GENERAL PATHOLOGY
INFLAMMATION
LAB (3)

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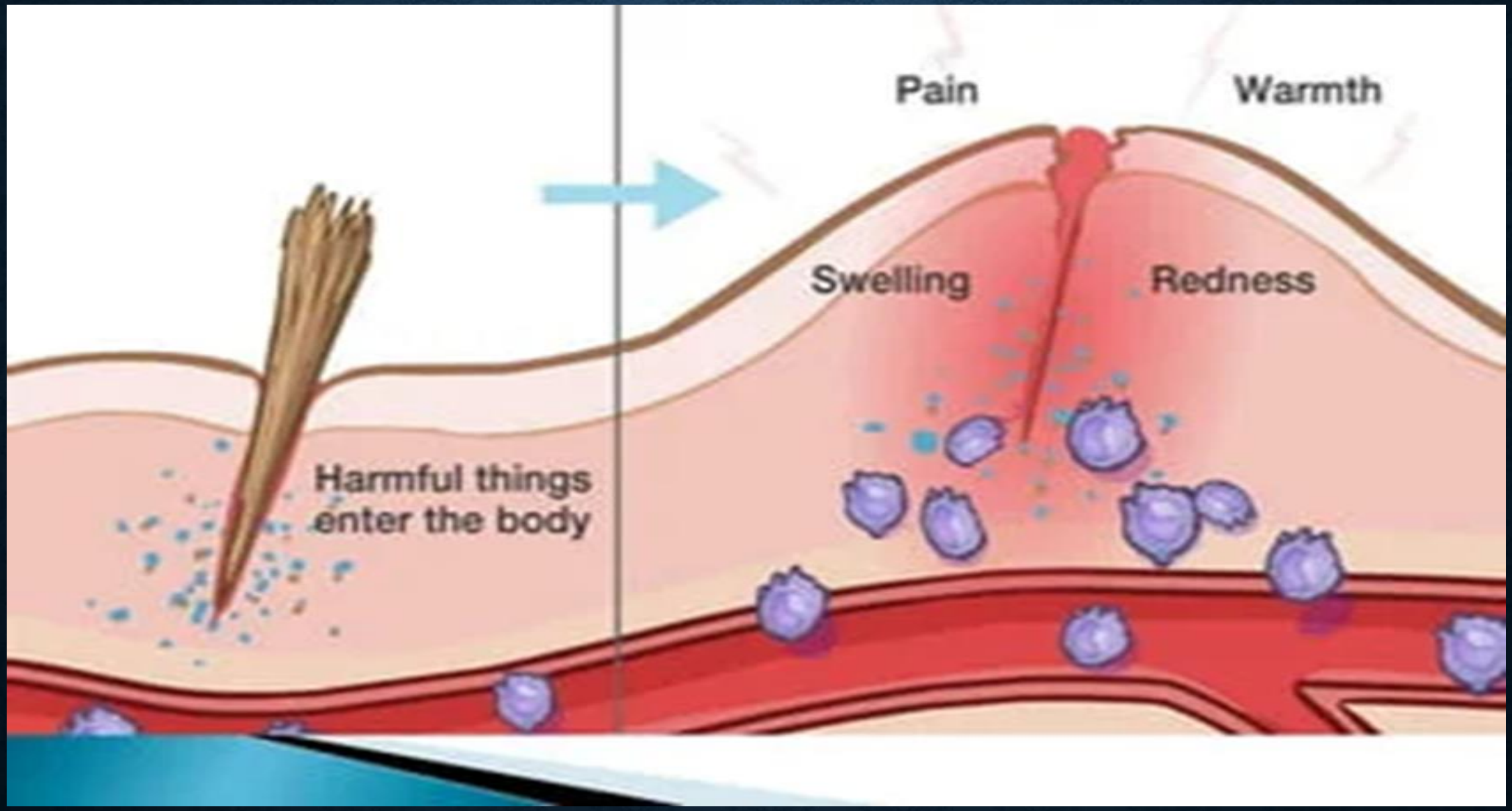
PHD. ORAL PATHOLOGY

Introduction

Defined as

"Local response of living tissues to injury due to any agent"

- Fundamentally protective response
- Ultimate goal is to get rid of intial cause cell injury(microbes, toxins)
- Consequence of such injury(necrosis, tissues)
- without inflammation wounds go unchecked, never heal


Pain
Warmth
Swelling
Redness
Harmful things
enter the body

WITHOUT INFLAMMATION??

- Infections would go unchecked.
- Wounds would never heal.
- Injured organs may remain permanently damaged.

CAUSES OF INFLAMMATION

LIVING

- Bacteria

- Viruses

- Mycoplasma

- Fungi

- Protozoa
- Parasites

Non living

Trauma - osteomyelitis

Heat – burns

Ionizing radiation

Uv light, infared

Chemicals , acids, alkalies

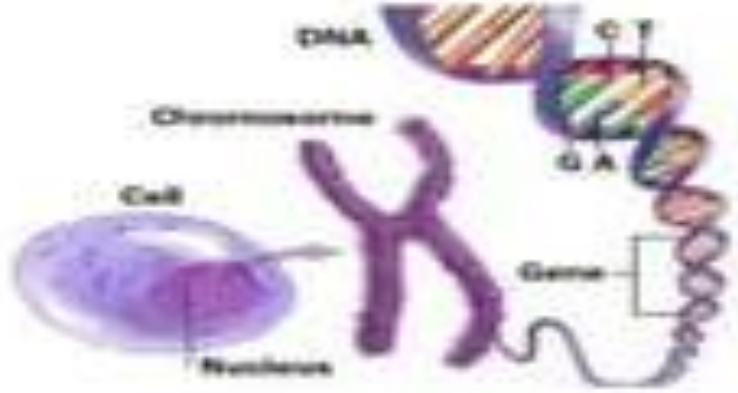
Foreign body

Idiopathic

GENETIC VARIATIONS

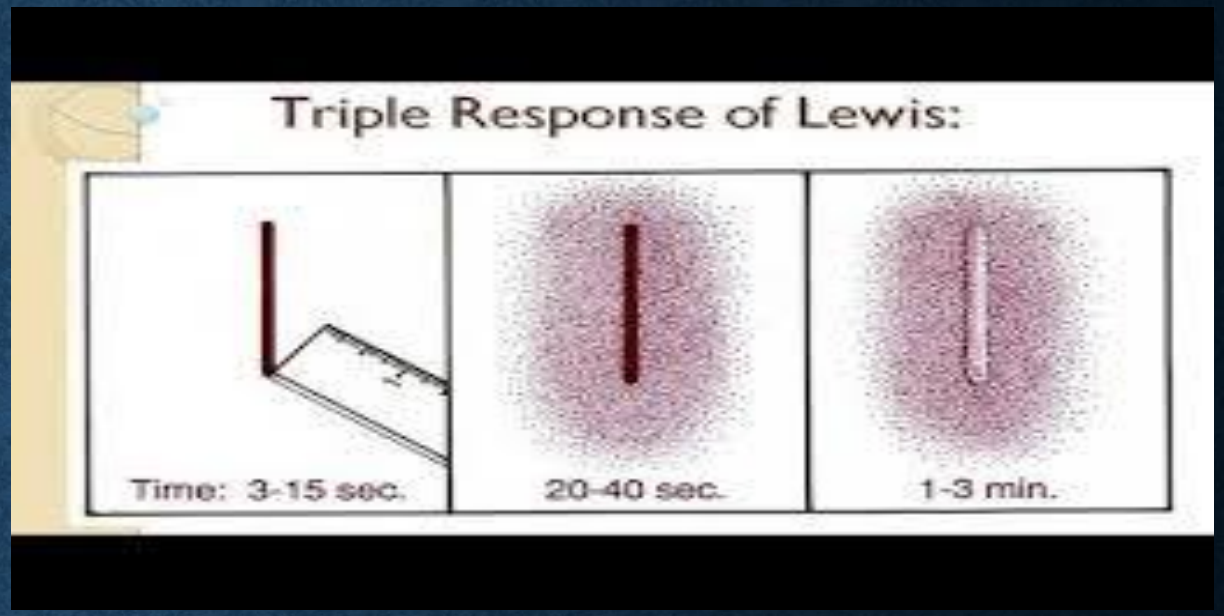
Variations in inherited genes can place them at risk/ predispose them to inflammatory Disease.

