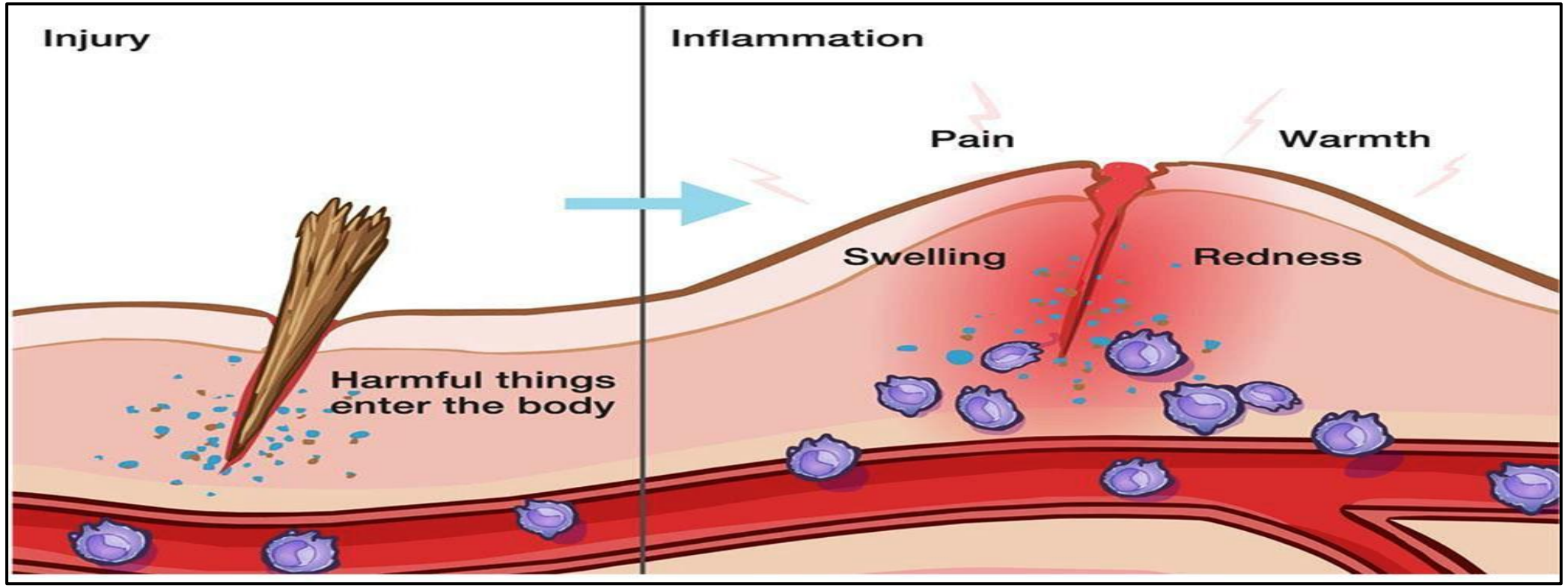
Inflammation and repair



Objectives

1. Define inflammation and explain its role and causes.
2. Compare between different types of inflammation.
3. Describe acute inflammatory reaction.
4. Differentiate between exudate and transudate.

