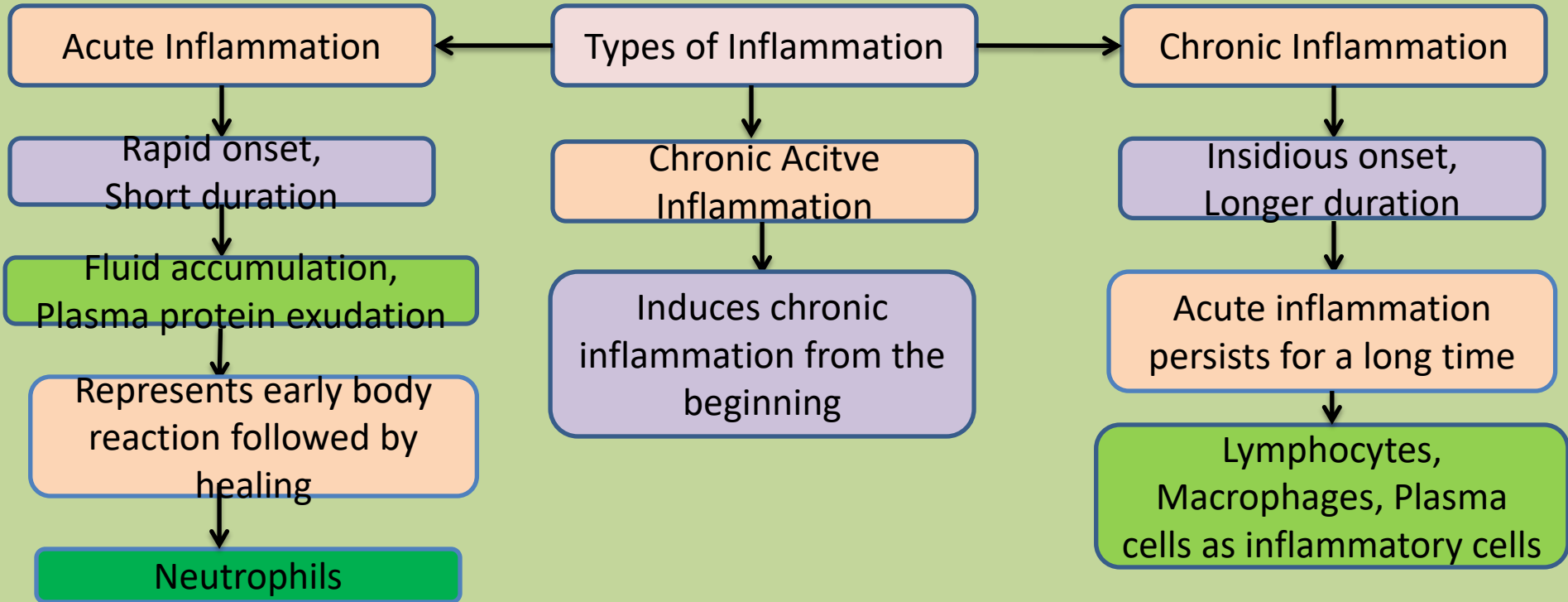
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graph TD; A[Outcomes of Acute Inflammation] --> B[Resolution]; A --> C[Healing by scar]; A --> D[Chronic inflammation];
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Resolution

