

Inflammation

It is the characteristic response of living vascularized tissue to injury.

Inflammatory process doesn't occur in dead tissue.

Causes

- Physical factors.
- Chemical factors.
- Infections
- Hypersensitivity.
- Necrotic tissue which induce inflammatory reaction in surrounding tissue.

Types of Inflammation

- **Acute Inflammation:** It is of relatively short duration “lasting from few minutes up to a few days”. It characterized by exudation and by predominantly neutrophilic leukocytes accumulation.
- **Chronic Inflammation:** It is of longer duration “several days to years” and it manifested histological by accumulation of lymphocytes and macrophages and by tissue destruction and repair.

Acute inflammation

Signs:

- Redness
- Heat
- Swelling
- Pain
- Loss of function

The arm at the bottom is swollen (edematous) and reddened (erythematous) compared to the arm at the top.

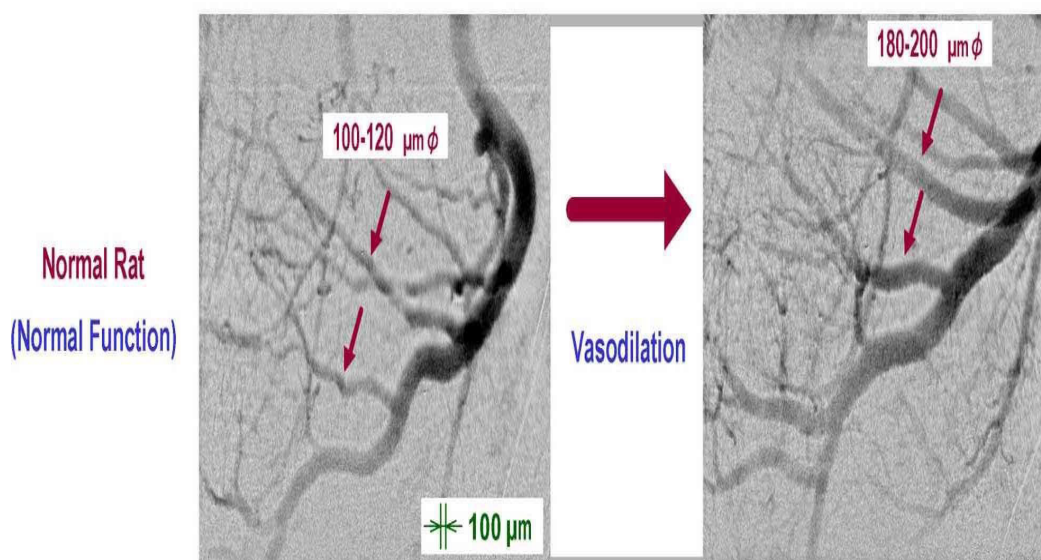