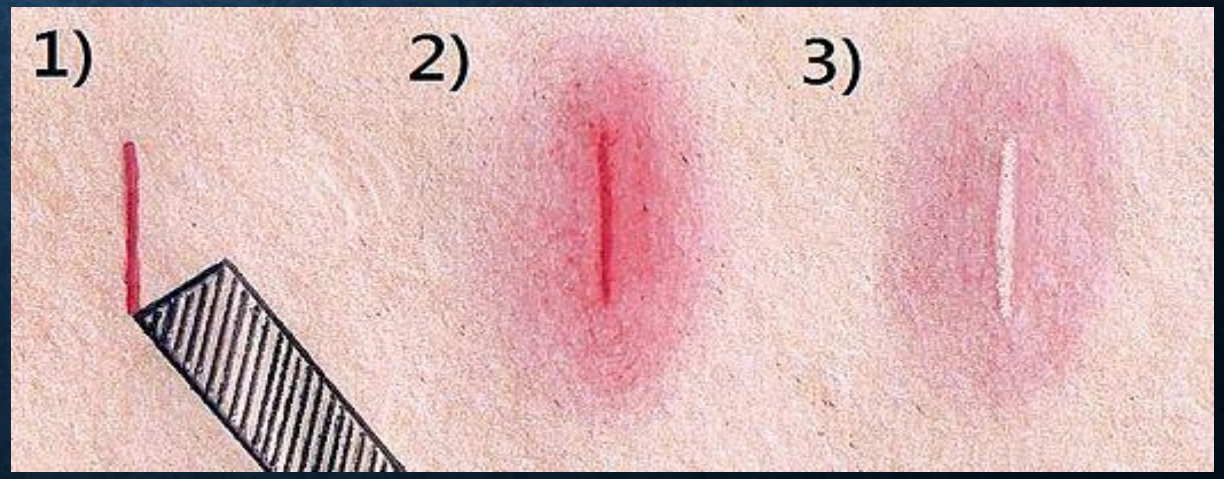
These usually don't cause disease by themselves Unless they are challenged by particular trigger

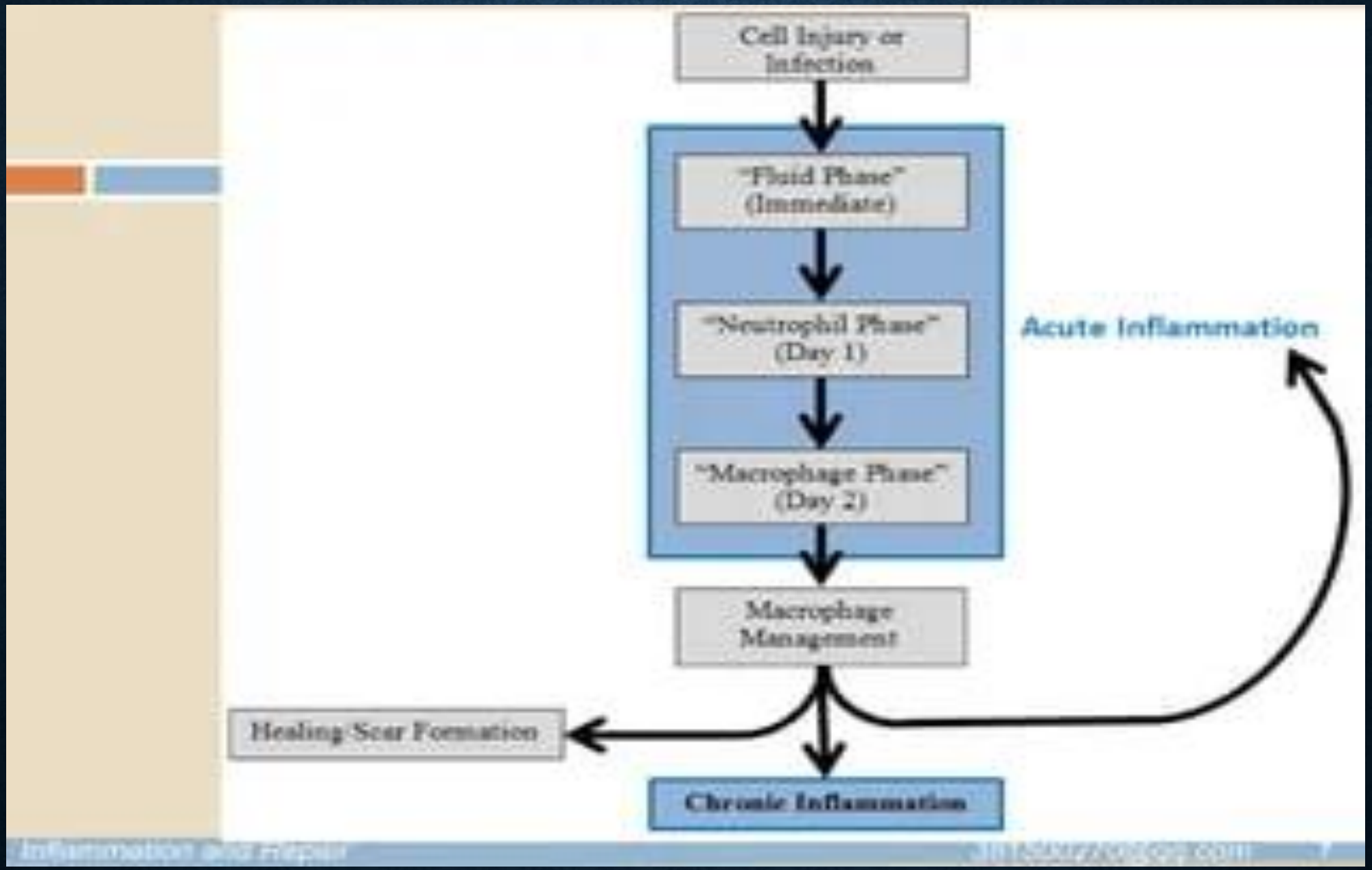


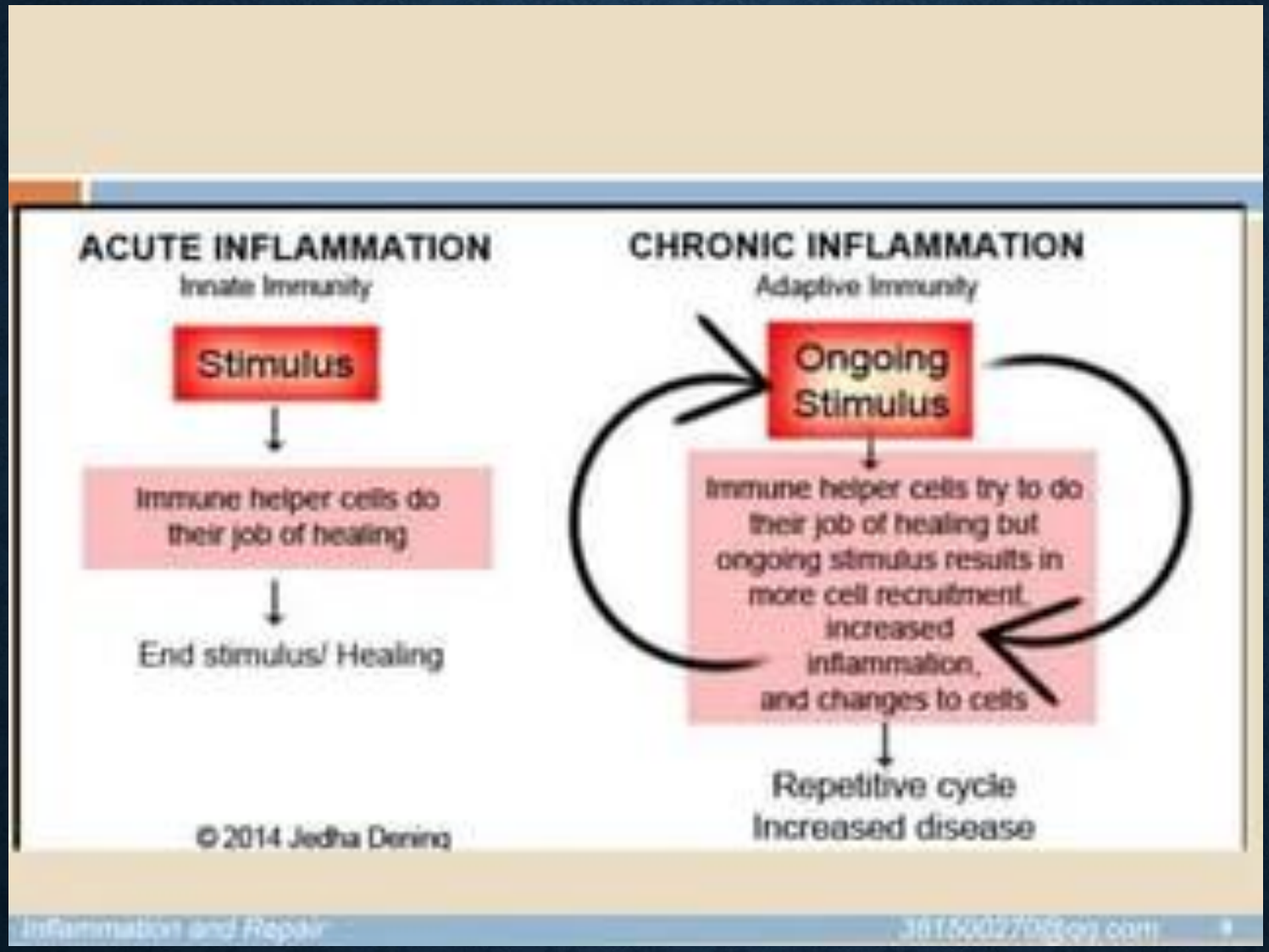
triple response of Lewis is a cutaneous response that occurs from firm stroking of the skin.

•Red spot, caused by capillary vasodilation.
•Flare, a redness in the surrounding area due to arteriolar dilation mediated by axon reflex.
•Wheal, caused by exudation of extracellular fluid from capillaries and venules.
The red spot emerges within 15 seconds.
Flare can take up to 45 seconds to begin.Wheal can take up to 3 minutes to begin






Cell Injury or Infection
"Fluid Phase" (Immediate)
"Neutrophil Phase" (Day 1)
"Macrophage Phase" (Day 2)
Acute Inflammation
Macrophage Management
Healing/Scar Formation
Chronic Inflammation


ACUTE INFLAMMATION
Innate Immunity
Stimulus
Immune helper cells do their job of healing
End stimulus/ Healing
© 2014 Jedha Dening
CHRONIC INFLAMMATION
Adaptive Immunity
Ongoing Stimulus
Immune helper cells try to do their job of healing but ongoing stimulus results in more cell recruitment, increased inflammation, and changes to cells
Repetitive cycle
Increased disease

Immunity

- Innate→
  - inborn cell immunity, cytokines, complements, activatiton of adaptive
- Adaptive →
  - acquired immune system specialized cell immunity
  - eliminate or prevent pathogen growth.
  - Active and Passive immunity
  - Two types
    - Humoral (antibody,complement pr, peptides)→blood tfx
    - and cell mediated(T cell immunity)

TYPES OF INFLAMMATION

ACUTE
"GOOD" INFLAMMATION

A serious threat triggers inflammation (a cut, bruise, infection, etc.)