Healing by scar

Chronic inflammation

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Chronic Inflammation

It can be caused in one of the following 3 ways

Chronic inflammation following acute inflammation

Recurrent attacks of acute inflammation

Chronic inflammation starting *de novo*

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Chronic Inflammation

Cancer

Cardiovascular
disease

Neurological
disorders

Metabolic
disorders

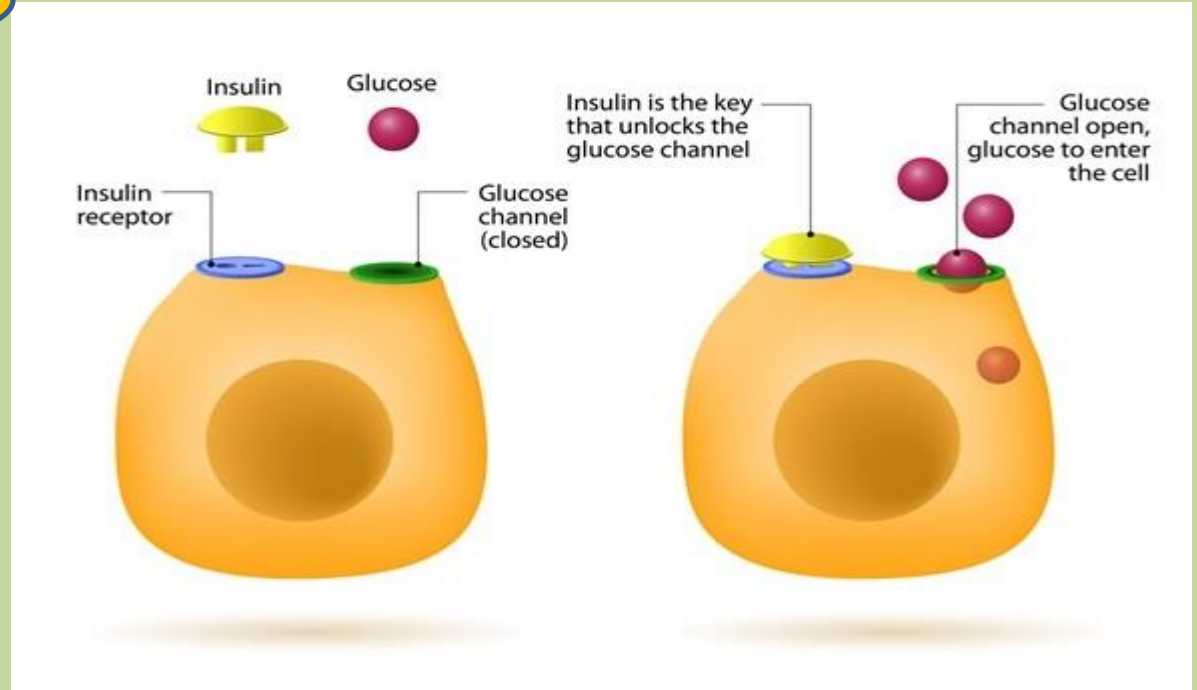
Pulmonary
diseases

Bone and joint
diseases

Autoimmune
disorders

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Metabolic disorders



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General characteristic features of chronic inflammation

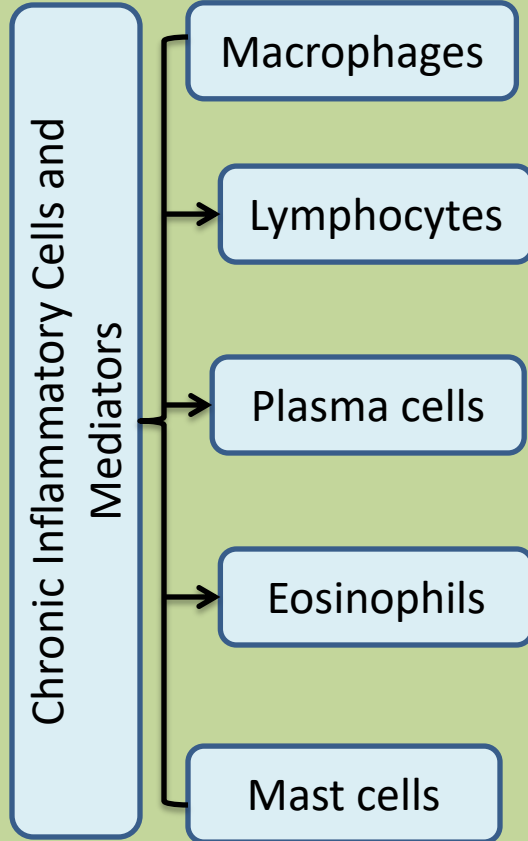
Infiltration with mononuclear cells- macrophages, lymphocytes

Tissue destruction

Healing by proliferation and connective tissue replacement of damaged tissue, Angiogenesis



