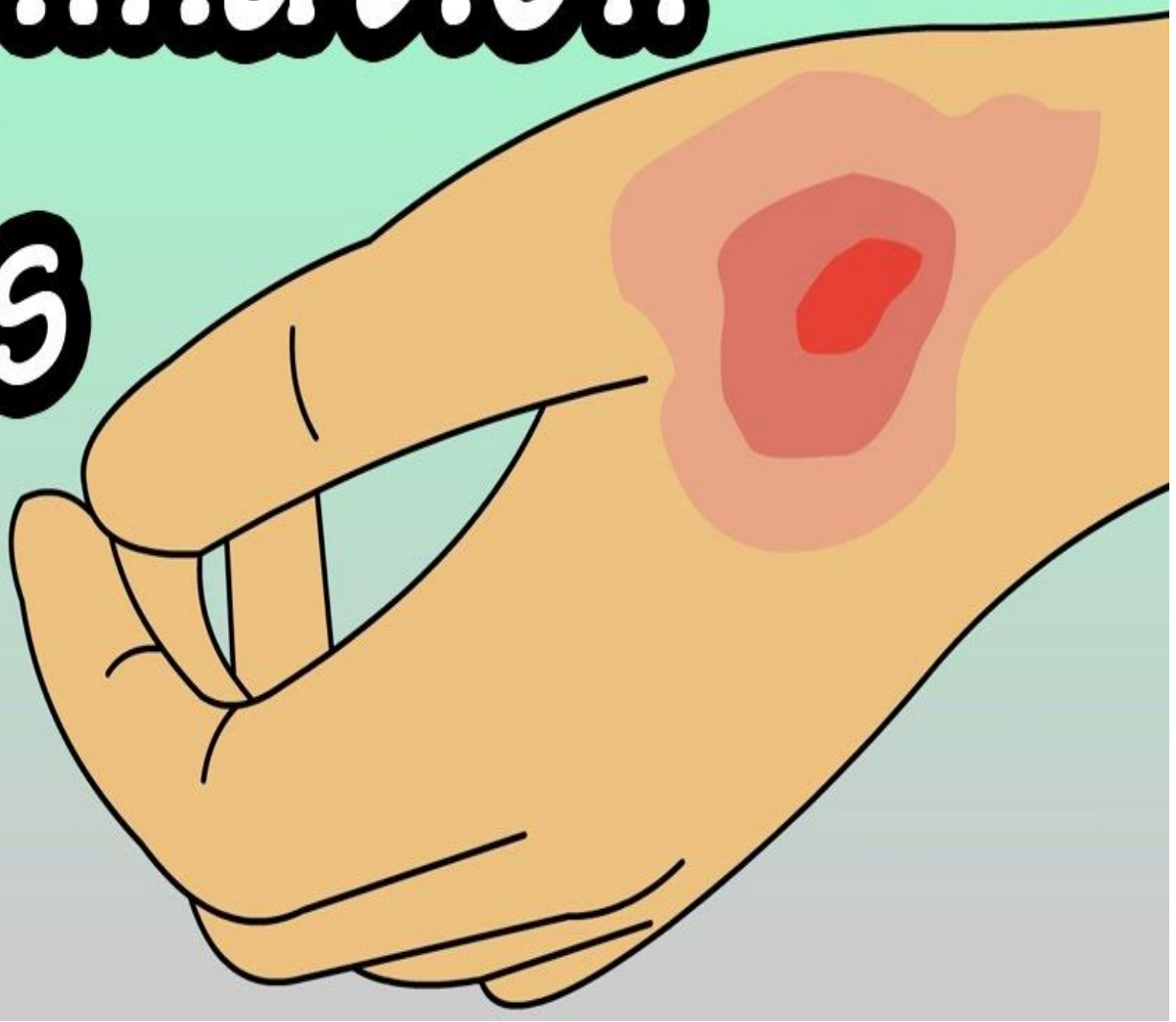
Inflammation

By
Dr. Raghed M. Jassem

