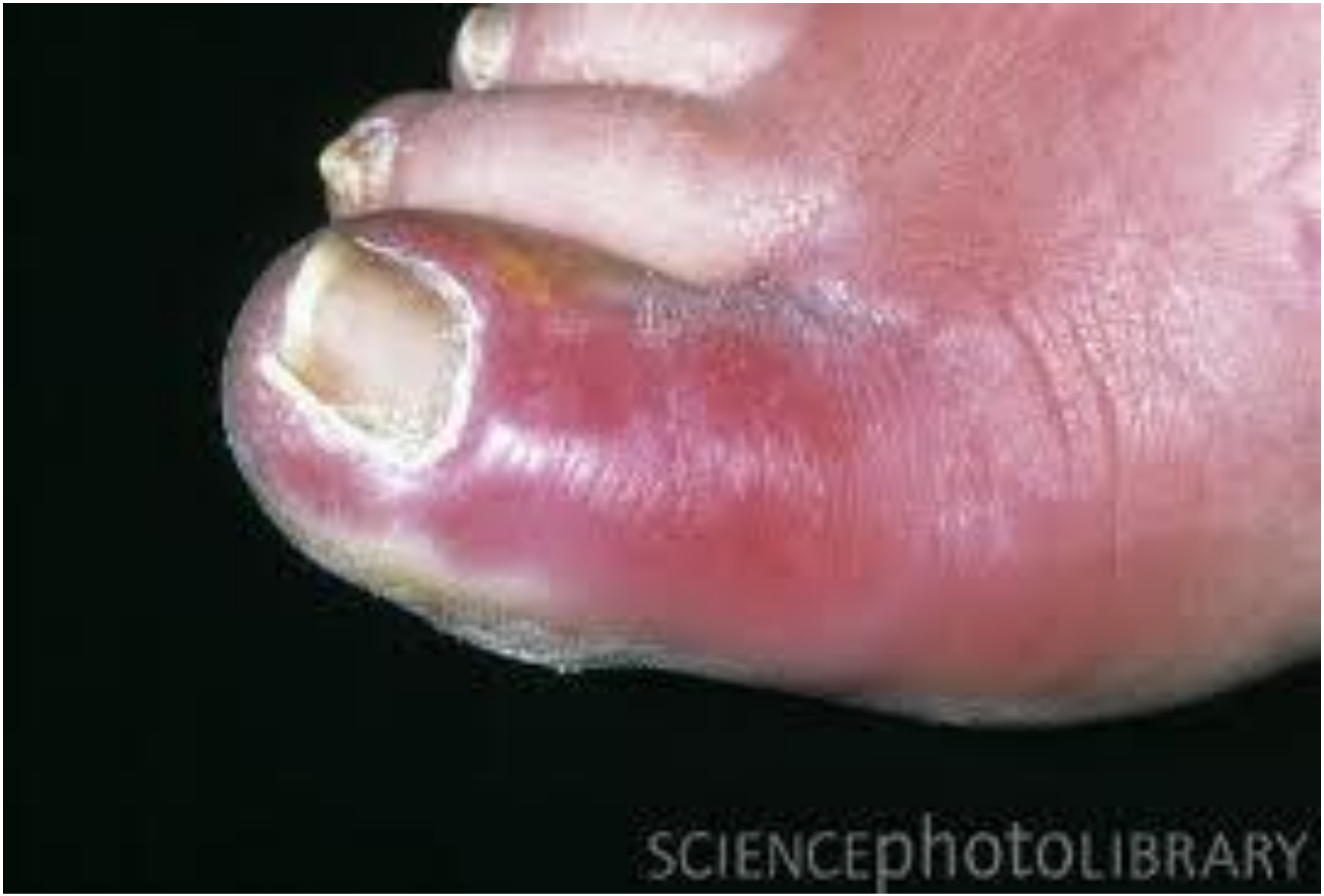
ACUTE INFLAMMATION

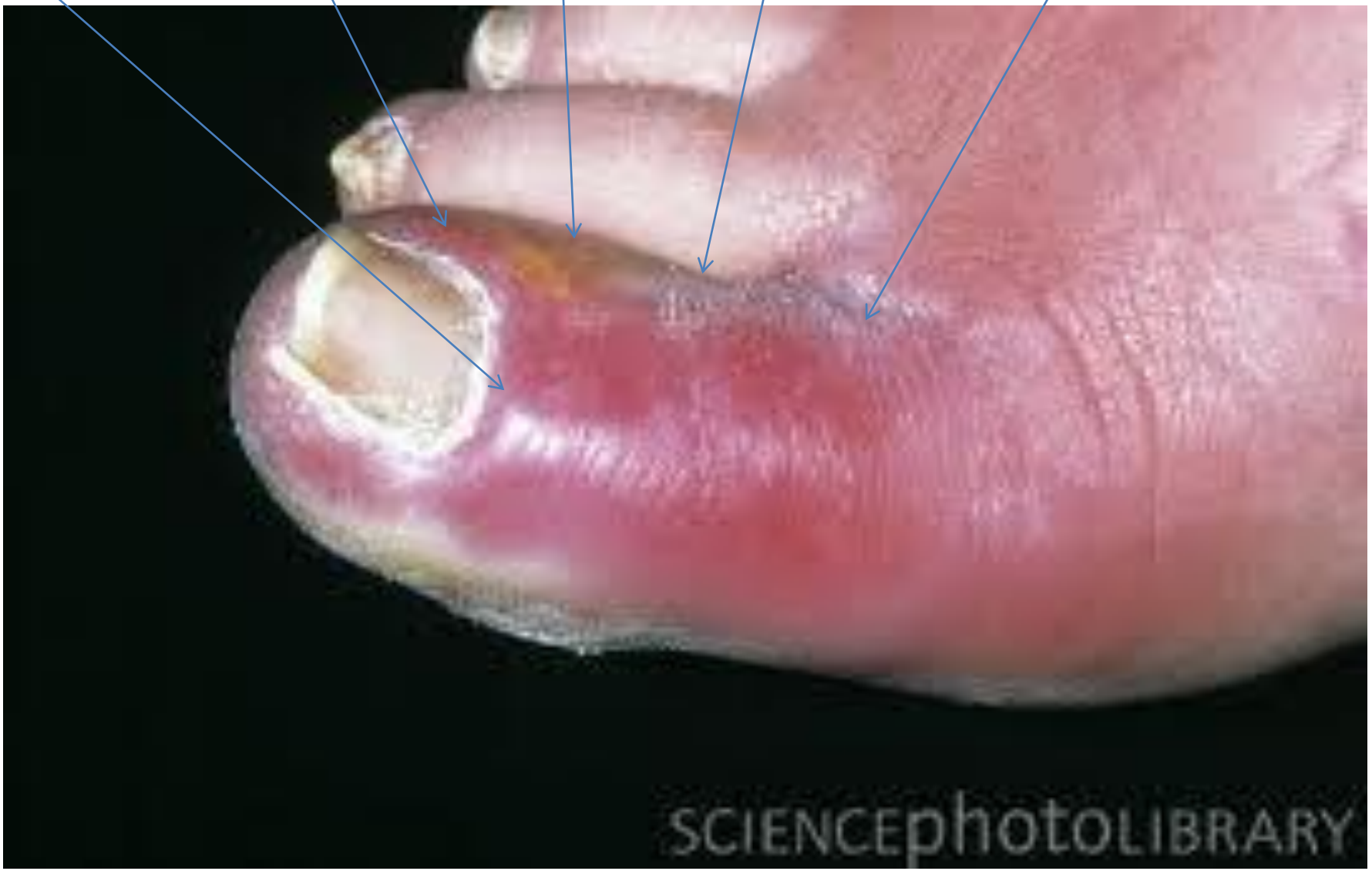
2020-2021

FLAME

WHAT ARE THE CRITERIA OF ACUTE INFLAMMATION?



Red, swollen, hot, painful, no function



GREEK

Calor, rubor, tumor, dolor, functio laesa

ACUTE INFLAMMATION

Definition: it is local response of living tissue to injury. Inflammation is the reaction of the vacular & supporting parts of a tissue to injury with the result of formation of protein and cells rich exaudate.

It is a process by which cells & plasma accumulate around an injurious substance and tend to remove or destroy it.

Add (itis) to organ it means inflammation weather acute or chronic like appendicitis, cholecystitis, tonsilitis, gingivitis, somatitis.

CAUSES OF ACUTE INFLAMMATION

1-Mechanical: cutting, crushing.

2-Living organism: bacteria, viruses, parasites.

3-Physical: radiation, cold, heat(burn).

4-Chemical.

5-Immunological: allergic dermatitis, asthma.

6-Tissue necrosis: as a result of hypoxia, trauma

e.g. infarction, like myocardial infarction.

CHANGES IN ACUTE INFLAMMATION

1- Changes in the caliber of the blood vessels. (arteriolar vasodialatation, so also capillaries & venules).

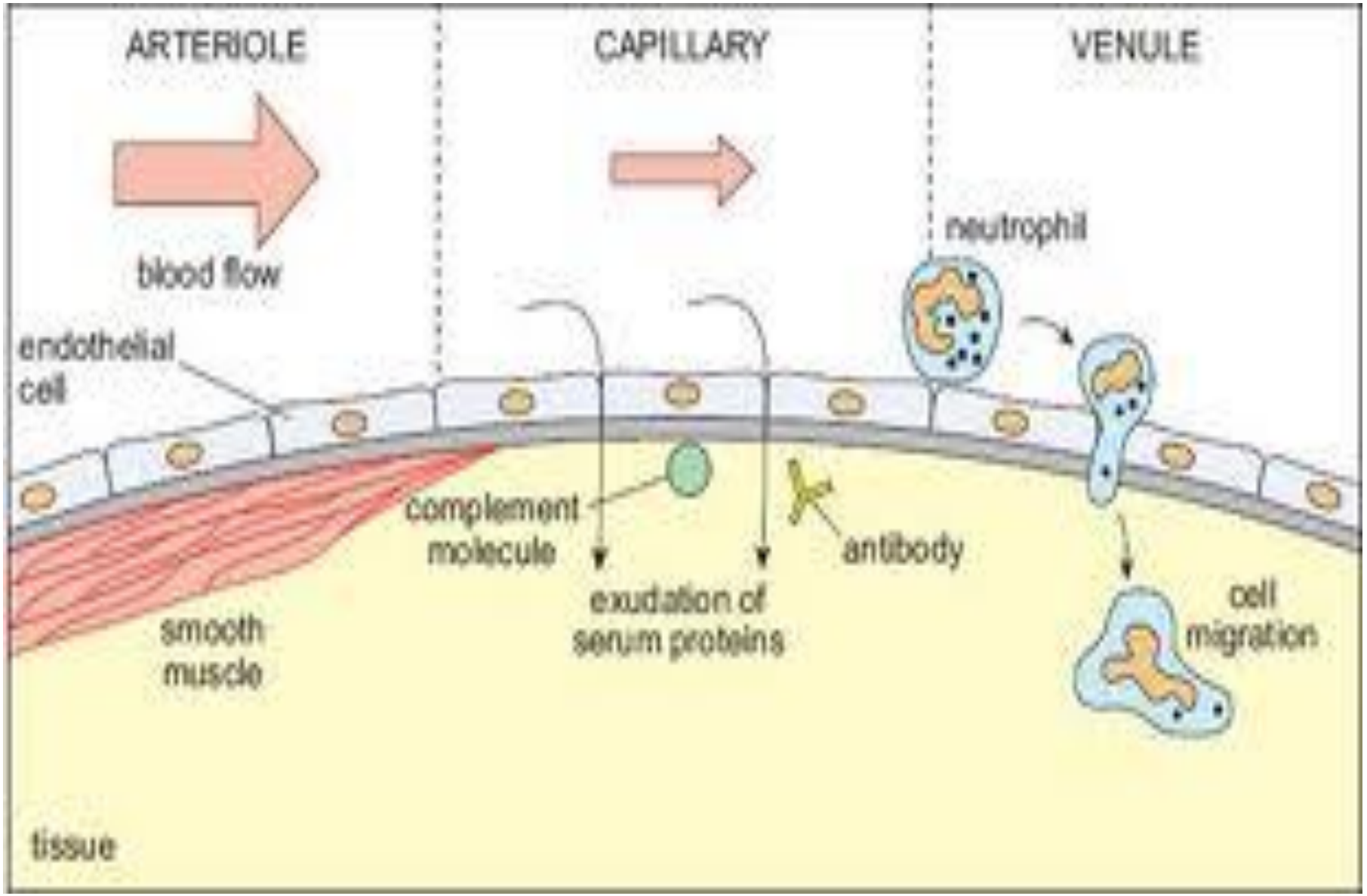
So increase blood flow and volume in the inflammed area because of the chemical mediators released as a result of the injury so also as a result of local nervous reflex, and this causes redness and raise of the tissue temperature i.e. hot area.