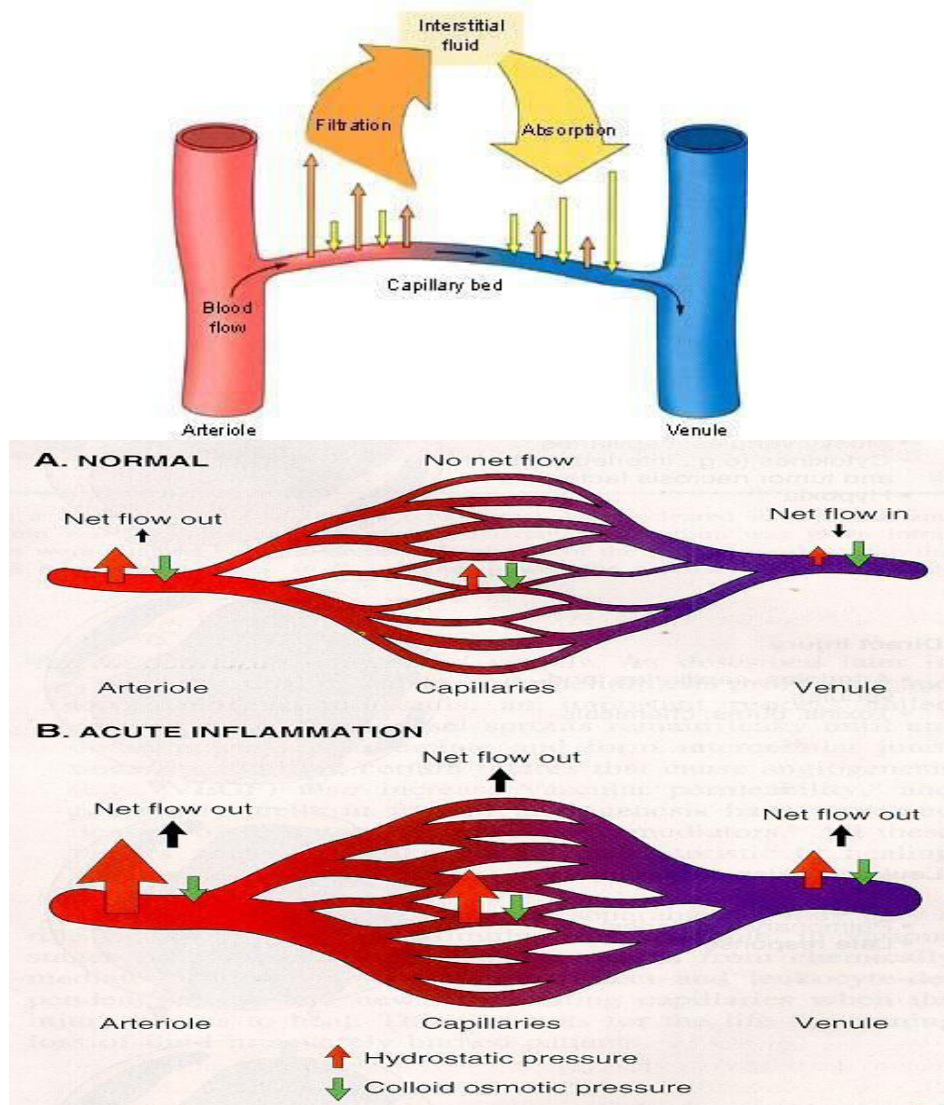
Components of acute Inflammation

- Changes In Vascular flow and caliber (hemodynamic changes).
- Increase vascular permeability.
- Accumulation of neutrophils leukocytes.

Changes in Vascular Flow and Caliber

- Immediate and transient vasoconstriction of arterioles.
- Relaxation and dilatation of small blood vessels and acceleration of blood flow “active hyperemia”.
- Increase permeability of blood vessels.
- Blood flow stasis.

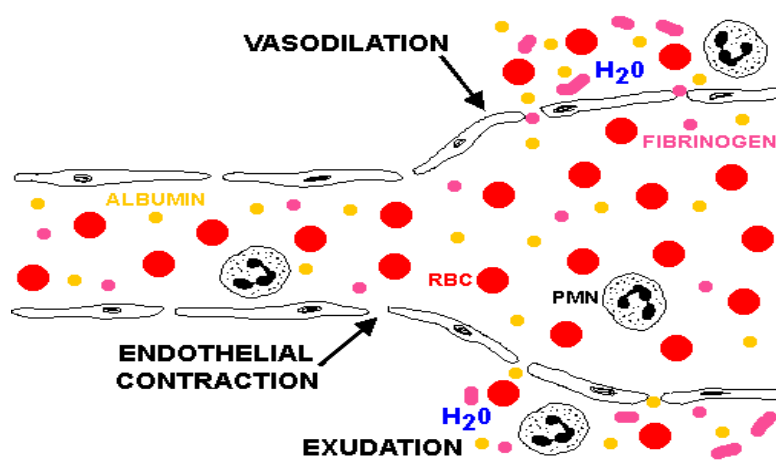




Increase vascular permeability

- ✓ Increase permeability permits the flow of protein rich fluid and even cells from blood into interstitial tissue.
- ✓ The movement of protein rich fluid from the plasma reduces the intravascular osmotic pressure.
- ✓ The net result is outflow of water and ions into the extra vascular tissue.
- ✓ Exudates are responsible for the swelling signs of acute inflammation.

