INFLAMMATION

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LESSION PLAN

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- DEFINITION
- CAUSES
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- ACUTE INFLAMMATION
- CHRONIC INFLAMMATION

INTRODUCTION

"The word inflammation means burning".

When a wound swells up, turns red and hurts, it may be a sign of inflammation.

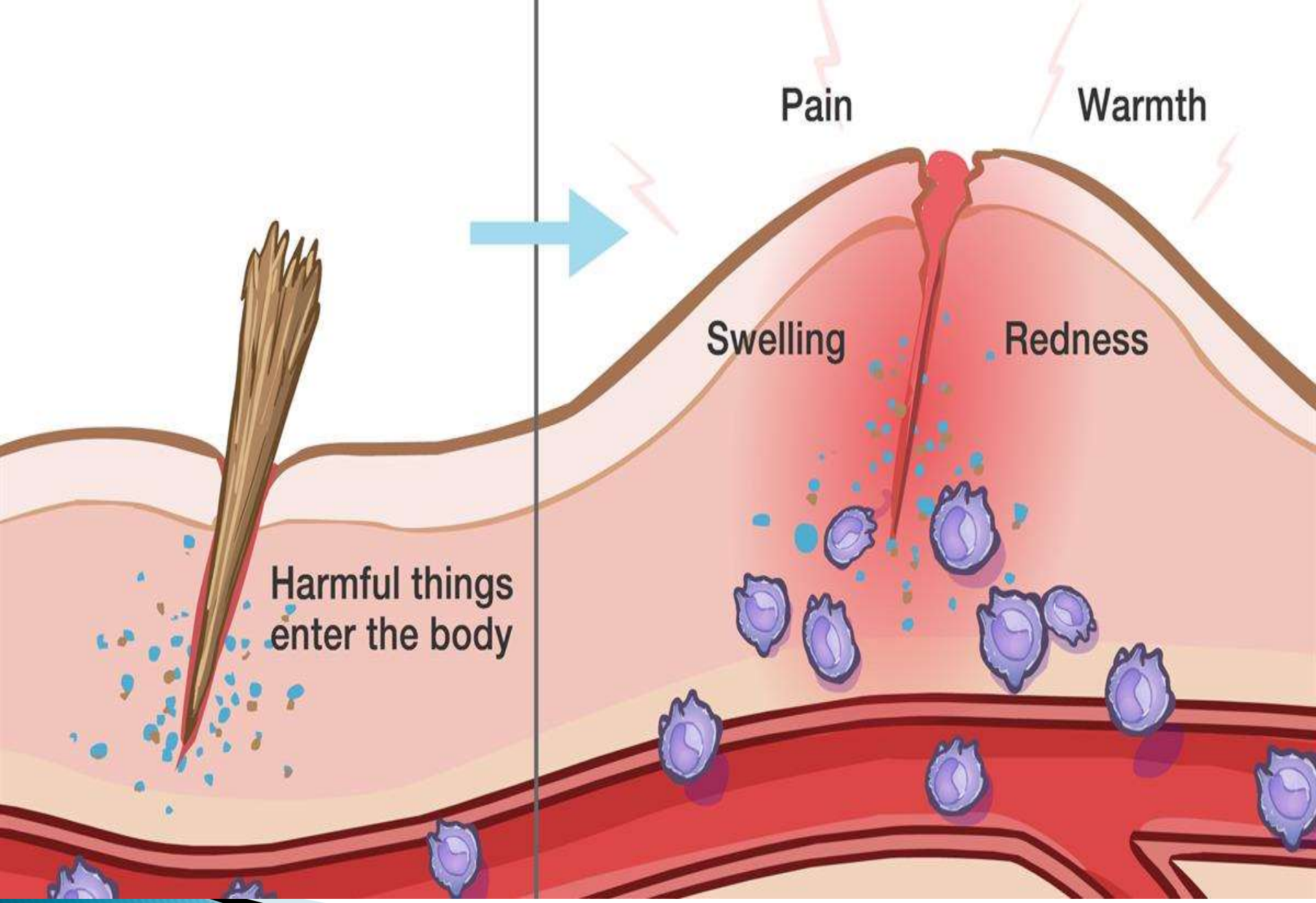
Very generally speaking, inflammation is the body's immune system's response to an irritant.

- inflammation is the visible response to an immune reaction, and activation of immune response is almost essential before inflammatory response appears.

- inflammation is a protective response by the body to variety of etiologic agents (infectious or non-infectious).

- *Inflammation involves 2 basic processes-*

early inflammatory response and later followed by healing


Pain
Warmth
Swelling
Redness
Harmful things
enter the body

DEFINITION

"Inflammation is defined as the local response of living mammalian tissues to injury due to any agent."

OR

It is a body defense reaction in order to eliminate or limit the spread of injurious agent, followed by removal of the necrosed cells and tissues.

