

Tissue Inflammation and Repair

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- I. Introduction
- II. Types of Inflammation
- III. Acute inflammation
- IV. Chronic inflammation
- V. Systemic effects of inflammation
- VI. Factors affecting healing
- VII. Treatment of inflammation

I. Introduction

Classical clinical signs

'calor'-----heat

(relevant primarily to inflammation of skin)

'rubor'-----redness

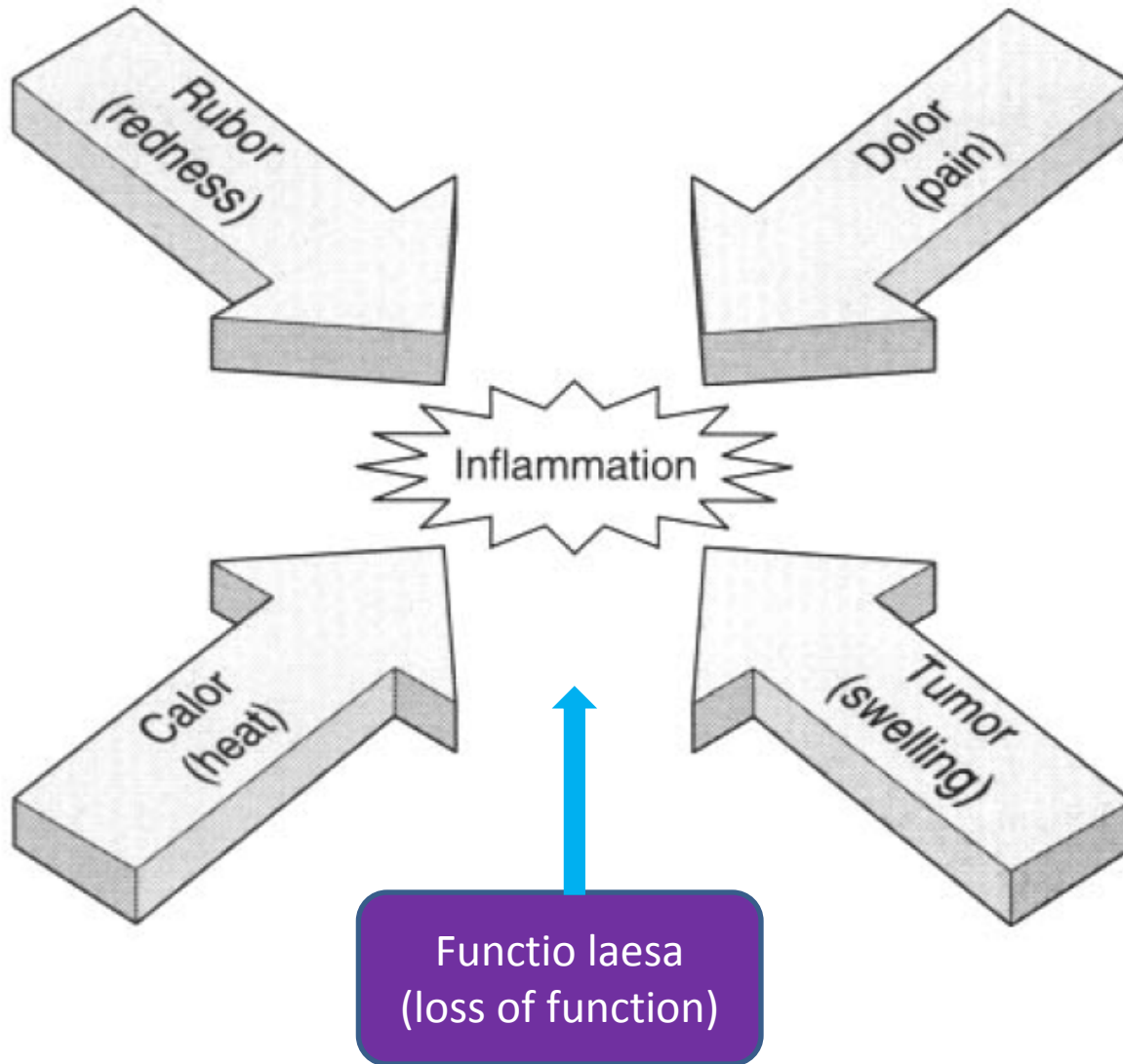
'dolor'-----pain

'tumor'-----swelling

They are indicative of the extravasation of plasma and infiltration of leucocytes at the site of inflammation.

'Functio laesa'---loss of function

Components of inflammation



Clinical features

- (a) Pain
- (b) Redness
- (c) Heat
- (d) Swelling
- (e) Loss of function



Trauma stimulating pain receptors



← Cell anoxia

Release of chemical substances (bradykinin, prostaglandin)
due to lack of oxygen & nutrients