Inflammation

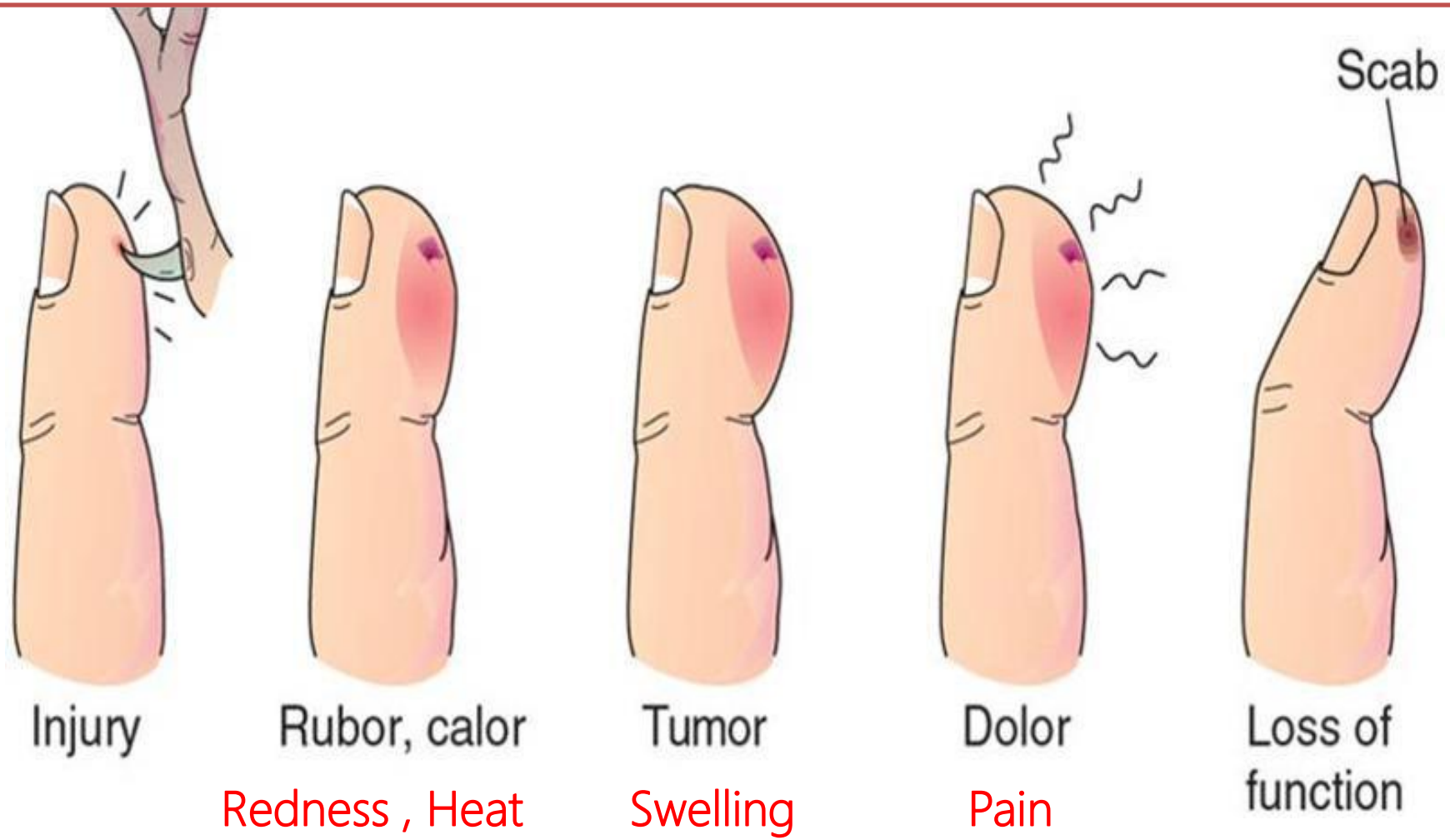
What Is It?



Inflammation:

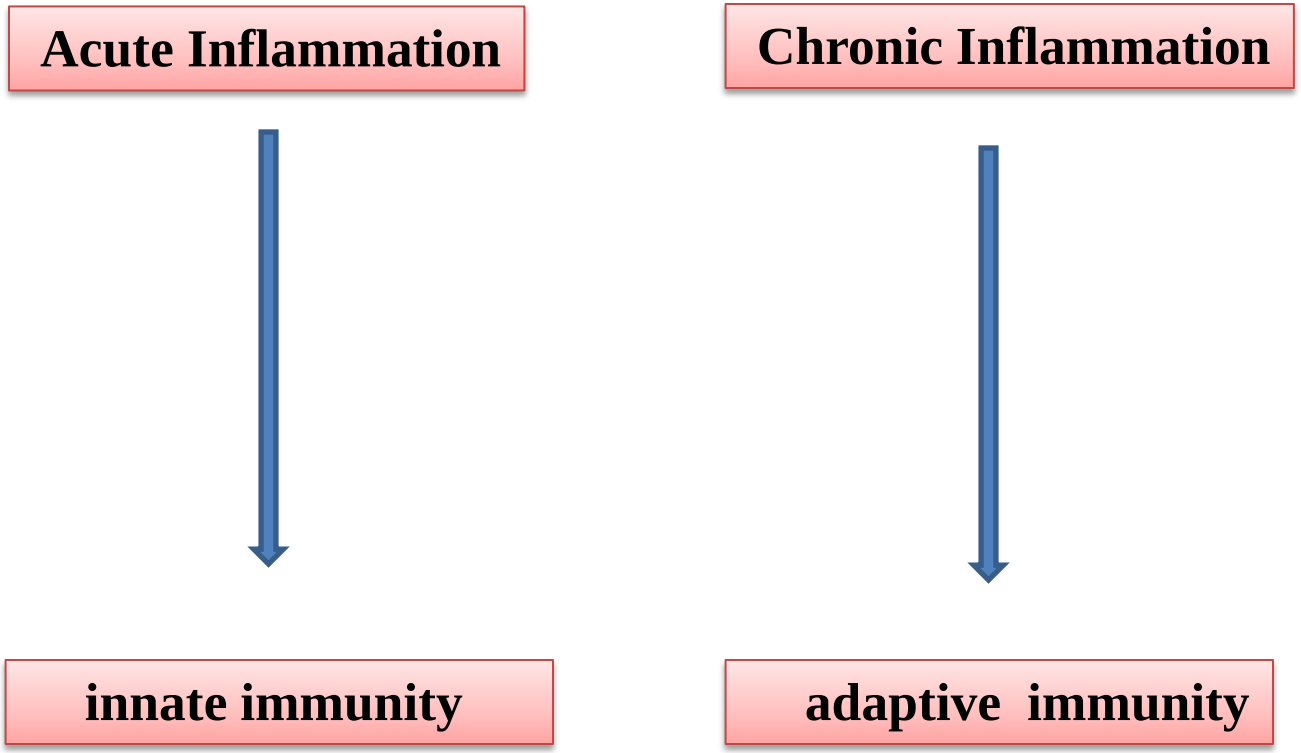
is the protective reaction of vascular tissue to local injury which is leading to the accumulation of fluid (Serum) and leukocytes in extra vascular tissue.

In the first century AD, the Roman physician Celsus described the "four signs of inflammation"



In the second century AD, another physician, Galen, added a fifth sign: *functio laesa* (loss of function).

Types of Inflammation



I - Acute inflammation

which occurs within seconds, minutes, hours, and days following the injury of tissues. The damage may involve the activation of some an immune response. During the acute inflammation the pathogen is often neutralized and eliminated by series of events

Stage of acute Inflammation

1- Vasodilation and increased vessel permeability (fluid phase / vascular stage)

2. Edema

3. Phagocyte mobilization (cellular phase)

4. Tissue repair

Stage of acute Inflammation

1- Vasodilation and increased vessel permeability (vascular stage)

It is immediately occur after tissue distraction or tissue injury , characteristic by