CAUSETIVE AGENTS

1. Physical: Heat, cold, radiation, mechanical trauma.

2. Chemical: Organic and Inorganic Substances.

3. Infective(Microorganism): Bacteria, viruses and their toxins, fungi, parasites.

4. Immunological: Cell-mediated and antigenantibody reactions.

5. Inert Materials: Foreign bodies.

SIGNS OF INFLAMMAION

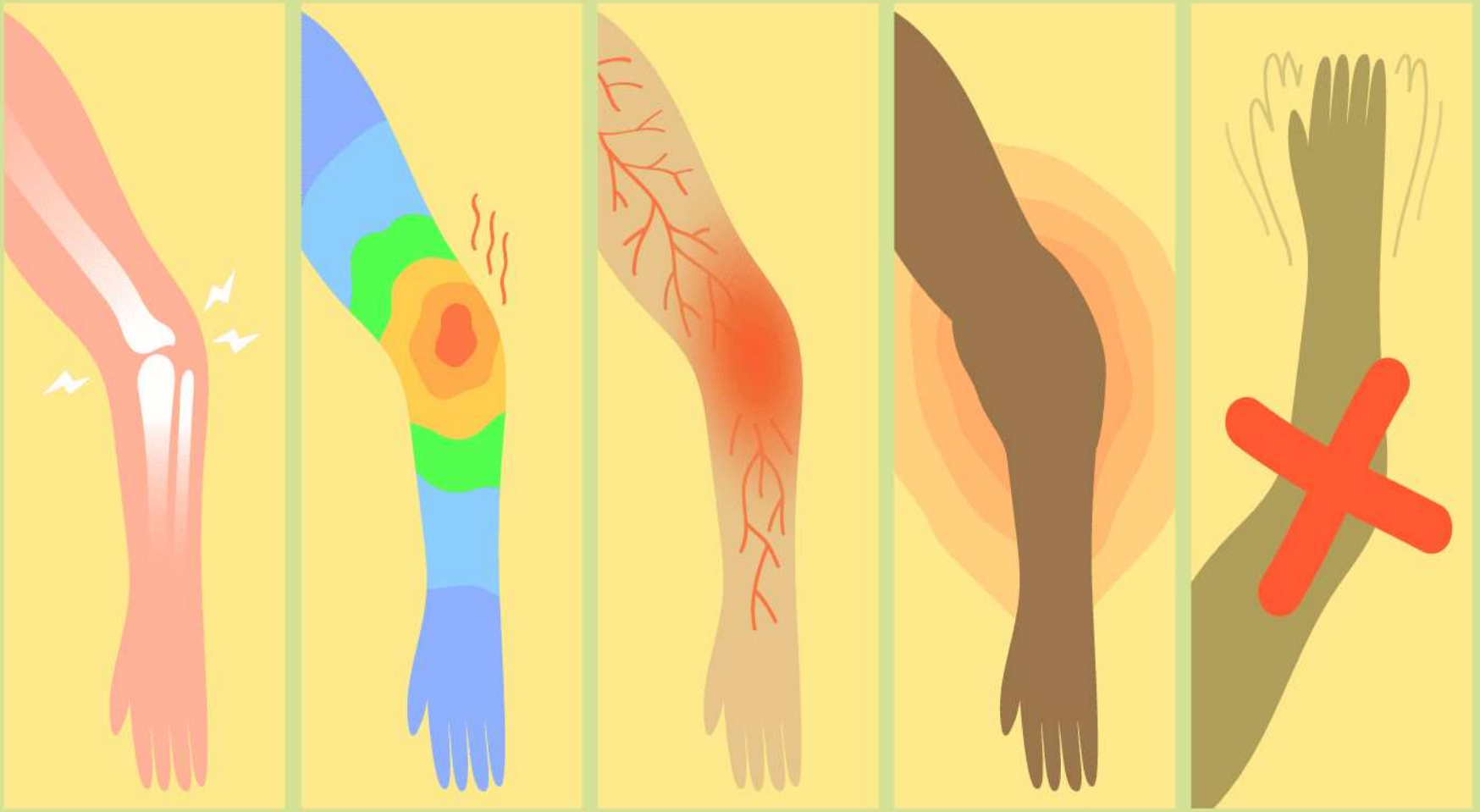
- The famous 4 cardinal signs of Inflammation as: (Roman writer Celsus in 1st century A.D.)

1. Rubor (redness)
2. Tumor (swelling)
3. Calor (heat)
4. Dolor (pain).

- The fifth sign was added by scientist ”Verchow”. Is-

5. Functio Leasa(Loss of Functions).

5 Cardinal Signs of Inflammation



Pain

Heat

Redness

Swelling

Loss of Function

TYPES

- Depending upon the **defense capacity of the host and duration of response**, inflammation can be classified as-

1. Acute Inflammation

2. Chronic Inflammation

ACUTE INFLAMMATION

.Short duration (lasting less than 2 weeks).