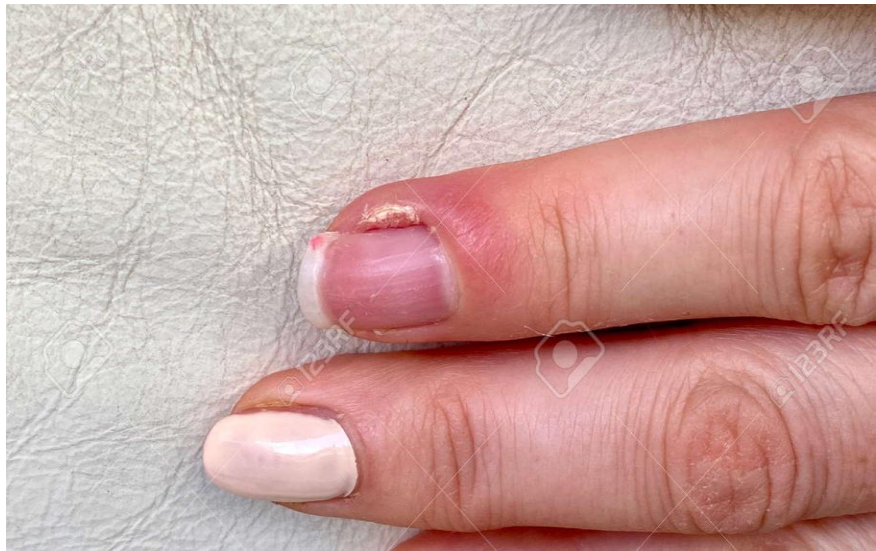
Inflammation

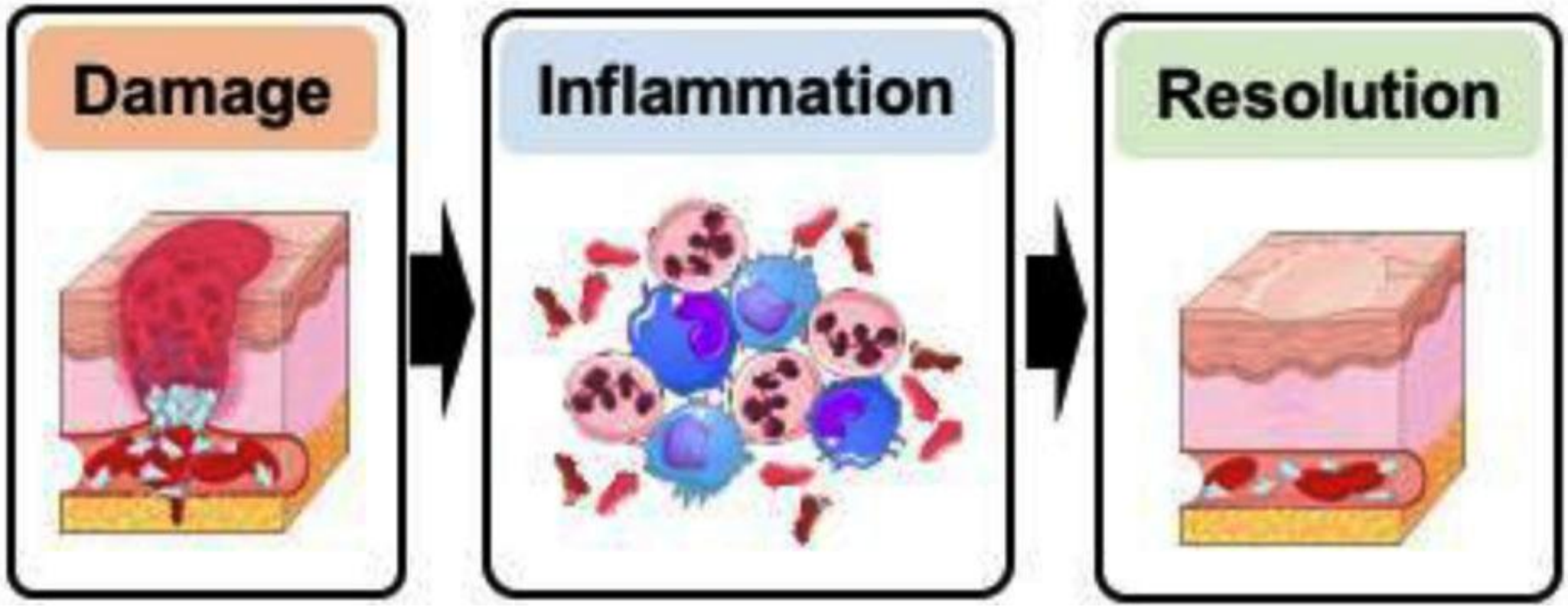
Definition:

Local changes (response) occur in vascularized living tissues when exposed to to an irritant (injury) in order to eliminate or limit the spread of injurious agent, remove necrotic cells and prepares the affected tissue for repair.

- **Local changes(response):**

in the form of vascular and cellular reactions.




Damage
Inflammation
Resolution

Roles of Inflammation:

1 - Isolation and elimination of the irritant

2 - Destruction of invading organisms and inactivation of toxins.

3-Achieving healing and repair.

Causes of inflammation

Inflammation is caused by injurious agents called irritants

Types of irritants:

- **Biological :** As bacteria and their toxins, viruses, fungi and parasites.

- **Physical agents:** Excessive heat or cold, radiation and electricity.
- **Chemical agents:** Acids, alkalis, organic and inorganic compounds.

- **Mechanical causes:** Wounds, crushing injuries, fractures and foreign bodies.
- **Immunological disturbances.**

Types of inflammation

Acute inflammation

Chronic inflammation

Subacute inflammation

Onset	**Rapid -gradual**
Course	**Regressive-progressive**
Duration	**Short –prolonged**

Types of inflammation

1- Acute inflammation: characterized by

- Rapid onset (Rapid tissue response).
- Short duration (days to weeks).
- High dose of irritant.
- presence of: Acute inflammatory cells + fluid exudate.

2- Chronic inflammation : characterized by

- Gradual onset(slow tissue response).
- Long duration.
- Low dose of irritant.
- presence of: Chronic inflammatory cells.

3-Subacute inflammation:

Grades in-between acute & chronic

Nomenclature of inflammation:

The Greek, Latin or English name of the organ + suffix “itis” as:

- **Gastritis:** inflammation of the stomach.
- **Glossitis**: inflammation of the tongue.
- **Hepatitis**: inflammation of the liver.
- **Appendicitis:** inflammation of appendix.

Exceptions:

- **Pneumonia =** inflammation of the lung
- **Pleurisy =** inflammation of the pleura.

Acute inflammation

- **Definition:**

Immediate response of living tissue to an injury. Its aim is to deliver leukocytes and mediators to the site of injury by the blood stream.

