INFLAMMATION

TYBSc PAPER -2, UNIT-4
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INFLAMMATION

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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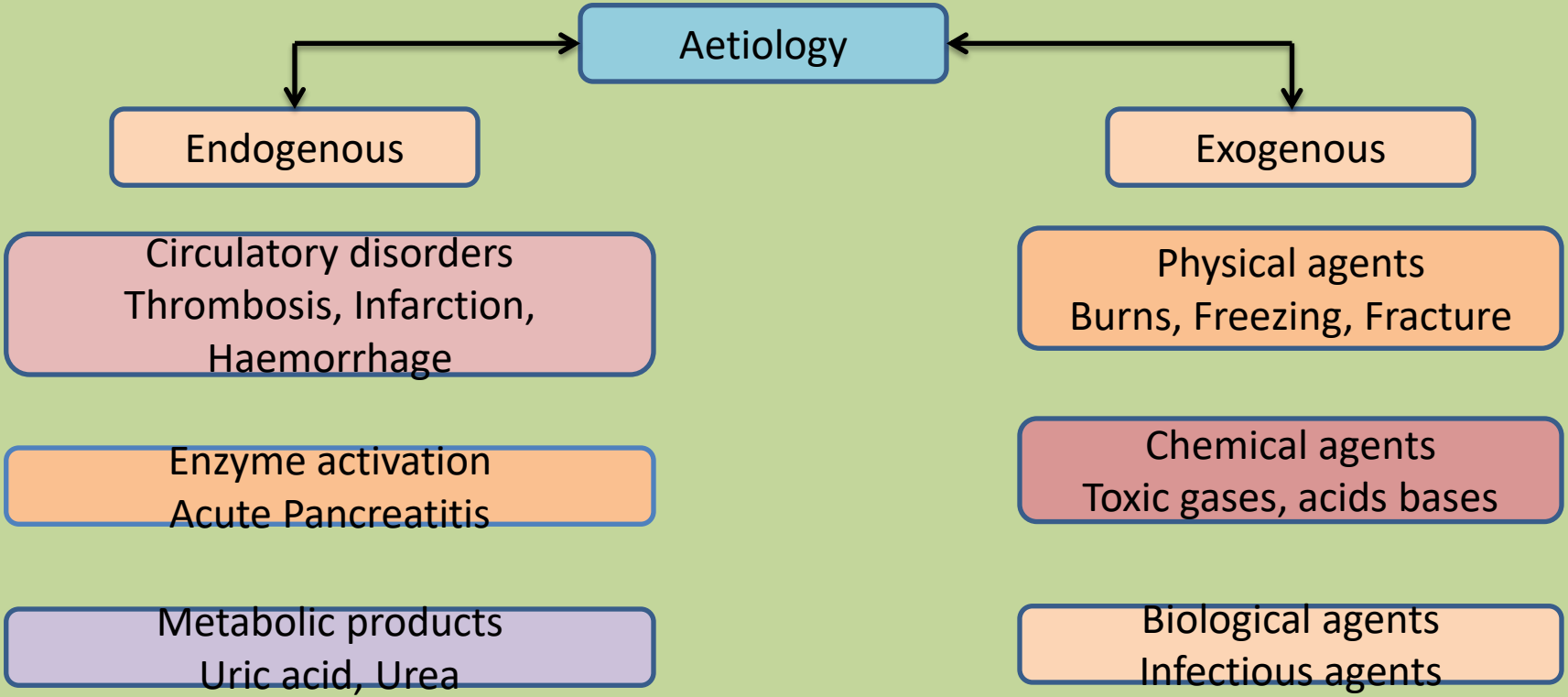

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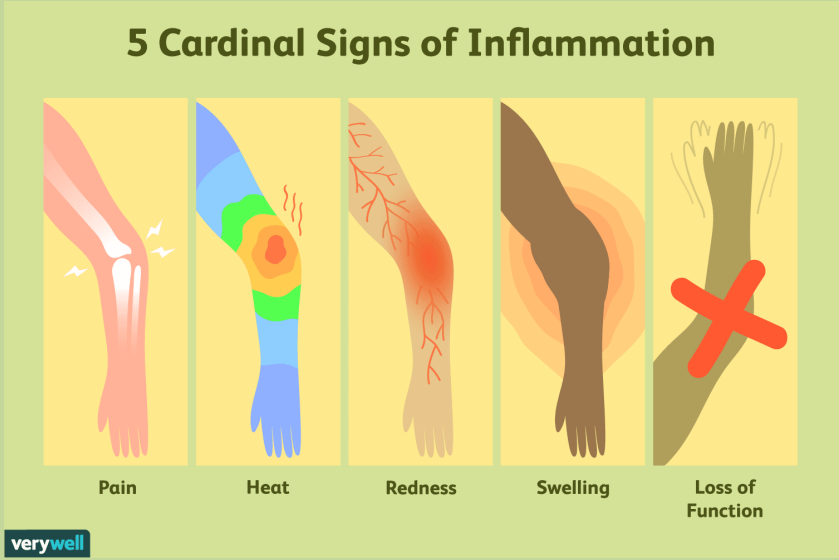
Signs of inflammation

Pain
Dolor

Heat
Calor

Redness
Rubor



Swelling
Tumor

Loss of function
Functio laesa

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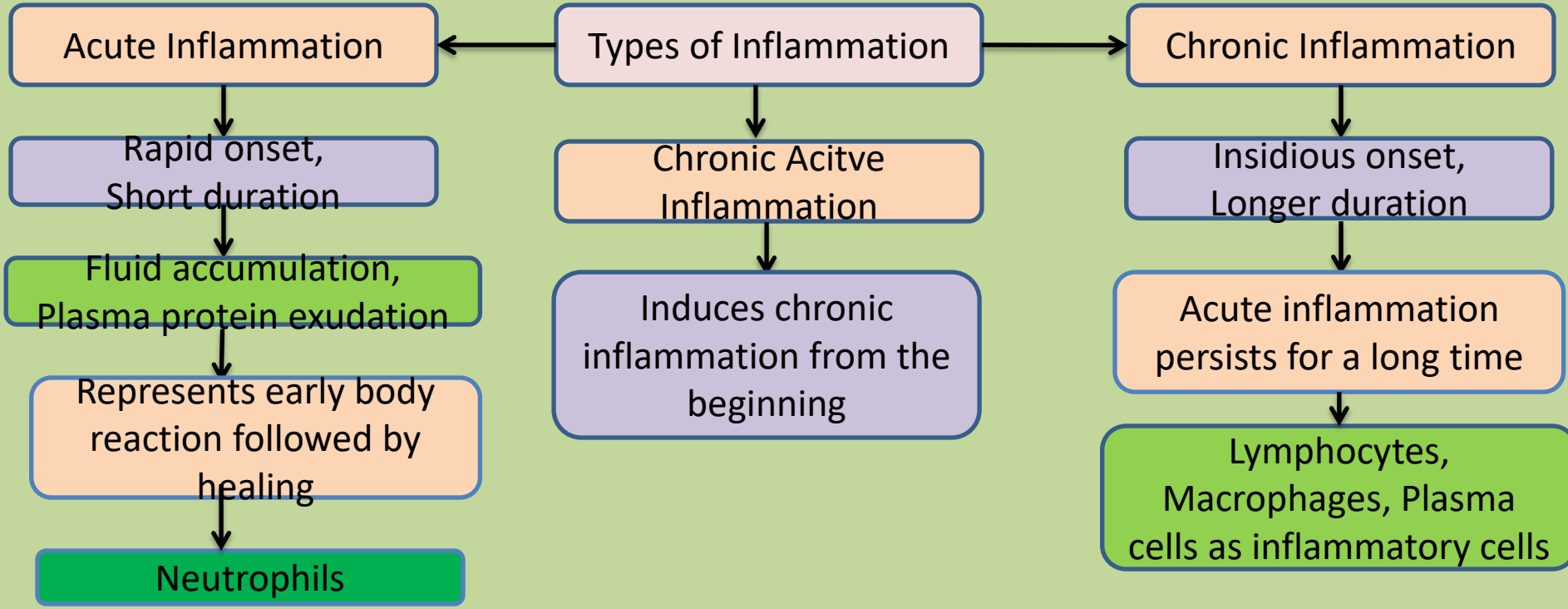