Bleeding



Leakage of
intracellular fluid



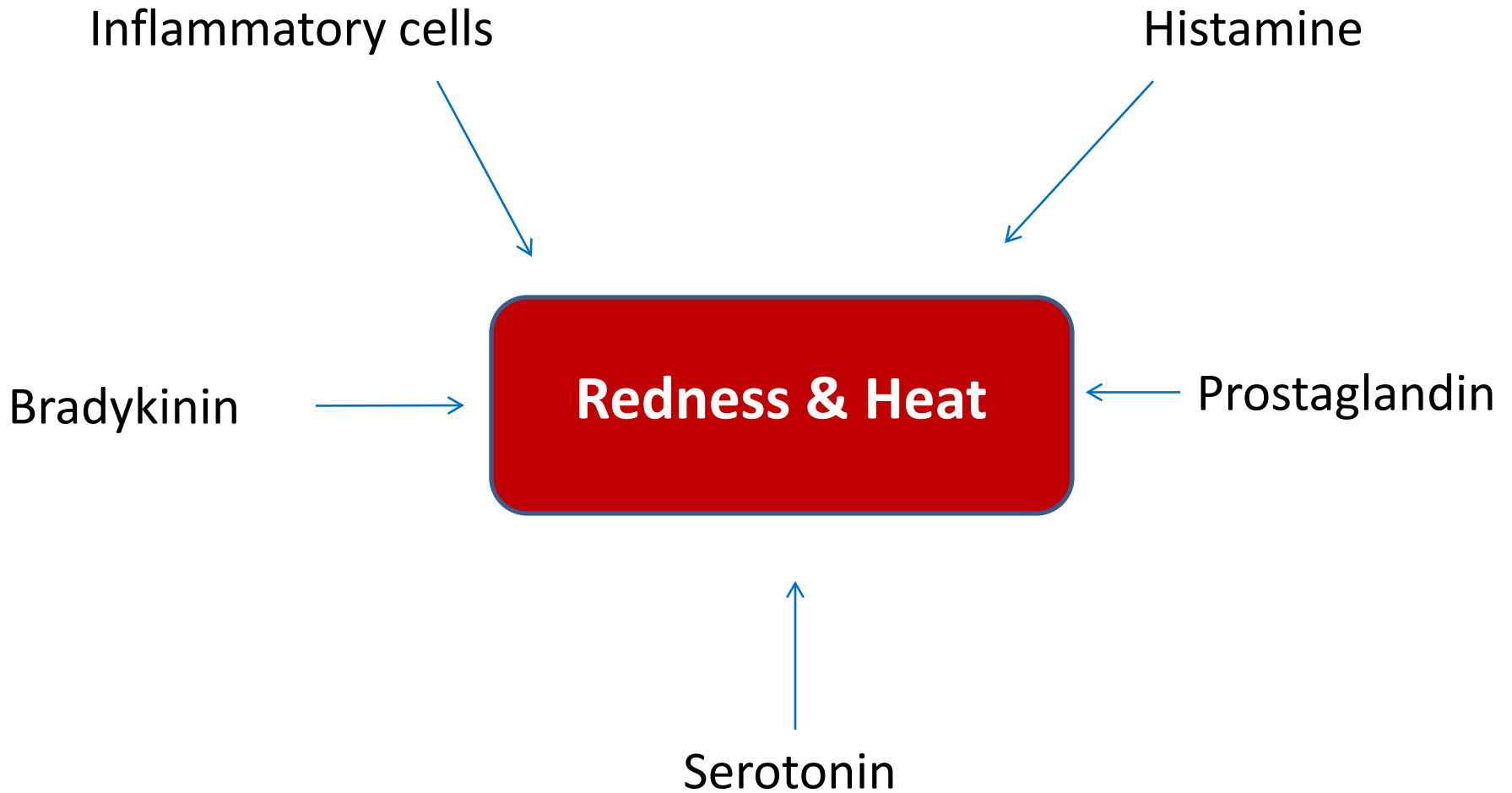
Swelling

Limb in
dependent
position



Increased osmotic pressure of
extracellular compartment





Purpose of inflammatory process



To heal the injured tissues

Inflammation

Complex stereotypical reaction of **vascularized living tissue** to local trauma

Inflammation \neq **Infection**

Inflammation is the immune system's response to tissue damage.

Damage is due to an exogenous source (a cut or burn) or to endogenous failures (a bone fracture).

The principal aim behind inflammation is to repair the tissue and bring it back to its original state.

Inflammation

Body's initial reaction to **an injury** is similar to its reaction to **an infection**.



This reaction is termed **inflammation**.



May manifest **macroscopically** (e.g. after an acute injury)
or
microscopically

Types of Injuries

- Macrotrauma
- Microtrauma



Micro vs. Macro Trauma

- Micro trauma - small repetitive traumas resulting in tissue breakdown NSCA
More Prevalent during Marathon Training



- Macro trauma - 1 large event or trauma resulting in injury NSCA
Training in Boston over the Winter
BEWARE!



An inflammatory condition ----- '-itis'

e.g. Tonsillitis

Bronchitis

Appendicitis

II. Types of Inflammation

Acute inflammation

usually lasts for only a few hours to a few days

By the end of this time

accumulated fluid & degraded proteins in extracellular spaces drained by lymphatic system

phagocytic cells remove exudates, debris & fibrin

inflammatory cells themselves will undergo cell death or 'apoptosis'

Tissues return to normal

Chronic inflammation

- Persistence of inflammation usually **beyond 10-14 days**
- accompanied by fibrosis (accumulation of synthesised collagen in the tissue)

Chronic inflammation(Contd.)