A) An increase in the diameter of blood vessels :-

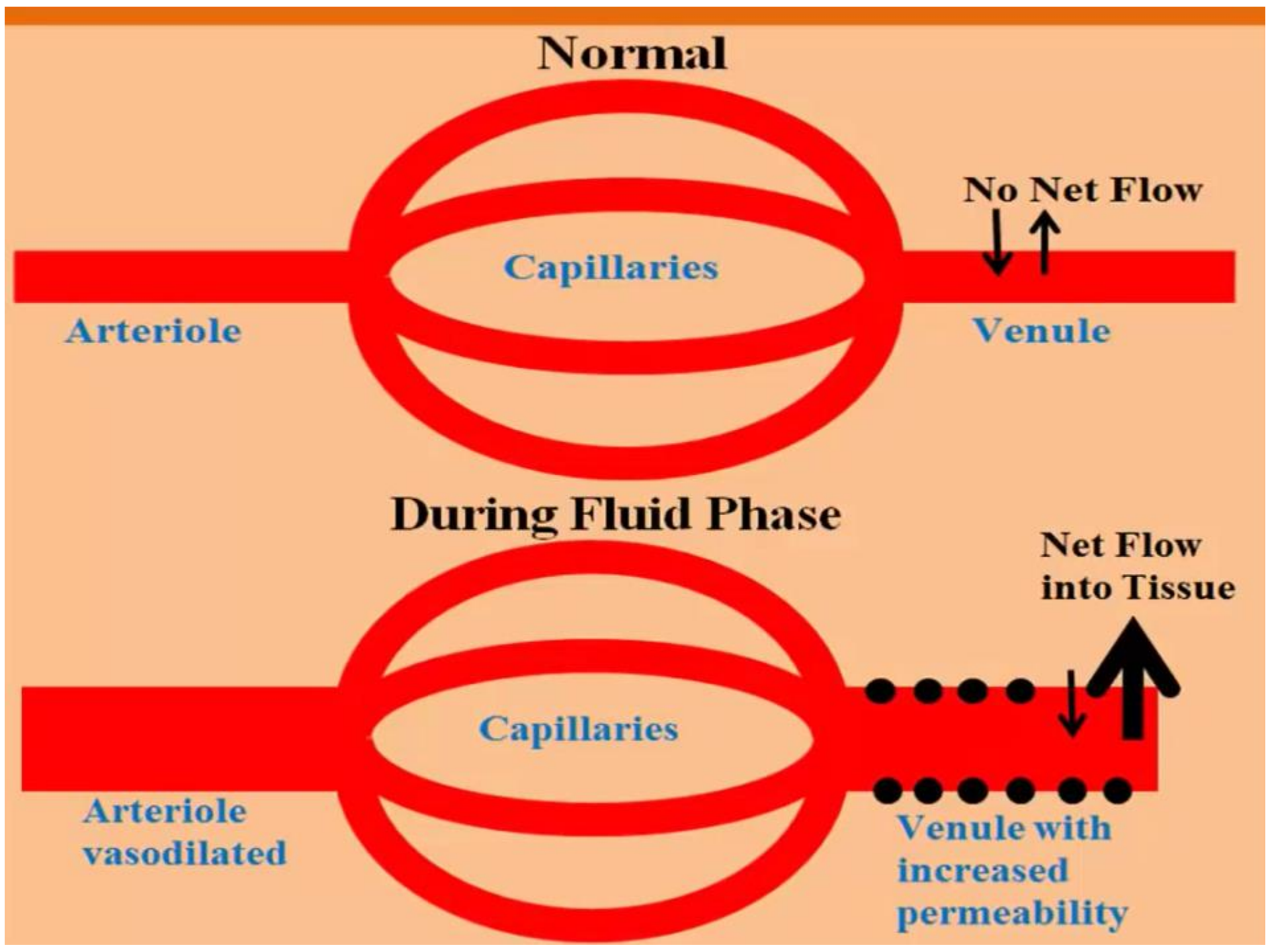
1- Arteriole dilation:- that will be carry more blood flow to the site of injury but the blood become more viscous because it moves very slowly

2- Venues permeability:-that cause more fluid will be engorged in to tissue resulting in engorgement of the capillary network.

The engorged capillaries and increased their permeability are responsible for tissue redness (*erythema*) and an increase in tissue temperature.

B) An increase in complement activation

C) An increase in cytokines releasing


Normal
No Net Flow
Capillaries
Arteriole
Venule
During Fluid Phase
Net Flow
into Tissue
Capillaries
Arteriole
vasodilated
Venule with
increased
permeability