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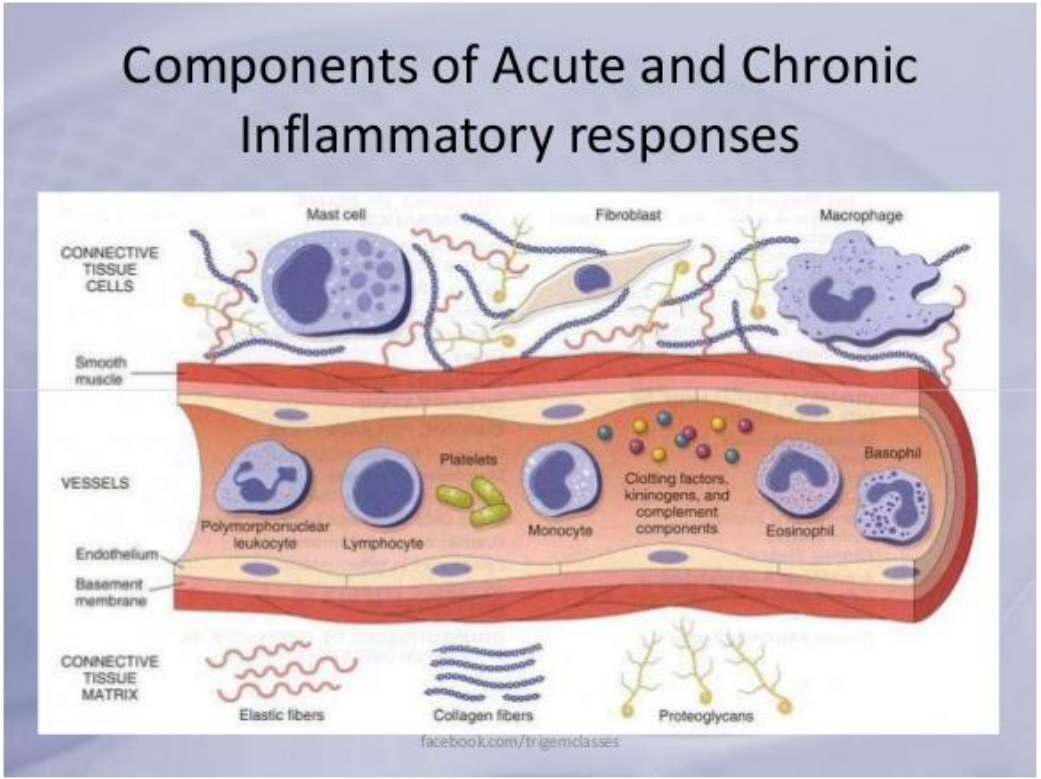

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Pathogenesis

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)

Leuckocytic infiltration

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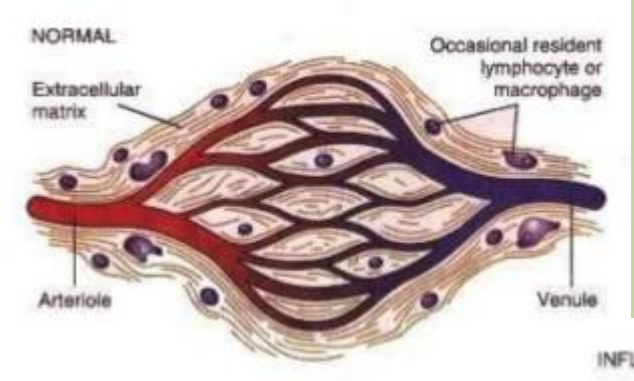
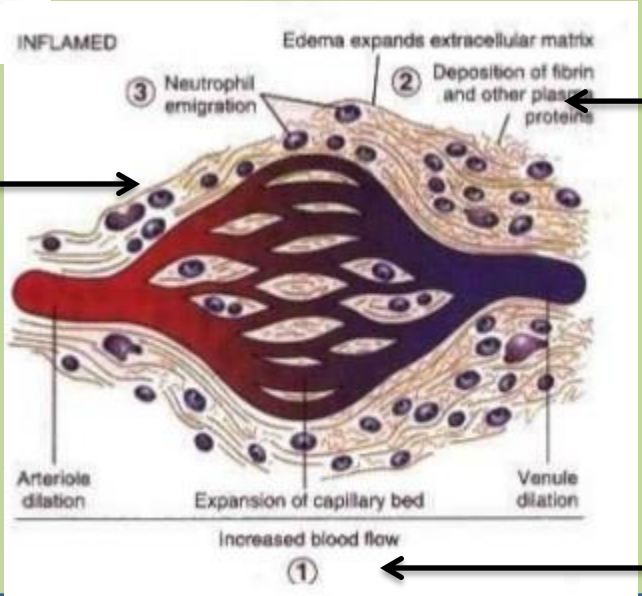
Mechanism of inflammation





Exudation
Edema

Emigration of cells

Chemotaxis

Vasodilation

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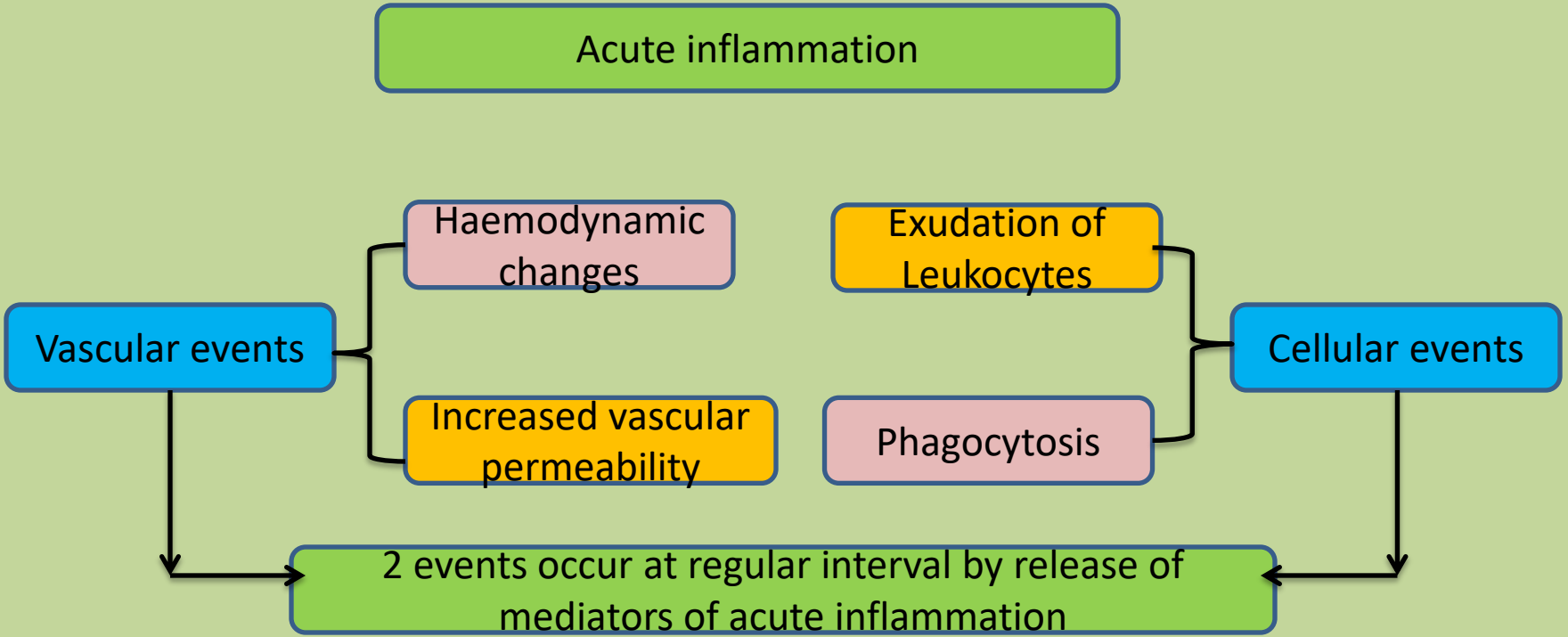