2-Changes in the blood flow. slow, stasis and

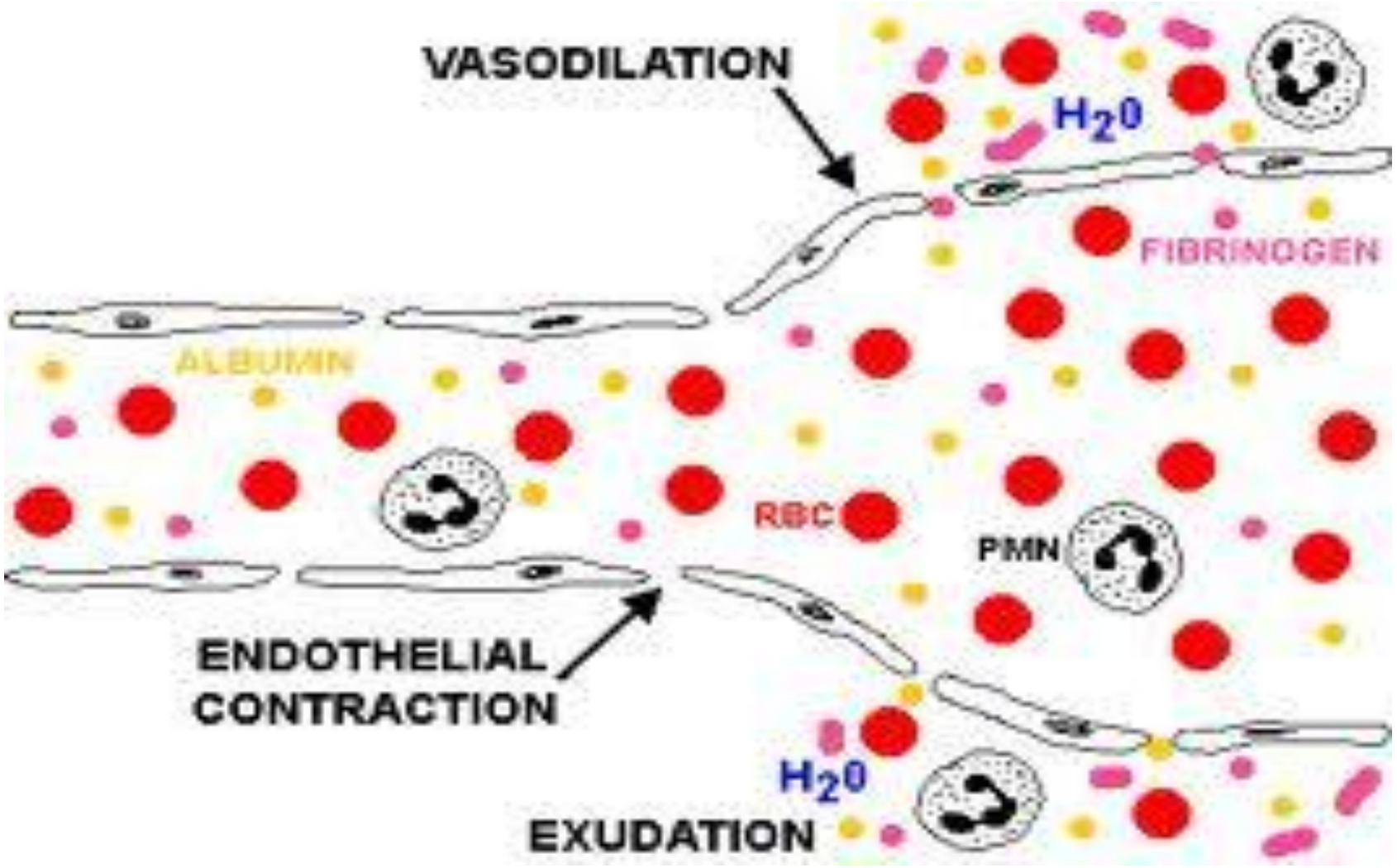
even thrombosis.

3-Changes in vessels permeability:

VESSEL CHANGES—FLUID&CELL EXUDATION



VASODIALATATION WITH CELLULAR EXUDATION



VASCULR CHANGES---EXUDATION

Changes associated with vascular changes:

1- Fluid exudation: this is passage of protein rich fluid from the vessels to the injuried

tissue, because of vasodialatation, increase

capillary pressure and increase vascular permeability. This fluid exudate causing the swelling and pain during inflammation.

Function of the fluid exudate:

a-Containing antibody.

CONT,D

b-Drugs & antibiotics.

c-Diluting the irritants.

d-High fibrinogen content which helps in:

a-Union & healing of the damaged tissue.

b-Barrier against bacterial spread.

c-It helps in trapping bacteria and subsequent phagocytosis by polymorphs & macrophages.

CONT,D EXUDATION

2- Cellular exudation :

Passages of blood cells out side the blood vessels. The W.B.C actively pass between endothelial cells of the vessel and it is called emigration.

First cell emigrates is polymorphs that is with in 2-9 mins. Then monocytes to be macrophage after several hours, their function to egulf bacteria or dead tissue and remove them from the field.

Lymphocytes coming late but in viral infection they come earlier, main action to stimulate

macrophage movement & activity, so also

producing antibody.

CONT,D ---CELLULAR EXUDATION

Eosinophils present in excess in allergic inflammation.

Red blood cells leaving the blood vessel in a passive way in between endothelial cells called diapedesis.

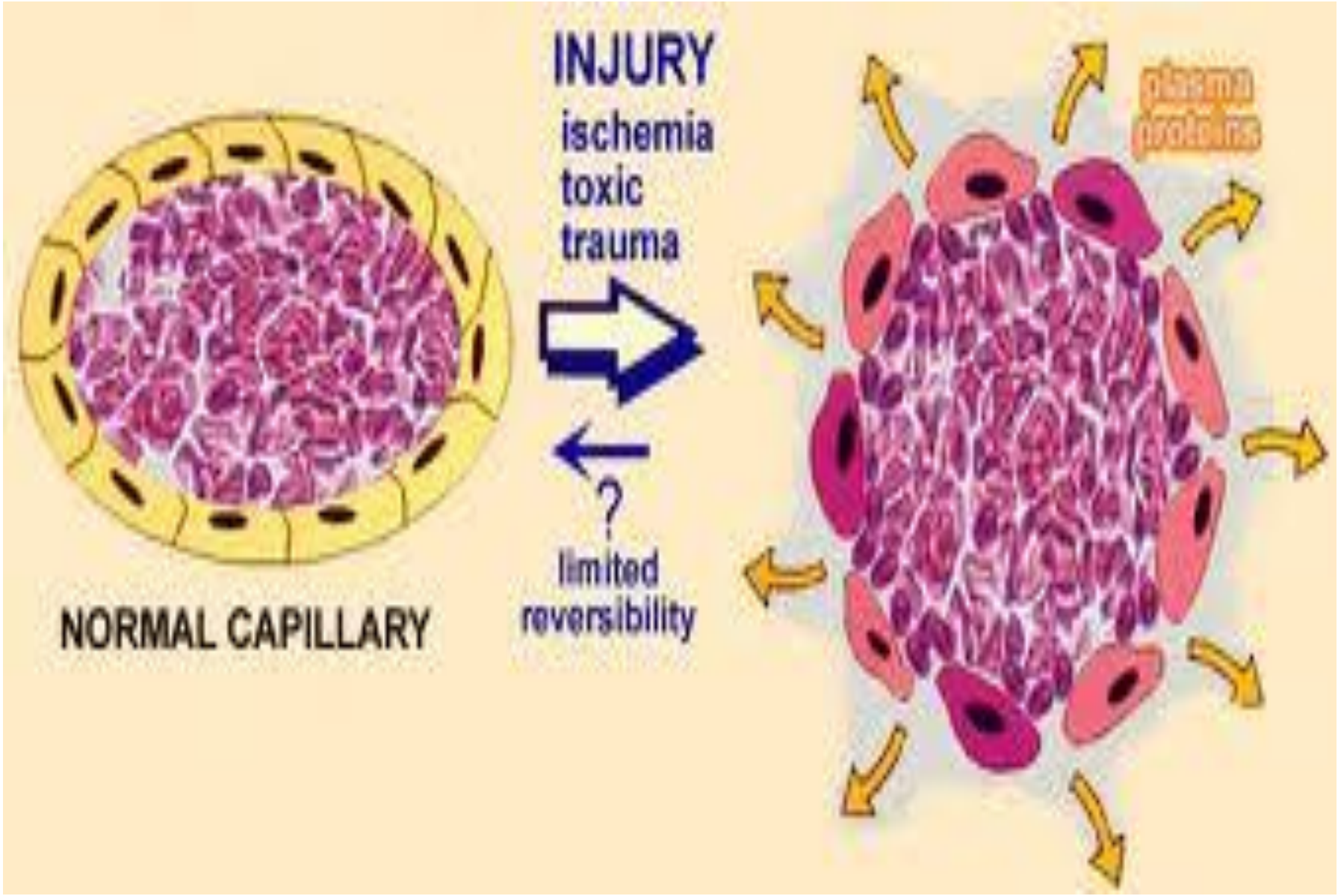
Mast cells (connective tissue cell), involved

in the inflammatory process, producing

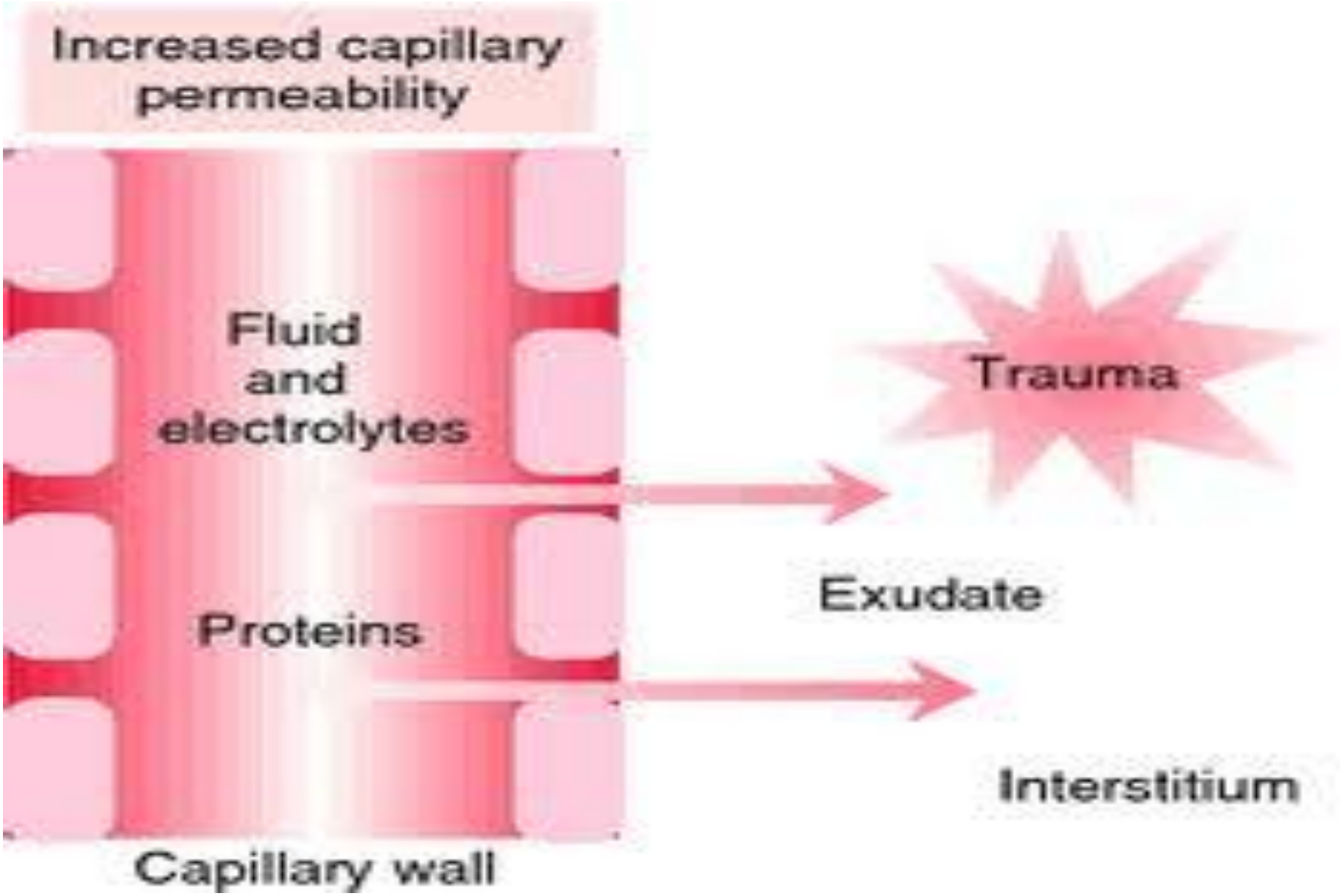
some chemicals e.g.histamine, heparin, 5 hydroxy tryptamine.