.Represents the early body reaction.

.Resolves quickly and is usually followed by healing."

FEATURES

1. Accumulation of fluid and plasma at the affected site.

2.Iintravascular activation of platelets.

3. Polymorphonuclear neutrophils as inflammatory cells.

RESPONSE

Acute inflammantory response is a progressive process and may be-

1. VASCULAR EVENTS

2. CELLULAR EVENTS

1. VASCULAR EVENTS

- Alteration in the microvasculature (arterioles, capillaries and venules) is the earliest response to tissue injury.

- These alterations include:

A. Haemodynamic changes.

B. Changes in vascular permeability.

A. Haemodynamic changes.

- The earliest features of inflammatory response result from changes in the vascular flow and calibre of small blood vessels in the injured tissue.

Such as-

1. Vasoconstriction of arterioles:

Irrespective of the type of injury, immediate vascular response.

i.e. transient vasoconstriction of arterioles.

- The blood flow may be re-established in 3-5 Seconds.

With more severe injury the vasoconstriction may last for about 5 minutes.

2. Persistent progressive vasodilatation:

- involves mainly the arterioles.
- Affects other components of the microcirculation like venules and capillaries.
- This change is obvious within half an hour of injury.
- Vasodilatation results in increased blood volume.

"Responsible for redness and warmth at the site of acute inflammation."

3. Local hydrostatic pressure :

Progressive vasodilatation, in turn, may elevate the **Local hydrostatic pressure** **resulting in transudation of fluid into** the extracellular space.

"This is responsible for swelling at the local site of acute inflammation."

4. Emigration:

Stasis or slowing is followed by **leucocytic margination** (mainly neutrophils).

The leucocytes stick to the vascular endothelium briefly, and then move and migrate through the gaps between the endothelial cells into the extravascular space.

B. Changes in vascular permeability.

In and around the inflamed tissue, there is accumulation of oedema fluid in the interstitial compartment .

which comes from blood plasma by its escape through the endothelial wall of peripheral vascular bed.

- In the initial stage, the escape of fluid is due to **vasodilatation** and consequent **elevation in hydrostatic pressure**.

. Increased vascular permeability of microvascular bed is explained on the basis of **Starling's hypothesis.**

"In normal circumstances, the fluid balance is maintained by two opposing sets of forces:"

1. Forces that cause **out ward movement of fluid from** microcirculation are intravascular hydrostatic pressure and colloid osmotic pressure of interstitial fluid.

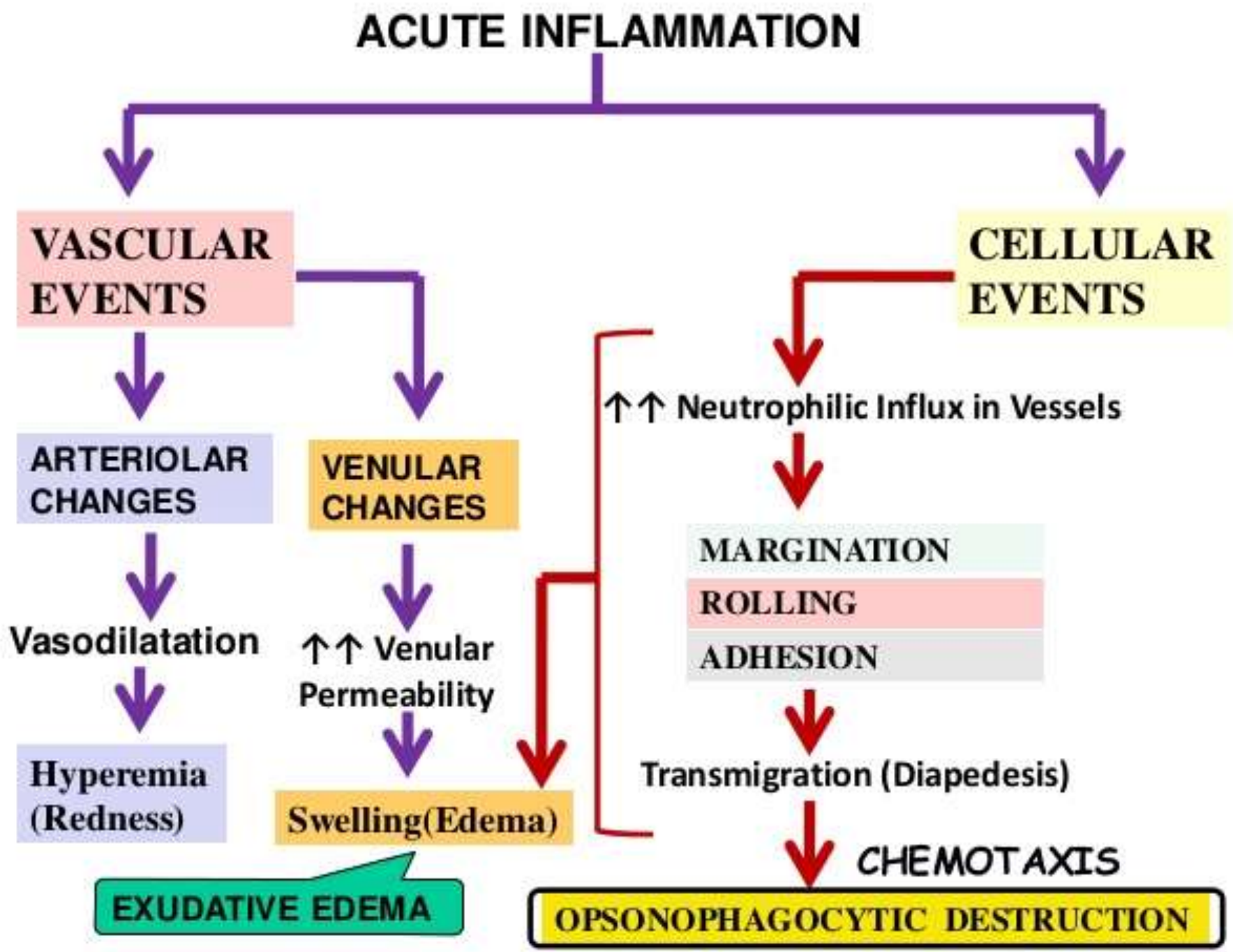
2. Forces that cause **inward movement of interstitial fluid** into circulation are *intravascular colloid osmotic pressure and hydrostatic pressure of interstitial fluid.*