- can occur when the resolution to acute inflammatory process is not achieved, possibly through **the causative agent not being removed** leading to prolonged inflammation
- Chronic inflammation can arise as **a low-grade inflammatory process** without a preceding acute phase.
(e.g. Rheumatoid arthritis)

III. Acute inflammation



Acute inflammation: The Mechanisms

Phases of acute inflammation

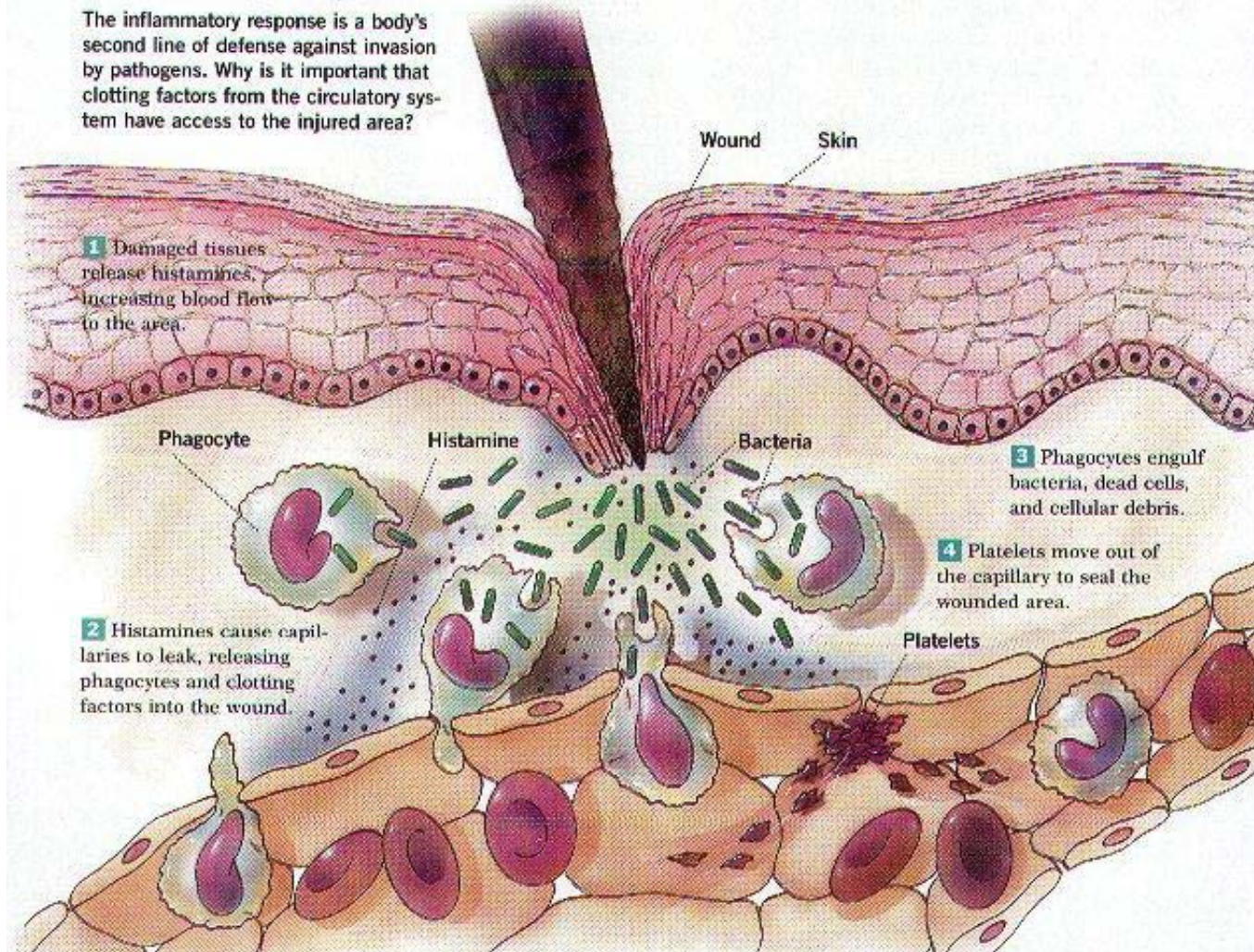
1. Vascular
2. Extravasation of plasma (Fluid exudate)
3. Cellular exudate