Platelets also involved in inflammatory process.

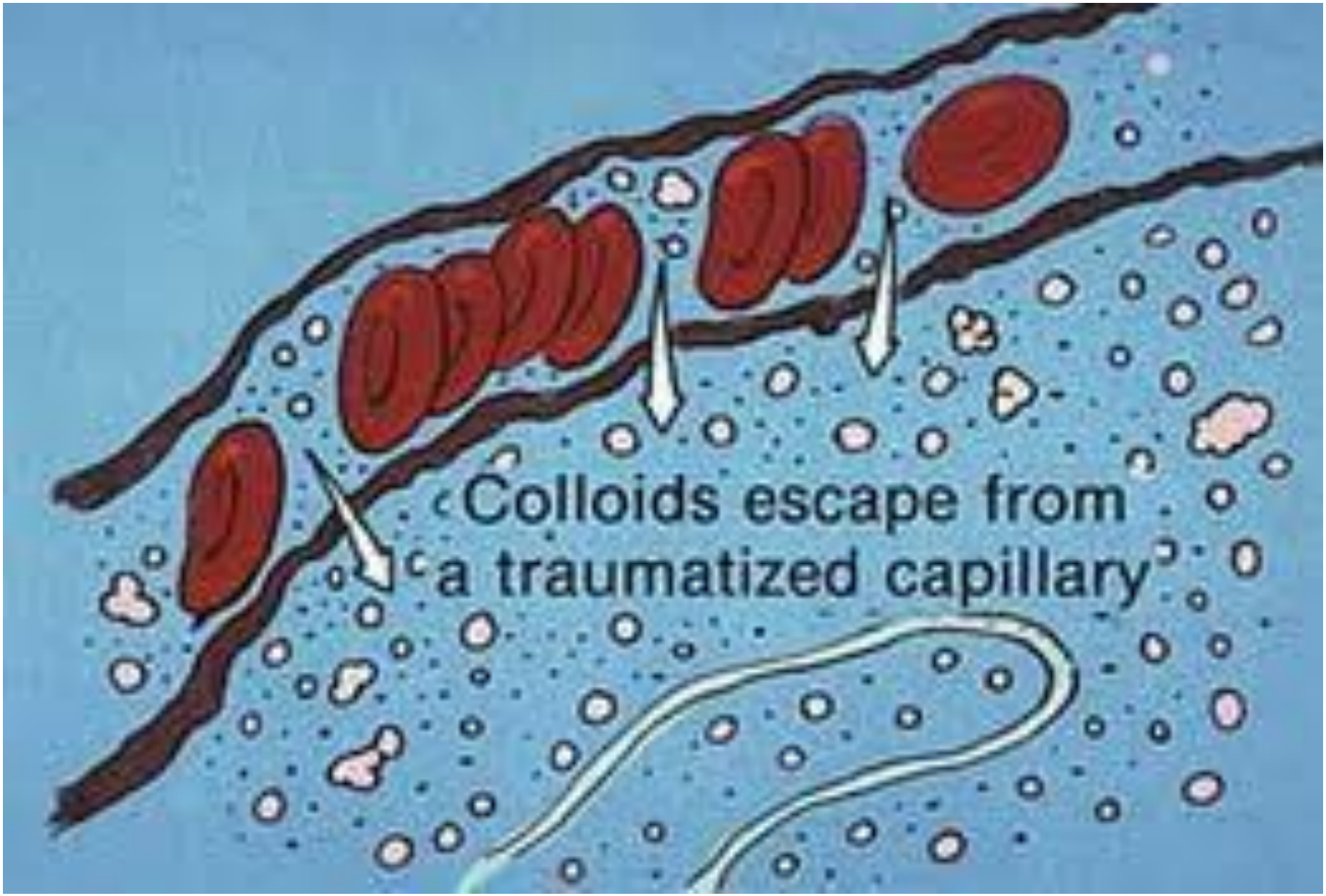
EXUDATION


INJURY
ischemia
toxic
trauma
?
limited
reversibility
NORMAL CAPILLARY
plasma proteins