- **Cardinal signs of acute inflammation:**

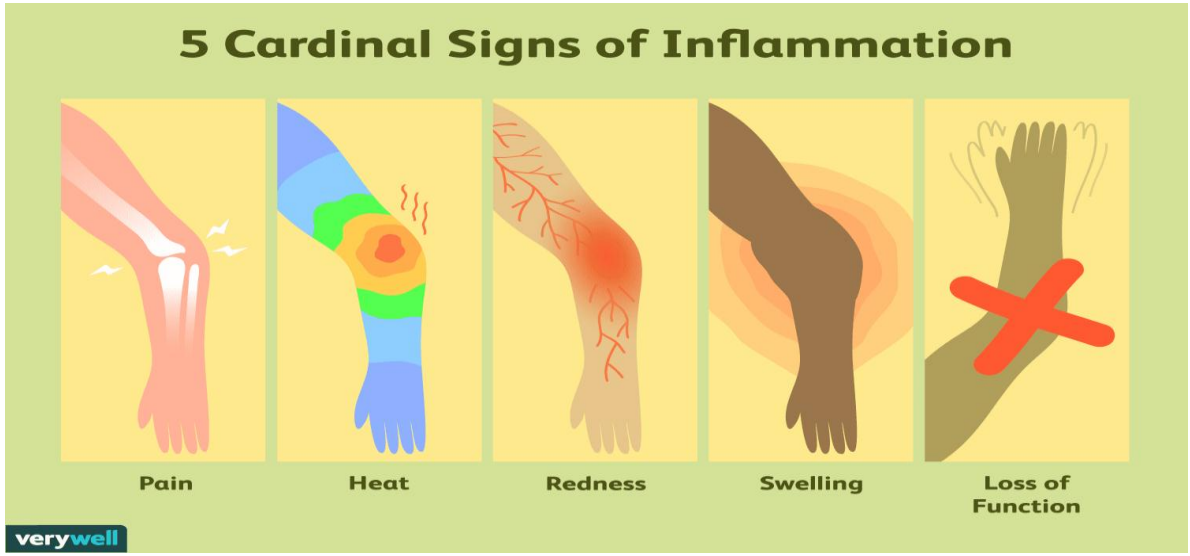
1- Redness

2- Hotness

3- Pain

4- Swelling

5- Loss of function



Acute inflammatory reactions

(A) **LOCAL REACTIONS:**

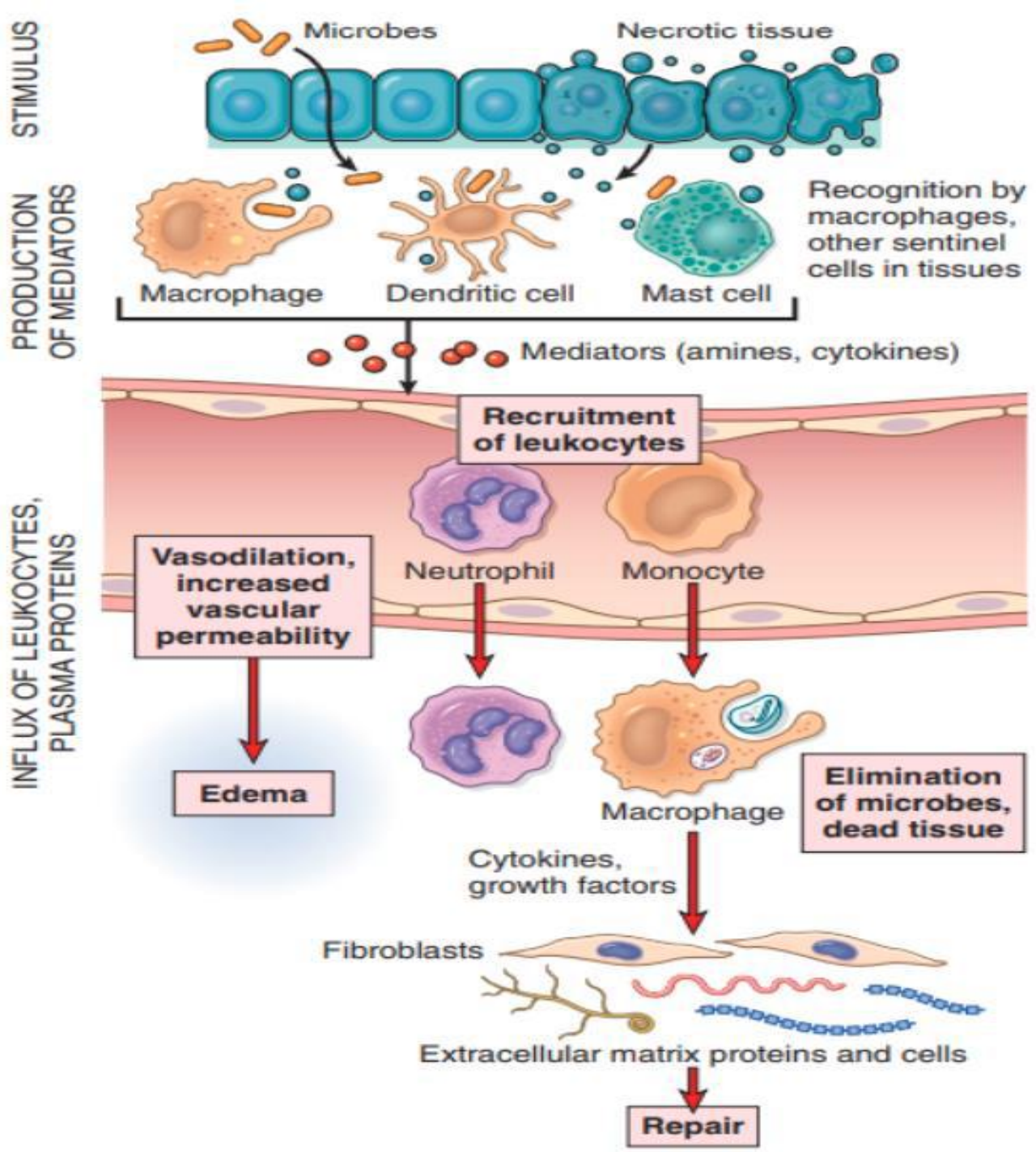
1. Local tissue destruction.
2. Vascular response.
3. Cellular responses.