The diagram shown here illustrates the process of exudation, aided by endothelial cell contraction and vasodilation, which typically is most pronounced in venules. Chemical mediators producing endothelial contraction include: histamine, and other mediators.

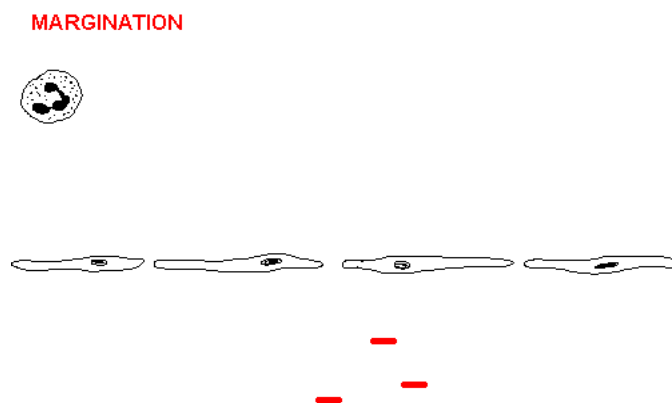


Cellular Response

The cellular stage of acute inflammation is marked by movement of leukocytes into the area of injury this stage includes:

1. The margination and pavementing of WBC.
2. Emigration of WBC.
3. Chemotaxis: A process in which leucocytes move towards certain substances termed “chemotactic agent or chemotaxines”
4. Phagocytosis and Degranulation:

The neutrophils move from blood toward the inflamed tissue, and then phagocytise the pathogen.



Phagocytosis

- Recognition and attachment of the particle to the ingesting leukocyte
- Engulfment of particle.
- Kill of particle.

Phagocytosis results in the release of lysosomal enzymes. Lysosomal enzyme also release into the extra cellular space where cell injury and matrix degradation result. This explains the pain and loss of function.

Inflammatory Mediators

These are endogenous substances released either from cells or derived from plasma. These mediators are responsible for both vasodilatation and increase permeability of blood vessels and some of them responsible for cellular emigration.

Mediators derived from the plasma

- Kinn system
- Clotting system
- Complement system
- Fibrinolytic system

Mediators released by cell

- Histamine
- Serotonin
- Prostaglandin
- Lysosomal component

Types of Inflammation According to the Exudate

- Serous inflammation
- Fibrinous inflammation
- Membranous inflammation
- Purulent inflammation
- Catarrhal inflammation

Exudation of a protein-rich fluid into a cavity leads to a transudate. The fibrin in this fluid can form a fibrinous exudate on the surfaces. Here, the pericardial cavity has been opened to reveal a fibrinous pericarditis with strands of stringy pale fibrin between visceral and parietal pericardium.



Effect of acute inflammation

- **Beneficial effects:**

Inflammatory exudate:

1. Limiting the harmful effect of toxic compounds by dilution of toxin.
2. May contains antibodies.
3. Fibrin formation.
4. Promotion of immunity
5. Cell nutrition
6. Phagocytosis

Harmful effects of Inflammation

- Swelling
- Rise of tissue pressure
- hypersensitivity

outcome of acute inflammation

- Resolution
- Progress to chronic inflammation
- Healing with scar formation (occurs when large amounts of tissue are damaged).
- Abscessation Pus formation.

Abscessation

- ❖ Pyogenic bacteria have ability to attract neutrophils.
- ❖ when large amount or numbers of neutrophils enter the infected area and when neutrophils die that allows lysosomal enzymes to release and digest the tissue, so we can see cavity containing fluids.

The contents of pus are:

- Pyogenic bacteria (dead and life)
- Nutrophils (dead and life)
- dead tissue.