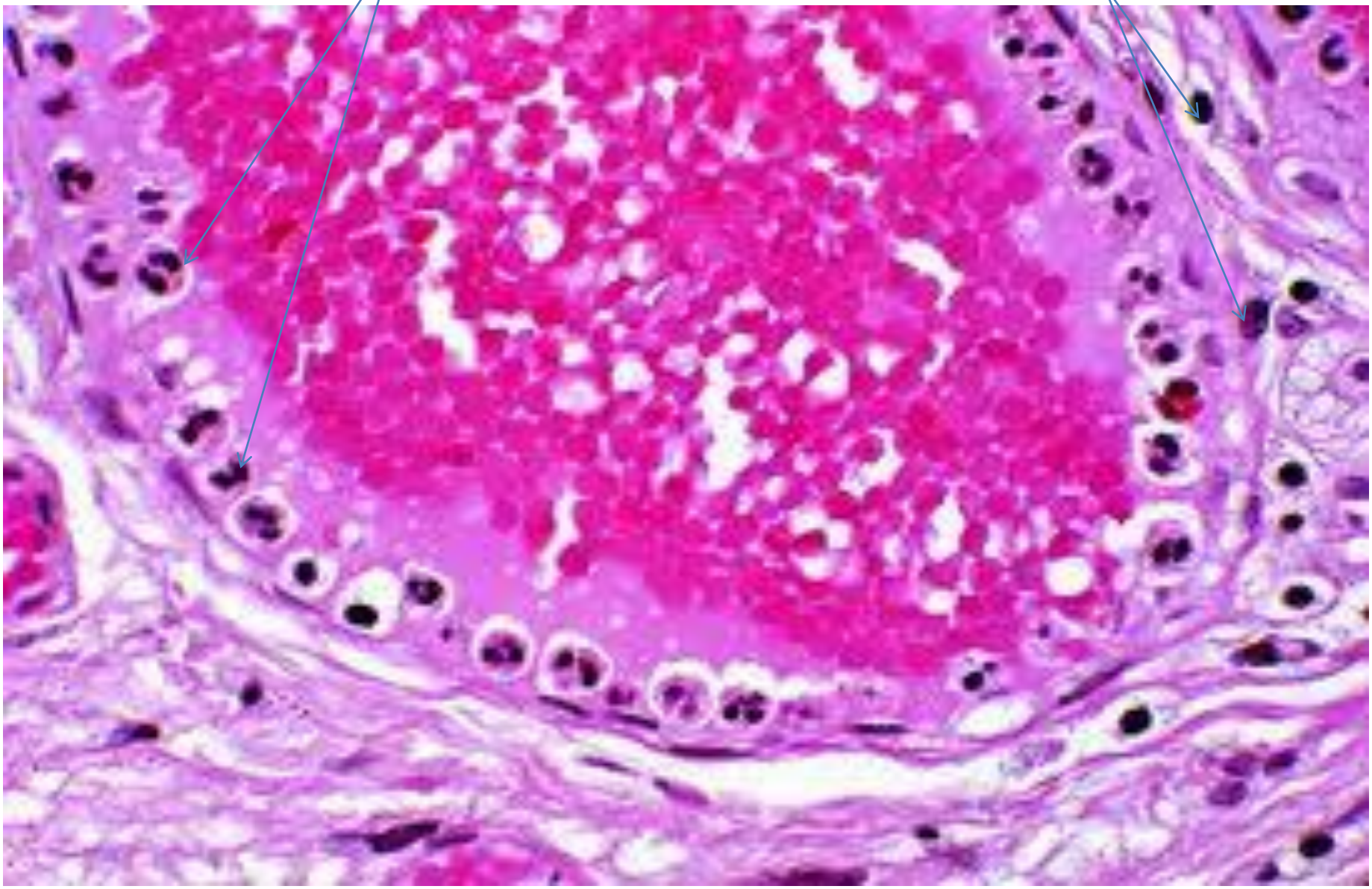
CAPILLARY PERMEABILITY



COLLOIDS EXUDATION

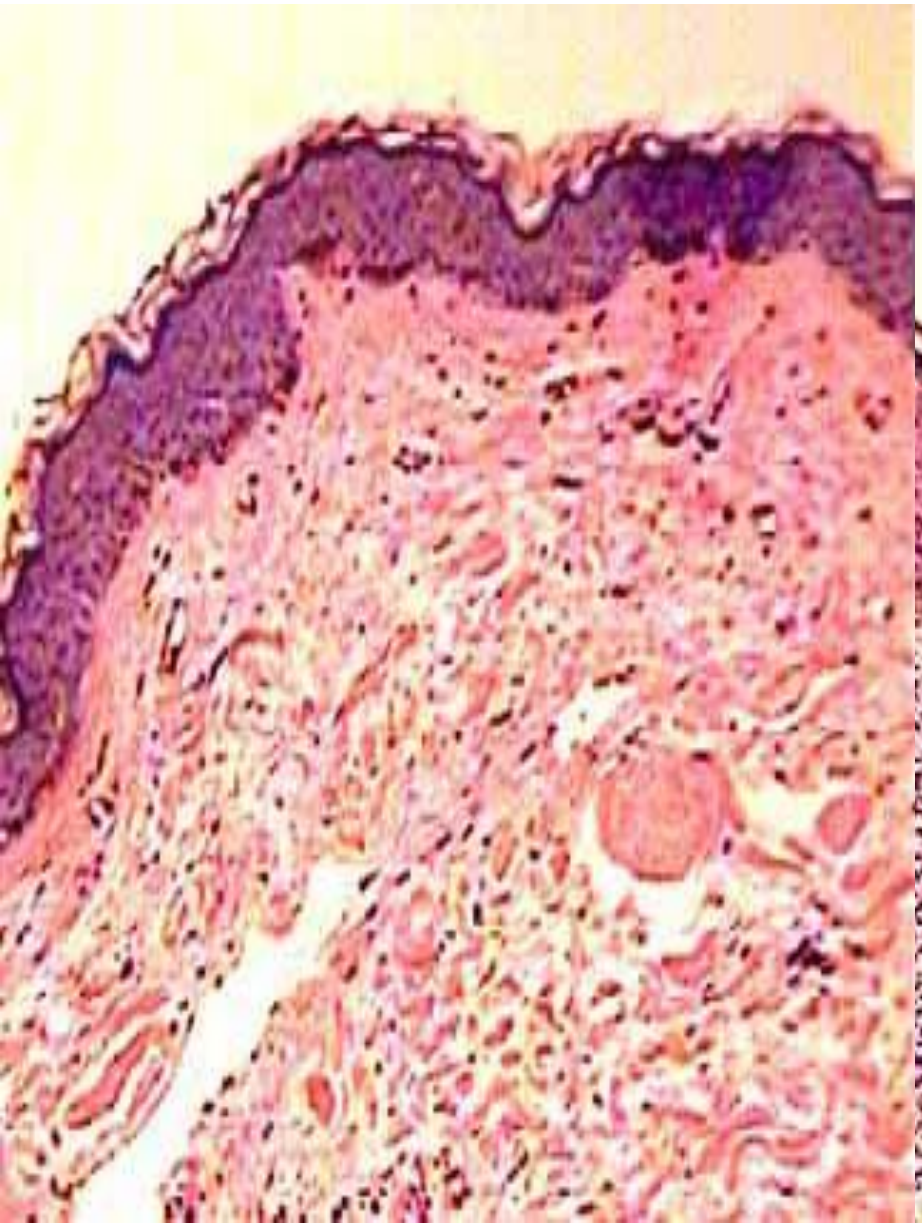


WBC MARGINATION BEFORE EMIGRATION

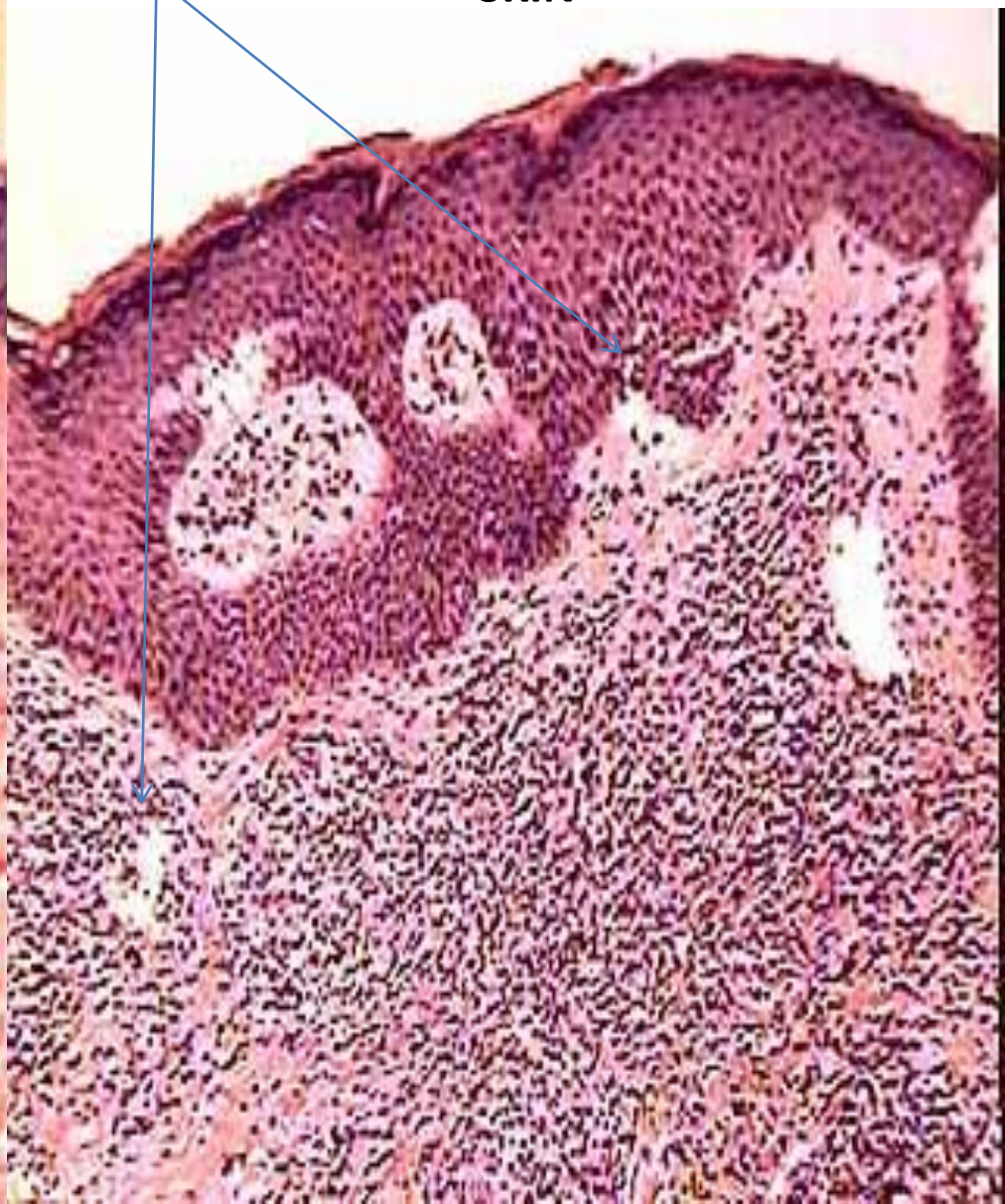


COMPARISON BETWEEN NORMAL SKIN&INFLAMMED ONE

NORMAL SKIN



WBC FILLING THE INFLAMMED AREA - SKIN

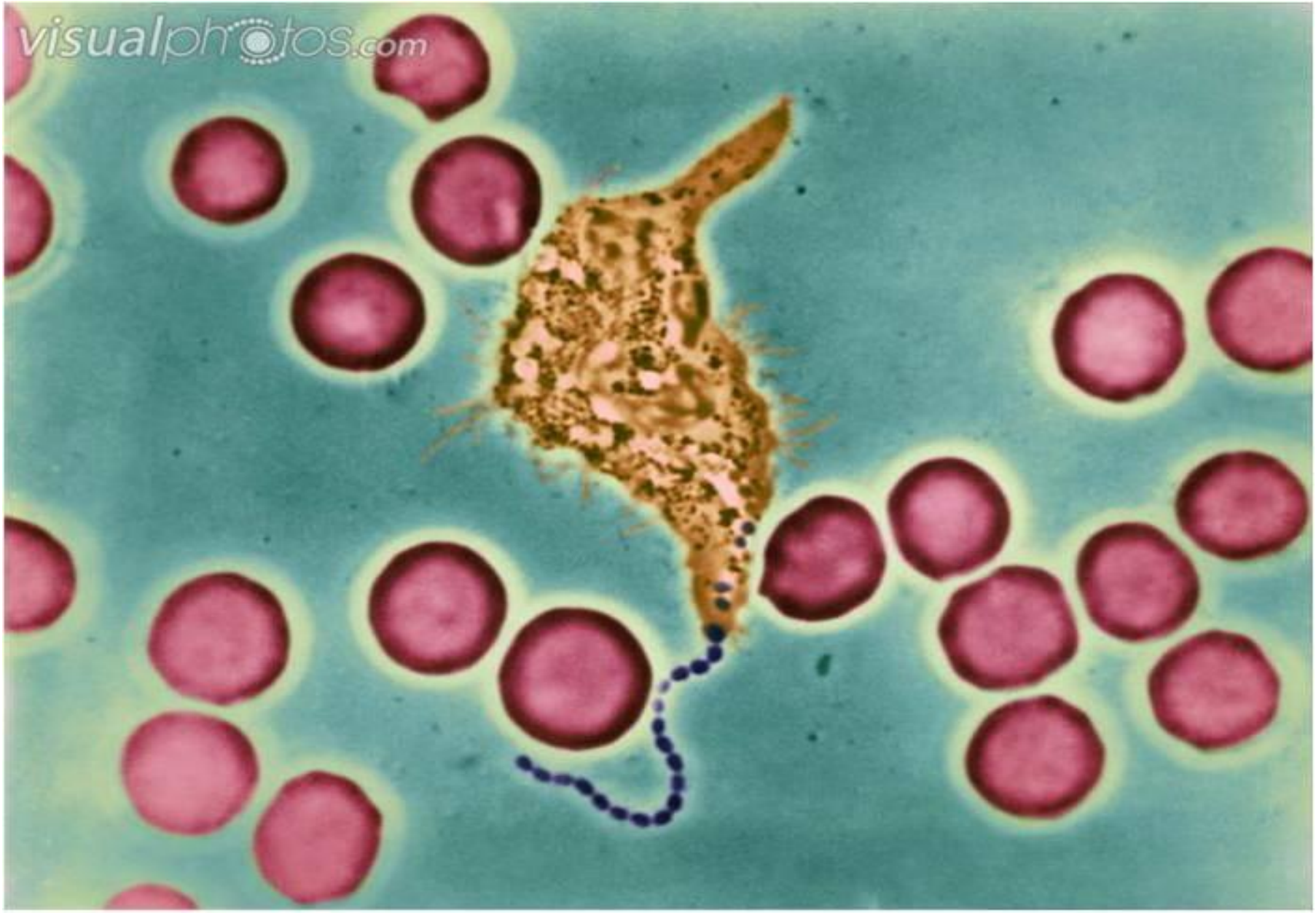


WBC FUNCTIONS

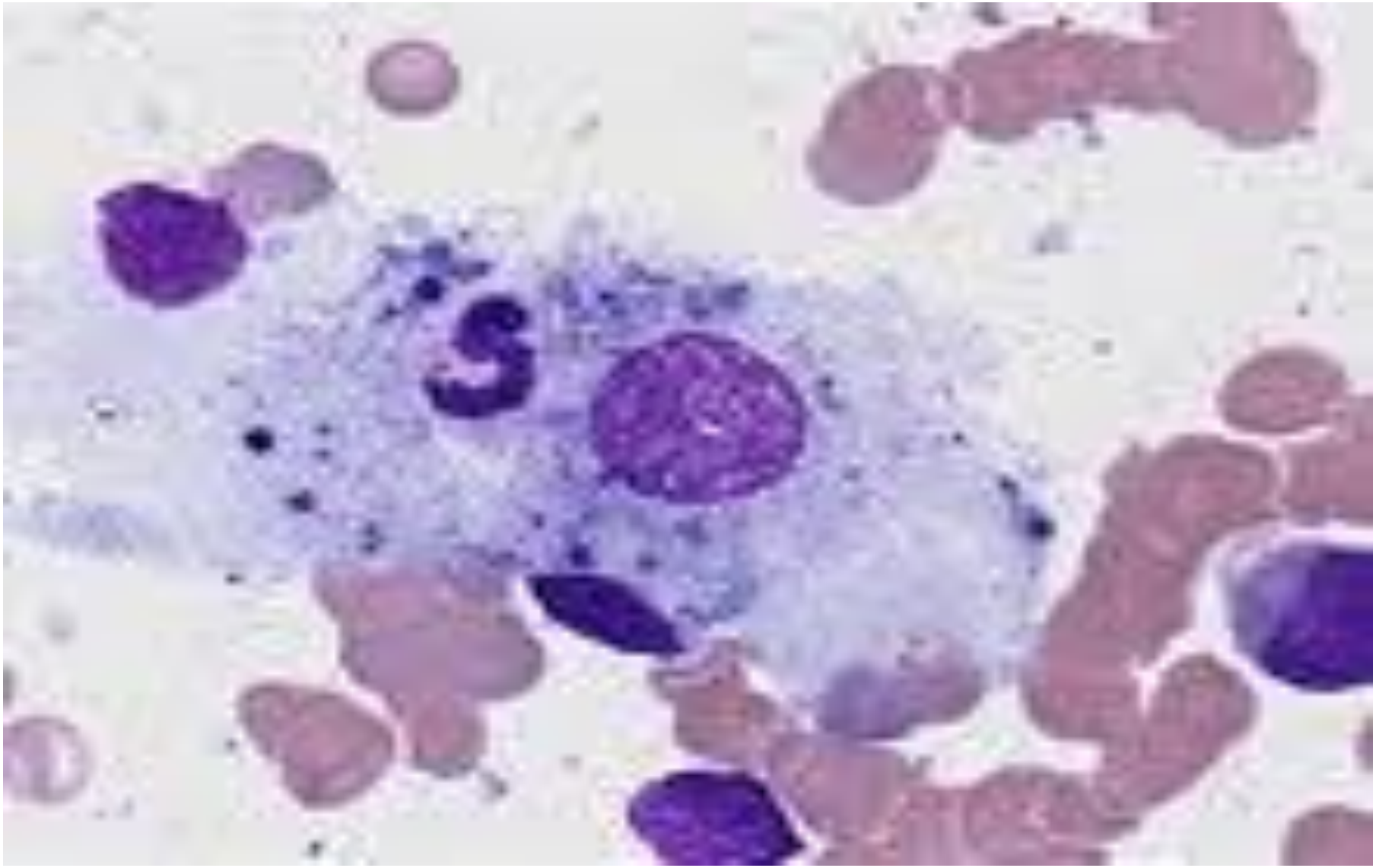
Phagocytosis of the bacteria. Opsonin it is natural non-specific antibody covering the bacteria to make it easily phagocytosed by polymorhs & macrophages. The process called opsonisation.

Phagocytosis without opsonisation of the bacteria called surface phagocytosis where WBC depend on entrapement of bacteria by fibrin.