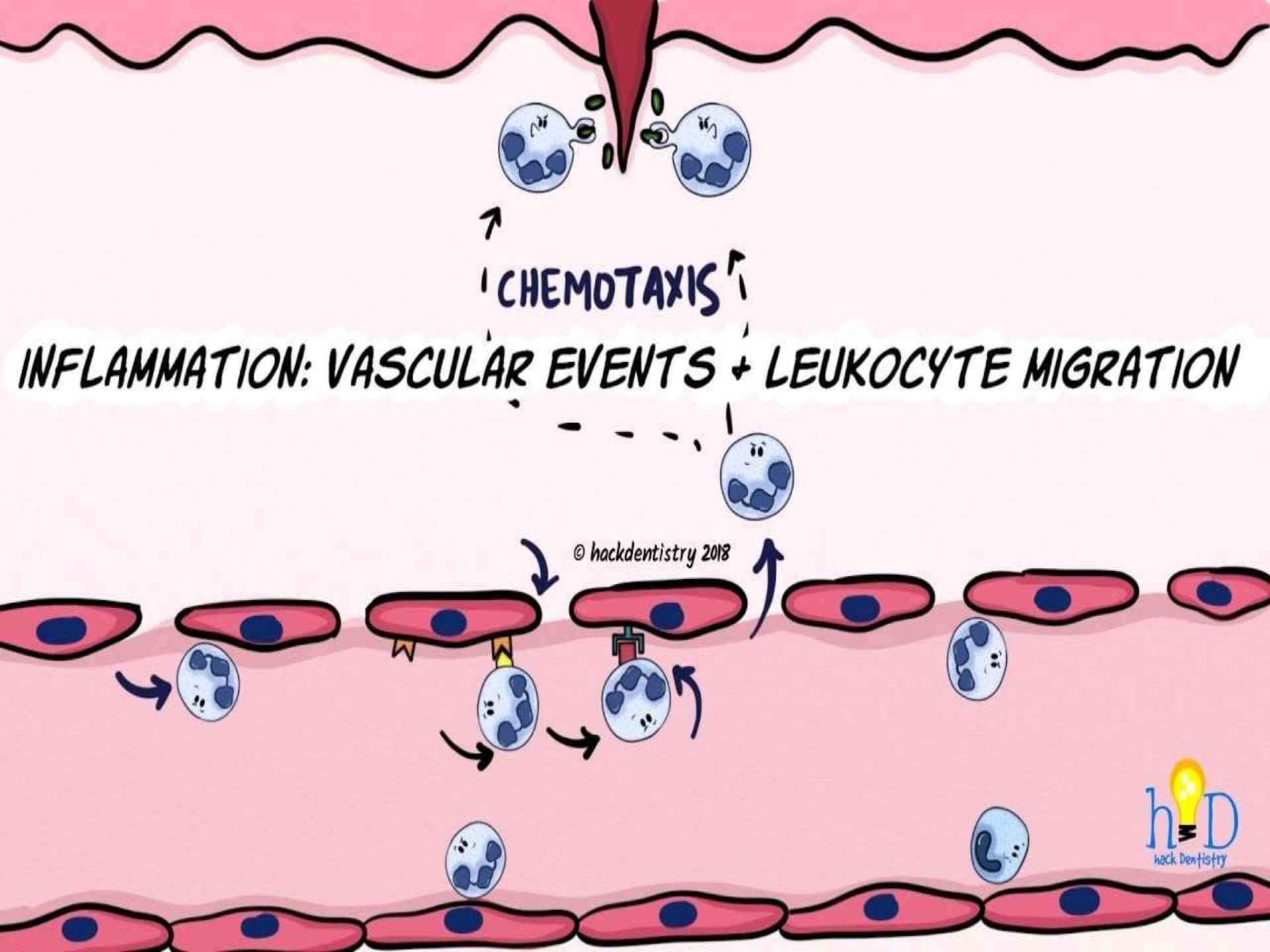
- However, in inflamed tissues, the endothelial lining of microvasculature **becomes more leaky**.