The body releases inflammatory compounds

The job gets done & anti-inflammatory compounds are released. The body goes back to business as usual

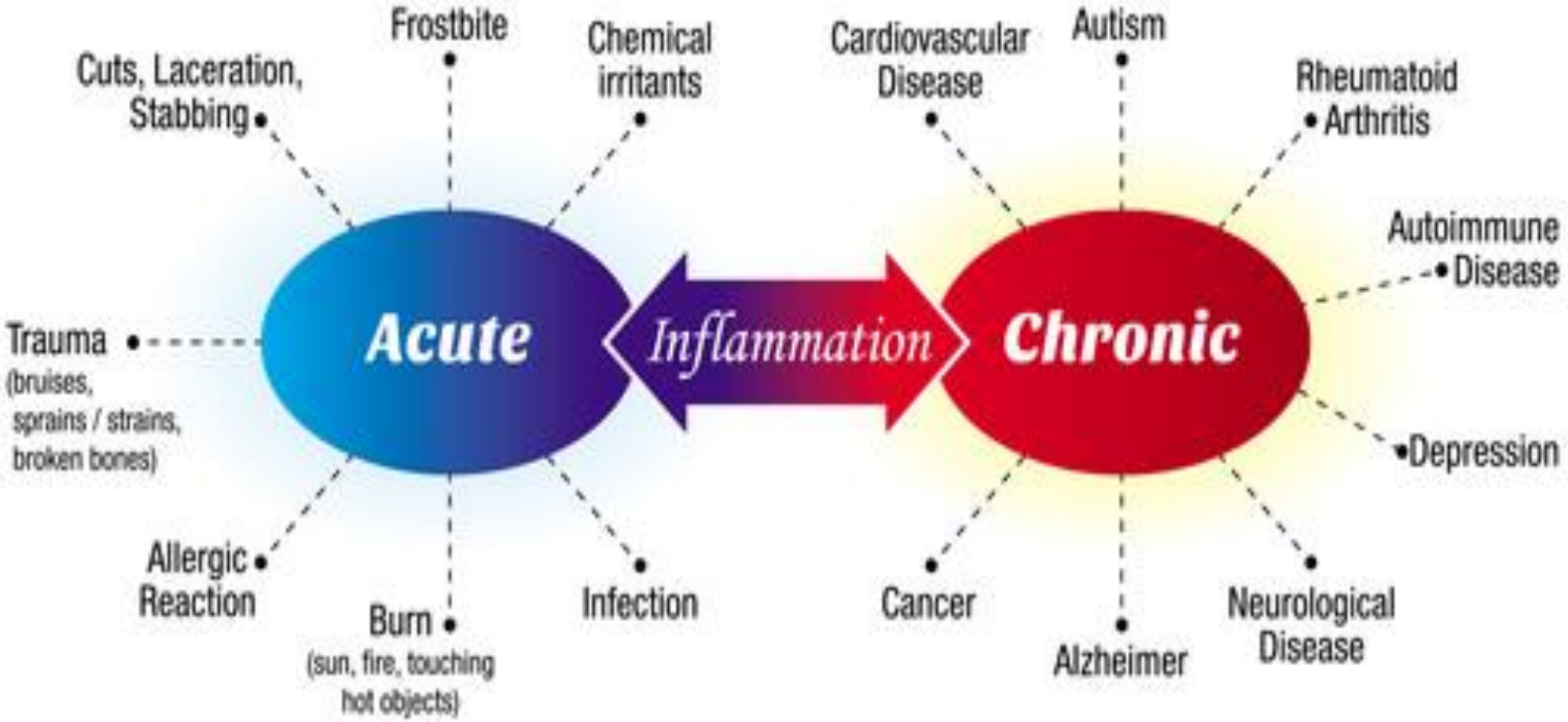
CHRONIC
"BAD" INFLAMMATION

A non-serious event triggers inflammation (eating a certain food, acne bacteria. etc.)

The body releases inflammatory compounds

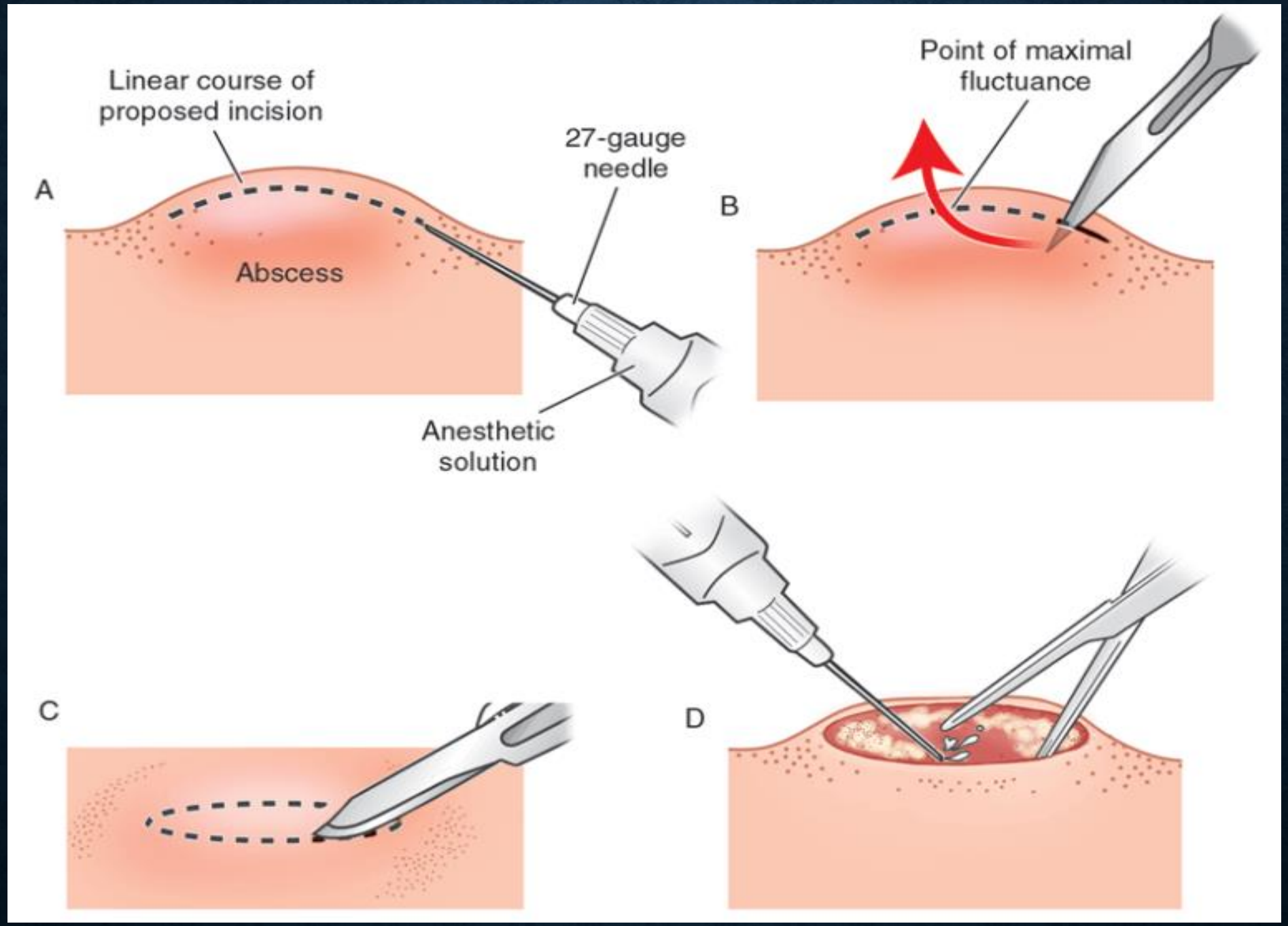
The body *doesn't* release anti-inflammatory compounds and keeps sending an inflammatory response