PHAGOCYTOSIS-WBC ENGULFING THE BACTERIA



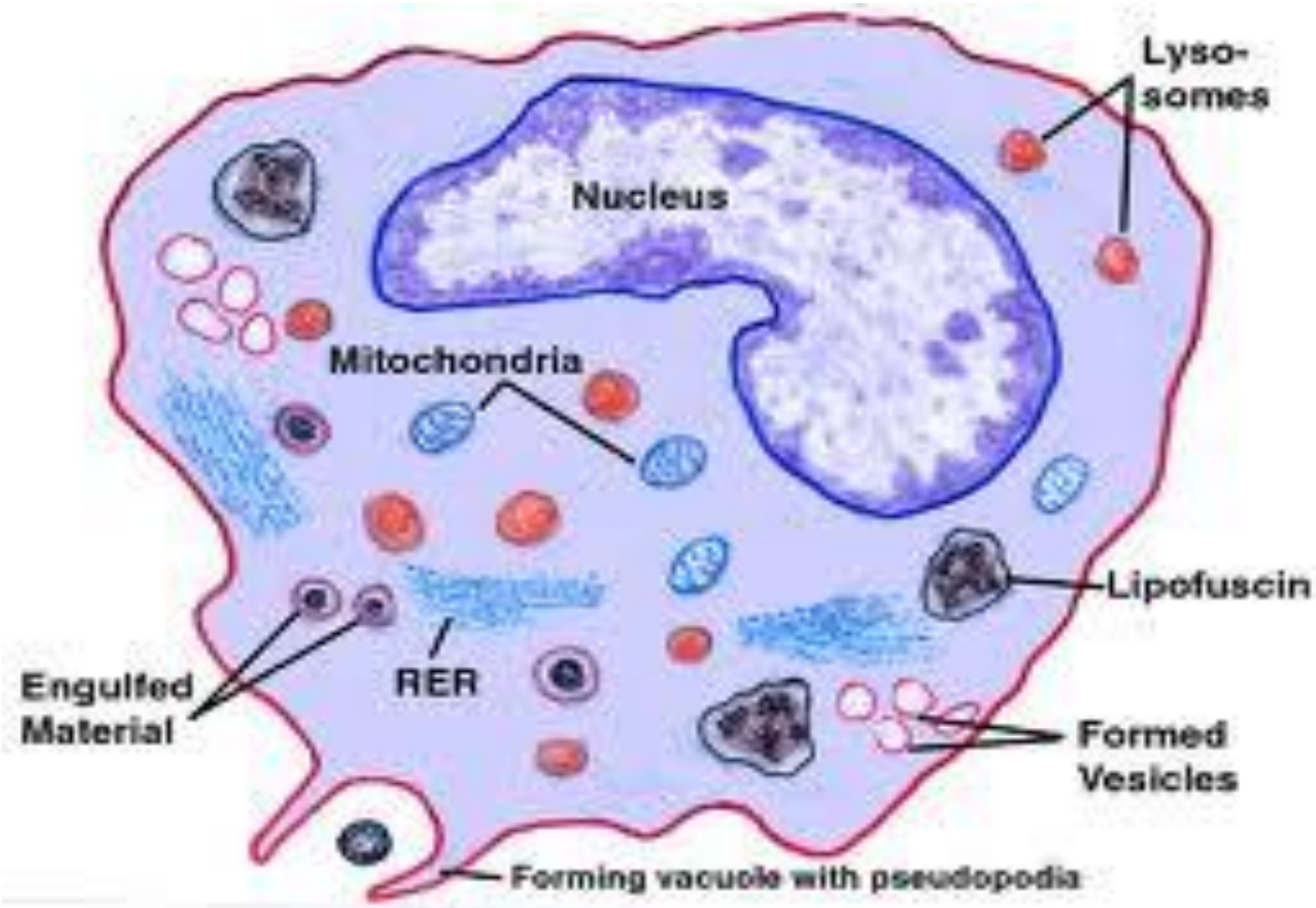
MACROPHAGE WITH BACTERIA INSIDE



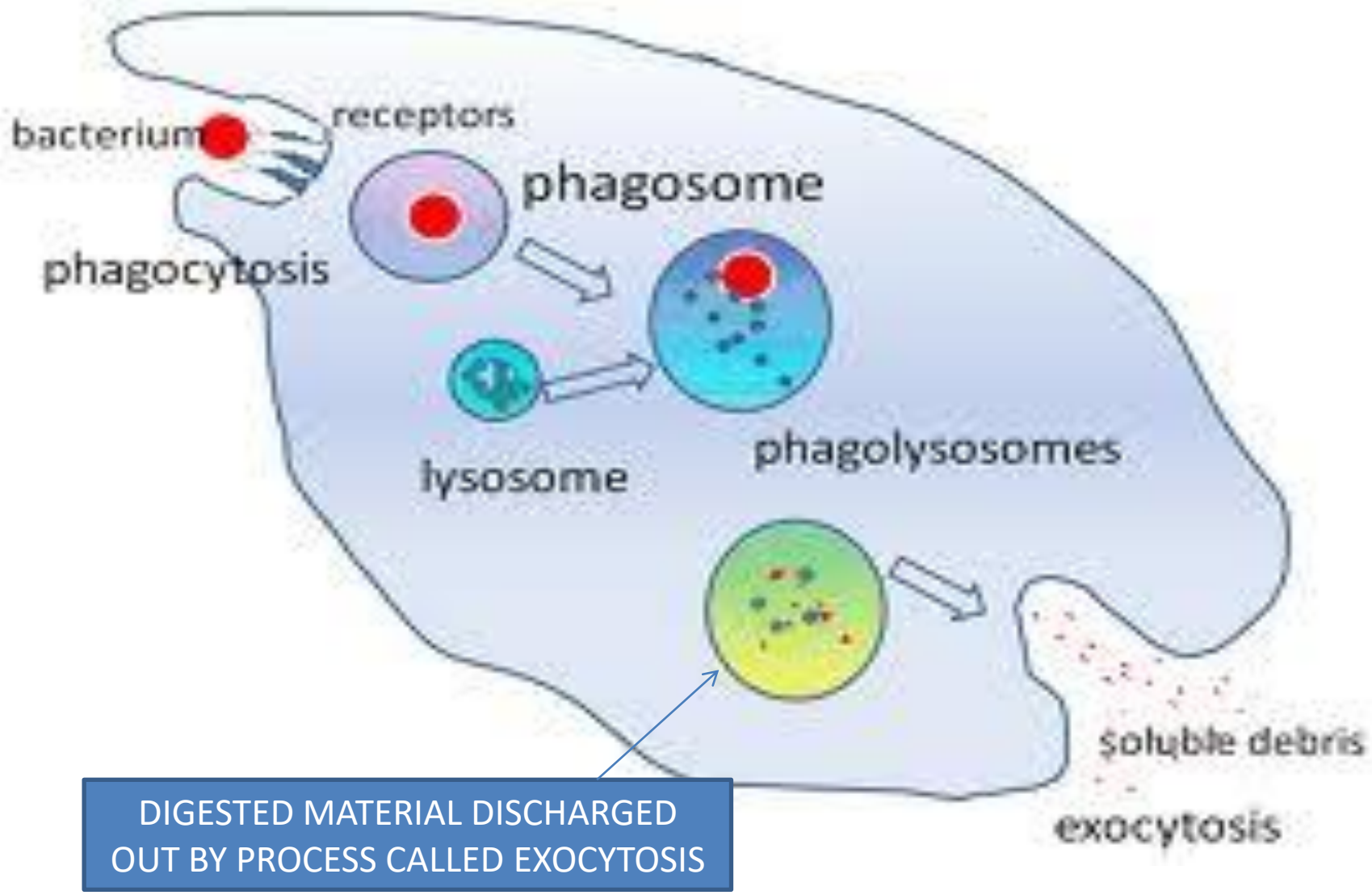
Fate of ingested bacteria & dead tissues inside WBC

The microrganism after ingestion will be digested by enzymes realesed from lysozymes like lysozomal enzymes, acid hydrolytic enzymes, lysozymes, acidity, lactoferrin, alkaline phosphatase, etc.

DIGESTION OF ENGULFED MATERIAL



INGESTED MATERIAL CALLED PHAGOSOME+LYSOZOME=PHAGOLYSOZOME



THE PATHOLOGICAL CHANGES DURING INFLAMMATION

The injurious agent affecting the tissue causing the acute inflammation by 2 factors :

1-local nervous reflex.

2-chemical mediators.

a-Amines: histamine, produced by mast cells , basophils, and platelets.

5- hydroxytryptamine(serotonin) produced by platelets.

b-The kinins(derived from plasma protein called kininogens): like bradykinin: causes vasodilatation, increase vascular permability and produce pain.

c-Polymorphs products.

d-Complement products.

CONT,D –CHEMICAL MEDIATORS IN ACUTE INFLAMMATION

e-Arachidonic acid derivatives:

Prostaglandins :thromoxane a2 prostacycline

Leukotriens:

Prostaglandins produced by all body cells:

In inflammation cause vasodialatation, increase vascular permeability,

it also causes pain. So any drug inhibits prostaglandins will inhibit inflammation and

decrease pain like aspirin, voltaren, ibuprufen.

CONT,D - ARACHIDONIC ACID DERIVATIVES

So they are called prostaglandins inhibitors-

anti inflammatory & analgesics.

Leukotriens: arachidonic acid derivative -chemical mediator, causing bronchospasm in asthma, and its release inhibited by glucocorticoids (cortisone).

f-Other chemical mediator called cytokines:
Interleukin 1 (IL1)

Interleukin 6 (IL6)

Interleukin 8 (IL8)

Tumour necrosis factor(TNF)

CONT,D

These cytokines produced by monocytes, polymorphs and t-lymphocytes.

Interleukin 1 causes inflammation and

fever by acting through prostaglandins, through heat regulating center in the hypothalamus

and so fever inhibited by aspirin, diclofenac (voltaren), ibuprufen, etc. by inhibiting prostaglandins.

Also relieve pain through prostaglandins

inhibition.

SYSTEMIC EFFECTS OF ACUTE INFLAMMATION

Fever: due to effect of the cytokines; IL1

and tumour necrosis factor(TNF), directly on heat regulating center in the hypothalamus.

C-reactive protein: increase production by the liver. So also other proteins.

Increase glucocorticoids production by adrenal (suprarenal gland).

Leucocytes production increase: as a result of IL1 (interleukin1) & TNF stimulation to the bone marrow. In longstanding inflammation leucocytosis depend on colony stimulating factor produced by macrophages.

SO IN SUMMARY:

ACUTE INFLAMMATION:

Red: vasodilatation.

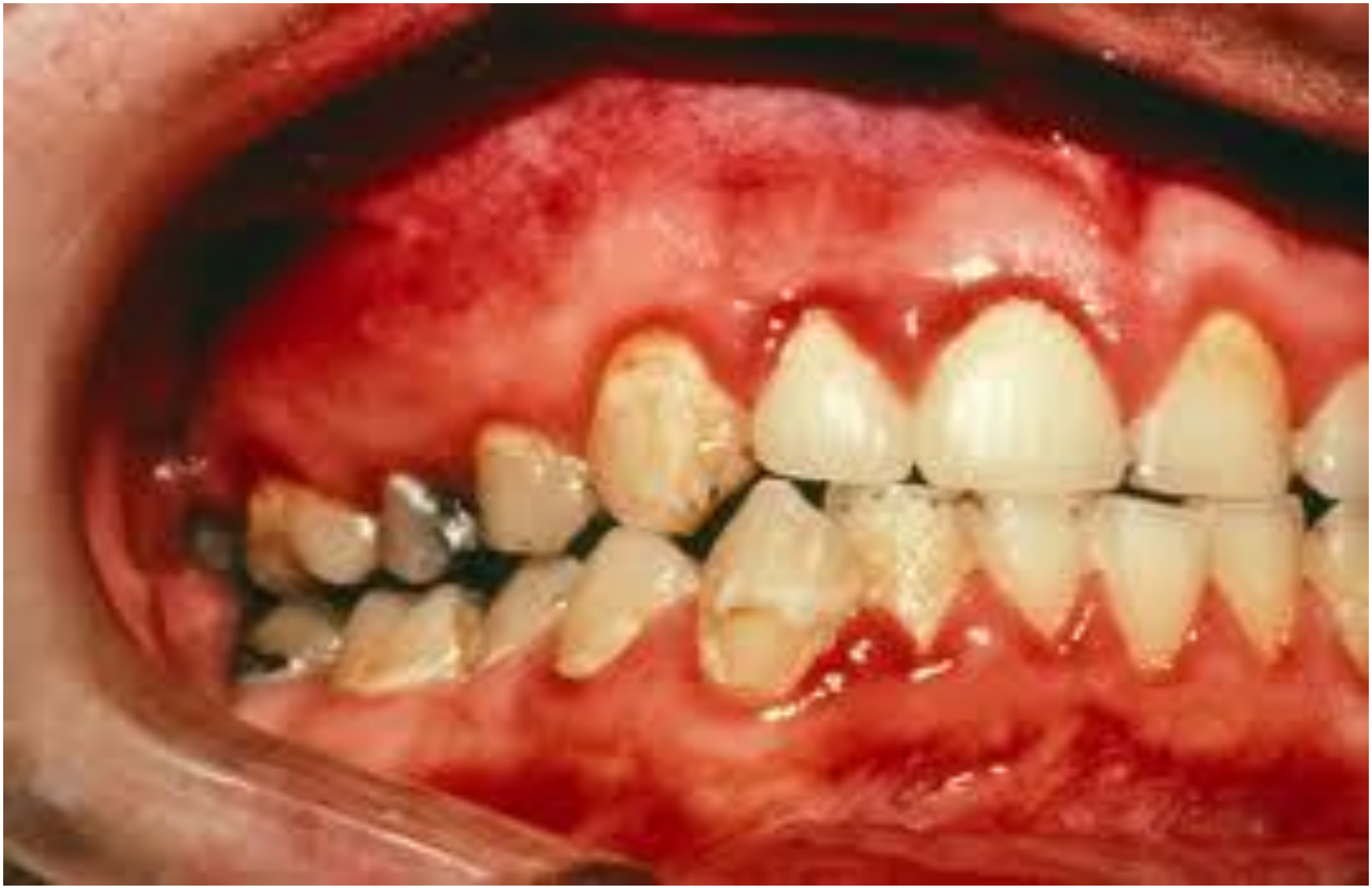
Hot: vasodialatation.

Swelling: exudation.

Pain: exudation-pressure on nerves and chemical mediators (kinin, prostaglandins, acid,.etc.). So aspirin, ibuprufen and voltaren inhibit release of those chemicals and so decrease the pain during inflammation.