(B) SYSTEMIC REACTIONS:

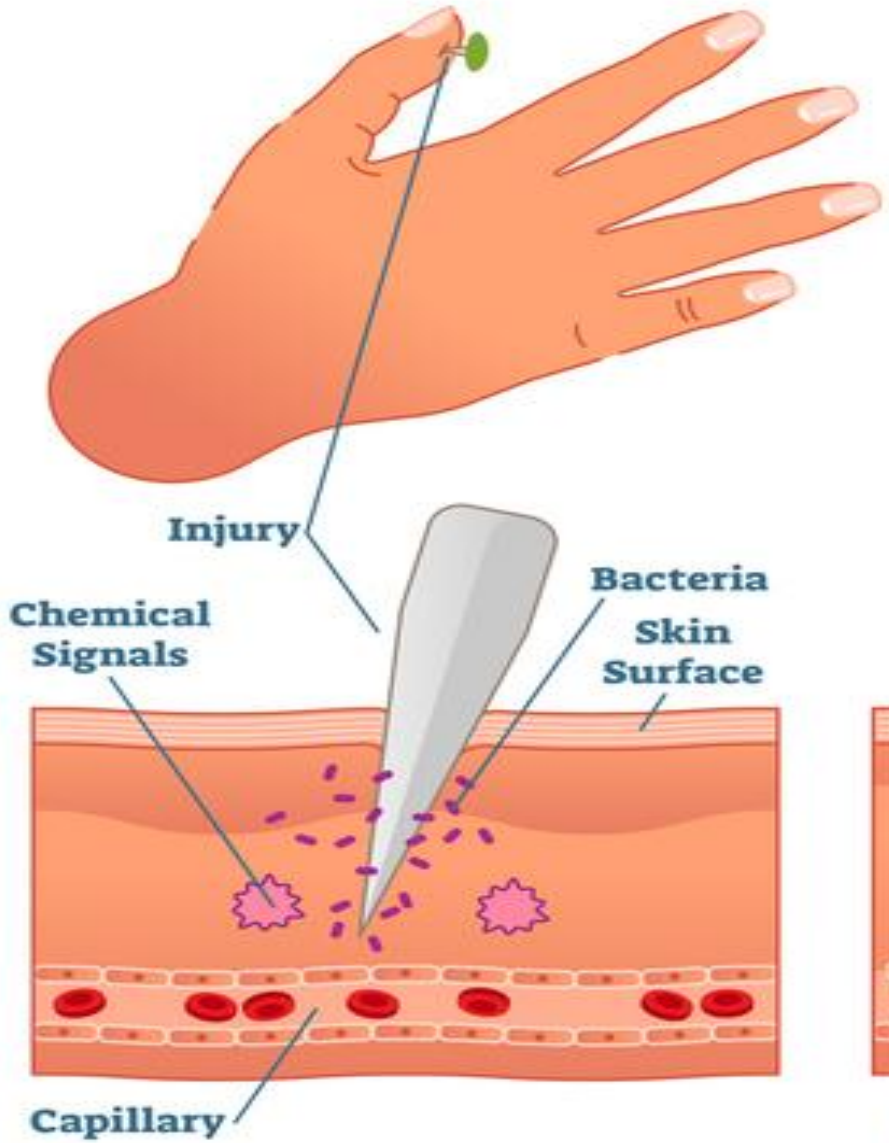
1. Changes in blood cells.
2. Acute phase reaction.
3. Changes in organs.

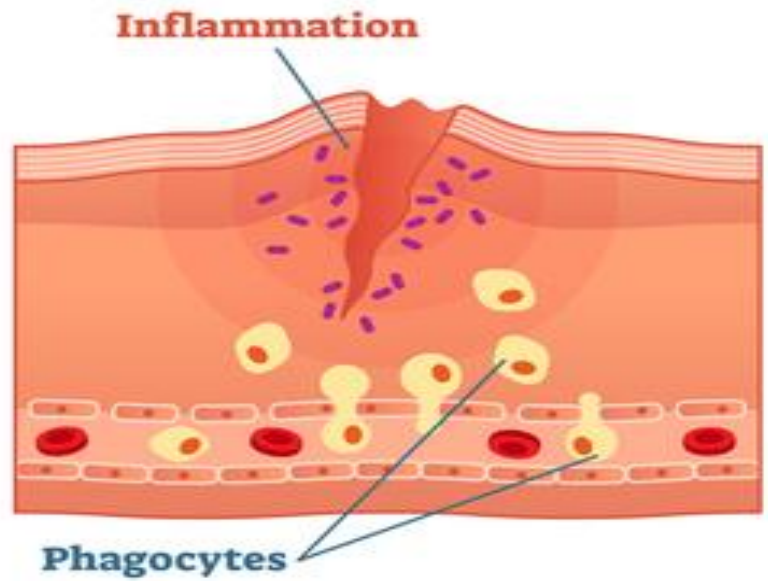
4 Stages of INFLAMMATION

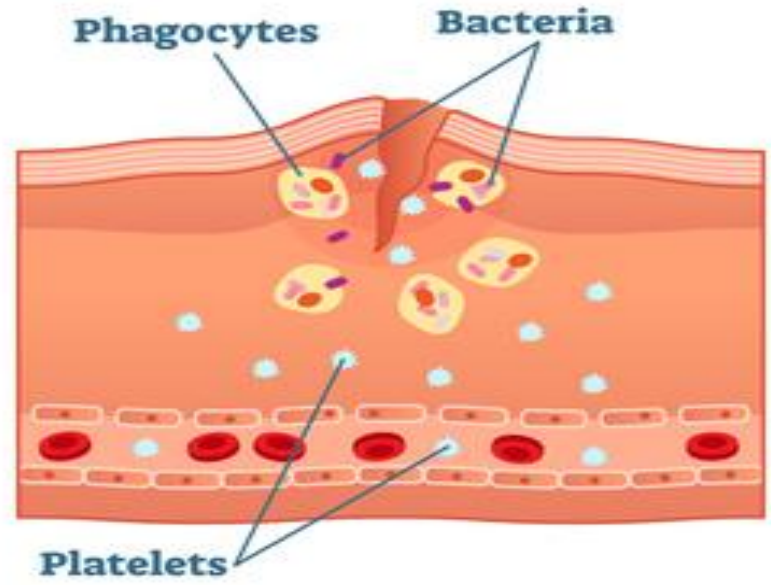
1. Trigger & Alarm
2. Vascular & Fluid Response
3. Cellular Cleanup
4. Resolution & Repair