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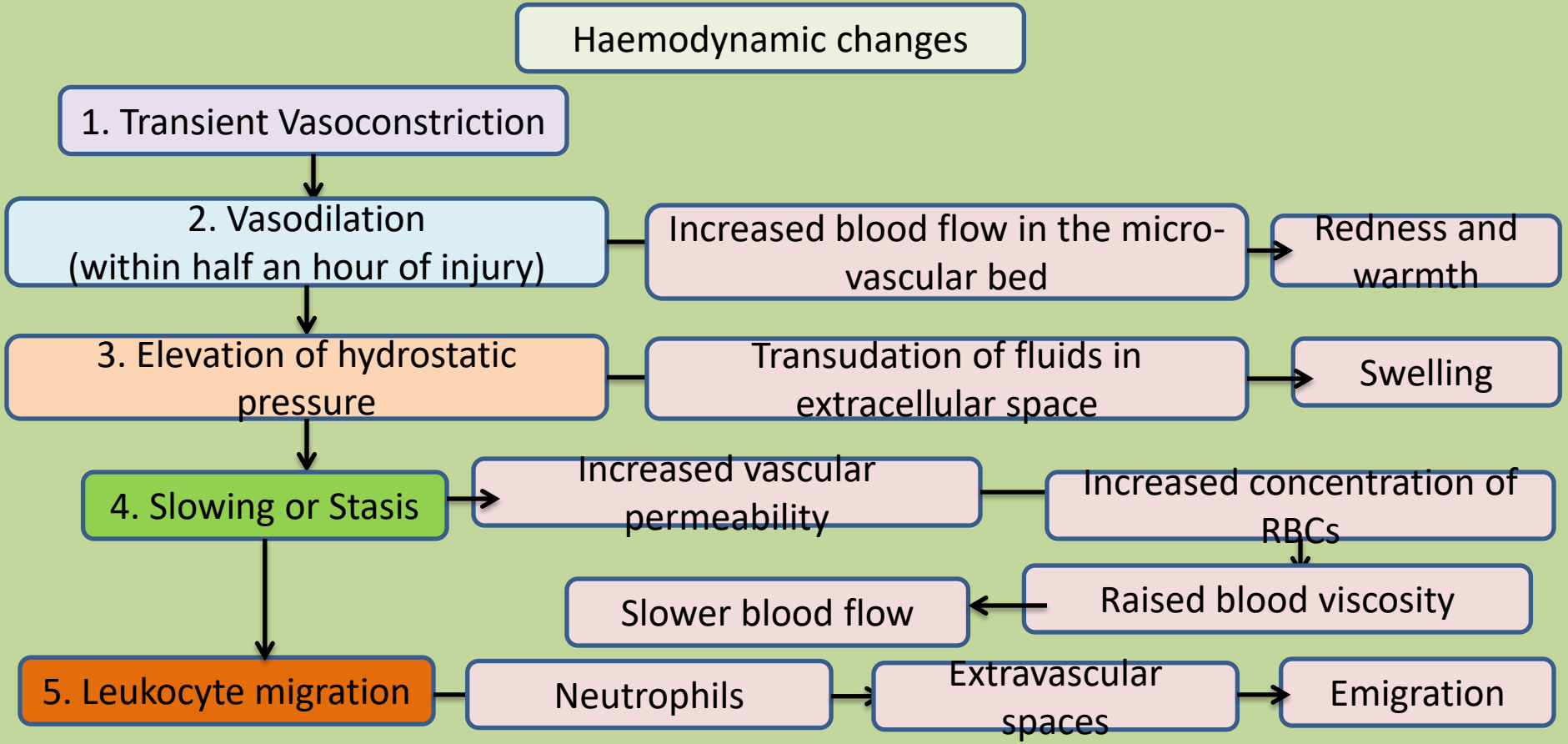

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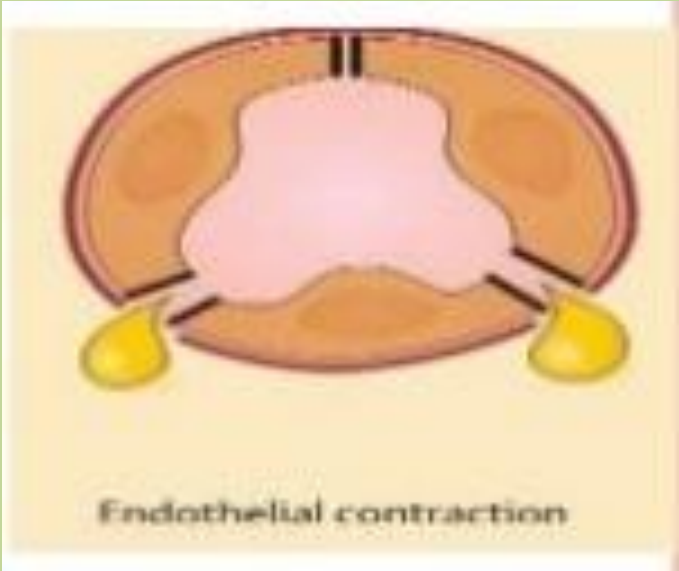
Mechanism Increased vascular permeability

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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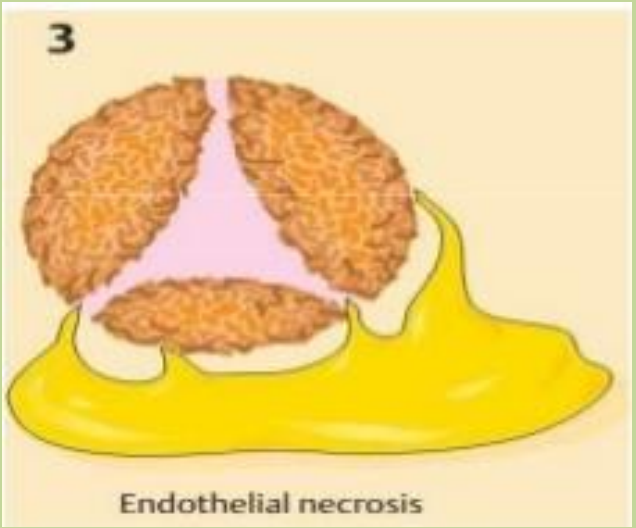
Mechanism Increased vascular permeability