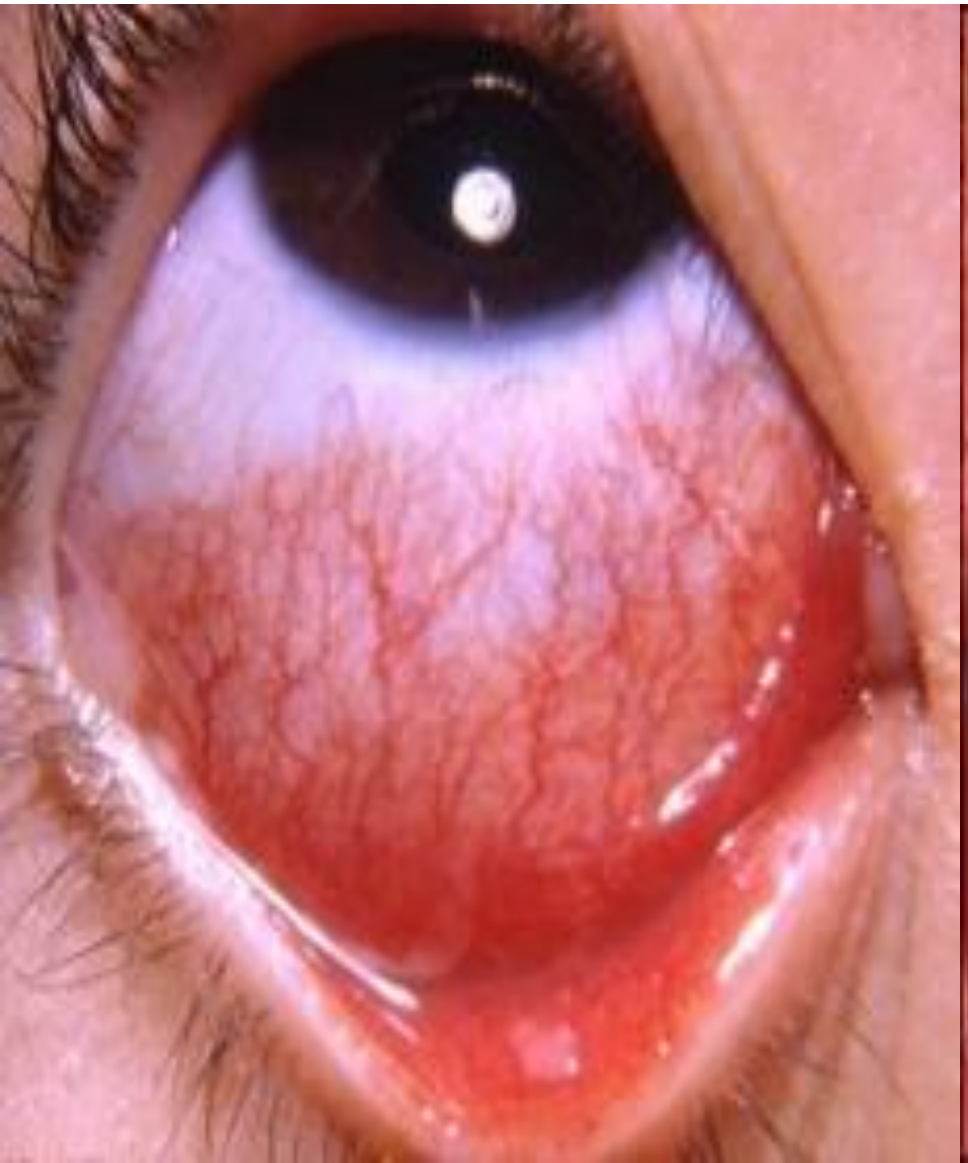
Loss of function:could you eat by inflammed gingiva?

EXAMPLES OF ACUTE INFLAMMATION
ACUTE GINGIVITIS



EXAMPLE OF ACUTE INFLAMMATION

ACUTE CONJUNCTIVITIS



ACUTE GINGIVITIS

ACUTE INFLAMMATION

ACUTE TONSILITIS

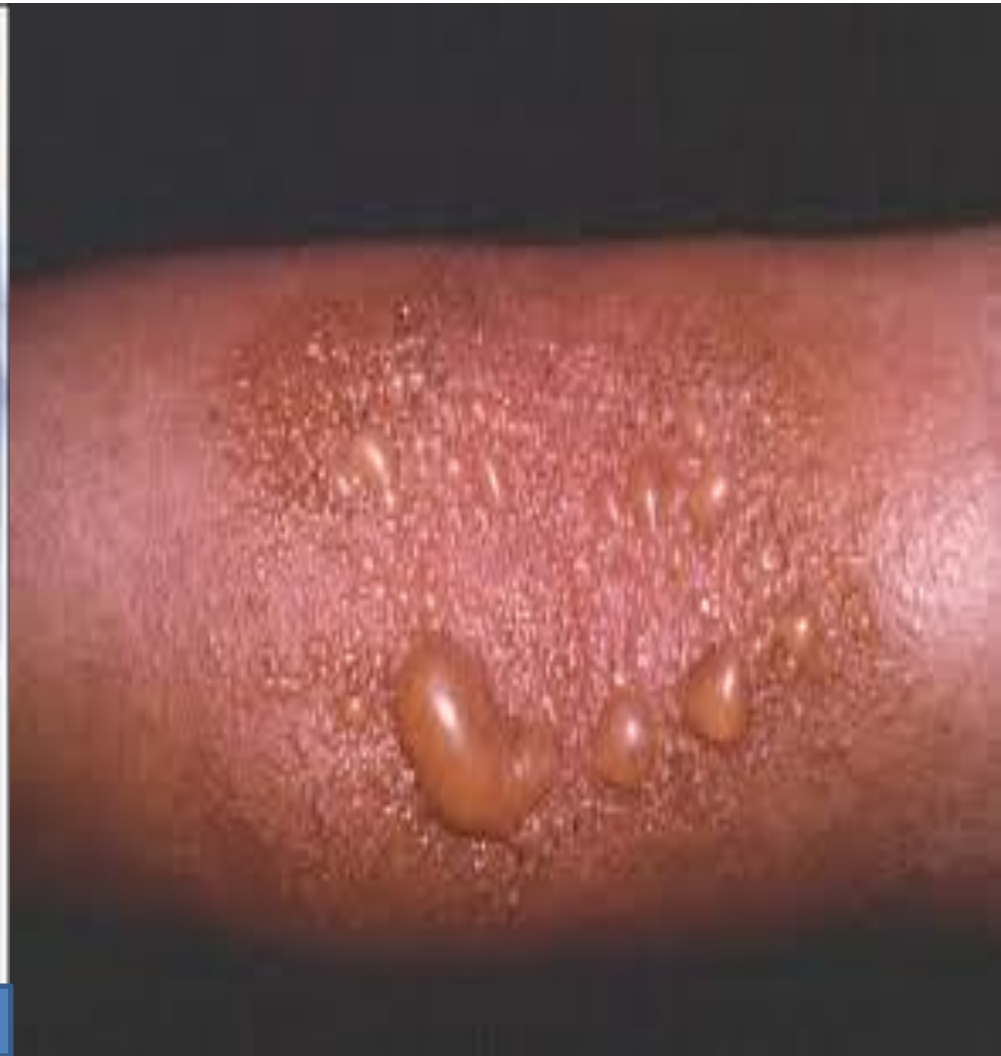
ACUTE APPENDICITIS

VARIETY OF ACUTE INFLAMMATION

CATARRHAL INFLAMMATION—ACUTE INFLAMMATION OF MUCOUS MEMBRANE LIKE RESPIRATORY,OR BODY CAVITY WITH EXCESSIVE MUCUS AND CELL EXUDATION

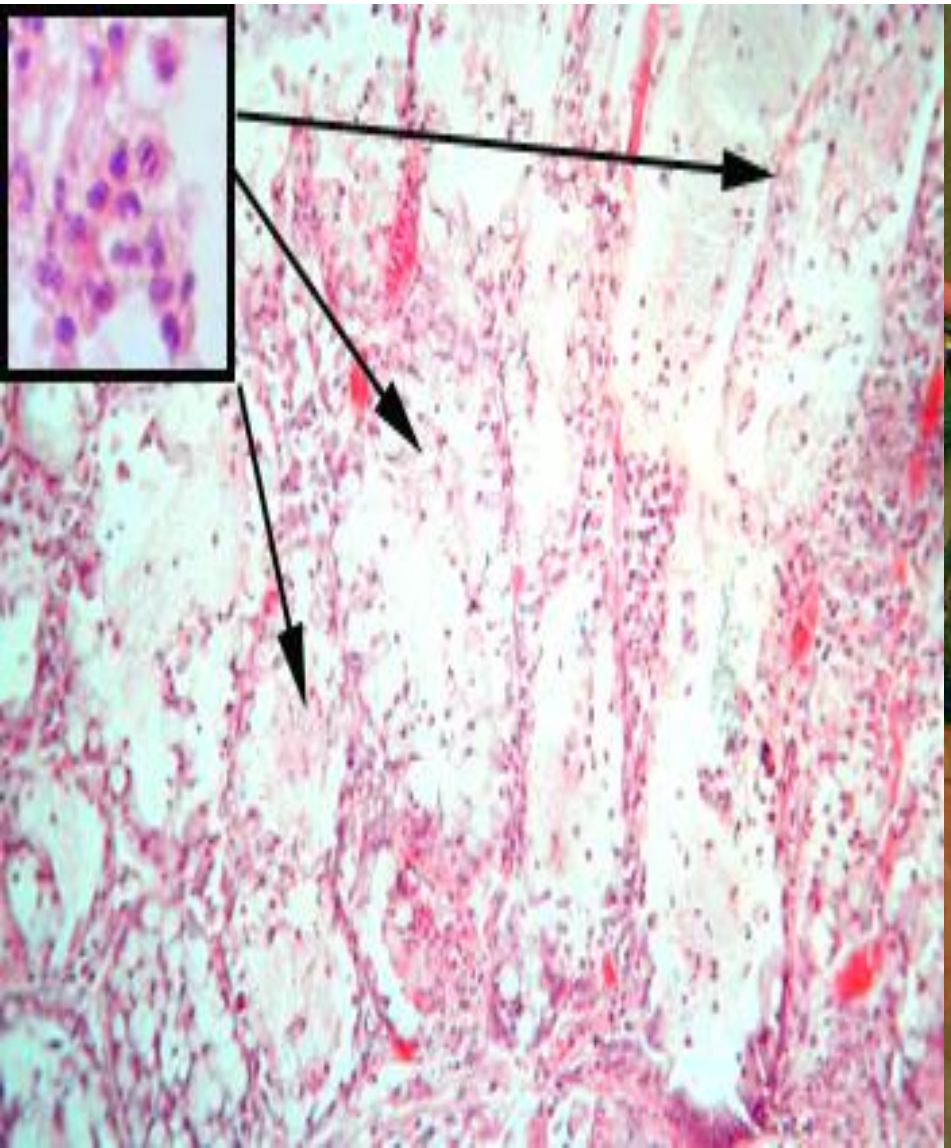
COMMON COLD

SEROUS INFLAMMATION—EXCESSIVE SEROUS NON-VISCID FLUID SECREATION

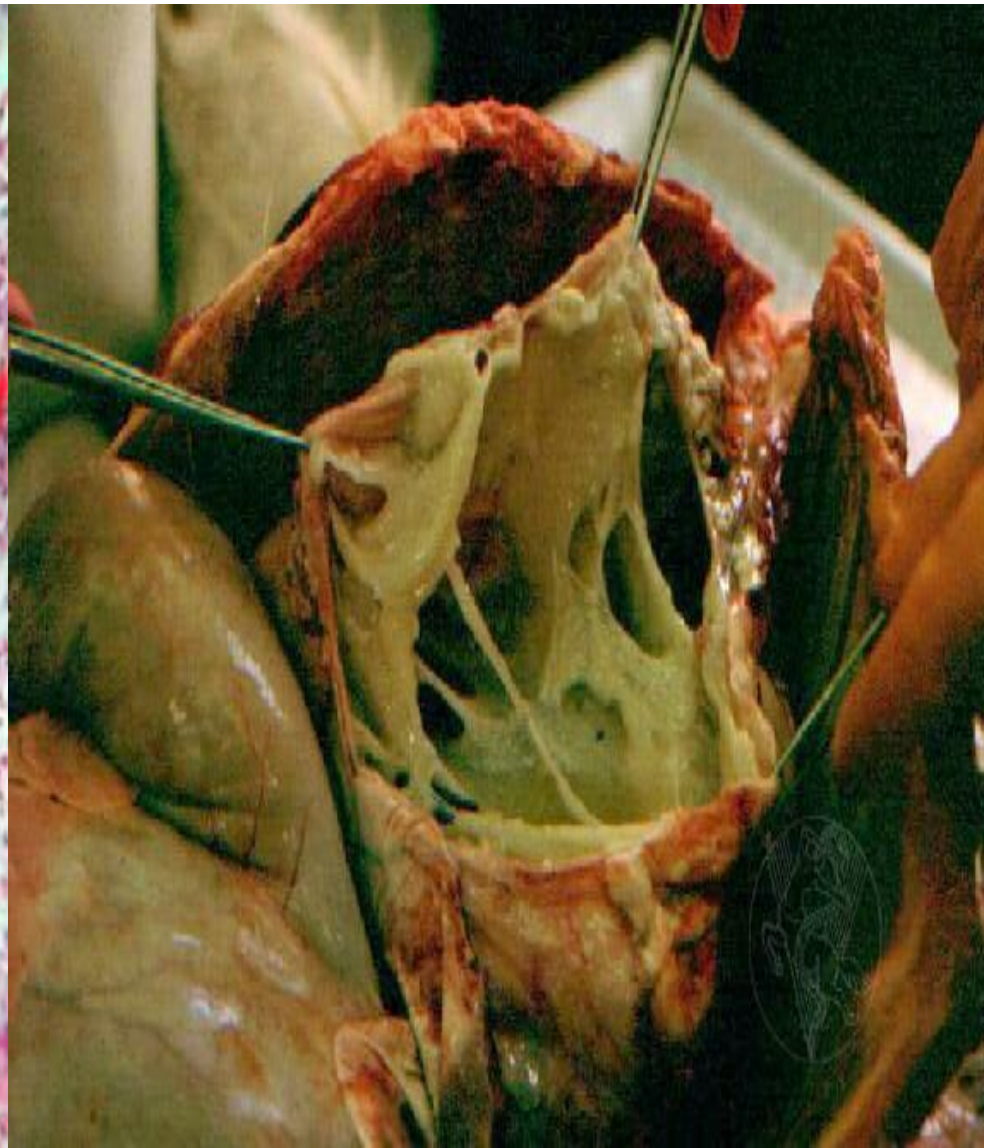


CONT,D

EXUDATIVE INFLAMMATION—TOO MUCH EXUDATE



FIBRINOUS INFLAMMATION—EXCESS OF FIBRIN-PERICARDITIS.