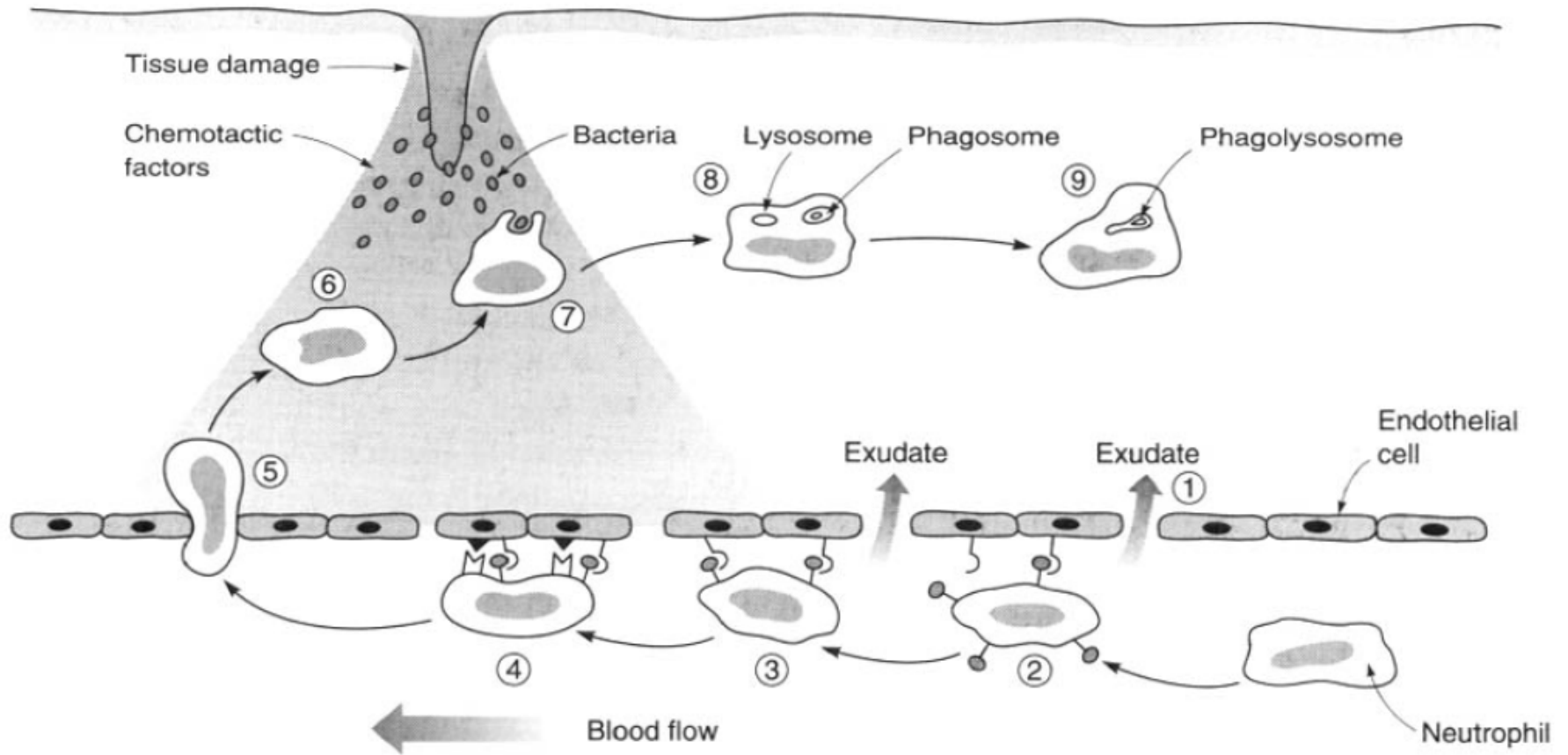
Acute inflammation: Detailed Consideration

- Initiating events
- Events at the site of damage
- Vasodilation
- Increased capillary permeability
- Cell migration
- Phagocytosis

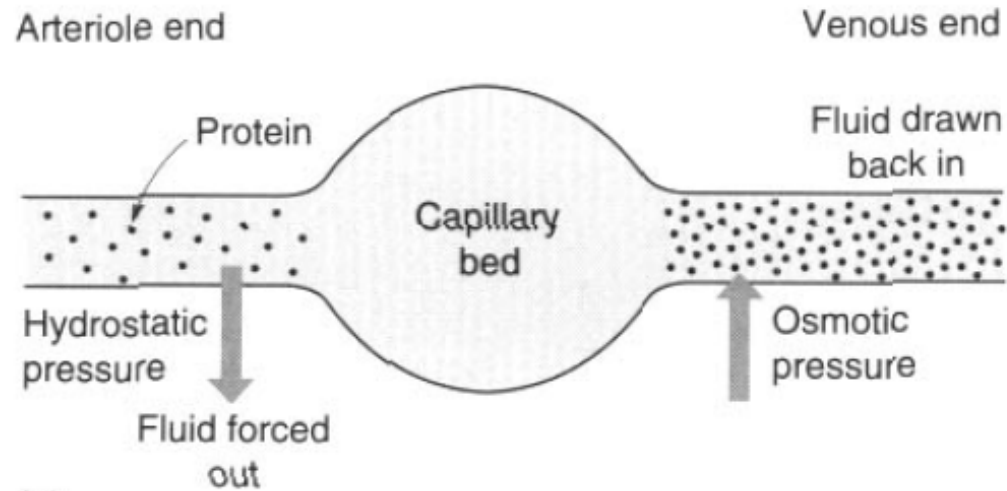
Steps of the Inflammatory Response

The inflammatory response is a body's second line of defense against invasion by pathogens. Why is it important that clotting factors from the circulatory system have access to the injured area?