- Consequently, intravascular colloid osmotic pressure **decreases** and osmotic pressure of the interstitial fluid **increases**.

- Resulting in **excessive outward flow** of fluid into the interstitial compartment.

- Which is **exudative inflammatory oedema**.


ACUTE INFLAMMATION
VASCULAR EVENTS
CELLULAR EVENTS
ARTERIOLAR CHANGES
VENULAR CHANGES
↑↑ Neutrophilic Influx in Vessels
Vasodilatation
↑↑ Venular Permeability
MARGINATION
ROLLING
ADHESION
Hyperemia (Redness)
Swelling(Edema)
Transmigration (Diapedesis)
CHEMOTAXIS
EXUDATIVE EDEMA
OPSONOPHAGOCYTIC DESTRUCTION


CHEMOTAXIS
INFLAMMATION: VASCULAR EVENTS + LEUKOCYTE MIGRATION
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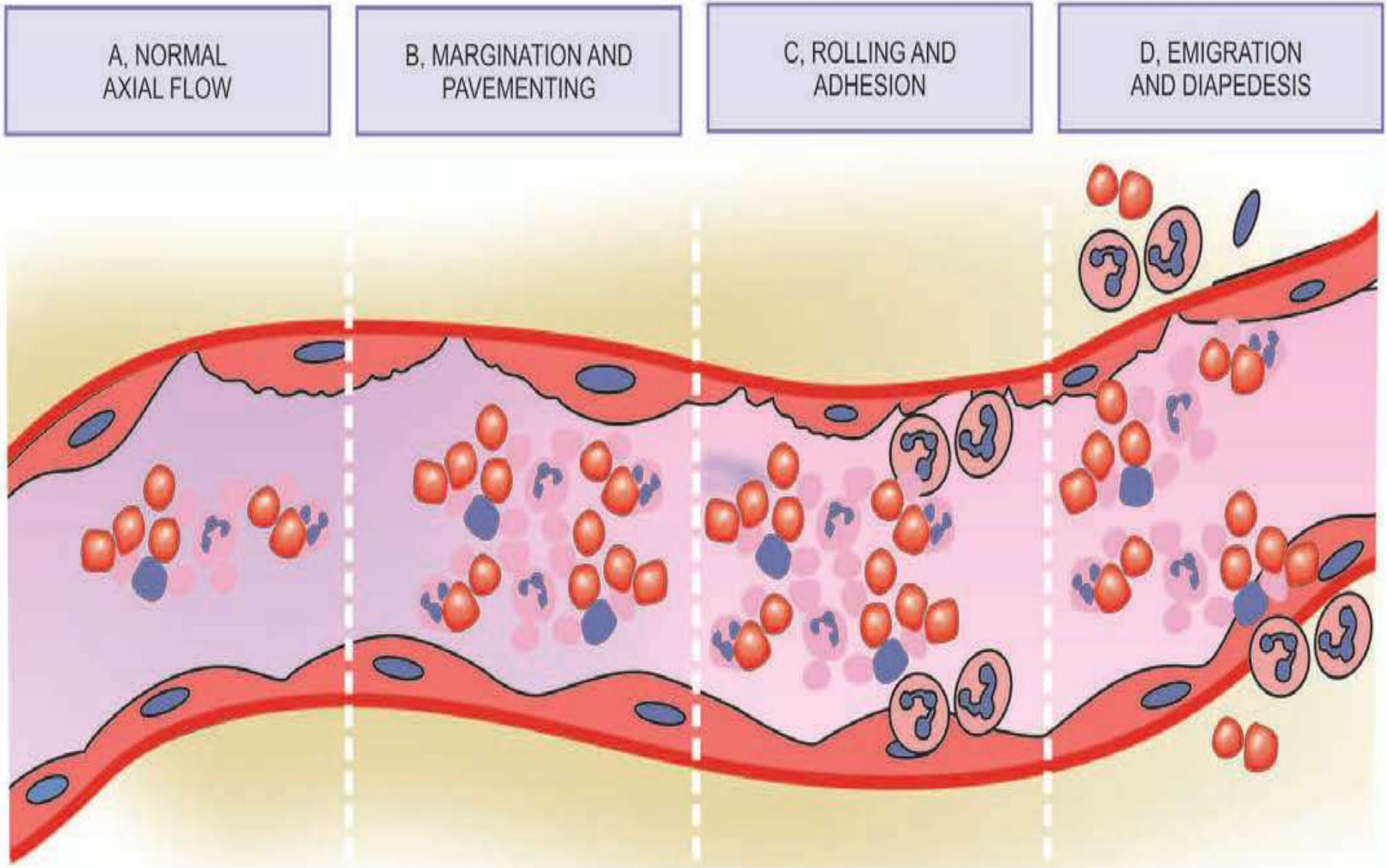
2. CELLULAR EVENTS