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Macrophages

Dominant cells of chronic inflammation

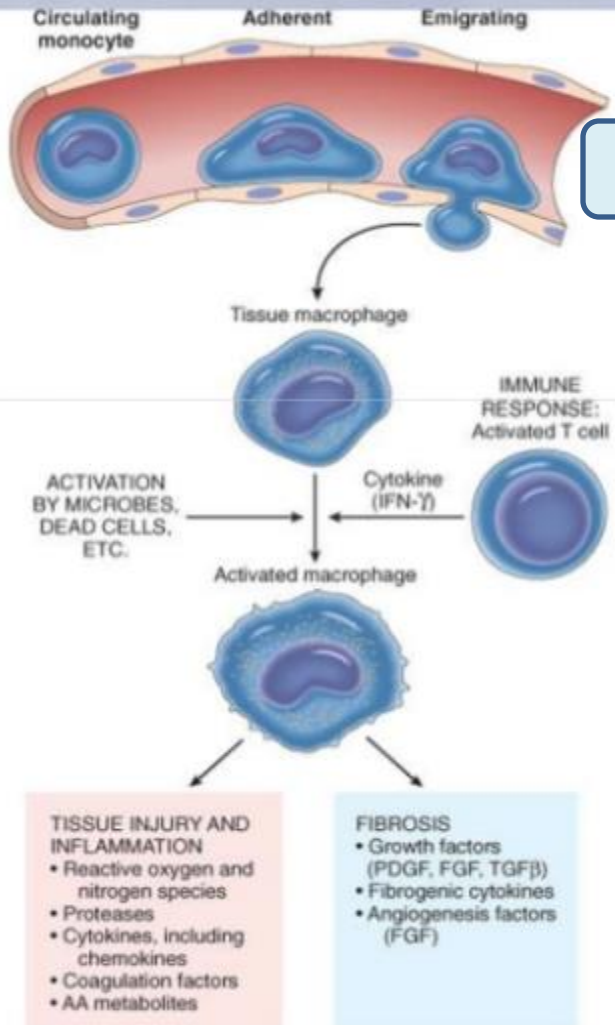
Derived from circulating blood monocytes