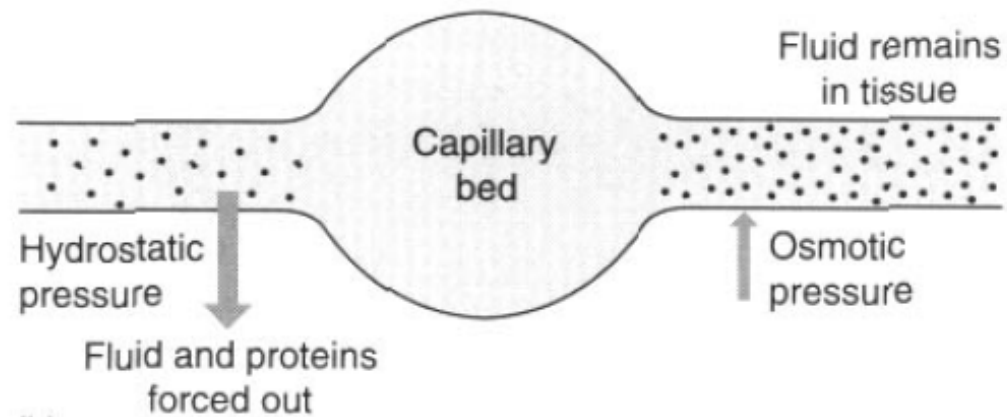




Schematic illustrating neutrophil margination, emigration, chemotactic attraction and phagocytosis in an inflammatory response



(a)



(b)

Generation of oedema in inflamed tissue

Benefits and Drawbacks of the Inflammatory Response

- Benefits
- Drawbacks

Forms of acute inflammation

- Serous inflammation
- Catarrhal inflammation
- Fibrinous inflammation
- Haemorrhagic inflammation
- Suppurative inflammation
- Pseudomembranous inflammation
- Gangrenous or necrotising inflammation