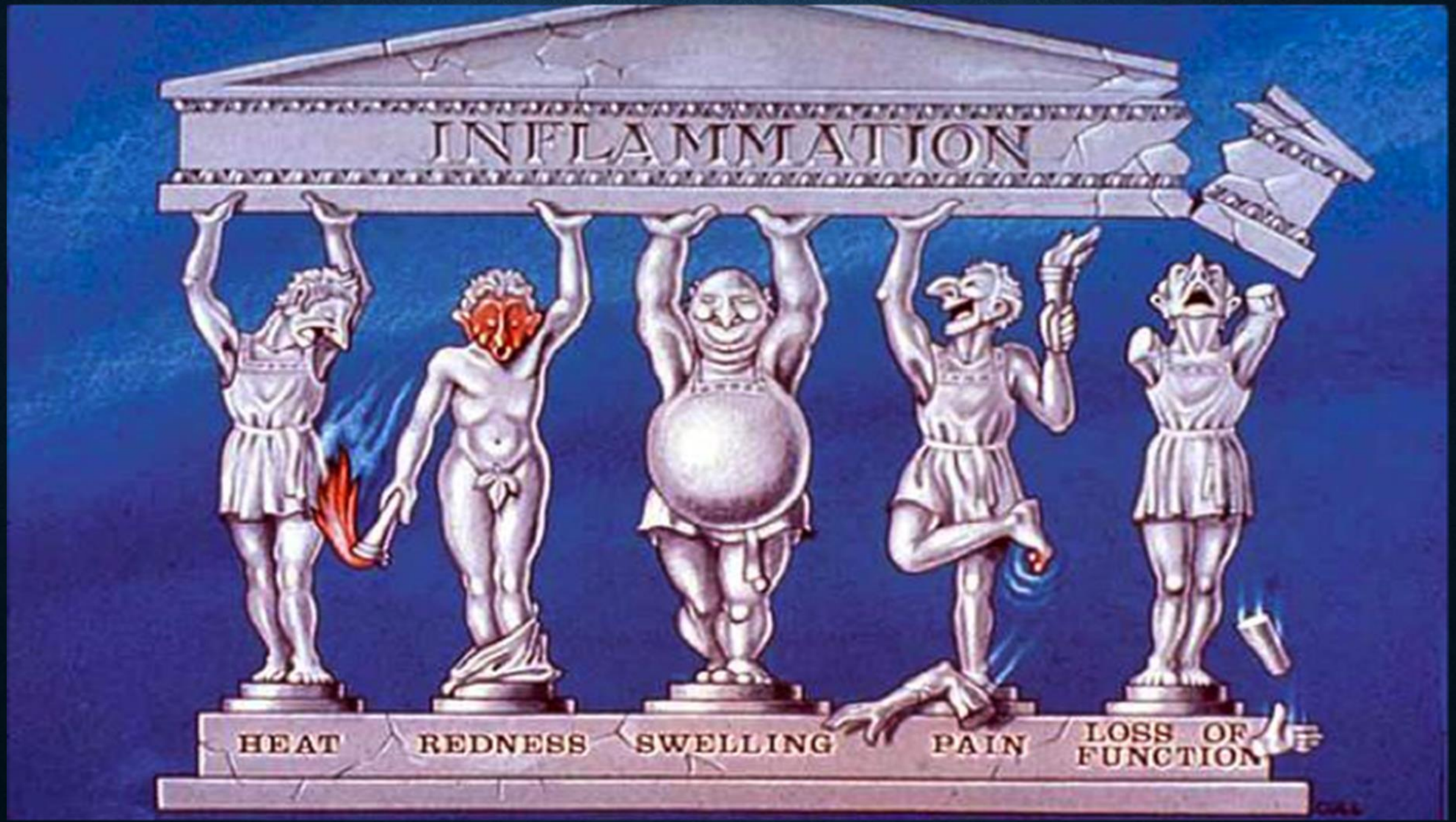
GoodGlow.co


Cuts, Laceration, Stabbing
Frostbite
Chemical irritants
Cardiovascular Disease
Autism
Rheumatoid Arthritis
Autoimmune Disease
Depression
Trauma (bruises, sprains / strains, broken bones)
Acute
Inflammation
Chronic
Allergic Reaction
Burn (sun, fire, touching hot objects)
Infection
Cancer
Alzheimer
Neurological Disease

ORO - ANTRAL FISTULA

- It is an abnormal epithelized communication between maxillary sinus and oral cavity through perforation in the sinus wall


A
Linear course of proposed incision
Abscess
27-gauge needle
Anesthetic solution
B
Point of maximal fluctuance
C
D


INFLAMMATION
HEAT
REDNESS
SWELLING
PAIN
LOSS OF FUNCTION

CARDINAL SIGNS OF ACUTE INFLAMMATION

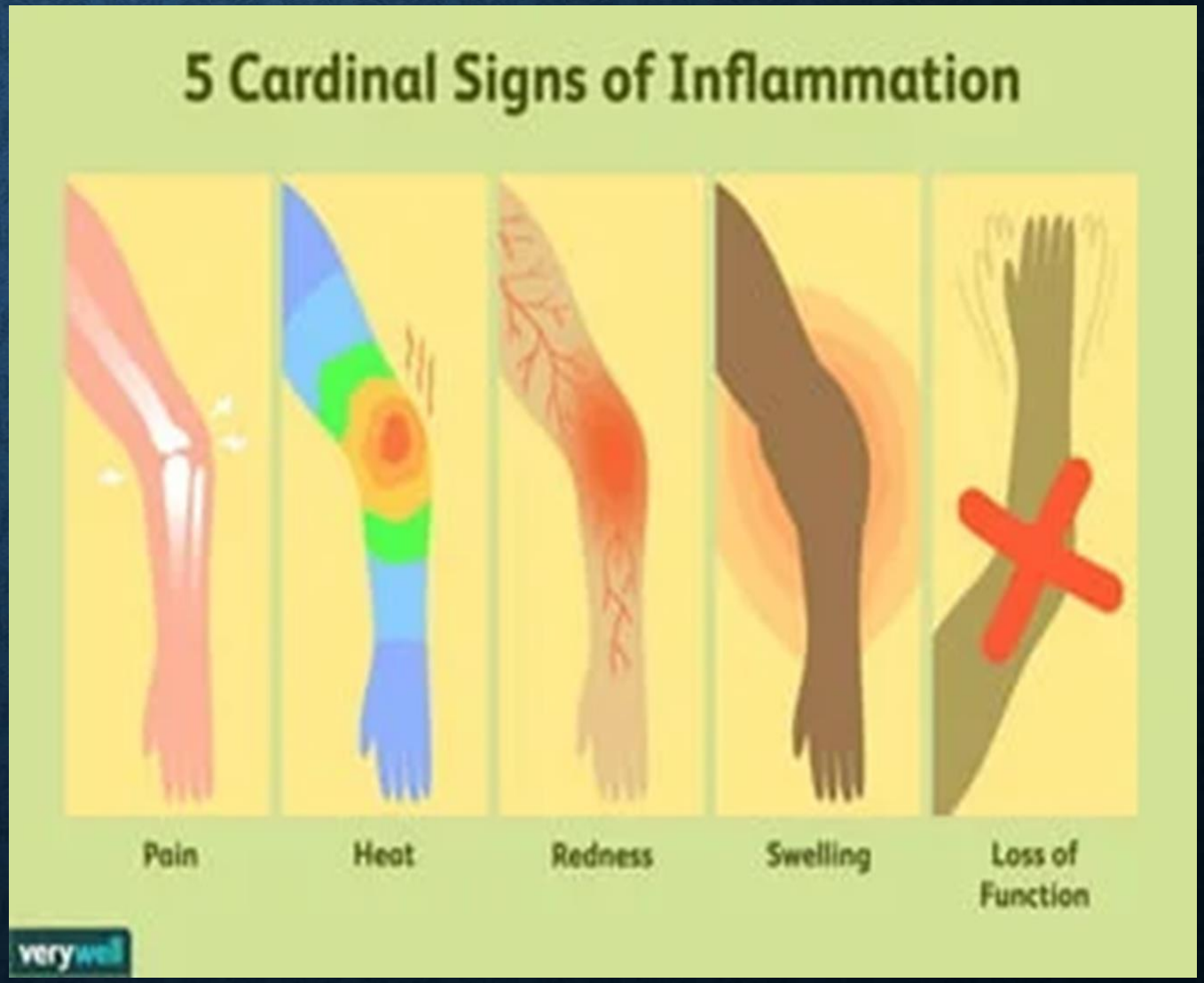
RUBOR (REDNESS)

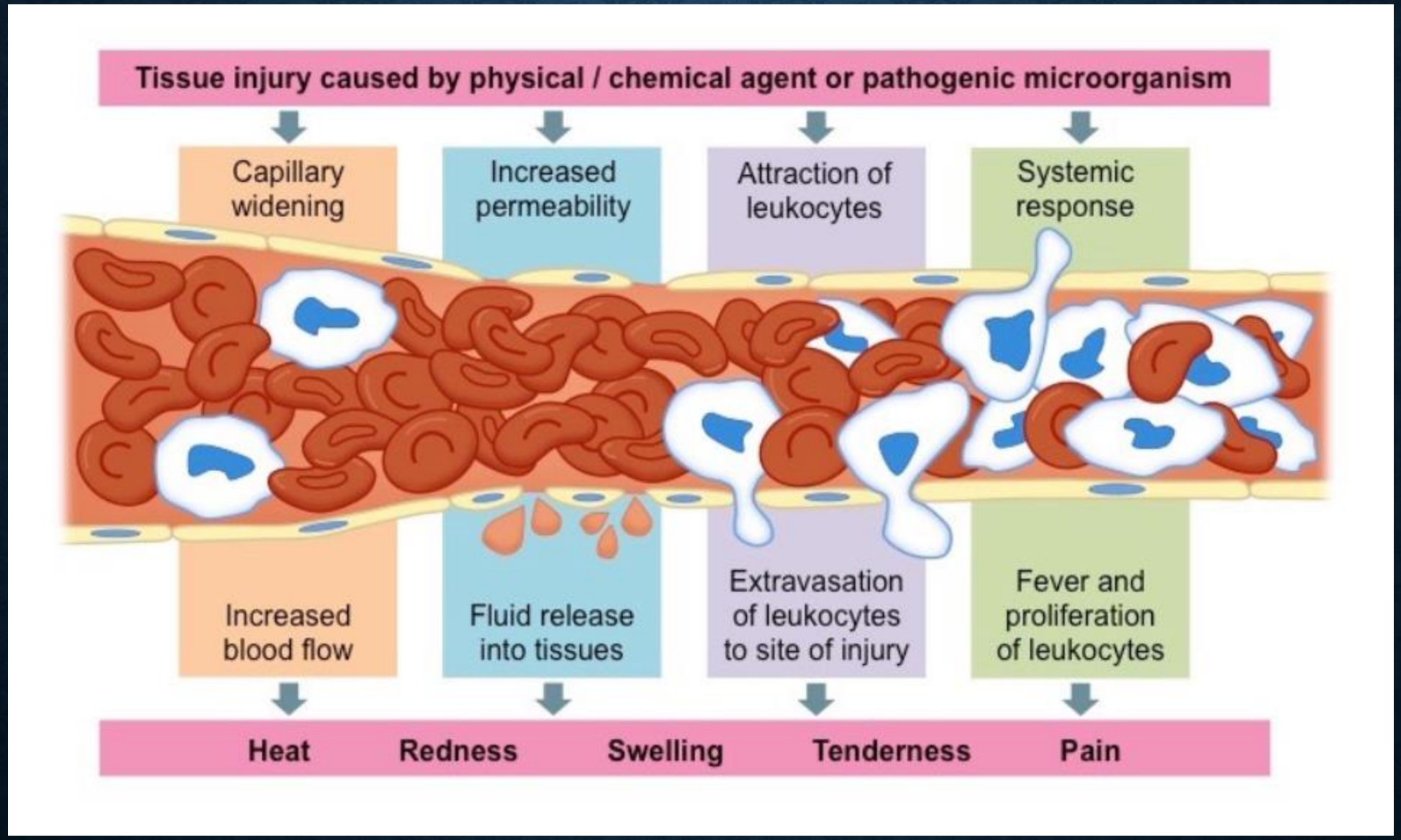
CALOR (HEAT)