INJURY INFLAMMATION







Tissue Injury
Release of chemical signals (Histamine)

Dilation and Increased Leakiness of Capillary
Phagocytes Migrate to the Area

Phagocytes Consume Bacteria and Cell Debris
Platelets Move Out of the Capillary to Seal the Wounded Area

Acute inflammatory reactions

LOCAL REACTIONS:

- **Local tissue destruction and release of chemical mediators :**

- The tissue damage is maximum at the site of the irritant and depends on its severity.

- Cells at the site of the irritant are killed (necrotic), The surrounding cells are less severely affected.

- Substances released from necrotic cells together with the inflammatory irritant trigger the *release* and *activation* of chemical substance called chemical mediators

-**Chemical mediators** may be produced locally by cells at the site of inflammation (e.g.; macrophages-mast cells).

-**Chemical mediators** as histamine are rapidly released from intracellular granules of mast cells, other mediators are synthesized de novo e.g., prostaglandins and leukotrienes, cytokines in response to a stimulus.

-The inflammatory reaction *is initiated* and regulated by chemicals that are produced at the site of the reaction.

-These chemical mediators play an important role in promoting the vascular and cellular changes in the inflamed area.

▪Vascular response:

1-Transient vasoconstriction:

-Due to *direct stimulation* of the vascular wall of arterioles by the irritant (e.g toxin).

-Vasoconstriction lasts for seconds to minutes only.

2-Vasodilatation: Occurs due to

-Local axon reflex .

-*Action of chemical mediators* as histamine (vasodilator substance) on vessel walls.

- It affects arterioles, capillaries and venules.
- Results in: increase the blood flow (hyperemia) to the inflamed area.

3-Increased vascular permeability and formation of exudate.

Mechanism:

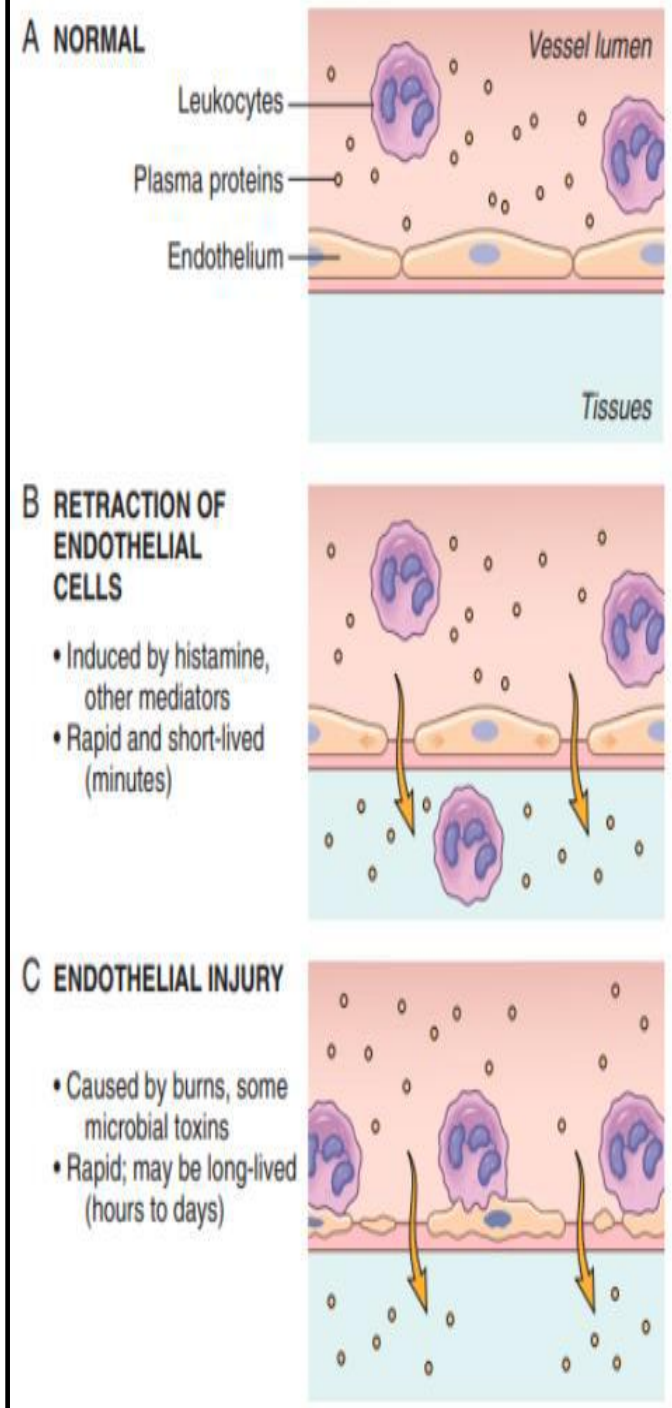
- Endothelial cell contraction and retraction *by the effect of chemical mediators* leads to widening of the intercellular junctions creating *gaps* between the endothelial cells.
- This leads to vascular leakage and extravasation of fluid followed by cellular exudate.