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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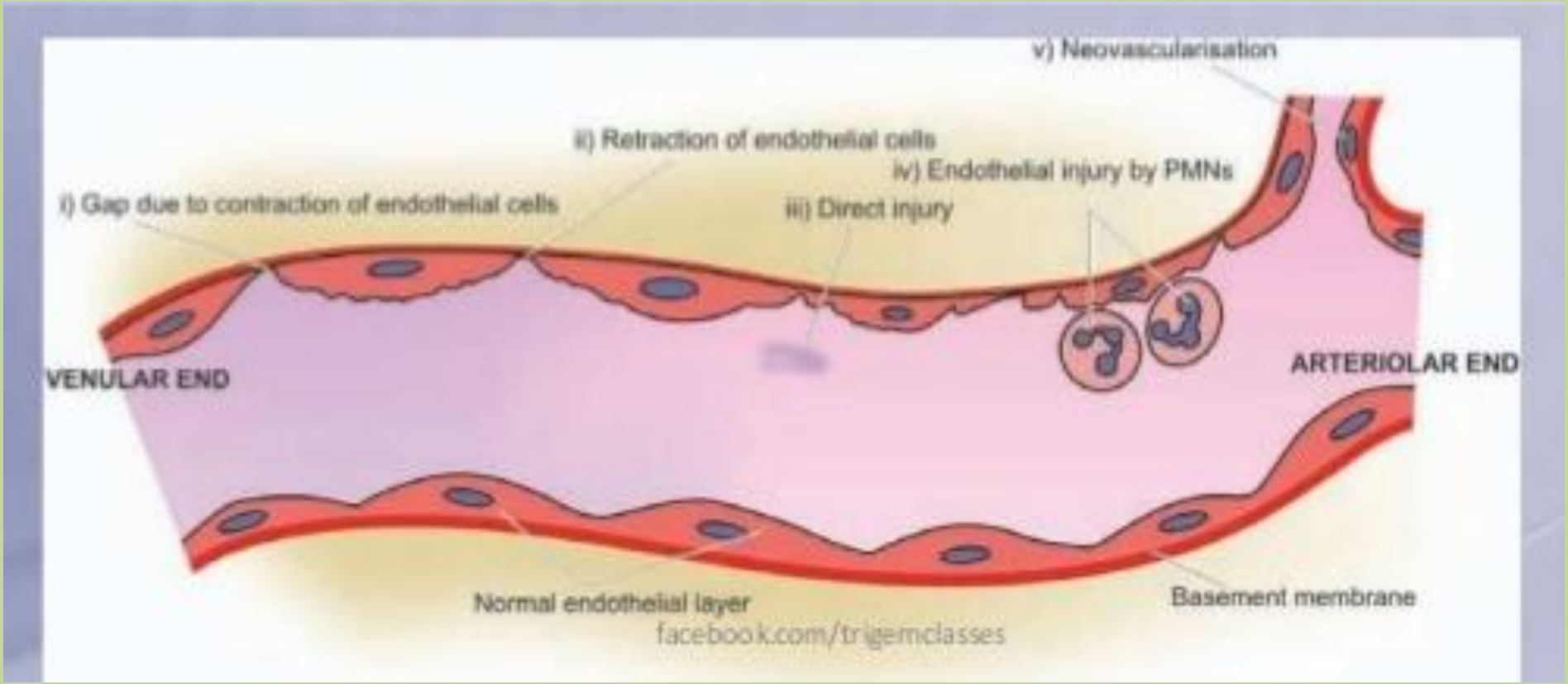

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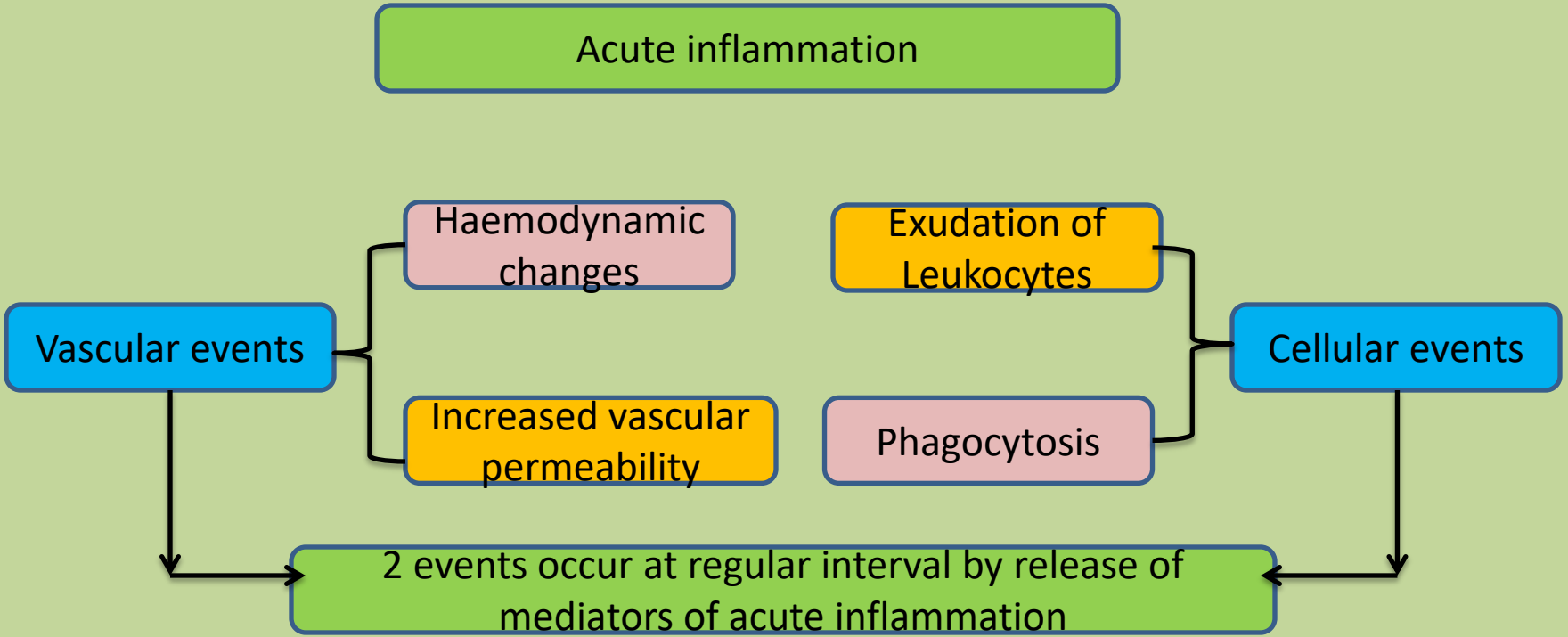

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Cellular events

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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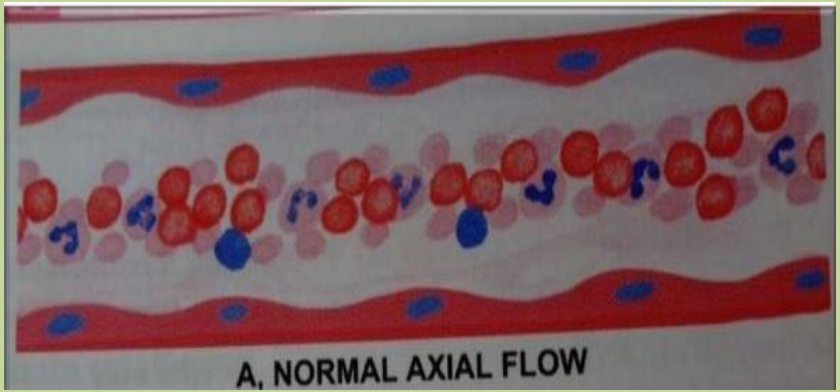