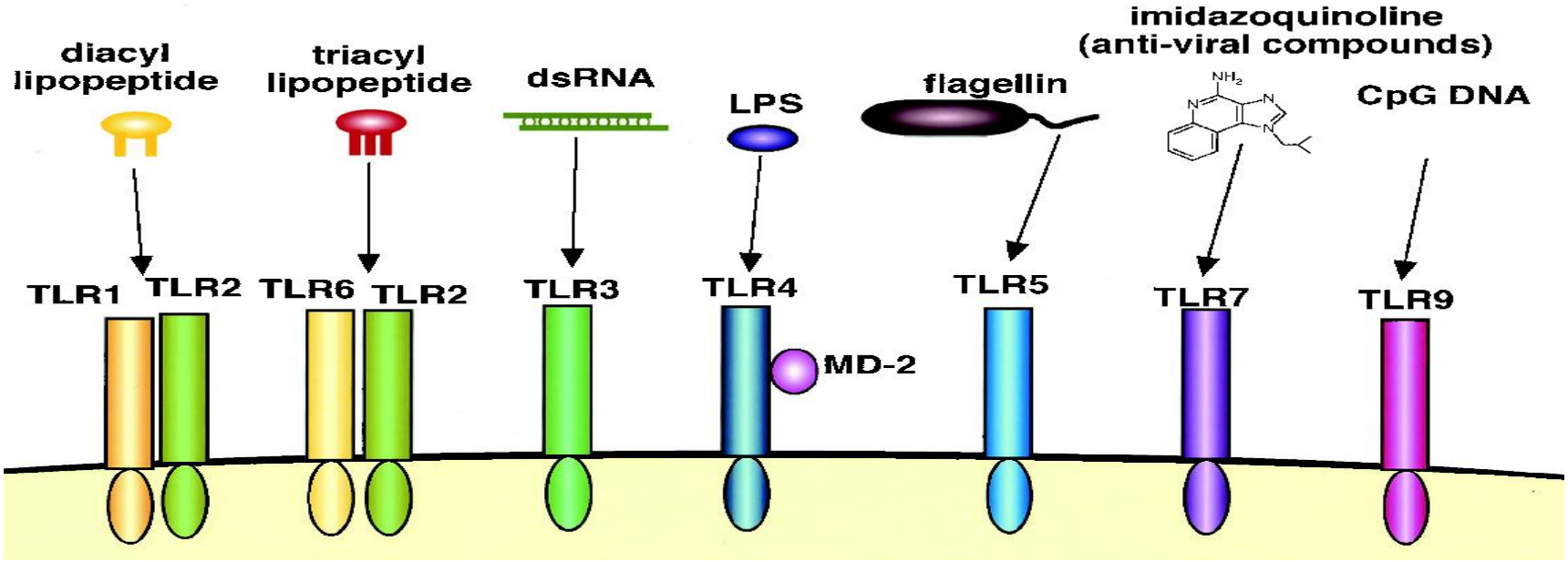
D) Macrophages and many other cells carry Toll-Like Receptors (TLRs) that recognize specific classes of microbes

Activation of TL-Receptor causes cytokine release that promotes inflammation & chemotaxis.

E) Mast cells secrete histamine.

Other cells and tissue damaged caused secrete various regulatory factors :- prostaglandins (PGE) , leukotrienes cause local vasodilation.

Bradykinin (Kinins system) and PGE2 cause pain (Dolor)



2. Edema – Accumulation of fluid from blood vessels exudate and that helps to

1- Dilute harmful substances

2- Increases supply of oxygen and nutrients needed for metabolism, inflammation and repair

3- Allows entry of clotting proteins, which reduces the spread of microbial agent. (A fibrin formation limit the spread of the invaders so that they remain localized).

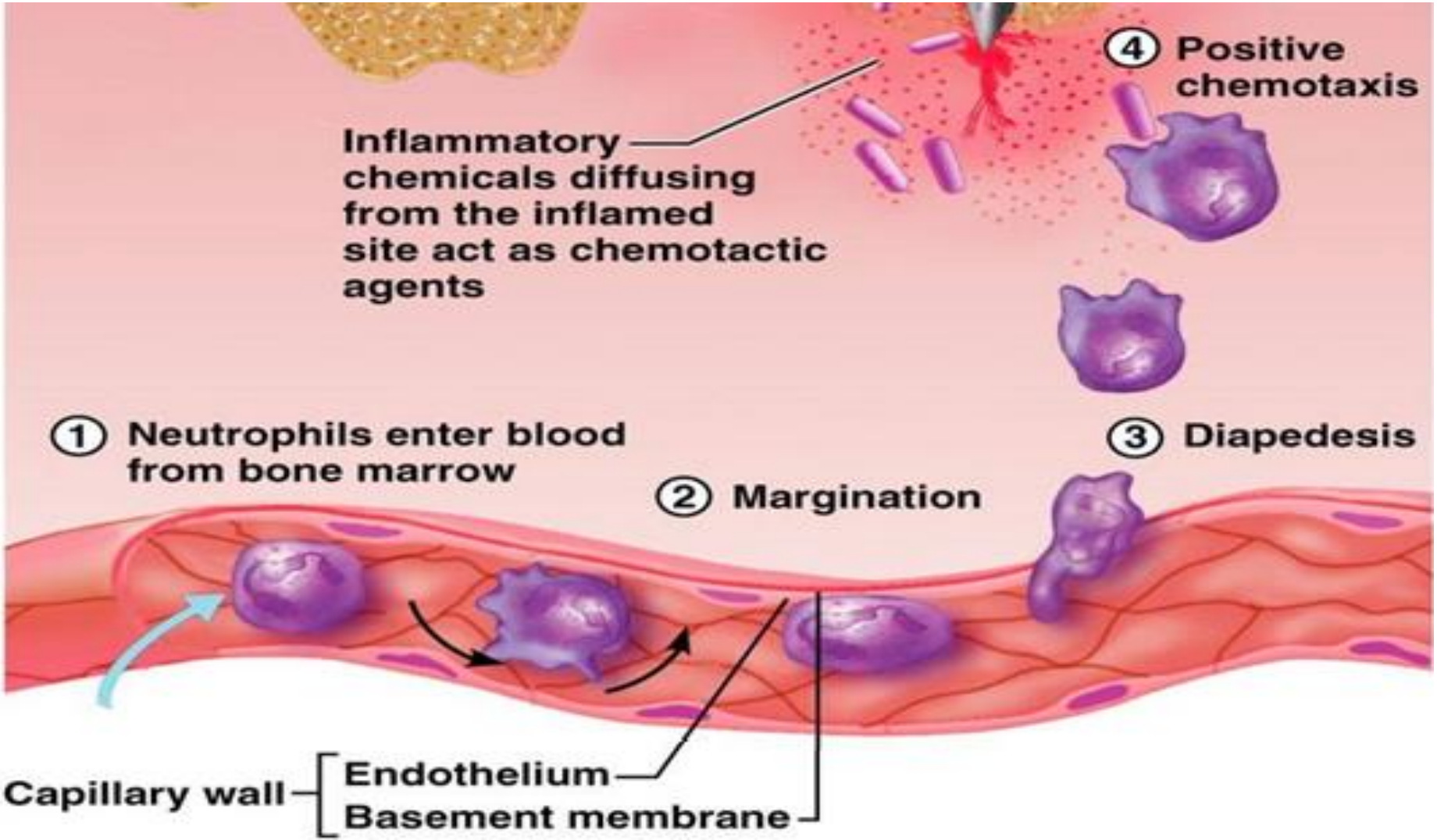
3. Phagocyte mobilization (cellular phase)