SUPPURATIVE INFLAMMATION

During acute inflammation as a result of tissue necrosis failure of complete resolution leading to pus formation.

Suppurative inflammation: it is inflammation associated with pus formation, could be acute or chronic.

Causes that lead to inflammation could cause suppuration.

Pus formation not only caused by microrganism, could be chemical cause like kerosene. Microrganism commonly is Staphylococci.

PUS

Fluid material which is composed of:

1-Dead & dying WBC.

2-Inflammatory exudate- oedema & fibrin.

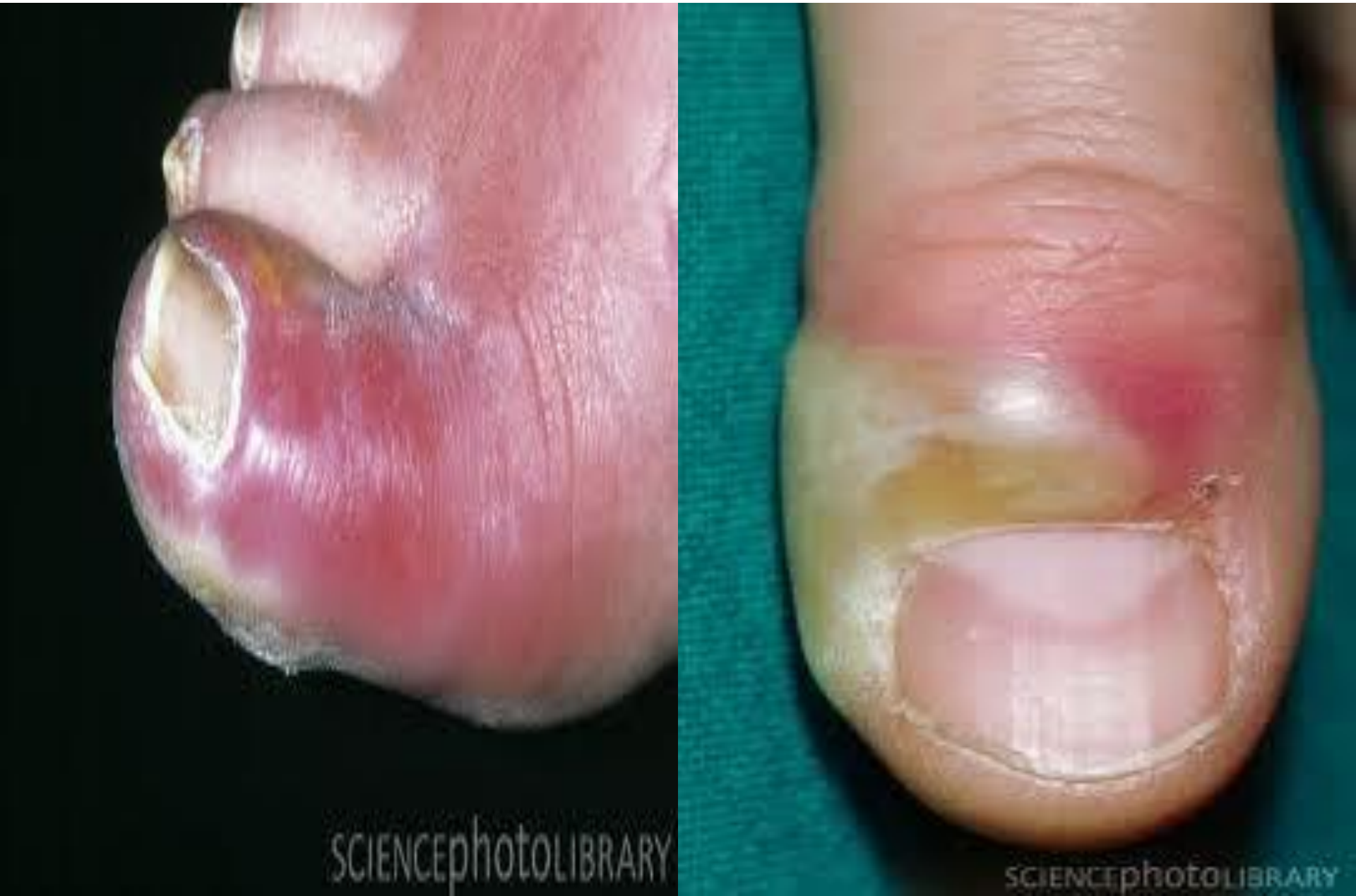
3-Tissue debris, nucleic acid & lipids.

4-Dead & dying bacteria if the cause is bacteria.

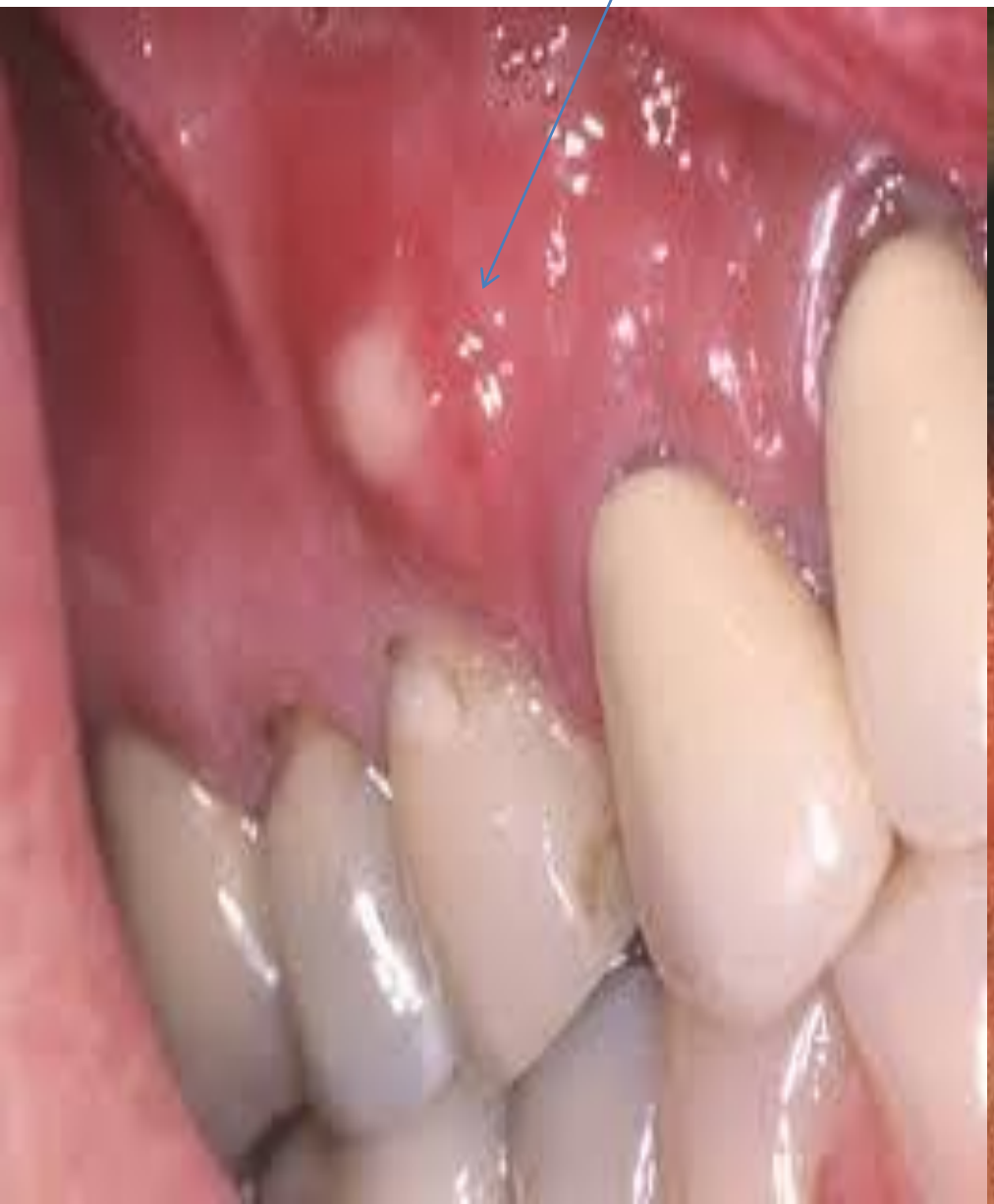
Pus cells: degranulated WBC .

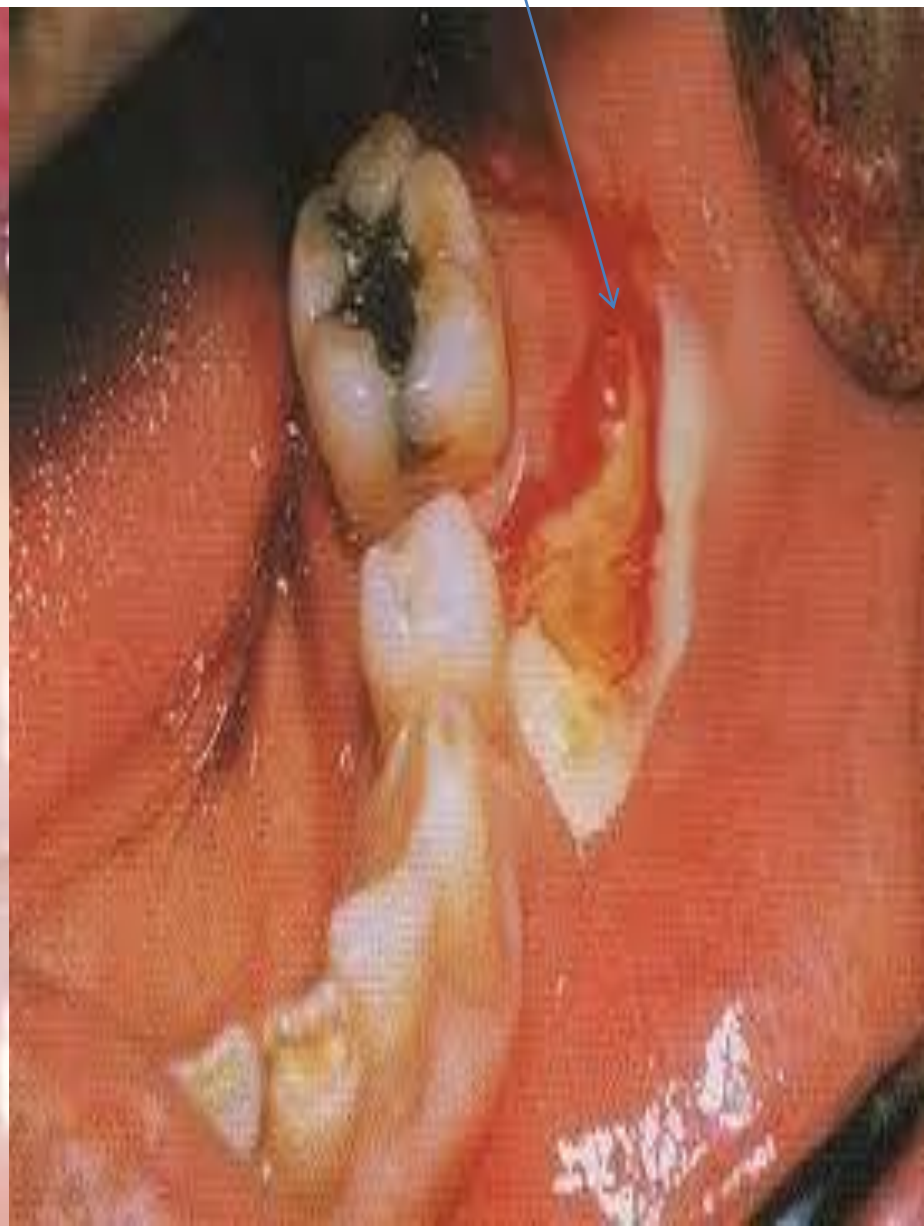
Abscess: pus in a cavity surrounded by inflamed tissue, and fibrinous material (pyogenic membrane) with heavy polymorphs infiltration. That is acute abscess cavity.