DOLOR (PAIN)

TUMOR (SWELLING)

FUNTIO LAESA (LOSS OF FUNCTION)

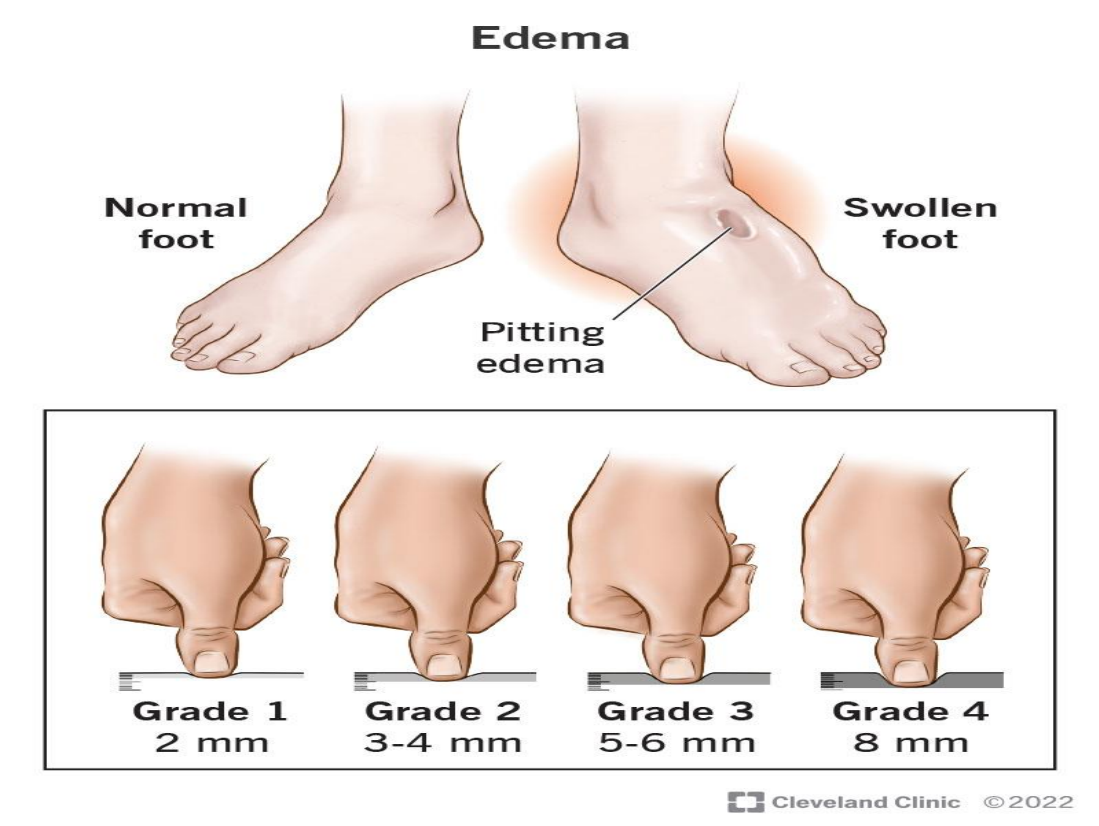


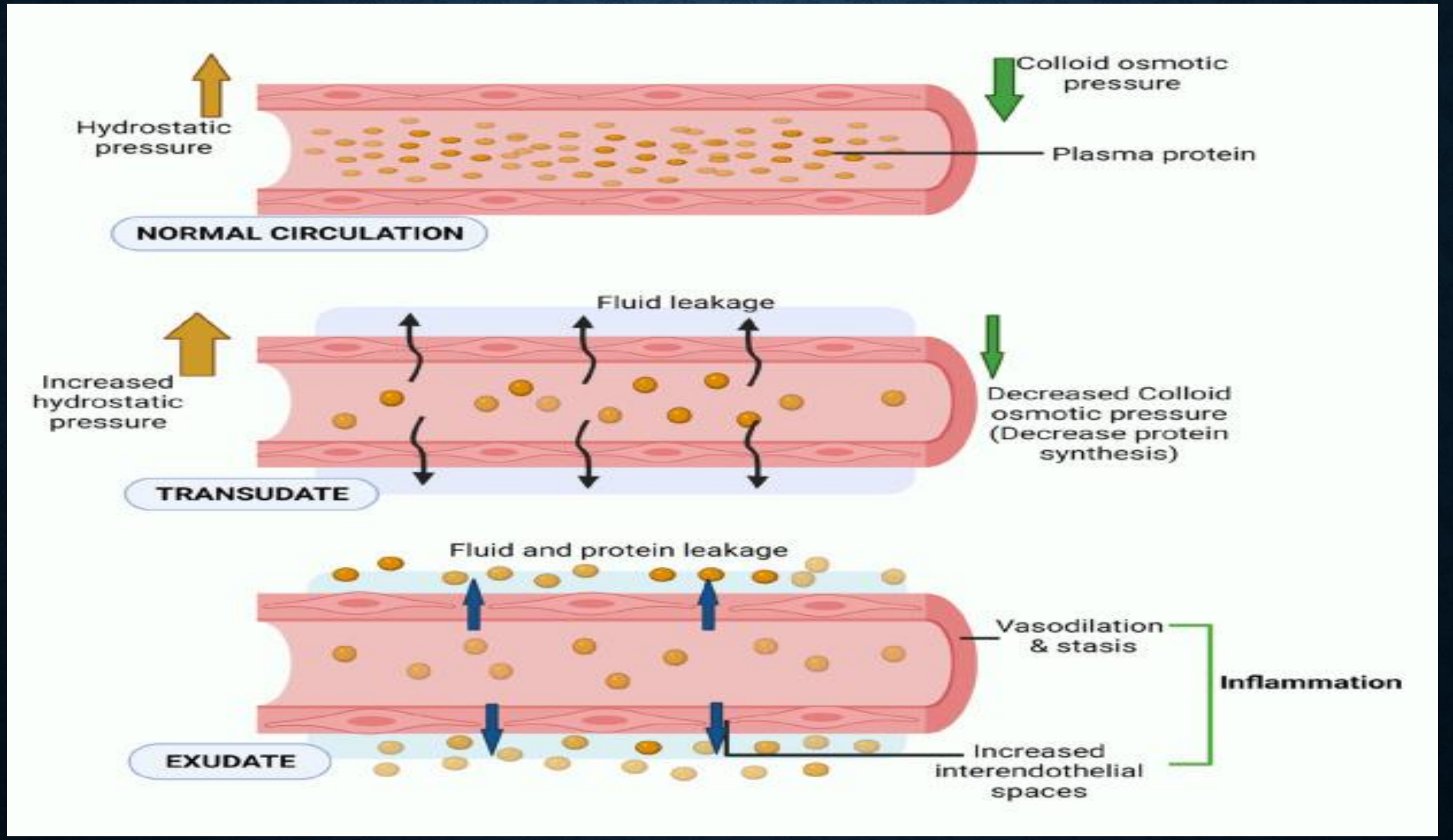

Tissue injury caused by physical / chemical agent or pathogenic microorganism
Capillary widening
Increased permeability
Attraction of leukocytes
Systemic response
Increased blood flow
Fluid release into tissues
Extravasation of leukocytes to site of injury
Fever and proliferation of leukocytes
Heat
Redness
Swelling
Tenderness
Pain

Edema (which is the cause of swelling) means an excess of fluid in the interstitial tissue or serous cavities; it can be either an exudate or a transudate.

Exudate is an extravascular fluid that has a high protein concentration and contains cellular debris. Its presence implies the existence of an inflammatory process that has, increased the permeability of small blood vessels. Pus, a purulent exudate, is an inflammatory exudate rich in leukocytes (mostly neutrophils), the debris of dead cells, and, in many cases, microbes.

Transudate is a fluid with low protein content (most of which is albumin), little or no cellular material, and low specific gravity. transudate is accumulating in non inflammatory conditions.