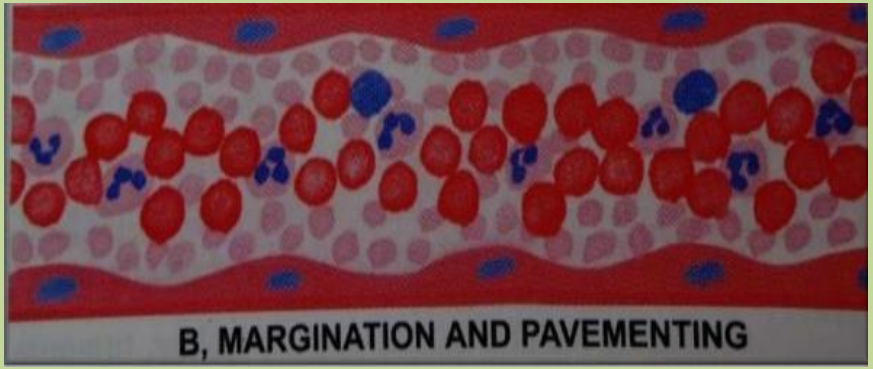

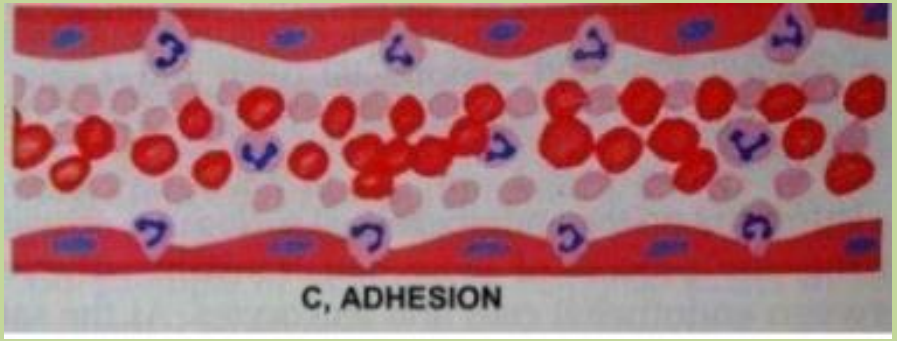

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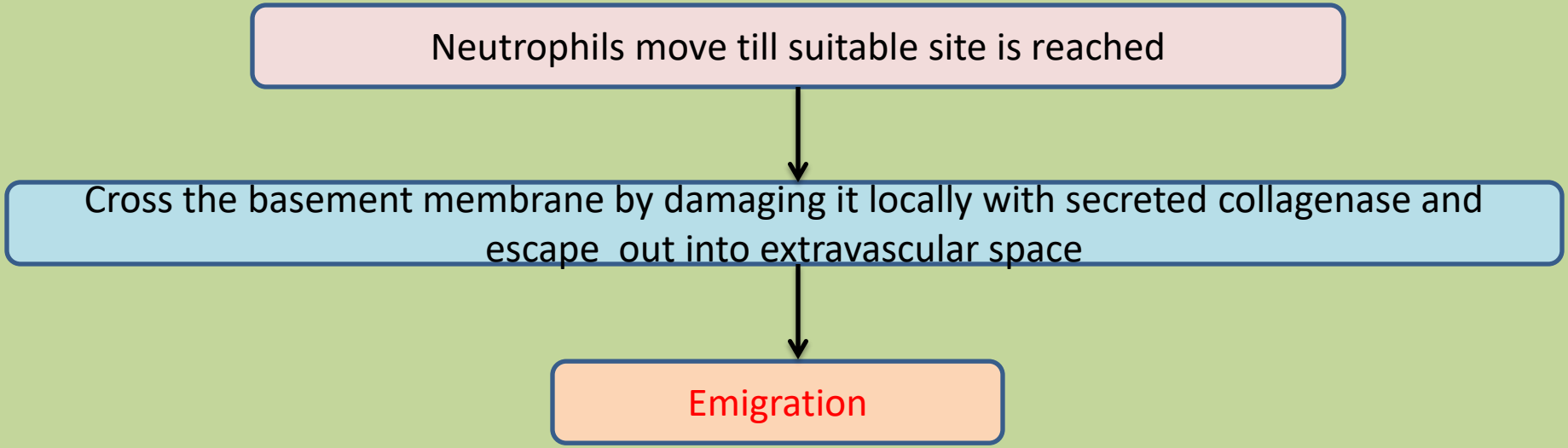

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Diapedesis: Simultaneously escape of RBCs takes place through the gaps between the endothelial cells

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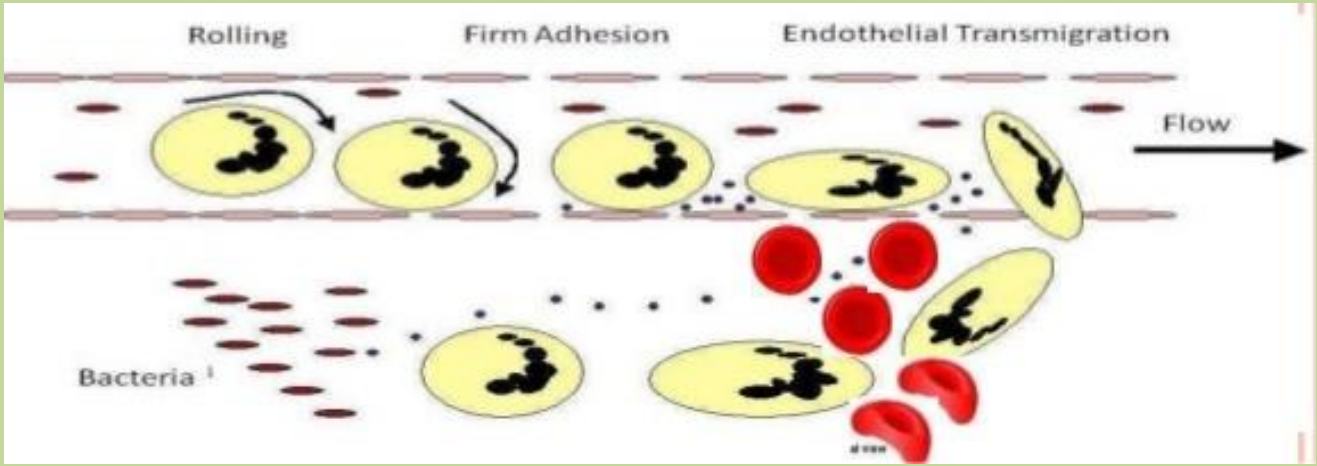
Diapedesis gives haemorrhagic appearance to the inflammatory exudate



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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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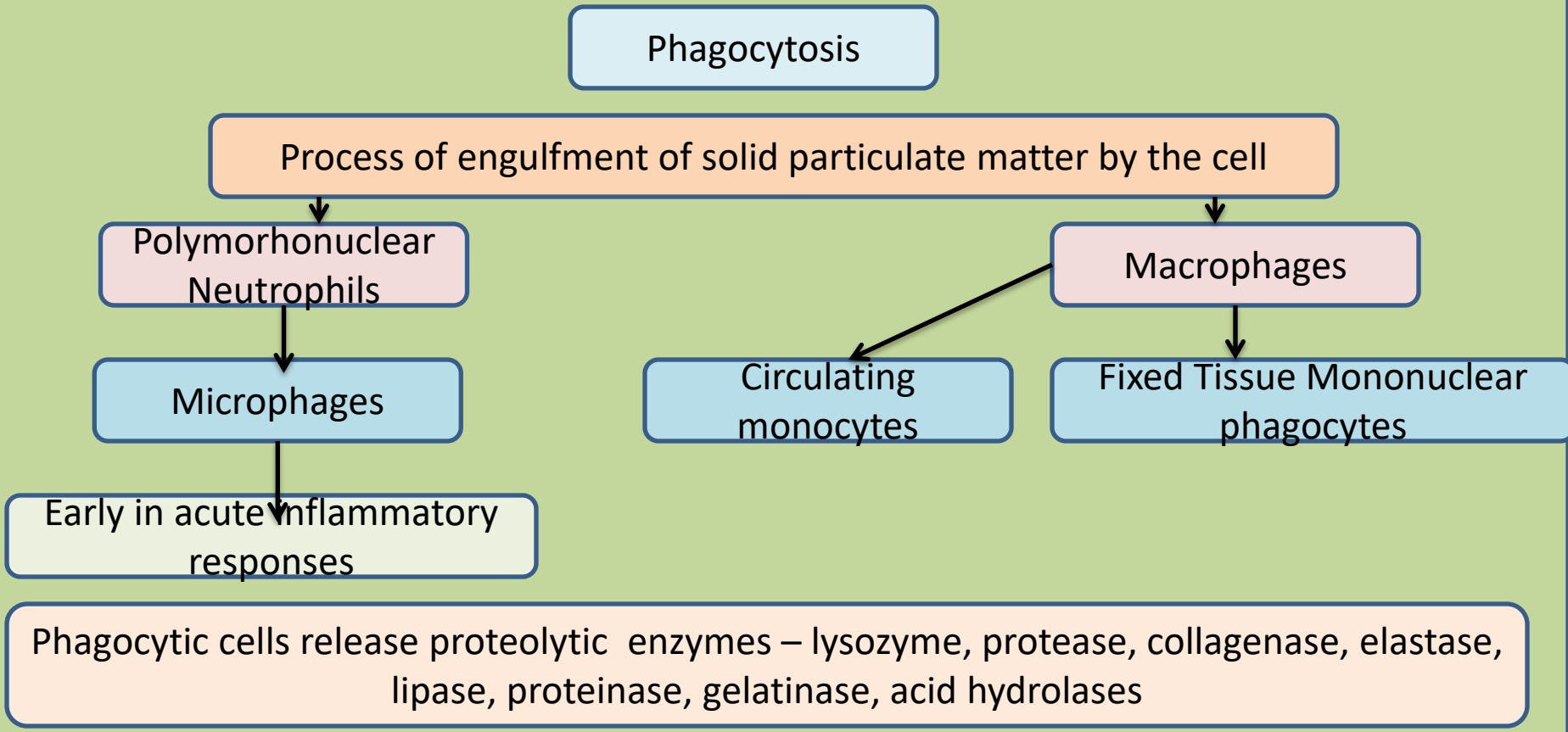