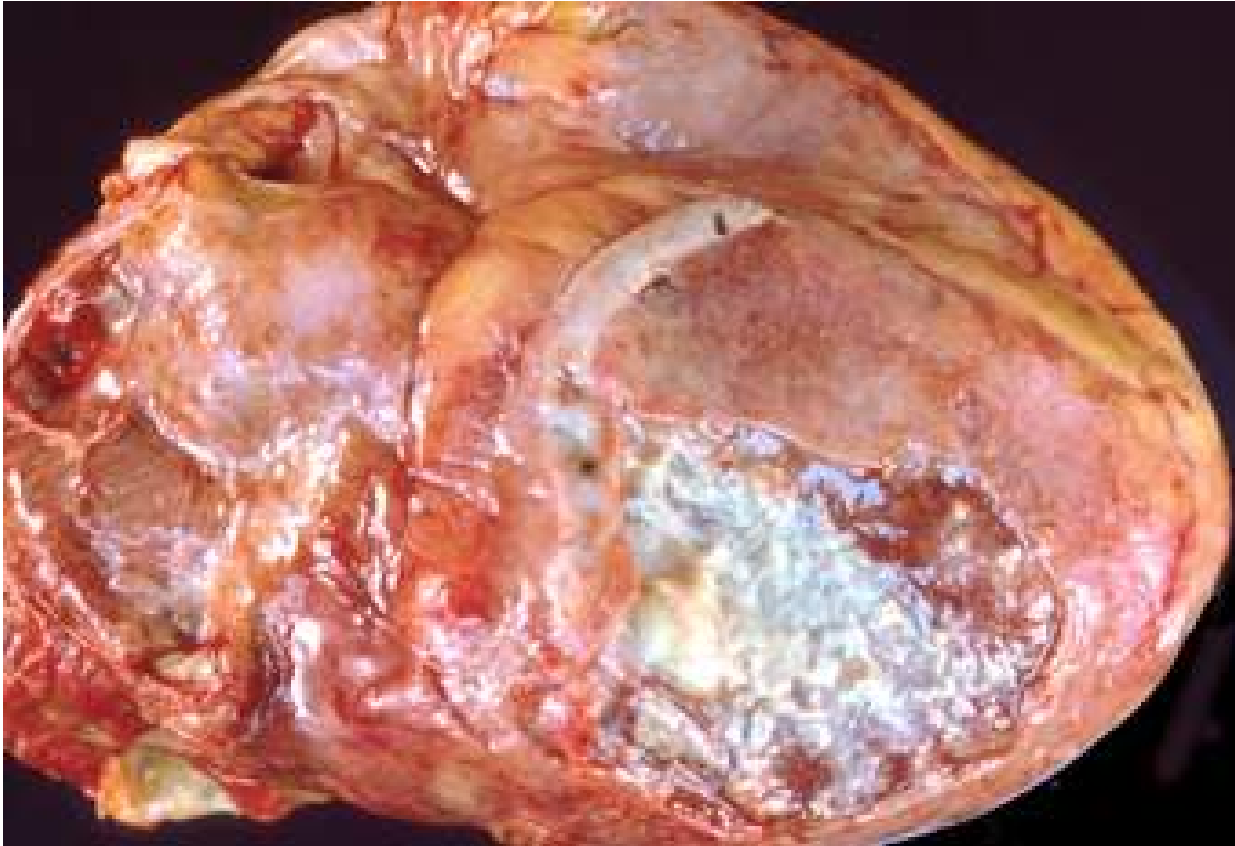
Serous inflammation



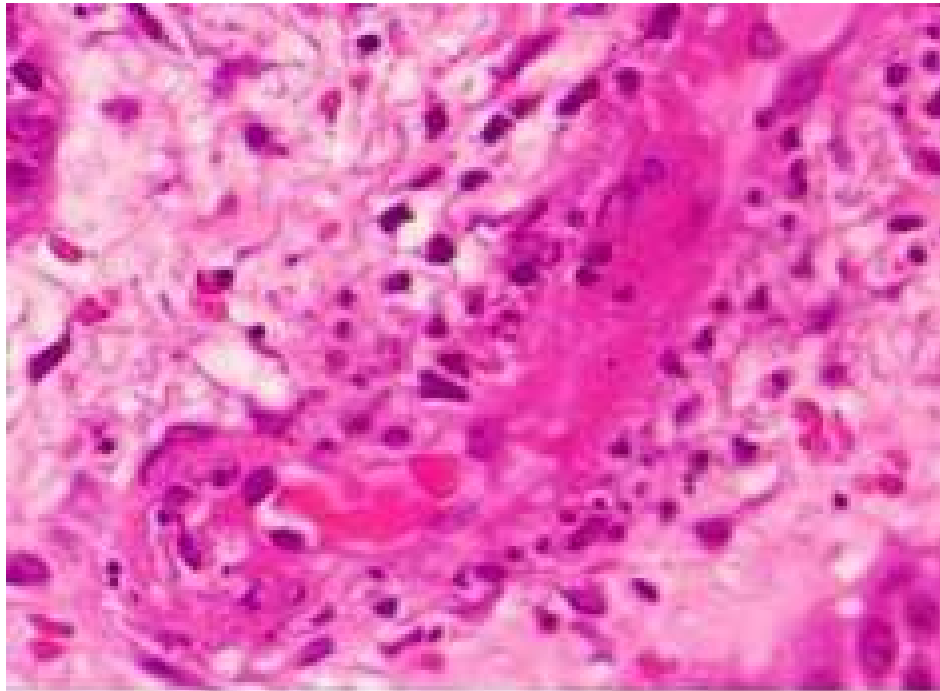
Catarrhal inflammation



Fibrinous inflammation **(Bread & Butter pericarditis)**



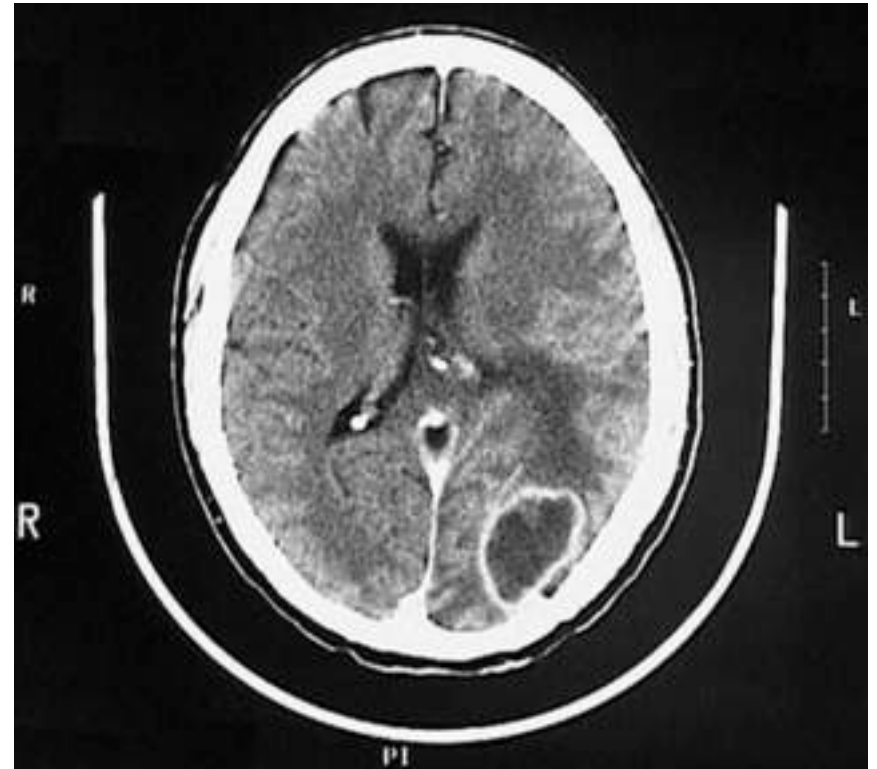
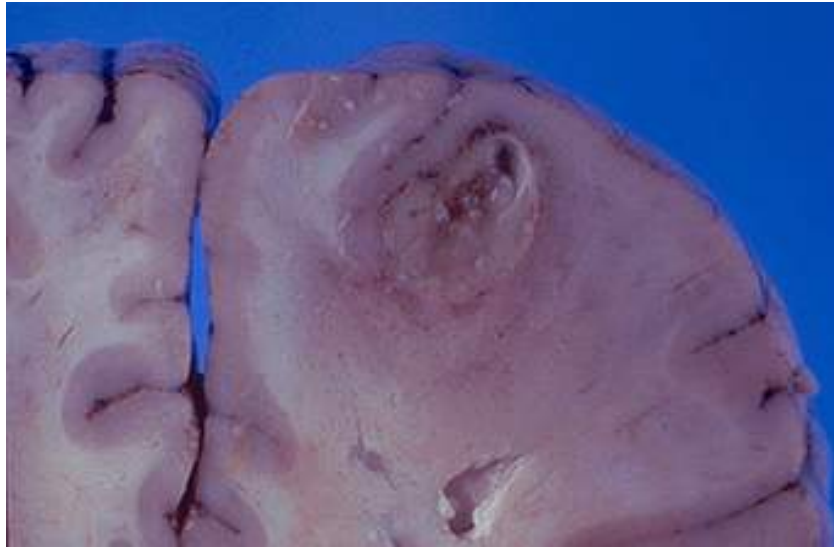
Haemorrhagic inflammation



Meningococcal Septicemia.
A vessel is occluded by a fibrin thrombus.
There are some neutrophils in the
surrounding dermis.