I. (Neutrophil phase):- Influx of phagocytes from the capillaries into the tissues including the following steps:-

1. Leukocytosis-inducing factors: increase neutrophil production form bone marrow
2. Margination :-adherence of the cells to the endothelial wall of the blood vessels
3. Diapedesis (amoeboid movement):- emigration between the capillary endothelial cells into the tissue
4. Chemotaxis of WBCs :- migration through the tissue to the site of the invasion ; neutrophils – rapid arrival within one day while monocytes – slower arrival.

The neutrophil will phagocytic of invading pathogen as well as the dead necrotic tissue causing clearance of injury site preparing it for healing stage

II. (Macrophage phase) :- macrophage arrived latter (after about 2 day) to the site of injury and it will be decided either go to the healing stage or continues to development of chronic inflammation (by acting as APCs for T- cells) and that will be depend on the type of pathogen and the releasing cytokines



4. Tissue repair

Tissue regrowth and repair of damage or scar formation ,pus cells will be form from dead phagocytes and other WBCs. If the Ags too numerous for effective removal by phagocytes, chronic inflammation will be develop.

II - Chronic inflammation

It is a response of prolong duration (weeks or months) in which inflammation, tissue injury and attempts at repair coexist in vary combinations

1- Infiltration by mononuclear cells.

The mononuclear cells are become predominant after **48 hours**. It is include (macrophages ,Lymphocytes , plasma cell and mast cells).

2- Tissue destruction (Necrosis) :- if the neutrophils and macrophages are unable to protect the host tissue and unable to destroy the M.O during the infection ,the body attempts to wall off and isolate the site by forming of granuloma

3- Proliferative changes, (attempt of healing) Occur by

- Proliferation of small blood vessels, (angiogenesis).
- Proliferation of fibroblast, (fibrosis-repair).

Cells of chronic inflammation :-

- Macrophages are the dominant cells in most of the chronic inflammation
- Functions of macrophages :-
  - Phagocytosis
  - Initiates tissue repair
  - Secrets inflammatory mediators
  - Displays antigen to T lymphocytes and responds to signals from T lymphocytes

Other cells of chronic inflammation

Lymphocytes ,plasma cells ,eosinophils ,mast cells ,neutrophils

Causes of Chronic inflammation :-

I. **I - Persistent infection**. ex:-Mycobacteria which have a cell wall with a very high lipid and wax content make them relatively insensitive to phagocytosis .

II. **Prolonged exposure to toxic agents:-** Some bacteria produce toxins that stimulate tissue damaging reaction even after bacteria death

III. **Autoimmunity and hypersensitivity**

IV. **Recurrent attacks of acute inflammation**