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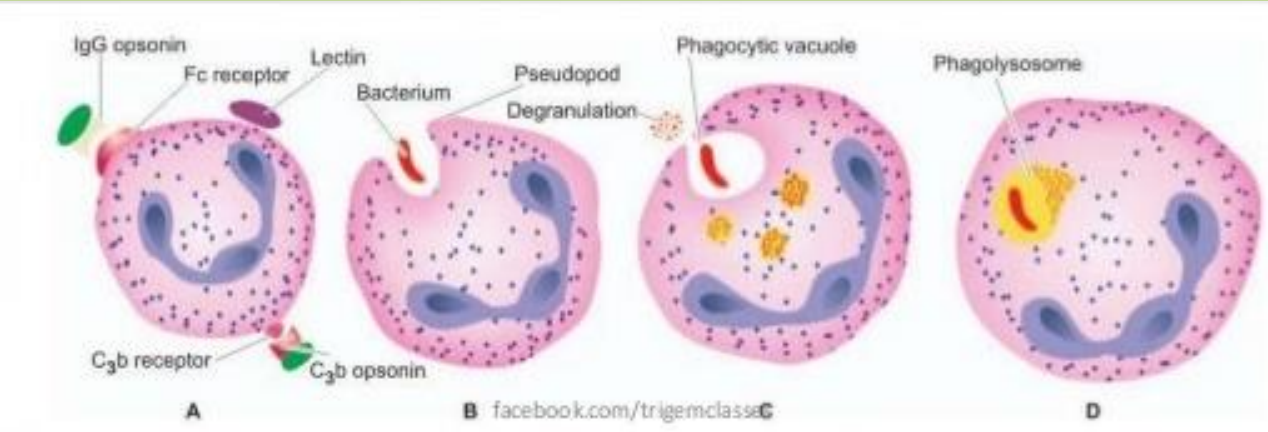
Phagocytosis

Recognition and Attachment

Engulfment

Killing and Degradation



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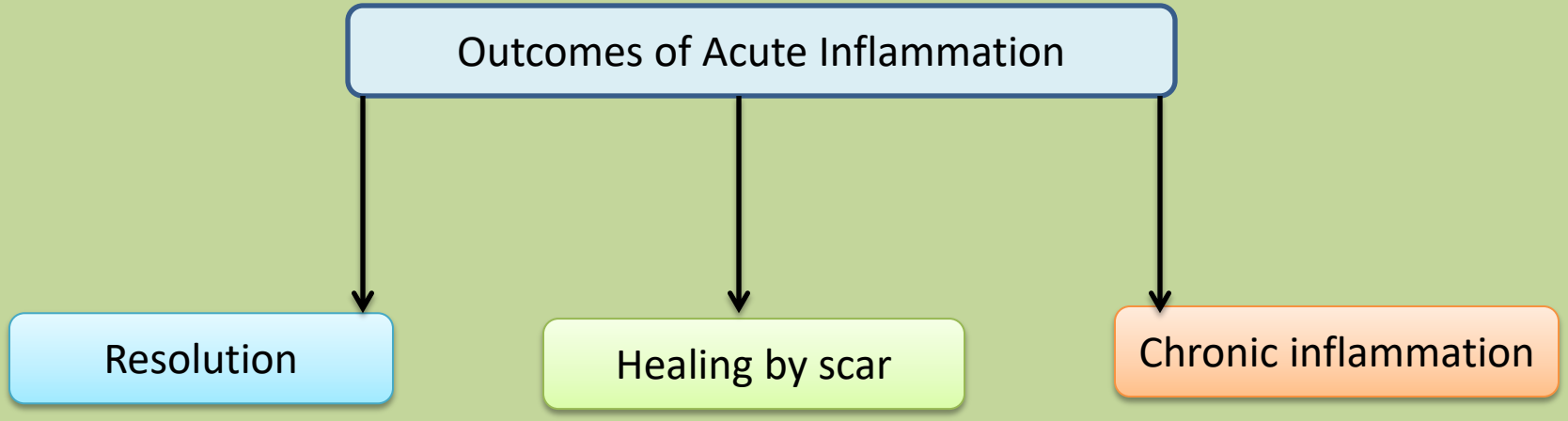

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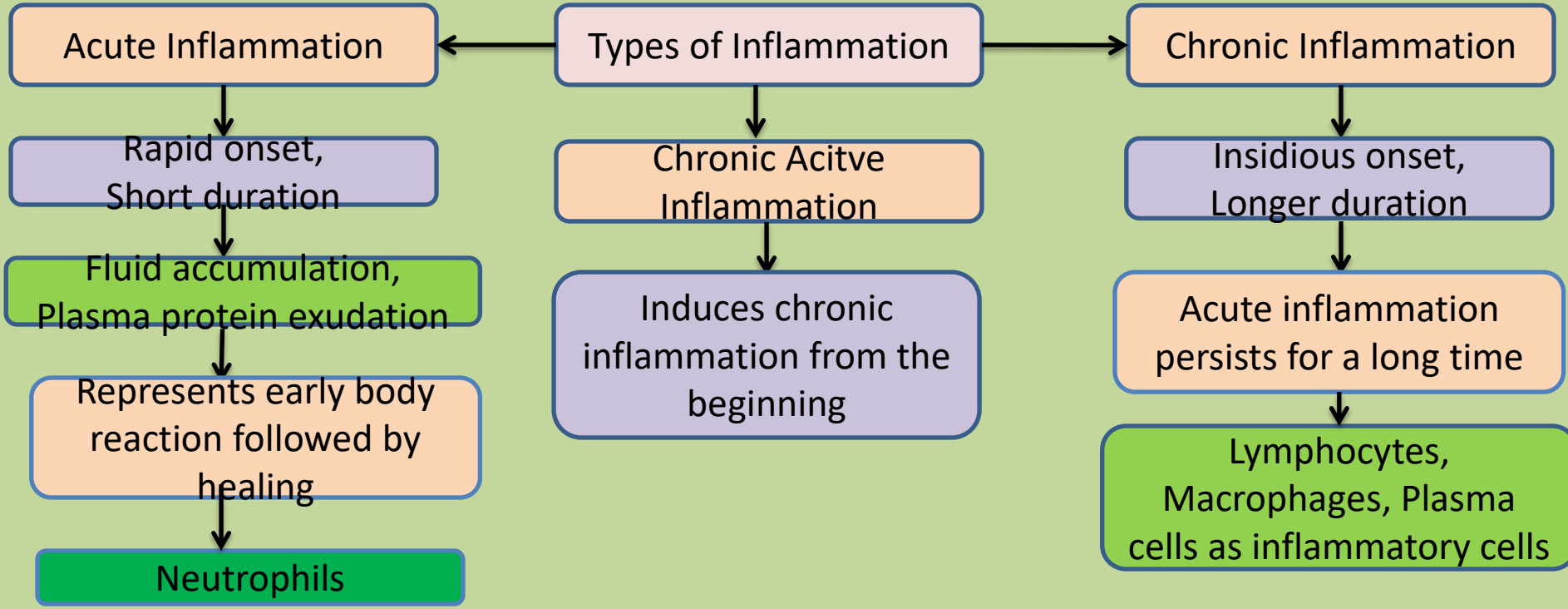