4- Slowing of blood stream (stasis):

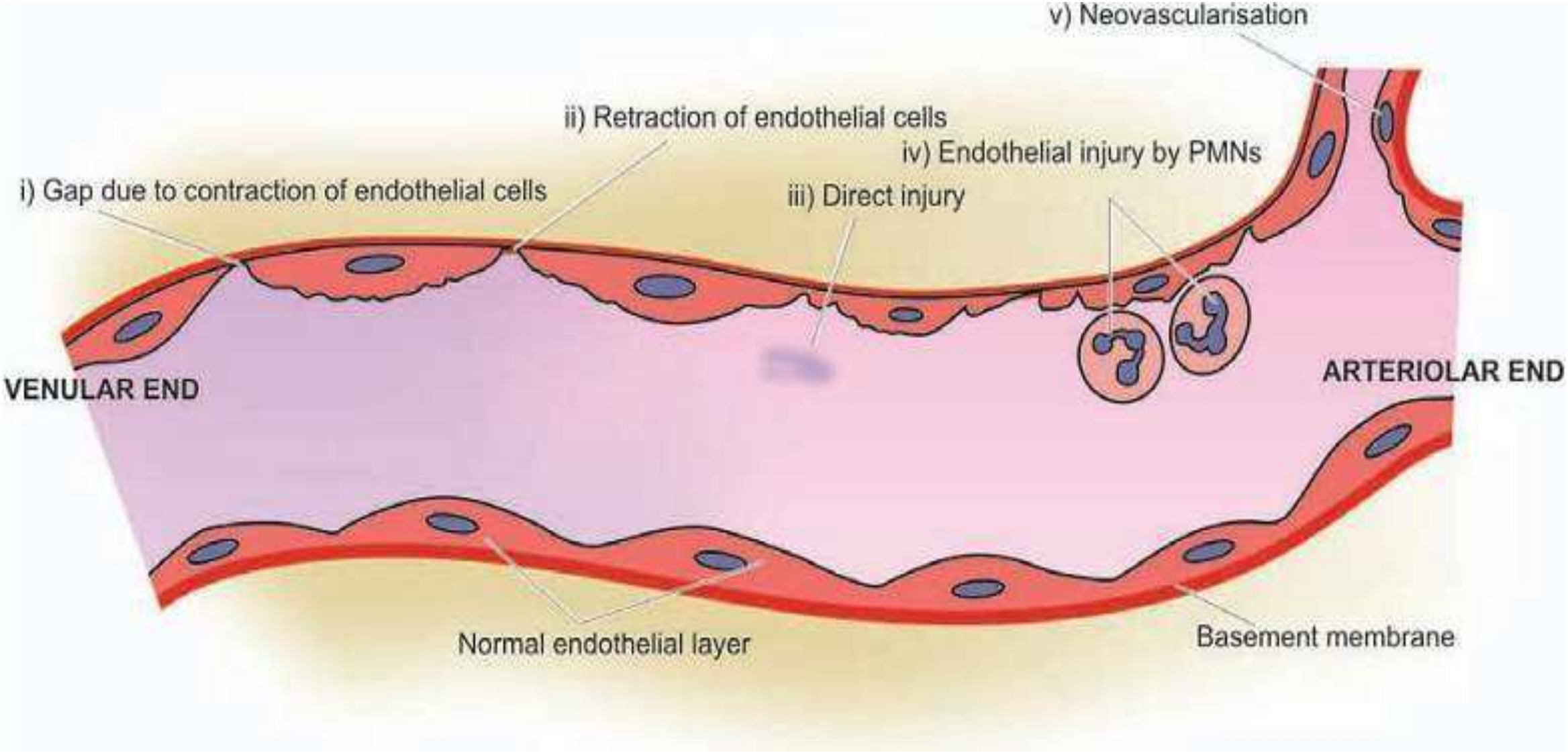
local slowing of the circulation in the injured due to:

- Increased blood viscosity due to vascular leakage (most important cause)

Aim: Allows leukocytes exudation (cannot leave circulation if the blood flow is rapid)