Suppurative inflammation



Pseudomembranous inflammation



Gangrenous or necrotising inflammation



Necrosis



Termination of inflammation

- Resolution
- Fibrosis
- Suppuration
- Necrosis

IV. Chronic inflammation

Chronic inflammation

- exudation is less obvious
- characterized more by changes in cell and connective tissue proliferation

Forms of Chronic inflammation

1. Diffuse interstitial inflammation
2. Granulomatous inflammation

1. Diffuse interstitial inflammation

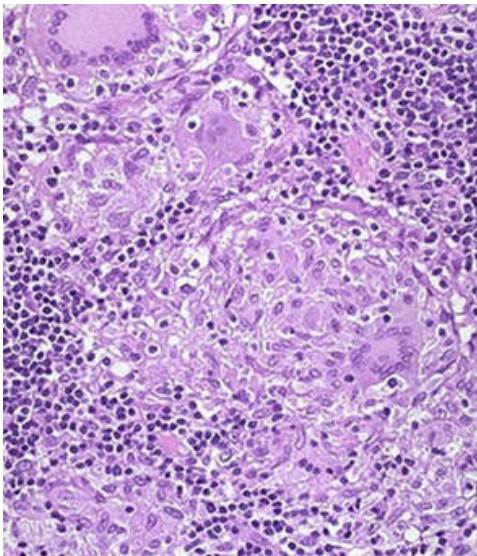
- has no particularly characteristic pattern of tissue reaction.
- The cells involved are monocytes, lymphocytes, plasma cells and fibroblasts (connective tissue cells).

2. Granulomatous inflammation

- There is an attempt to **wall-off** and so isolate the affected site.
- The cells involved are **reticuloendothelial cells** and their derivatives (largely macrophages).

Granuloma

A granuloma is a focal area of granulomatous inflammation which consists of a spherical accumulation of activated macrophages (epithelioid histiocytes) surrounded by lymphocytes & occasional plasma cells and giant cells and usually connective tissue.



Granuloma(Contd.)

- occur in relatively few diseases
- e.g. tuberculosis, syphilis, rheumatic fever
rheumatoid arthritis (as subcutaneous nodules)
foreign-body inflammation

Causes of Chronic Inflammation

- Persistence of infection with microorganisms
- Autoimmunity
- Prolonged exposure to either exogenous or endogenous toxins
- Persistence of acute inflammation

V. Systemic effects of inflammation

Systemic effects of inflammation

- Constitutional symptoms
- Haematological changes
- Pyrexia
- Reactive hyperplasia of the reticuloendothelial system
- Amyloidosis

VI. Factors affecting healing

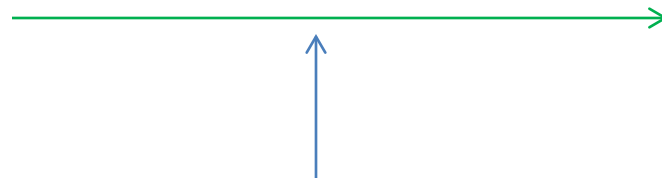
Factors affecting healing

- Many factors affect the healing of an individual, from their own hormonal status through to the use of deliberate interventions such as the use of a cold compress, ultrasound or drugs.

Healing of a skin wound

1. Primary union(First intention)
2. Secondary union(Second intention)

Proline



Hydroxyproline

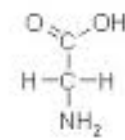
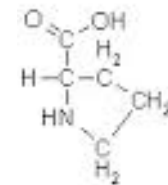
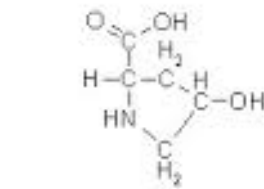
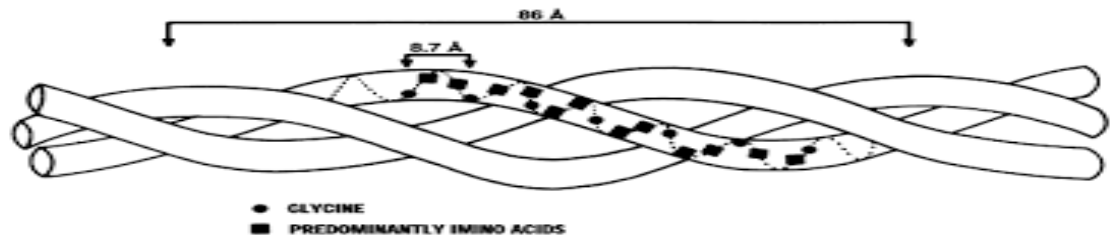
Proline hydroxylase