Hydrostatic pressure
Colloid osmotic pressure
Plasma protein
NORMAL CIRCULATION
Fluid leakage
Increased hydrostatic pressure
Decreased Colloid osmotic pressure (Decrease protein synthesis)
TRANSUDATE
Fluid and protein leakage
Vasodilation & stasis
Inflammation
Increased interendothelial spaces
EXUDATE

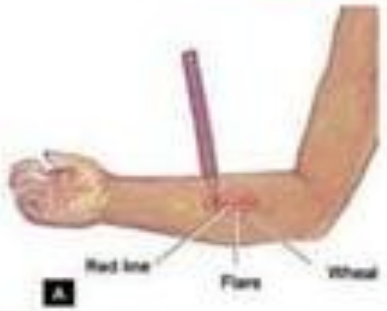
Rubor

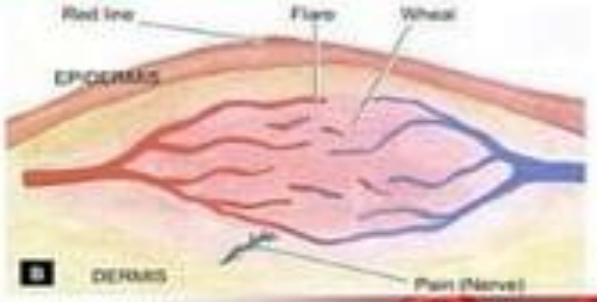
- *Redness is due to*

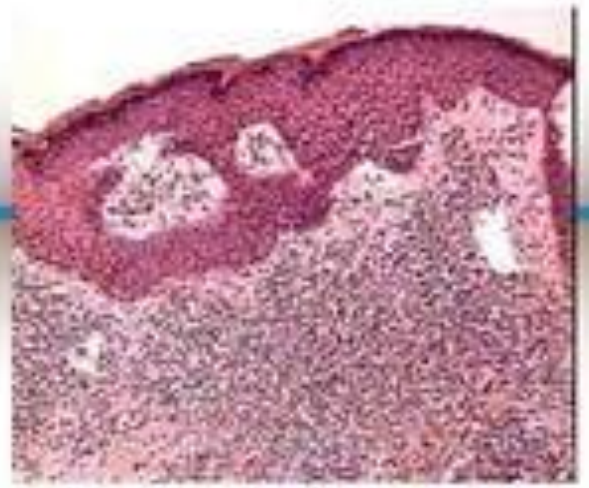
  VASODILATION (DUE TO RELEASE OF MEDIATORS)

  INCREASED BLOOD SUPPLY

TRIPLE RESPONSE OF LEWIS(1924)

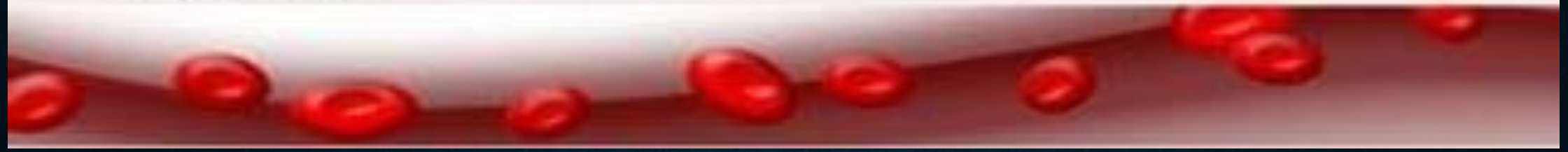






Internal View & External View Of Rubor.

RED LINE	APPEARS IN A FEW SECONDS	DUE TO VASODILATION OF CAPILLARIE AND VENULE
FLARE FLUSH AROUND RED LINE	APPEARS SLOWLY	DUE TO AXON REFLEX CAUSING DILATION OF ADJACENT ARTERIOLES
WHEAL SWELLING OEDEMA	APPEARS IN MINUTES	DUE TO TRANSUDATION OF FLUID INTO EXTRA VASCULAR SPACE



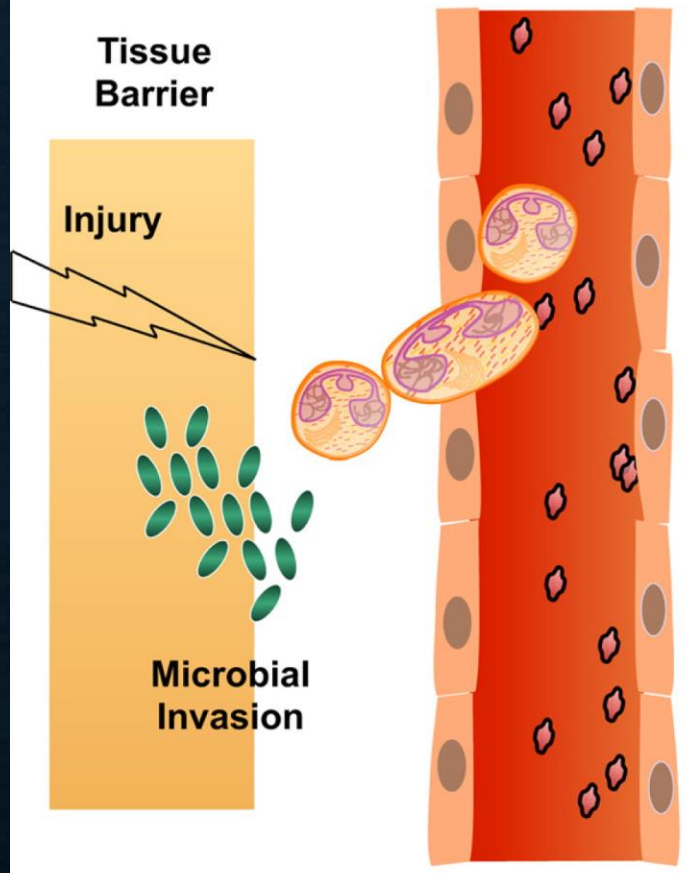
CALOR

Due to increase in blood supply

Due to increase in metabolic activity in that area

Appreciation of heat is possible in superficial areas with heat receptors... e.g : skin subcutaneous tissues

In deeper organs although associated with heat in the area , it is not associated with perception of increased heat



Cardinal signs of Inflammation:

Calor (heat)
Rubor (redness)
Tumor (swelling)
Dolor (pain)
Loss of function

Self-Limited

Resolution

Acute Inflammation

Abscess Formation

Wound Healing Scarring

Chronic Inflammation

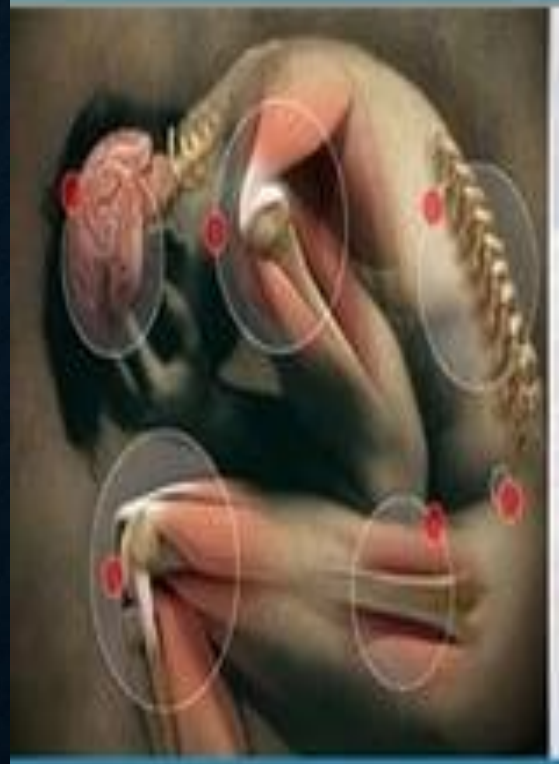
Rheumatoid Arthritis

Periodontal Disease

Diabetes
Cardiovascular diseases
Asthma

- Due to release of mediators which stimulate nerve endings
- Occurs only when appropriate sensory N. endings exists in inflamed area
- Type of pain depends on extent of stimulus rather than type of causative agent

DOLOR



Tumor

FUNCTIO LAESA

REFERRED PAIN:

- Due to spread of pain stimuli along the nerves to relevant spinal segment and relay of pain sensation to other areas served by same segmental distribution

- E.g Pain of myocardial ischemia referred to left shoulder

PAIN CAUSED BY INFLAMMATION OF	CHARACTER
SKIN	BURNING , ITCHING
DENSE TISSUES LIKE PERITONIUM BONE , ENCAPSULATED ORGANS	DULL , BORING , ACHING
PROGRESSED TO SUPPURATION	THROBBING
PUS REACHES TO SURFACE	SHARP , DARTING ,LANCINATING
NERVE TRUNK	BORING , TINGLING