- The cellular phase of inflammation consists of 2 processes:

I. Exudation of leucocytes.

II. Phagocytosis.

I. Exudation of leucocytes.



- The most important feature of inflammatory response is escape of leucocytes from the lumen of microvasculature to the interstitial tissue.

"In acute inflammation, polymorphonuclear neutrophils (PMNs) comprise the first line of body defense.
followed later by monocytes and macrophages."

The changes leading to migration of leucocytes are as follows:-

1. **MARGINATION:** As a result of this redistribution, the neutrophils of the central column come close to the vessel wall; this is known as *pavementing.*

2. ROLLING AND ADHESION:

i. Peripherally marginated and pavemented neutrophils slowly roll over the endothelial cells lining the vessel wall *(rolling phase).*

ii. This is followed by the transient bond between the leucocytes and endothelial cells becoming firmer *(adhesion phase).*

3. EMIGRATION: After sticking of neutrophils to endothelium, the former move along the endothelial surface till a suitable site between the endothelial cells is found where the neutrophils throw out cytoplasmic pseudopods.

II. Phagocytosis.

"Phagocytosis is defined as the process of **engulfment of solid particulate** material by the cells **(cell-eating).**"

- The cells performing this function are called **phagocytes**.

There are **2 main** types of phagocytic cells:

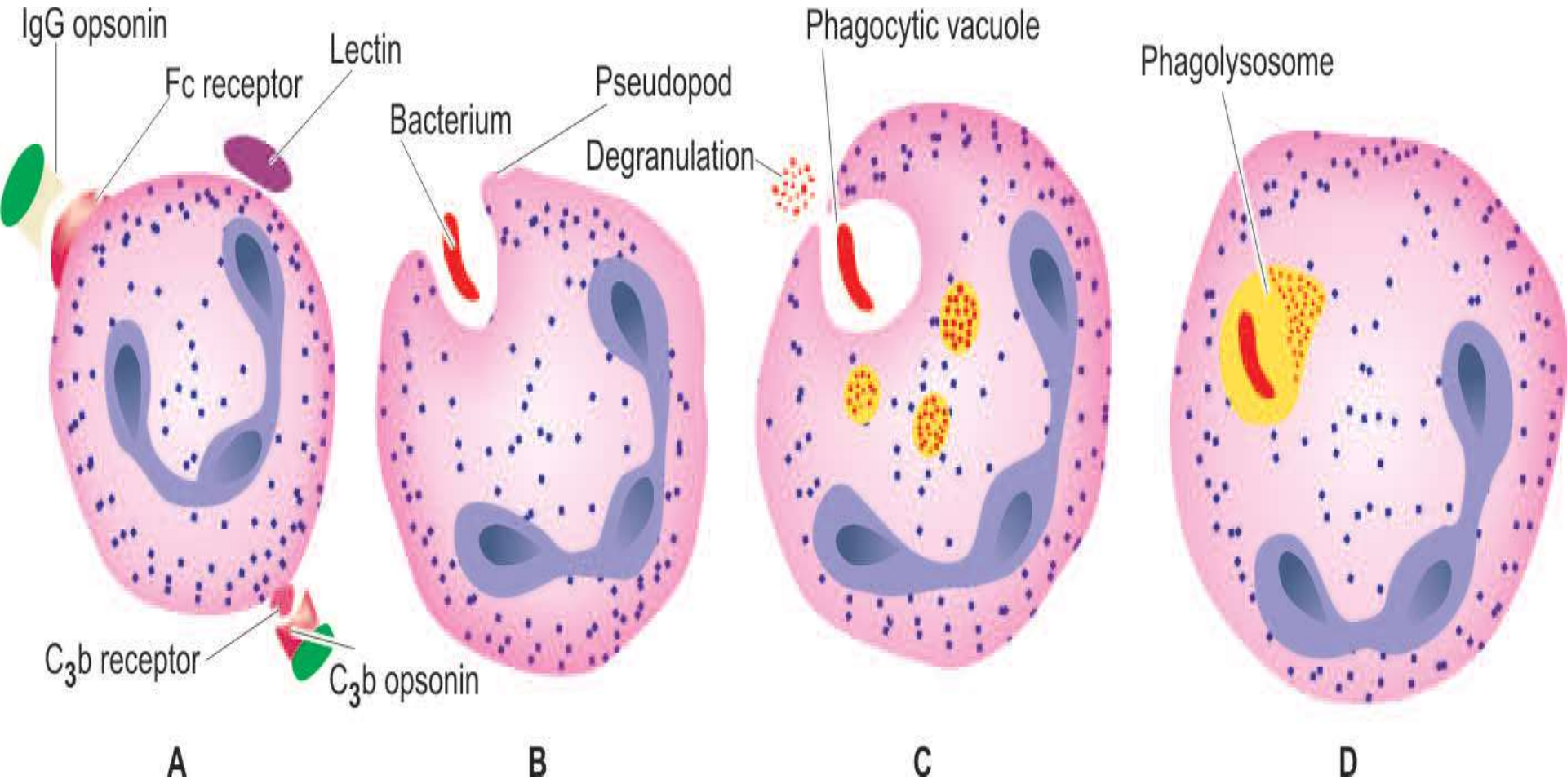
i. Polymorphonuclear neutrophils (PMNs): Commonaly called **Microphage.**