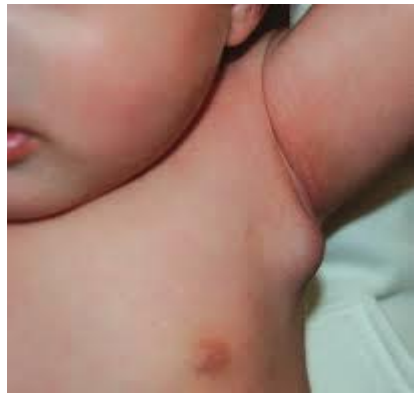


Endothelial cell contraction and retraction



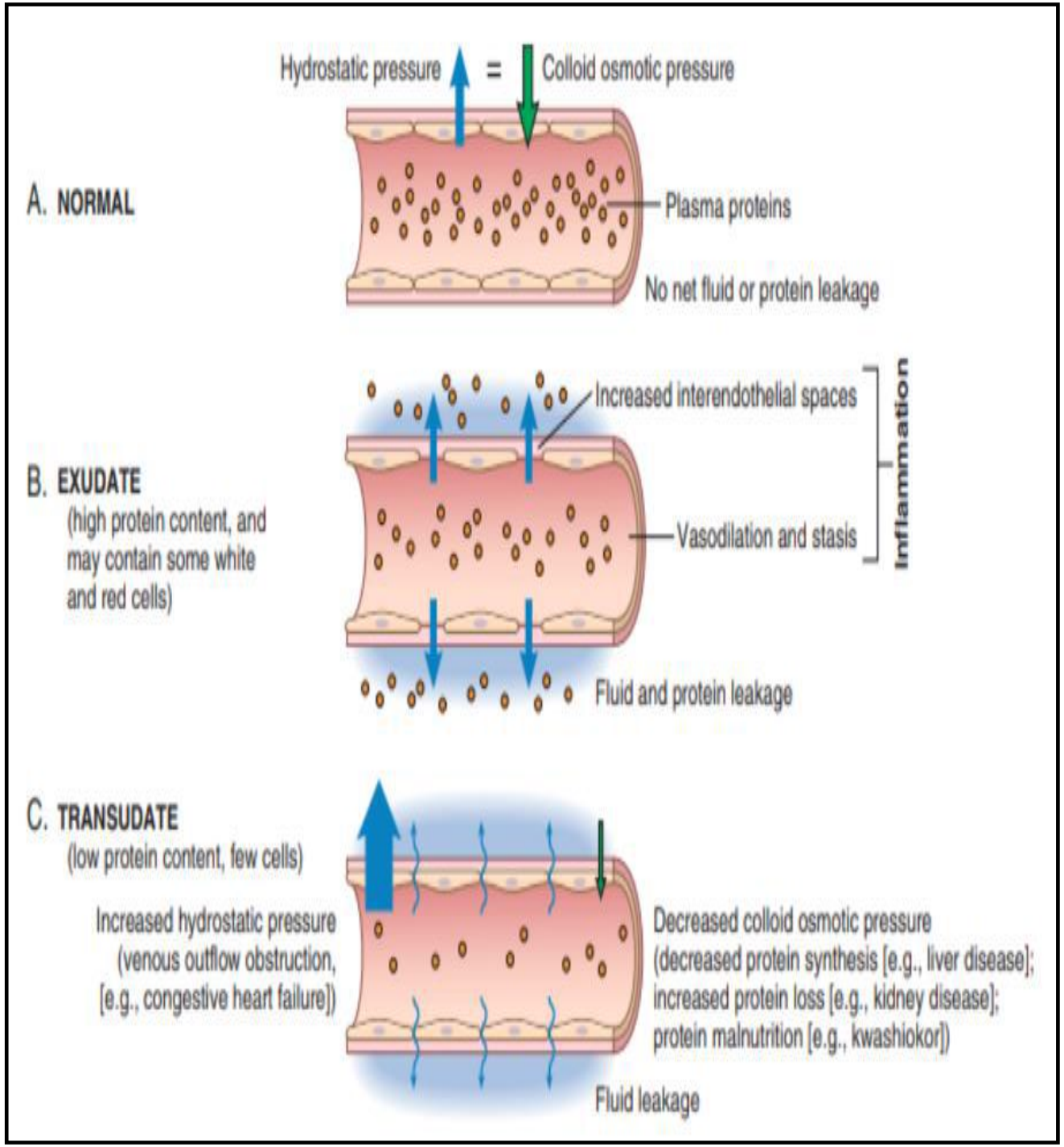
5.Dilatation of lymphatics:

- Because of increased vascular permeability. lymph flow is increased to help drainage of excess extravasated fluid escaped to the interstitial tissue back to blood.
- In addition to fluid, leukocytes and cell debris, as well as microbes, may find their way into lymph.
- The lymphatics may become secondarily inflamed (lymphangitis), as may the draining lymph nodes (lymphadenitis).