Reticuloendothelial system (RES) / Mononuclear Phagocytic system

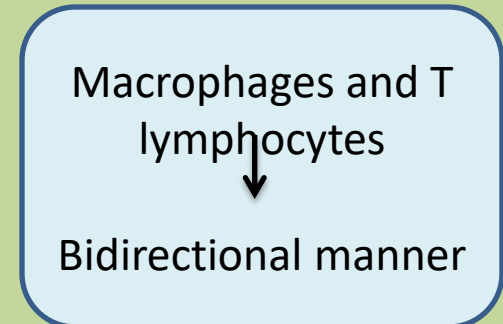
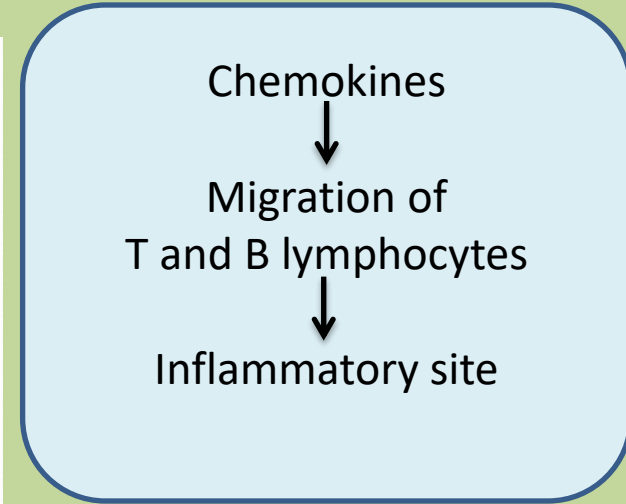
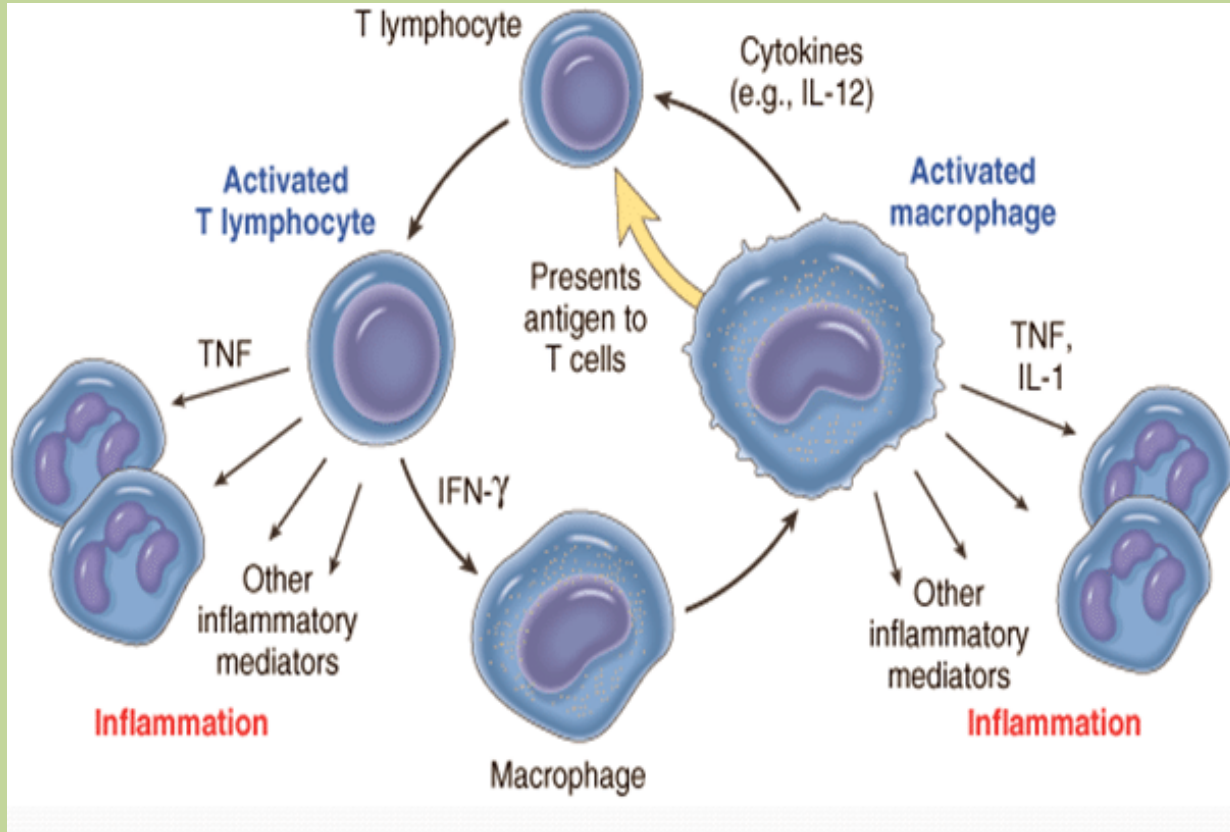
Kupffer cells in Liver, Sinus histiocytes in Lymph nodes, Microglial cells in CNS, Alveolar macrophages in lungs