SUPPURATION



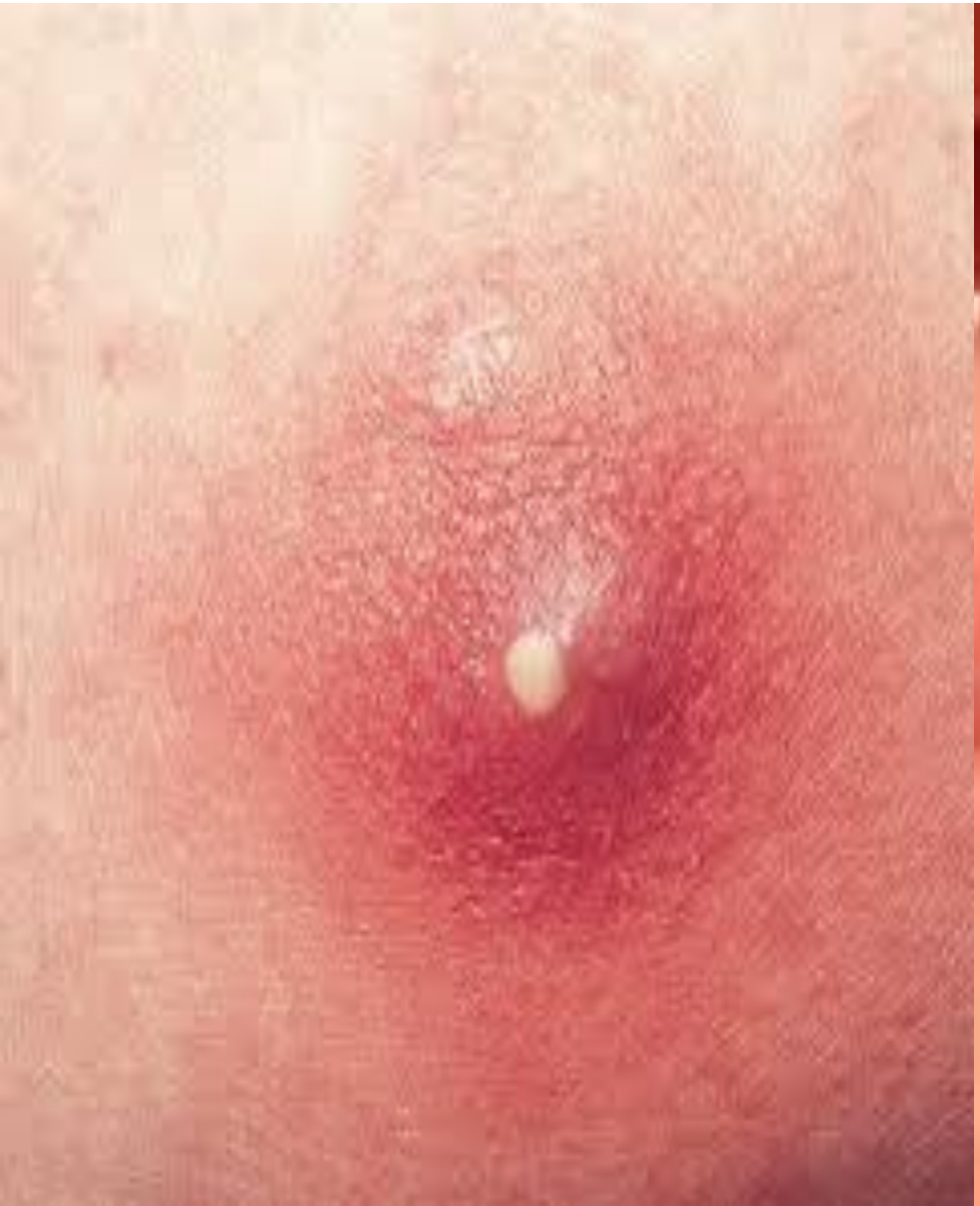
ACUTE APICAL ABSCEES& PERIODONTAL ABSCESS



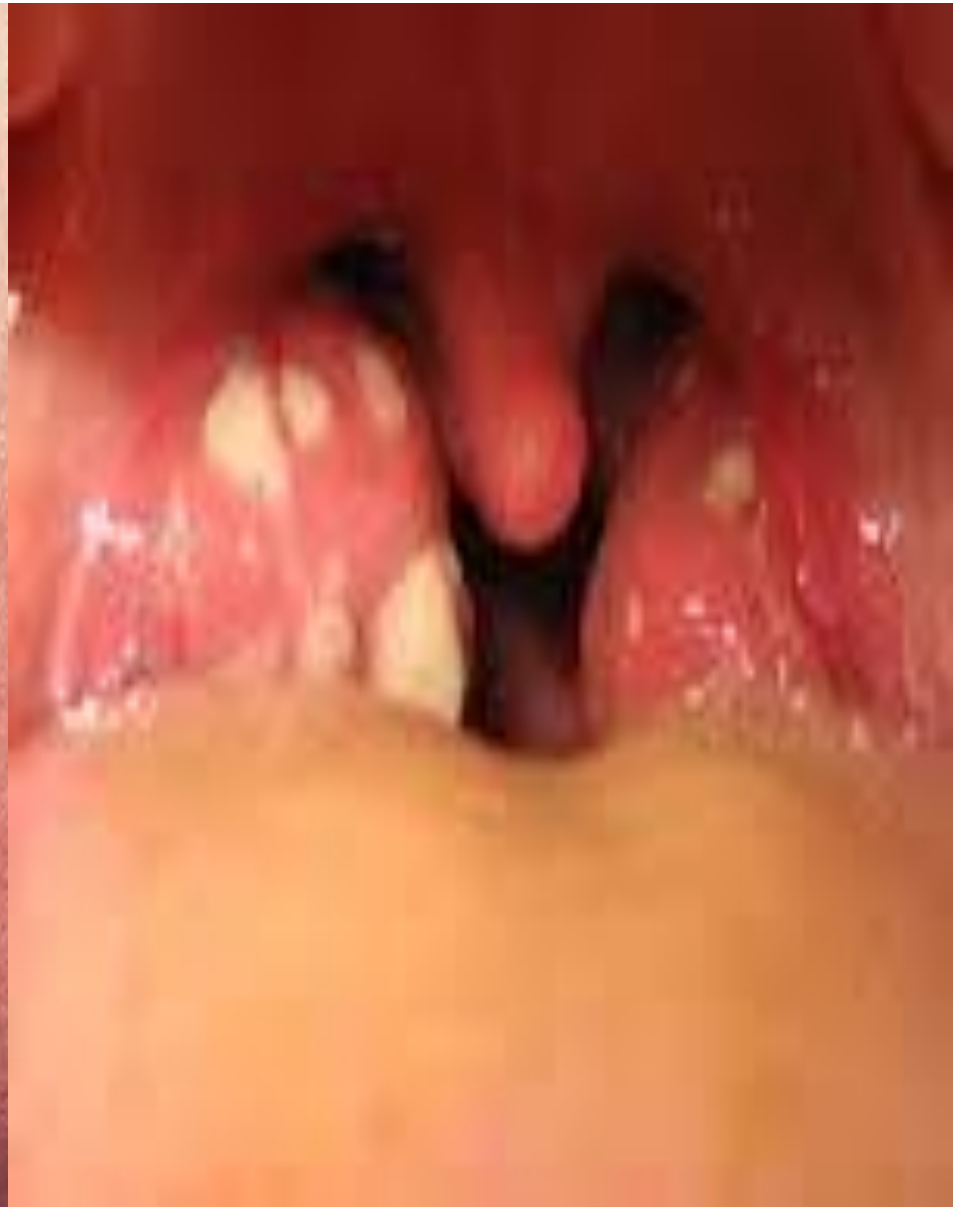


ACUTE SUPPURATIVE INFLAMMATION

ACUTE DERMATITIS---ABSCESS.



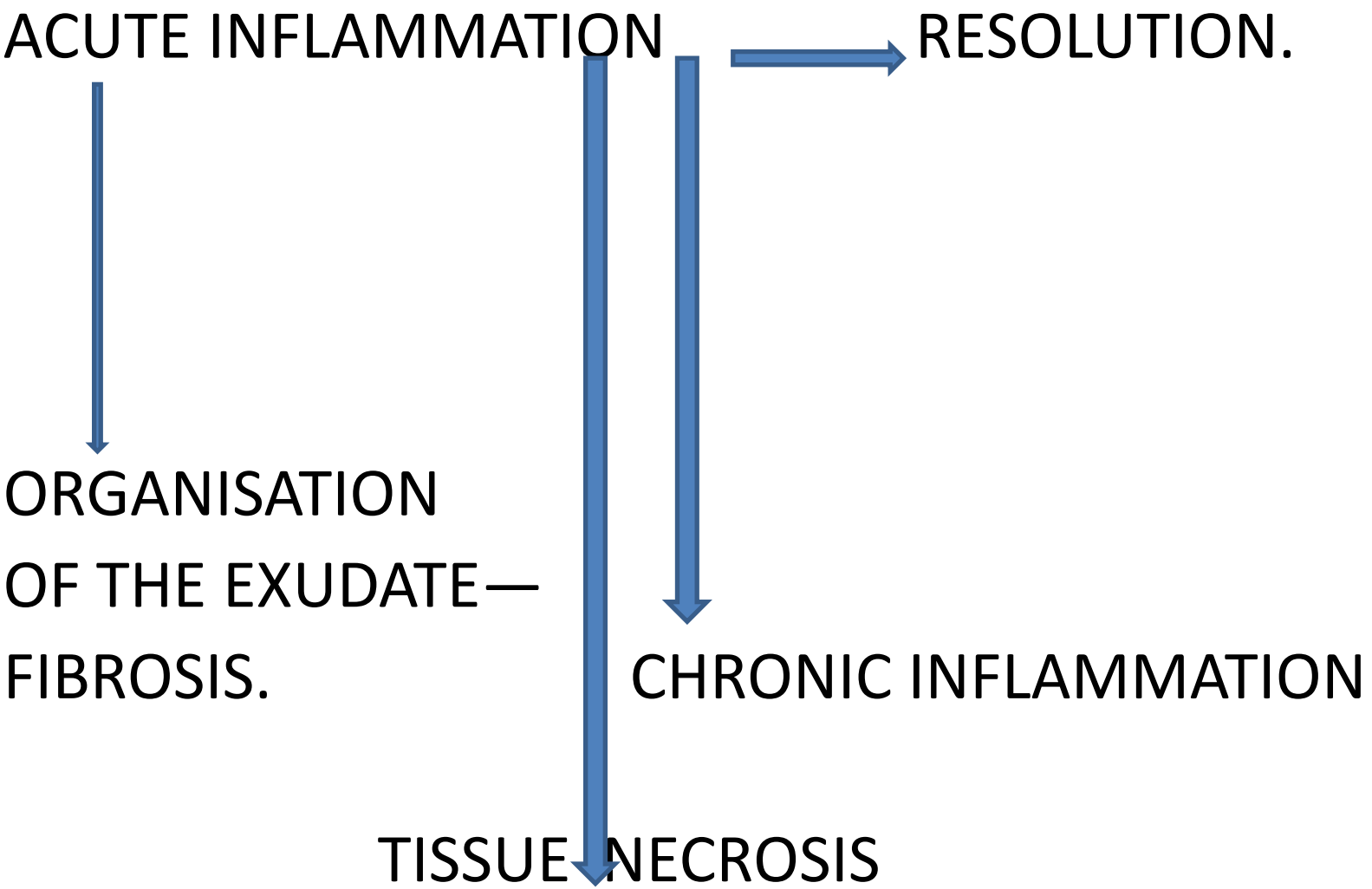
ACUTE TOSILLITIS &PUS FORMATION



CHRONIC ABSCESS

It is a cavity containing pus. The wall of the cavity composed of fibrous tissue infilterated by different types of cells, polymorphs, macrophage, lymphocytes and plasma cells.

FATE OF ACUTE INFLAMMATION



ORGANISATION

It is conversion of fibrin in a fibrinous inflammation in to granulation tissue which later becomes fibrous tissue.

Granulation tissue= capillary buds + fibroblast.

Fibroblast= connective tissue cells capable of producing collagens.

FATE OF INFLAMMATION