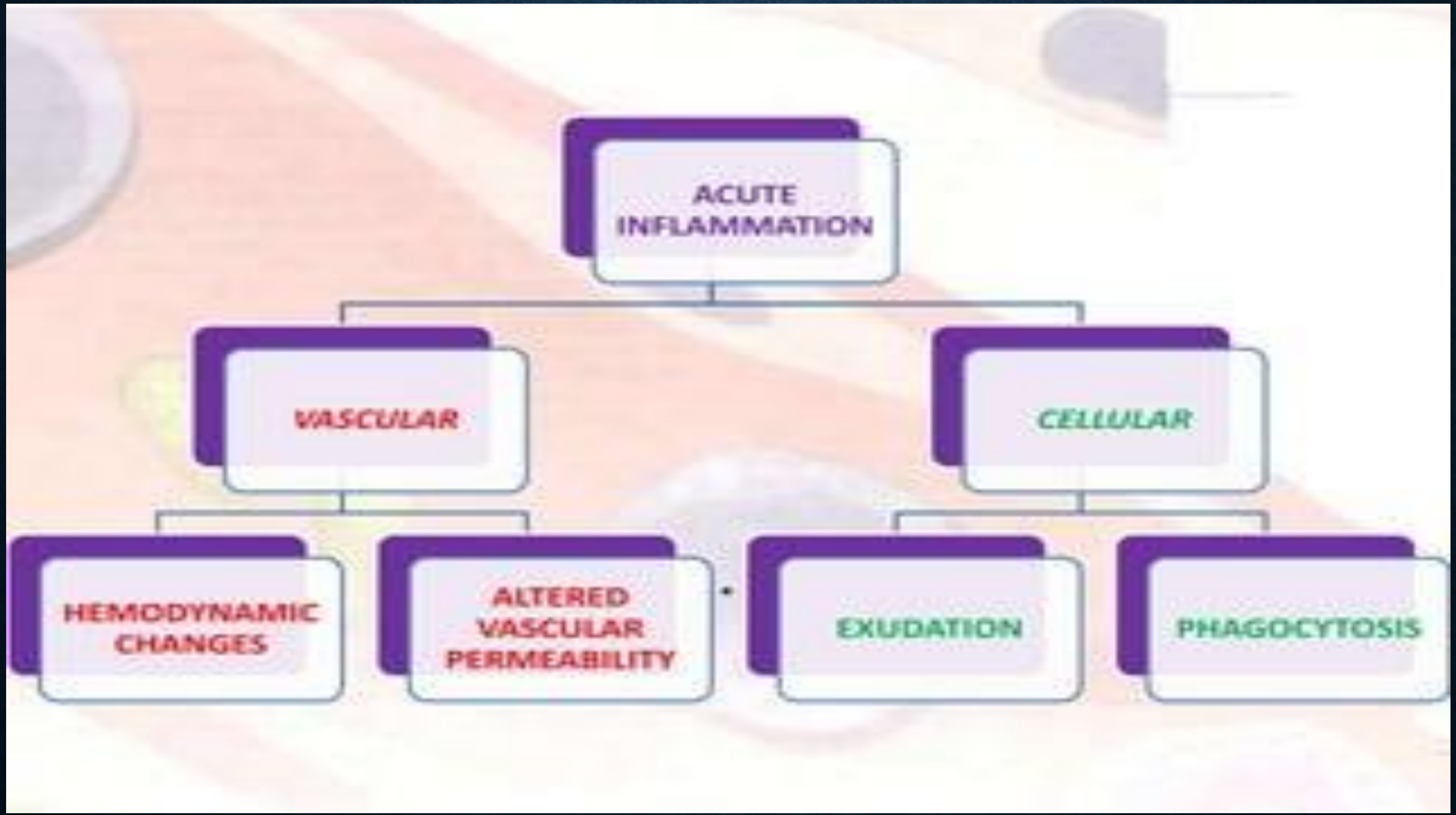
TUMOR/SWELLING

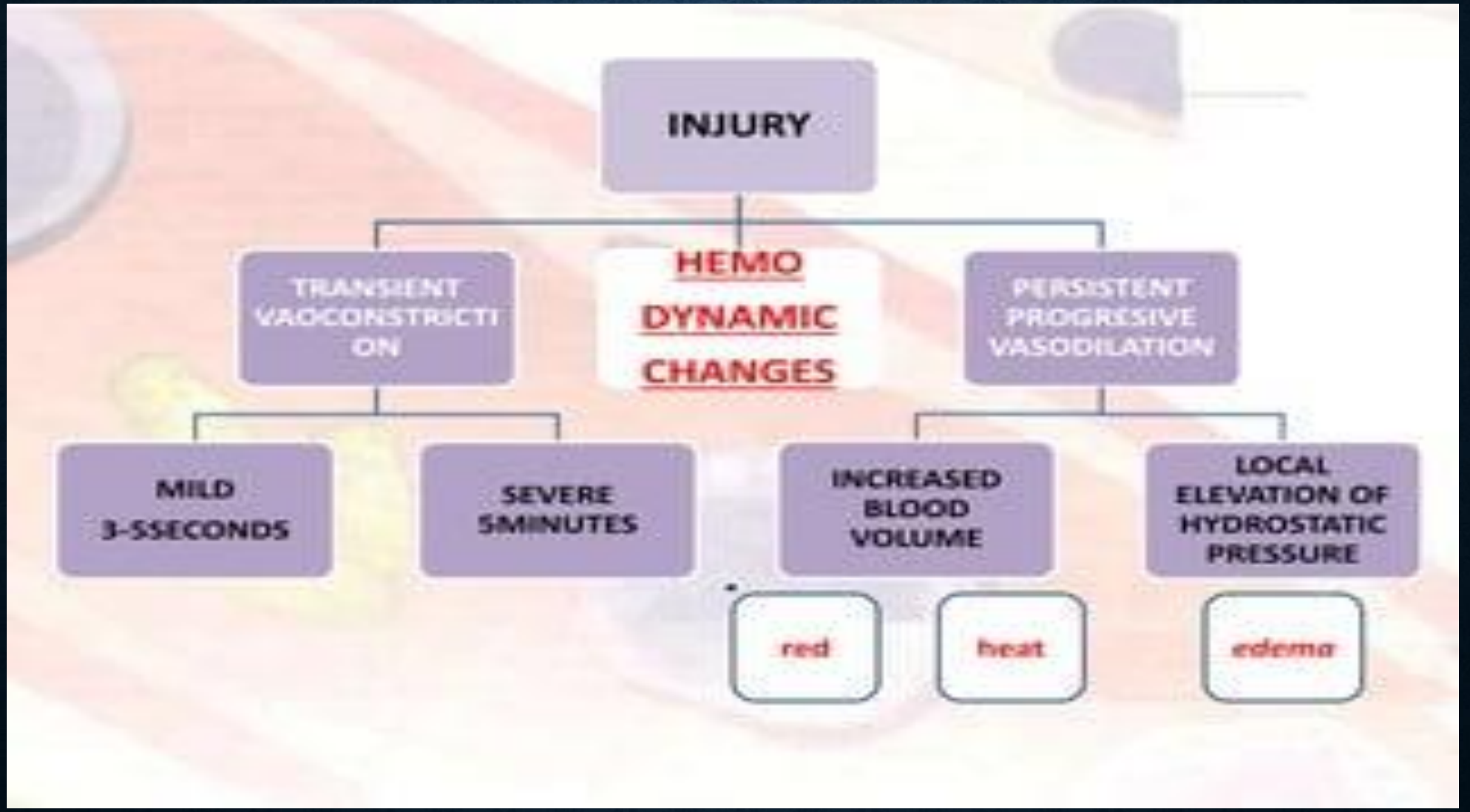
Due to increased vascularity

Accumulation of fluid and cells in damaged part

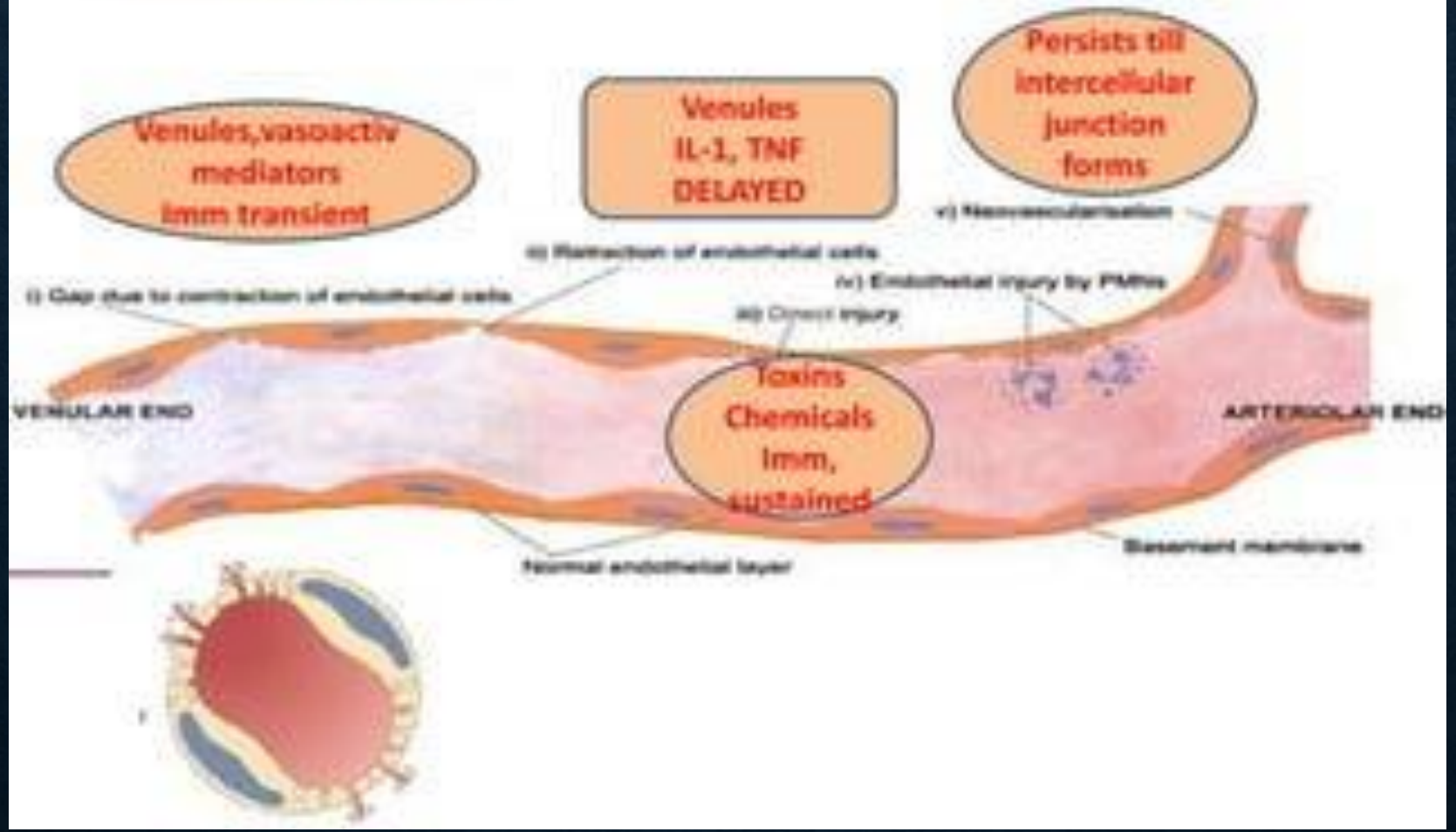
EDEMA- Excess fluid in interstitial / serous cavities it may be exudate / transudate in nature