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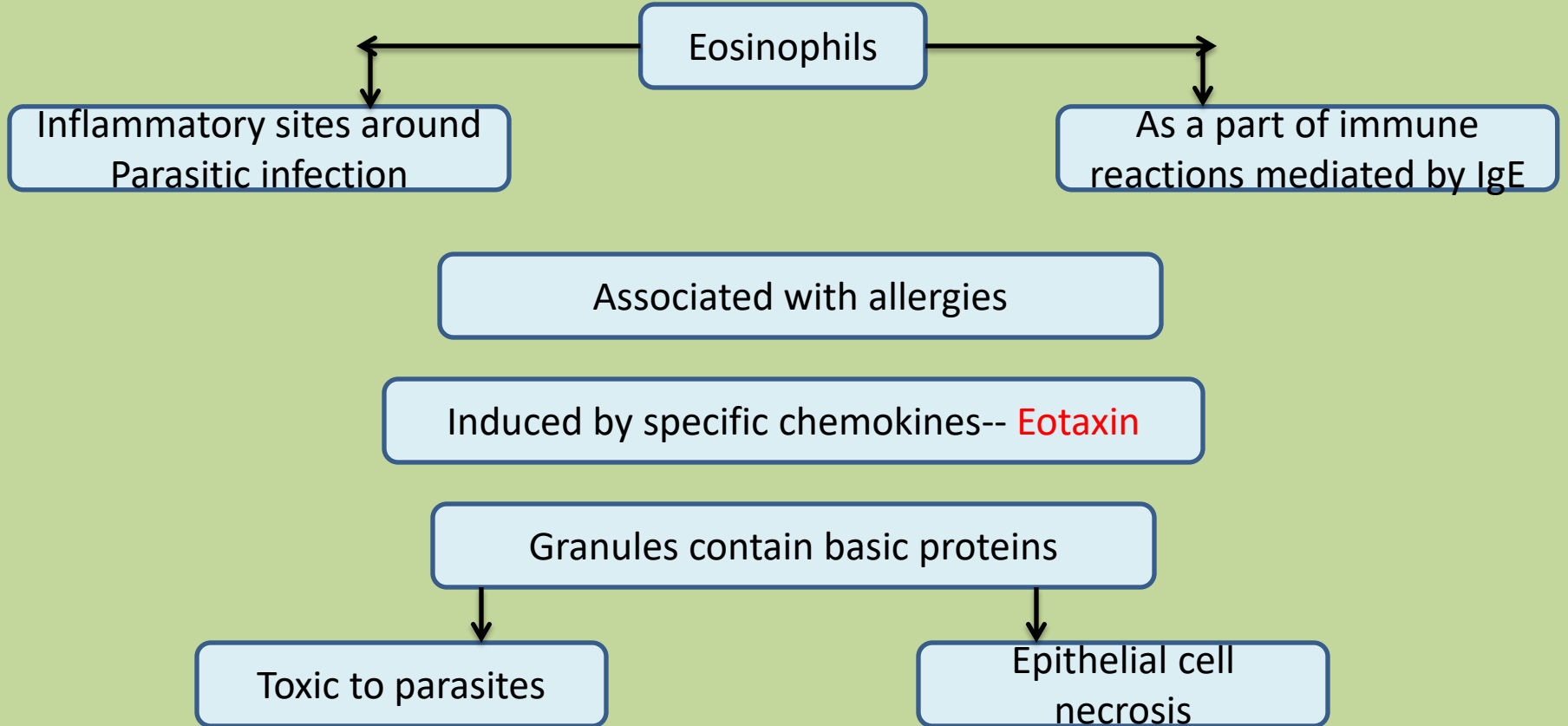
Macrophages



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Mast cells

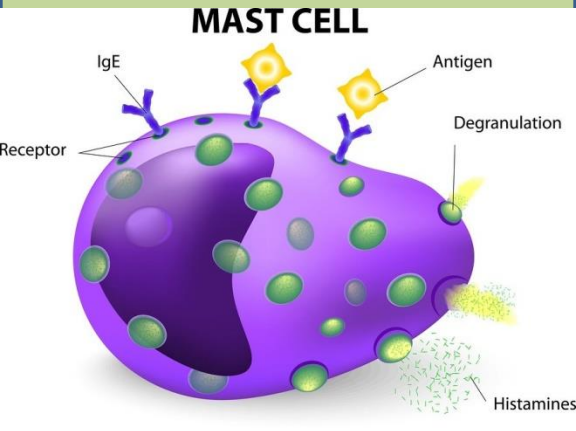
Mastocytes/ Labrocytes

Migrant cells of connective tissue

Granules rich in histamine and heparin

Binds with the fc portion of IgE antibody

Observed in both chronic and acute inflammatory responses



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Plasma cells

Plasma B cells

Produced in bone marrow and produce Antibodies in response to antigen

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Systemic effects of chronic inflammation

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graph TD; A[Systemic effects of chronic inflammation] --> B[Fever]; A --> C[Leucocytosis]; A --> D[Elevated ESR]; A --> E[Amyloidosis];
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Fever

Leucocytosis

Elevated ESR

Amyloidosis

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- <https://www.slideshare.net/DeepakKumarGupta2/inflammation-51778799>
- https://www.life.illinois.edu/mcb/458/private/lectures/ppt_pdf/Path_ggf_13_2017.pdf

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- <https://www.slideshare.net/NimraIqbal/chronic-inflammation-212>