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Chronic Inflammation

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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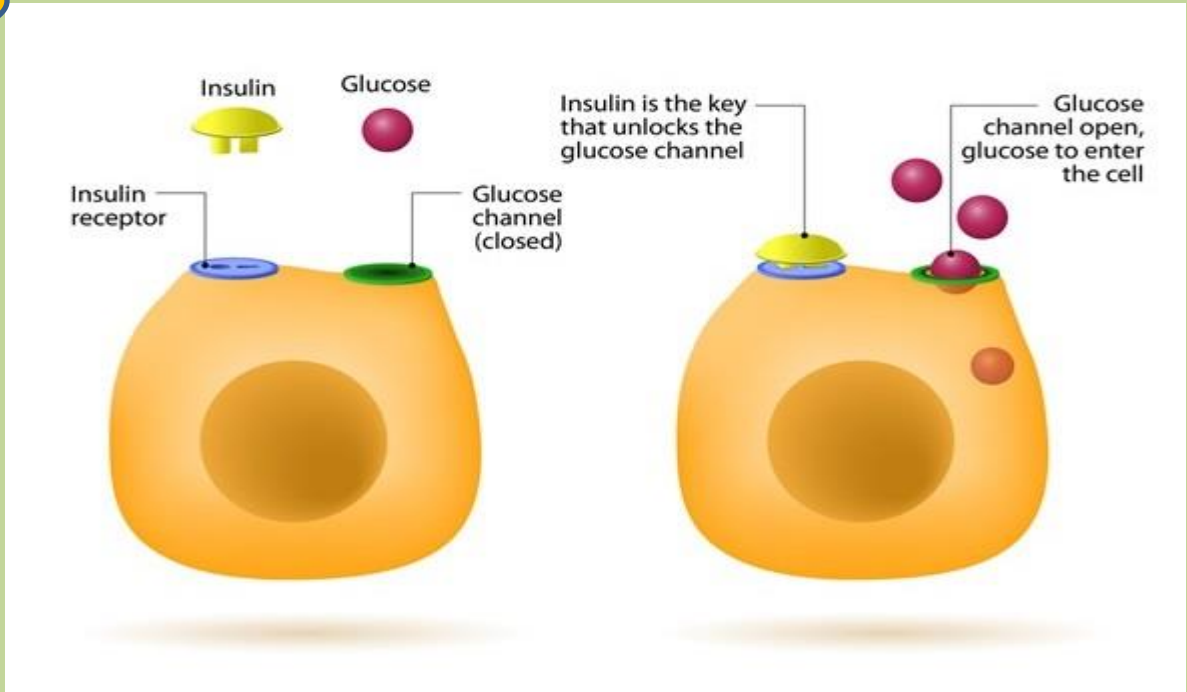
Metabolic disorders



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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, Angiogennesis

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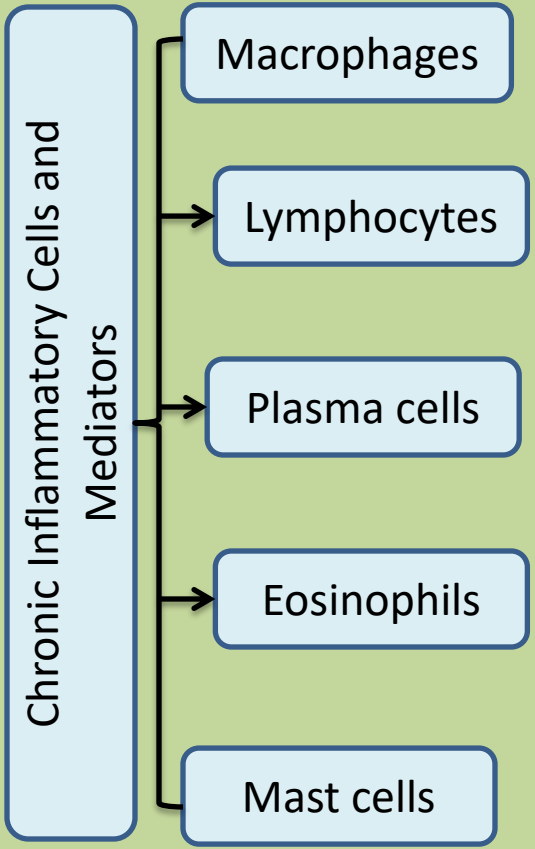

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Macrophages

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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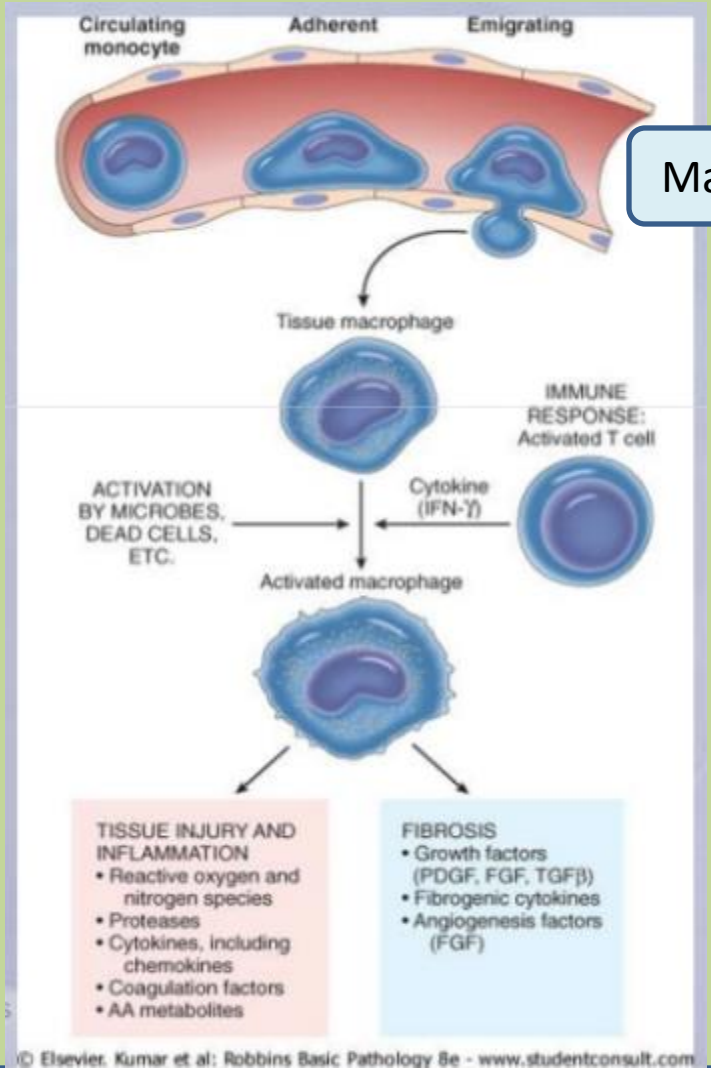
Macrophages



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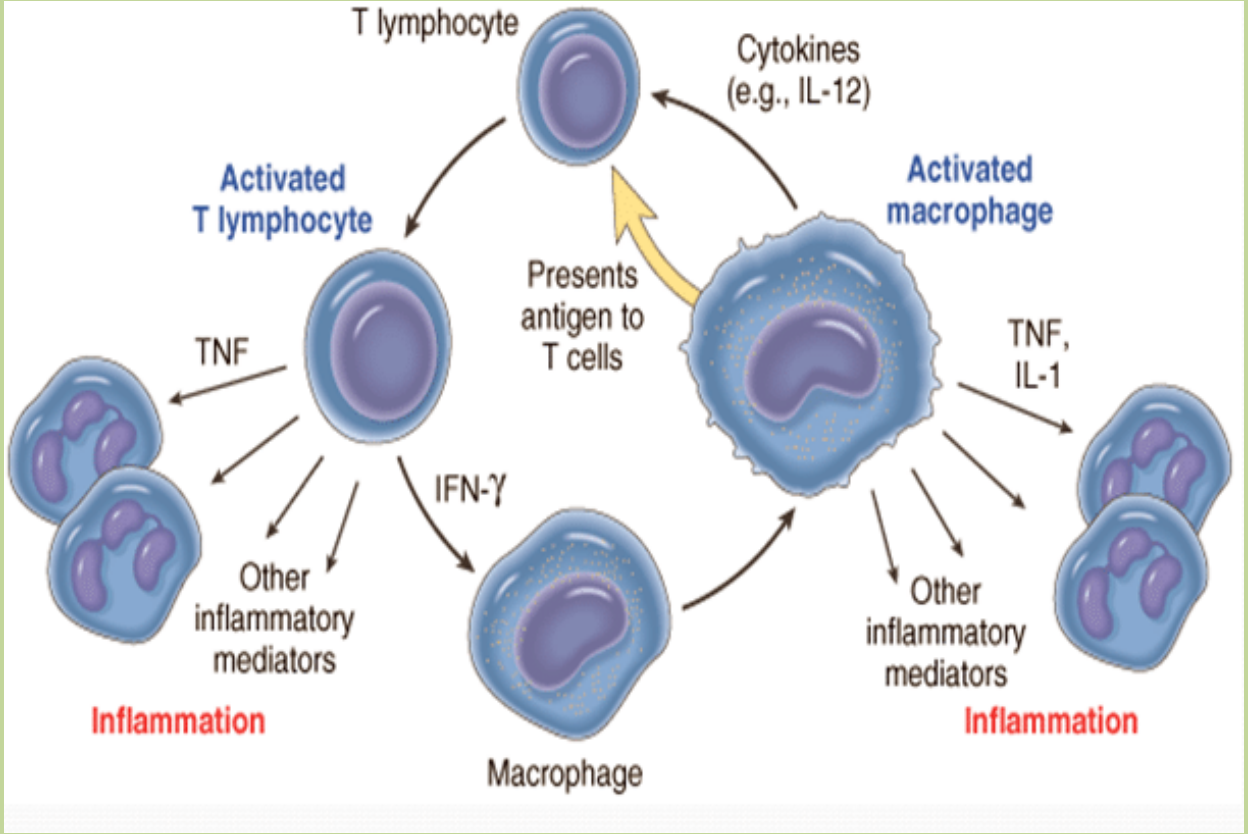