ii. Circulating monocytes: Commonaly called **Macrophage.**

STEPS OF PHAGOCYTOSIS

The microbe undergoes the process of phagocytosis by polymorphs and macrophages are involves the following 3 steps:

- 1. Recognition and attachment
- 2. Engulfment
- 3. Killing and degradation



Stages in phagocytosis of a foreign particle. A, Opsonisation of the particle. B, Pseudopod engulfing the opsonised particle.
C, Incorporation within the cell (phagocytic vacuole) and degranulation.
D, Phagolysosome formation after fusion of lysosome of the cell.

2. CHRONIC INFLAMMATION

- Chronic inflammation is of longer duration and occurs either after the causative agent of acute inflammation persists for a long time.


8 Ways Chronic Inflammation
Can Damage Your Body
Memory loss and cognitive decline
Increased cardiovascular risk
Abnormal growth of healthy cells
Compromised digestive function
Loss of muscle tone
Weight gain
Accelerated skin aging
Joint pain and loss of mobility
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DEFINITION

"Chronic inflammation is defined as prolonged process in which tissue destruction and inflammation occur at the same time."

The characteristic feature of chronic inflammation is-

- Presence of chronic inflammatory cells such as lymphocytes, plasma cells and macrophages.

- Granulation tissue formation, and in specific situations as granulomatous inflammation.

REASON OF CHRONIC INFLAMMATION

1. Chronic inflammation following acute inflammation.

When the tissue destruction is extensive, or the bacteria survive and persist in small numbers at the site of acute inflammation

e.g. in osteomyelitis, pneumonia terminating in lung abscess.

2. Recurrent attacks of acute inflammation.

When repeated bouts of acute inflammation culminate in chronicity of the process.

e.g.

I. in recurrent urinary tract infection leading to chronic pyelonephritis.

II. Repeated acute infection of gallbladder leading to chronic cholecystitis.

3. Chronic inflammation starting *de novo.*

When the infection with organisms of **low pathogenicity** is chronic from the beginning

e.g. infection with *Mycobacterium tuberculosis.*

SYSTEMIC EFFECTS OF CHRONIC INFLAMMATION

Chronic inflammation is associated with following systemic features:

1. **Fever.** **Invariably there is mild fever, often with loss of** weight and weakness.

2. **Anaemia.** **chronic inflammation** is accompanied by anaemia of varying degree.

3. Leucocytosis. generally there is relative lymphocytosis in these cases.

4. ESR. ESR is elevated in all cases of chronic inflammation.

5. Amyloidosis. **Long-term cases of chronic** inflammation may develop secondary systemic (AA) amyloidosis.

SYMPTOMS


SYMPTOMS OF
CHRONIC INFLAMMATION
FATIGUE
ALLERGY SYMPTOMS
DIGESTIVE PROBLEMS
WEIGHT GAIN
JOINT PAIN
HEADACHES AND BRAIN FOG
Wellbeing Center