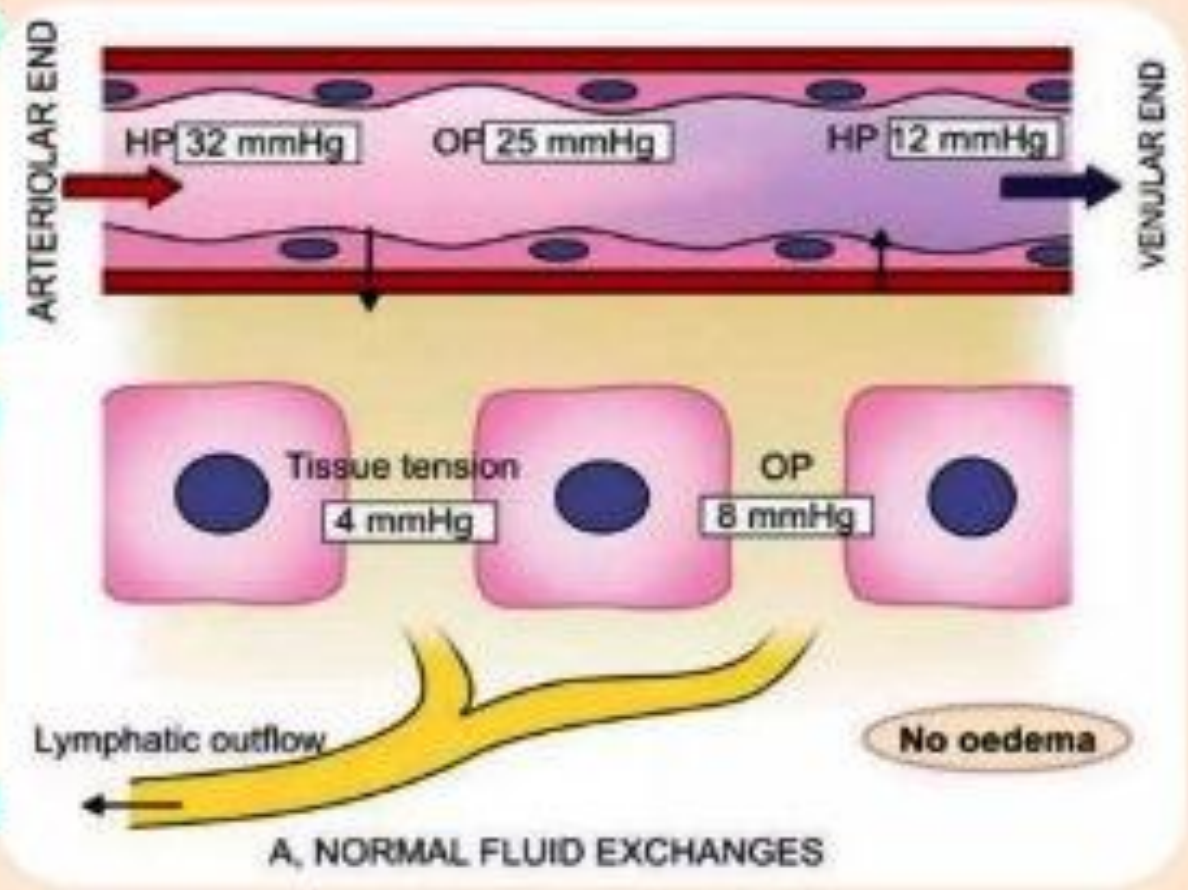


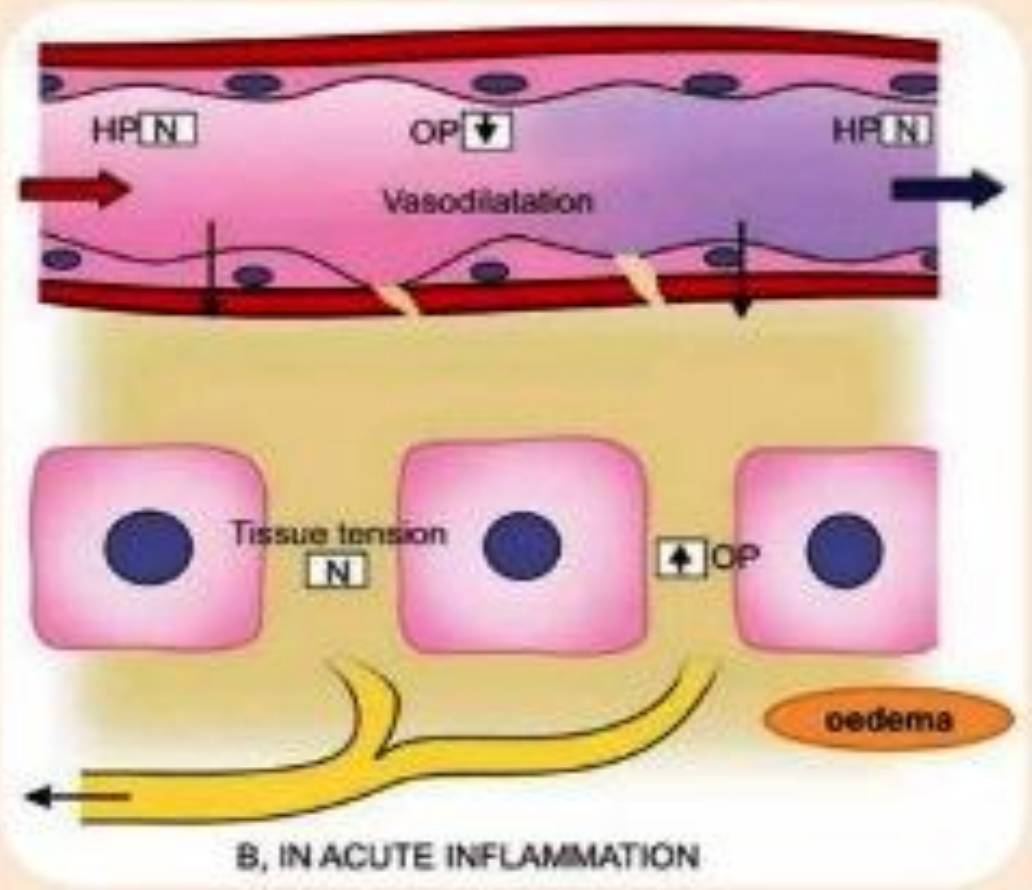
Reactions of Blood Vessels in Acute Inflammation

- Changes in vessel size (and consequently, flow).
- Increased vascular permeability.
- Formation of the fluid exudate .



Fluid exchange between blood & extracellular fluid;





Inflammatory fluid exudate

MECHANISM OF FORMATION:

1.Increase capillary hydrostatic pressure due to increased blood flow.

2. Increased capillary permeability due to:

- vasodilatation
- Endothelial retraction (pores) Leads to leakage of fluid rich in plasma proteins .

3. Increased osmotic pressure of interstitial tissue due to tissue protein breakdown by the irritant.

Composition of Inflammatory fluid exudate

1. High protein content (4-8 gm%).

2. High specific gravity (above 1018).

3. Turbid and yellowish because of the presence of large numbers of leukocytes.

Difference between exudate & transudate

Item	Exudate	Transudate
specific gravity	above 1018	Below 1018
colour	Straw ,yellow	Pale yellow
Aspect	Turbid	Clear
protein content	4-8 gm%.	Below 4 gm%
Fibrin	Rich	Absent
Coagulation	Present	Absent
cells	Rich in inflammatory cells	Minimal or absent

Functions of Inflammatory fluid exudate :

1. **Dilution** of the toxins to minimize their effects on the cells.

2. Brings **fibrinogen**, that changes into a fibrin network, which:
   - Helps movement of acute inflammatory cells to the site of the irritant.
   - **Localization** of the area of inflammation thereby localizes infection.
   - Acts as a framework for proliferation of fibroblasts to start repair.

3. **Transport** antibodies of different types to the site of inflammation

- https://www.youtube.com/watch?v=9bvMv5dQ7RU

Thank you