Oxygen

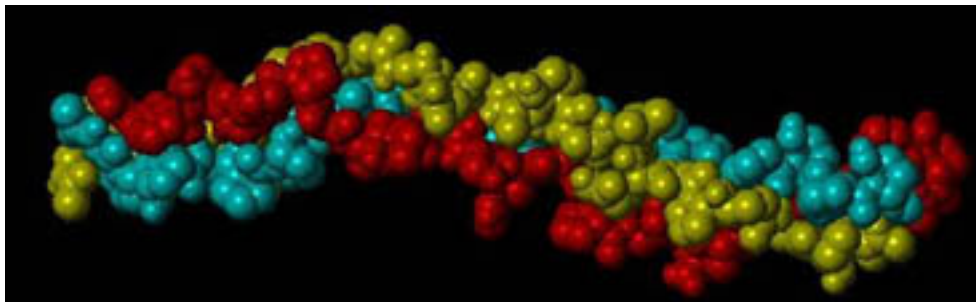
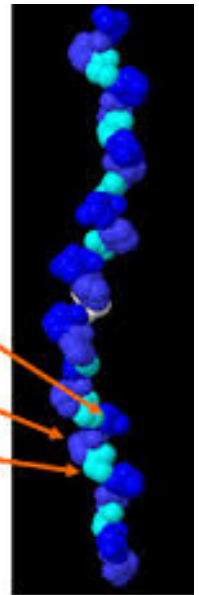
Vitamin C



Triple helix structure of collagen

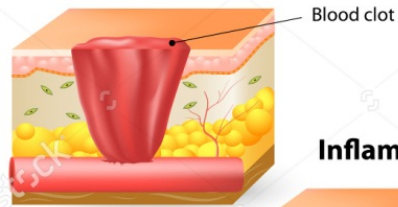


Hydroxyproline
Proline
Glycine

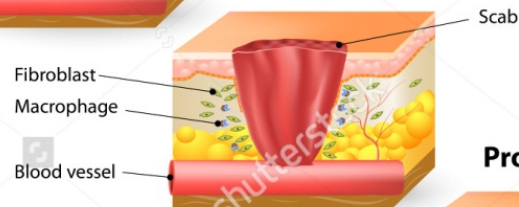


WOUND HEALING

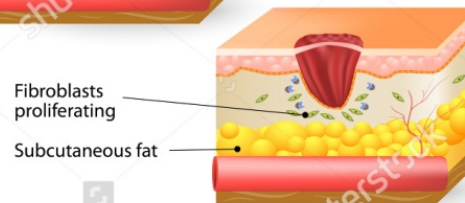
Bleeding



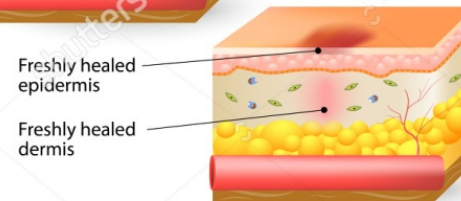
Inflammatory



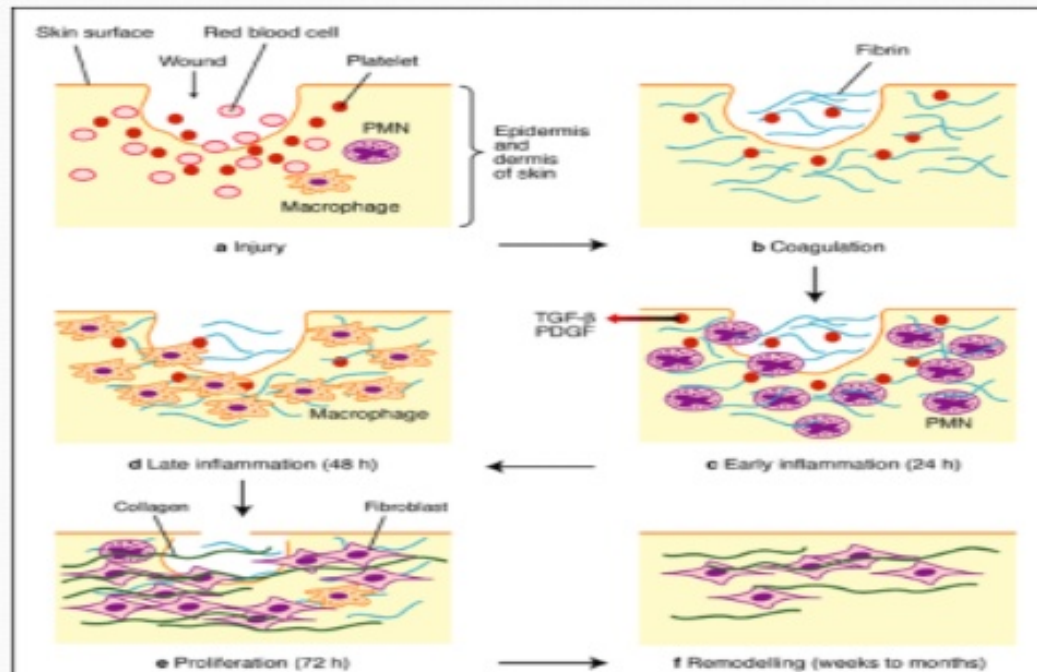
Proliferative



Remodeling



Stages of Wound Healing



The phases of cutaneous wound healing

Expert Reviews in Molecular Medicine © 2003 Cambridge University Press

VII. Treatment of inflammation

Treatment of inflammation

- **Temperature Therapy**
 - Cold therapy (cryotherapy)
 - Heat therapy
- **Mechanical Manipulation**
- **Electrotherapy**
- **Ultrasound Therapy**
- **Drug Therapy**

Review

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QUESTIONS?

THANK YOU