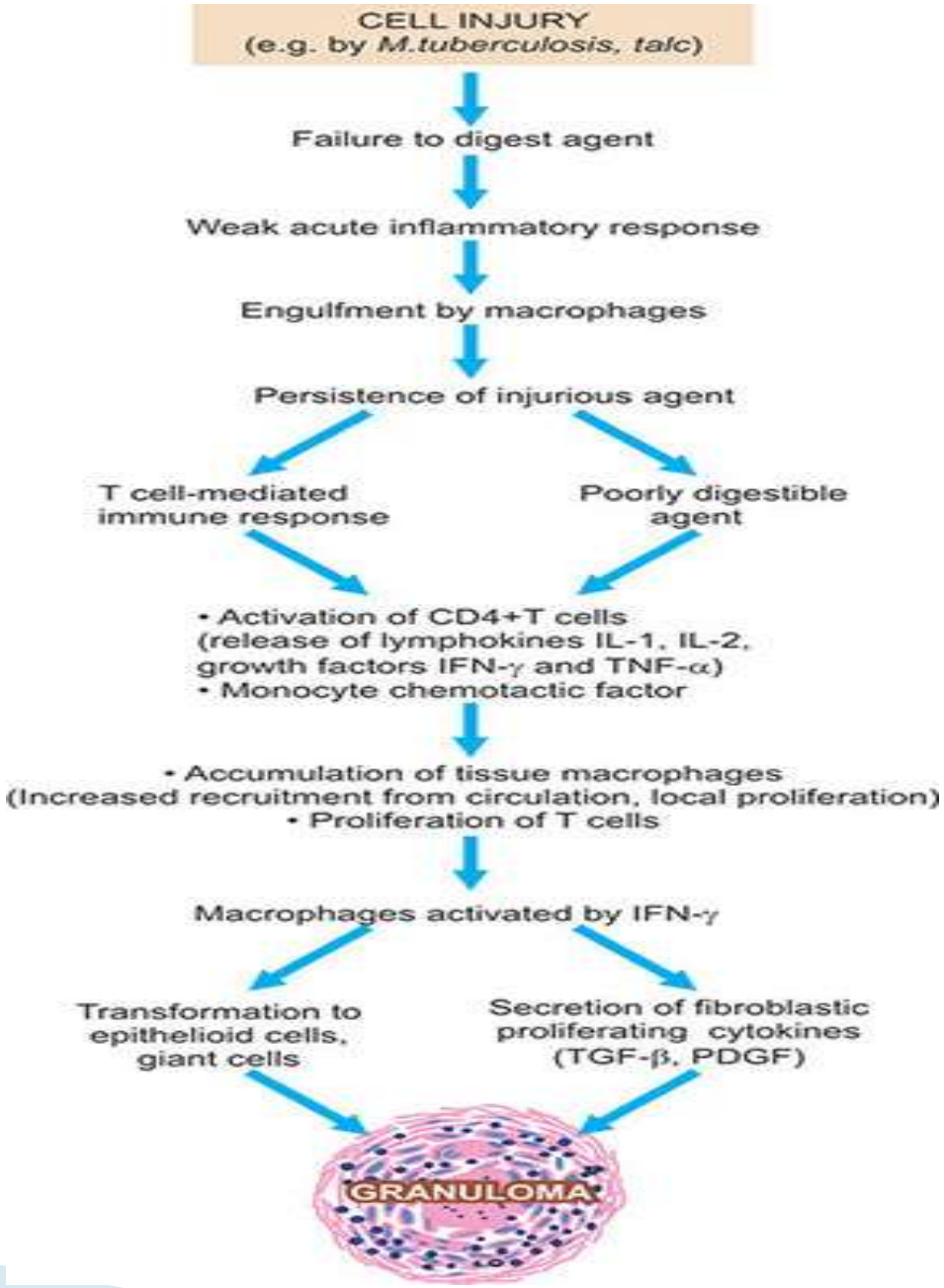
TYPES

1. Non-specific: **When the irritant substance produces a nonspecific** chronic inflammatory reaction with formation of granulation tissue and healing by fibrosis.

e.g. chronic osteomyelitis, chronic ulcer.

2. Specific: **When the injurious agent causes a characteristic** histologic tissue response.

e.g. tuberculosis, leprosy, syphilis.


CELL INJURY
(e.g. by M.tuberculosis, talc)
Failure to digest agent
Weak acute inflammatory response
Engulfment by macrophages
Persistence of injurious agent
T cell-mediated immune response
Poorly digestible agent
• Activation of CD4+T cells (release of lymphokines IL-1, IL-2, growth factors IFN-γ and TNF-α)
• Monocyte chemotactic factor
• Accumulation of tissue macrophages (Increased recruitment from circulation, local proliferation)
• Proliferation of T cells
Macrophages activated by IFN-γ
Transformation to epithelioid cells, giant cells
Secretion of fibroblastic proliferating cytokines (TGF-β, PDGF)
GRANULOMA

DIFFERENCE

	ACUTE	CHRONIC
ONSET	IMMEDIATE	DELAYED
DURATION	FEW DAYS	UPTO MONTHS, YEARS
CAUSATIVE AGENTS	BACTERIA INJURED TISSUES	PERSISTENT ACUTE , FOREIGN BODY , VIRAL AUTOIMMUNE
MAJOR CELLS	NEUTROPHILS ,BASOPHILS MONOCYTESMACROPHAGES	MONONUCLEAR CELLS MONO,LYMHPHOCYTES PLASMA CELLS FIBROBLASTS
PRIMARY MEDIATORS	VASOACTIVE AMINES	EICOSANOIDS
OUTCOMES	RESOLUTION ABSCESS FORMATION CHRONIC INFLAMMATION	TISSUE DESTRUCTION FIBROSIS NECROSIS

Thanks……..