Chemokines

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Migration of
T and B lymphocytes

↓

Inflammatory site

Macrophages and T lymphocytes

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Bidirectional manner

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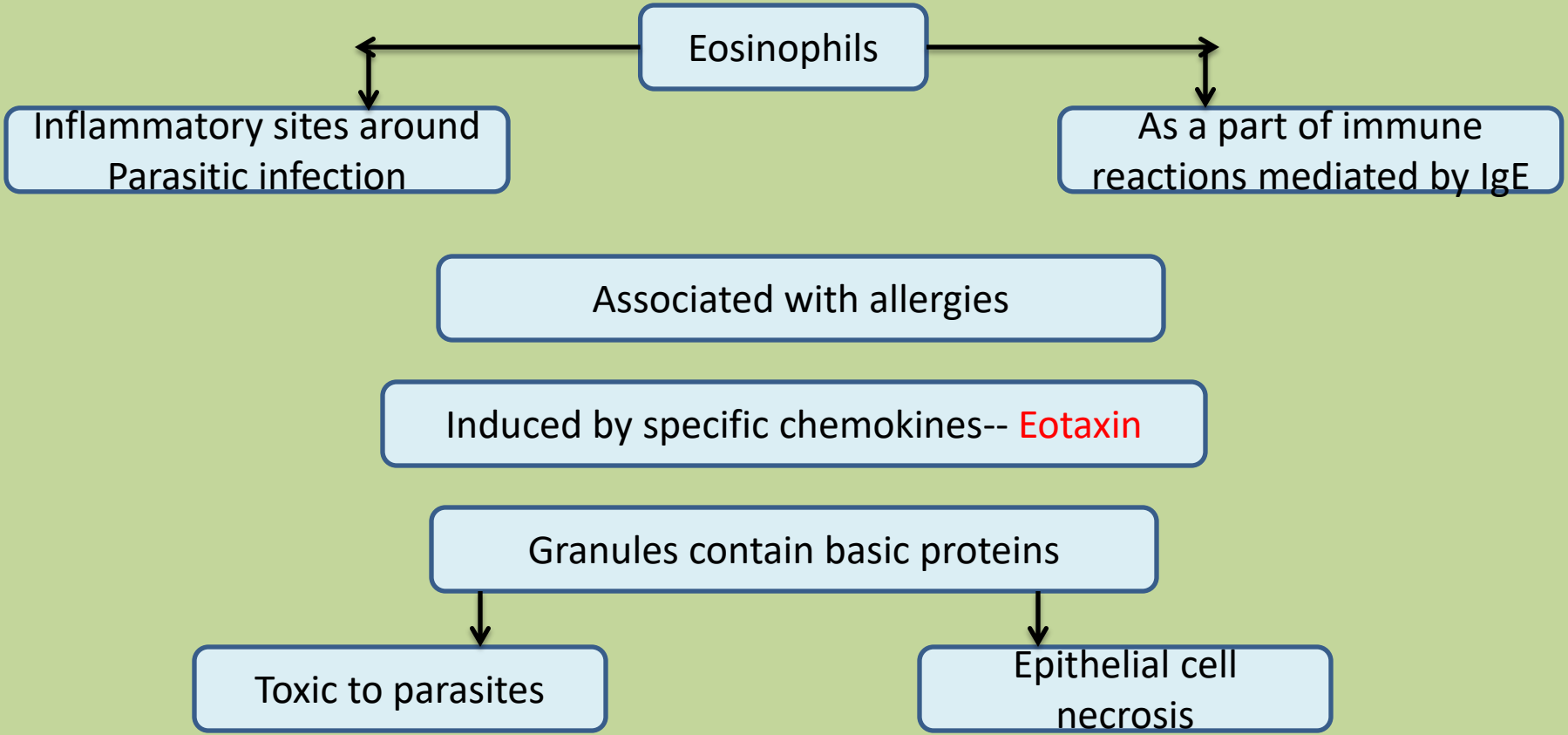

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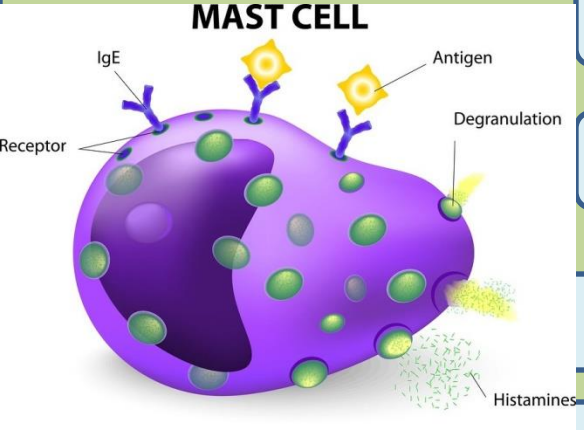
Mast cells



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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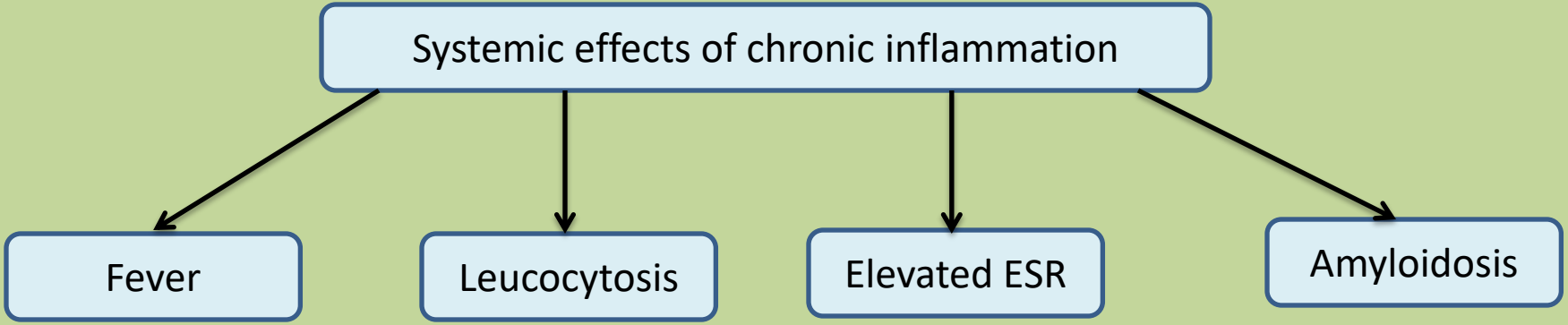

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- https://www.slideshare.net/DeepakKumarGupta2/inflammation-51778799

- 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