PUS: Purulent exudate rich in leukocytes(mostly neutrophils), debris of dead cells and in many cases microbes


ACUTE INFLAMMATION
VASCULAR
CELLULAR
HEMODYNAMIC CHANGES
ALTERED VASCULAR PERMEABILITY
EXUDATION
PHAGOCYTOSIS


INJURY
HEMO DYNAMIC CHANGES
TRANSIENT VASOCONSTRICTION
PERSISTENT PROGRESSIVE VASODILATION
MILD 3-5SECONDS
SEVERE 5MINUTES
INCREASED BLOOD VOLUME
LOCAL ELEVATION OF HYDROSTATIC PRESSURE
red
heat
edema

Five mechanisms of increased vascular permeability

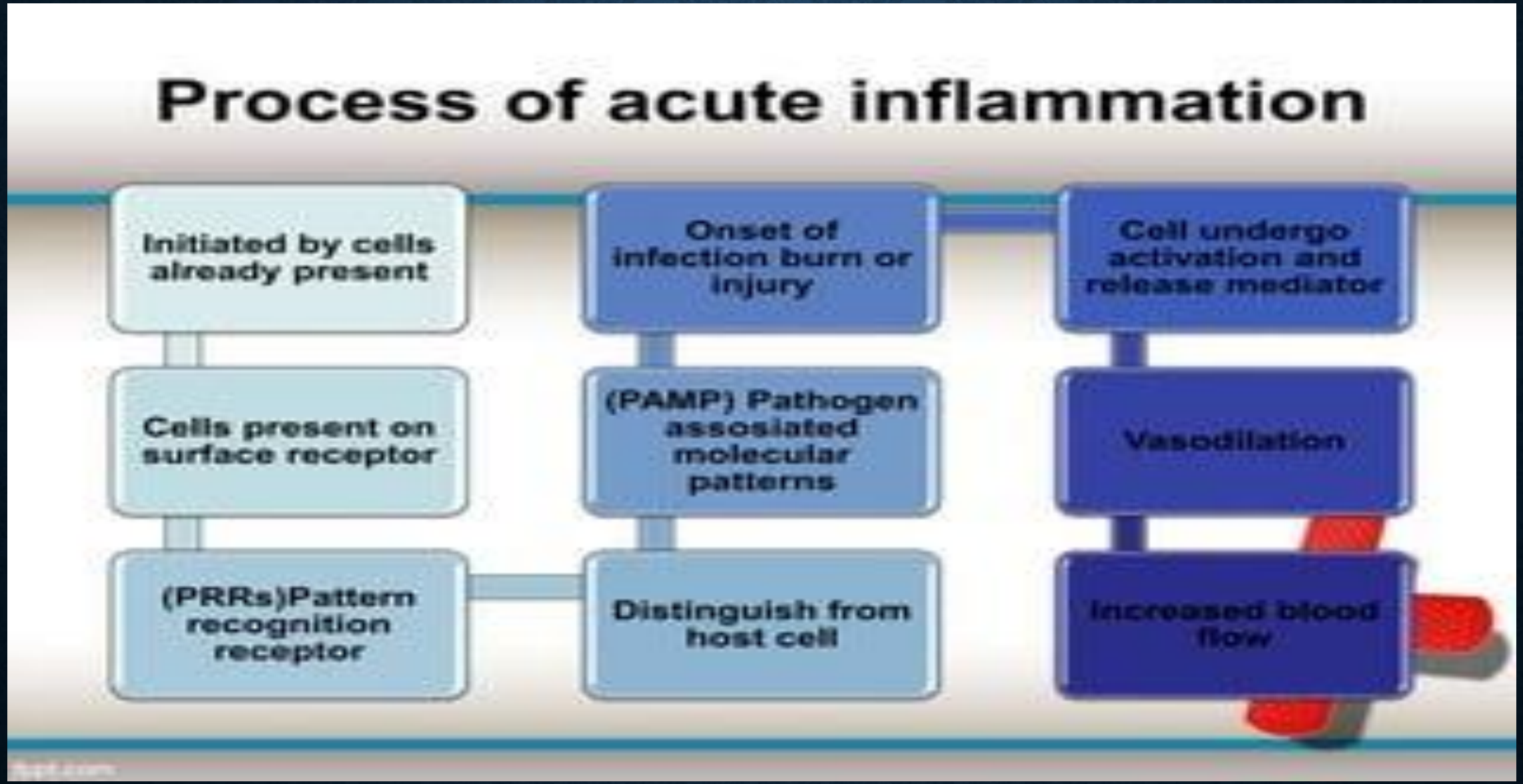


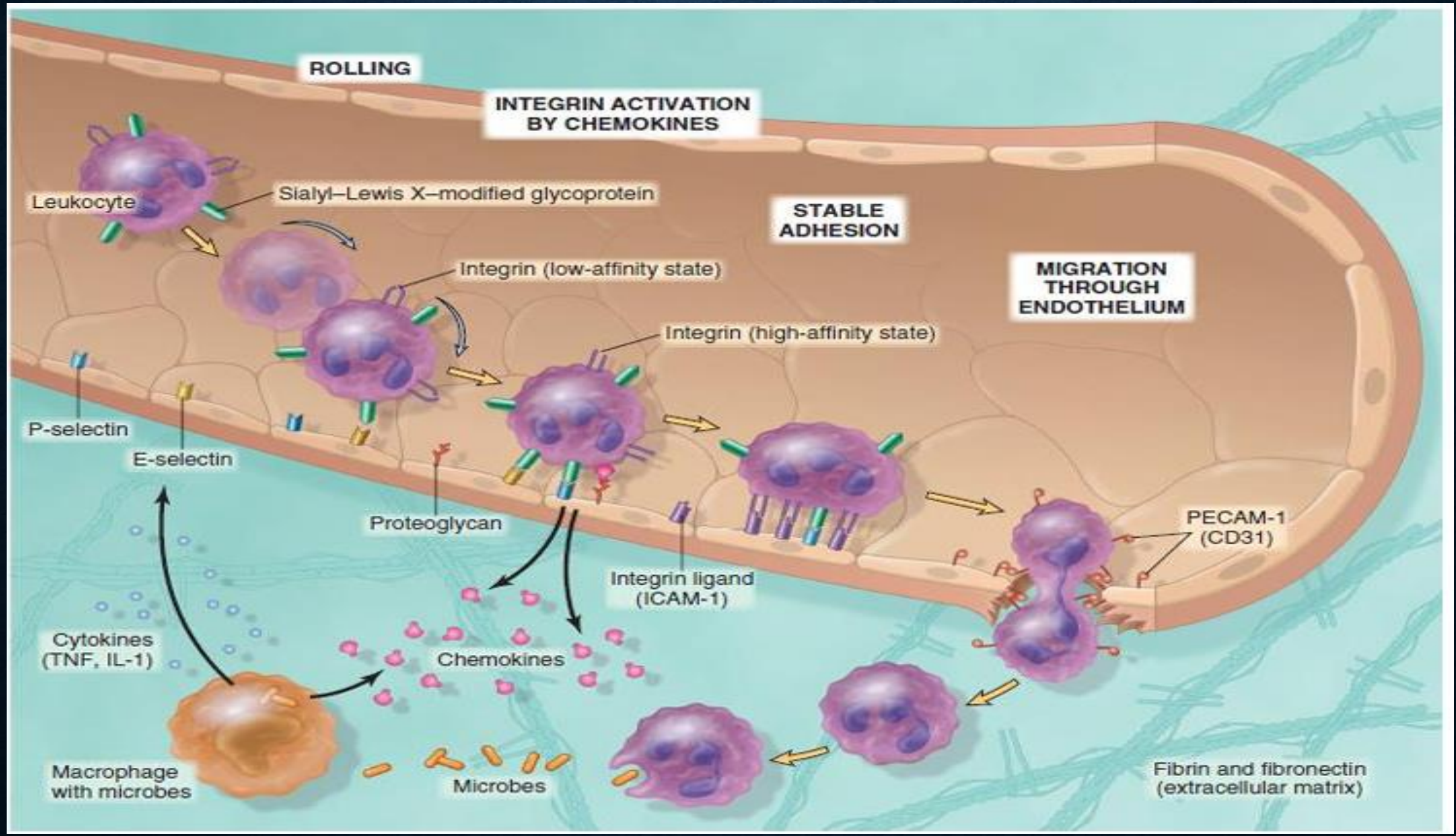
FUNCTIO LAESA – loss of function (later added by galen and virchow in 3rd century)

CELLULAR EVENTS OF ACUTE INFLAMMATION

- Vascular changes (Vasodilation, inc. vascular permeability)
- Cellular events takes place such as;

❖ MARGINATION
❖ ADHESION
❖ EMIGRATION
❖ CHEMOTAXIS
❖ PHAGOCYTOSIS


Process of acute inflammation
Initiated by cells already present
Cells present on surface receptor
(PRRs)Pattern recognition receptor
Onset of infection burn or injury
(PAMP) Pathogen assosiated molecular patterns
Distinguish from host cell
Cell undergo activation and release mediator
Vasodilation
Increased blood flow


ROLLING
INTEGRIN ACTIVATION BY CHEMOKINES
STABLE ADHESION
MIGRATION THROUGH ENDOTHELIUM
Leukocyte
Sialyl–Lewis X–modified glycoprotein
Integrin (low-affinity state)
Integrin (high-affinity state)
P-selectin
E-selectin
Proteoglycan
Integrin ligand (ICAM-1)
PECAM-1 (CD31)
Cytokines (TNF, IL-1)
Chemokines
Macrophage with microbes
Microbes
Fibrin and fibronectin (extracellular matrix)

Serous Inflammation:

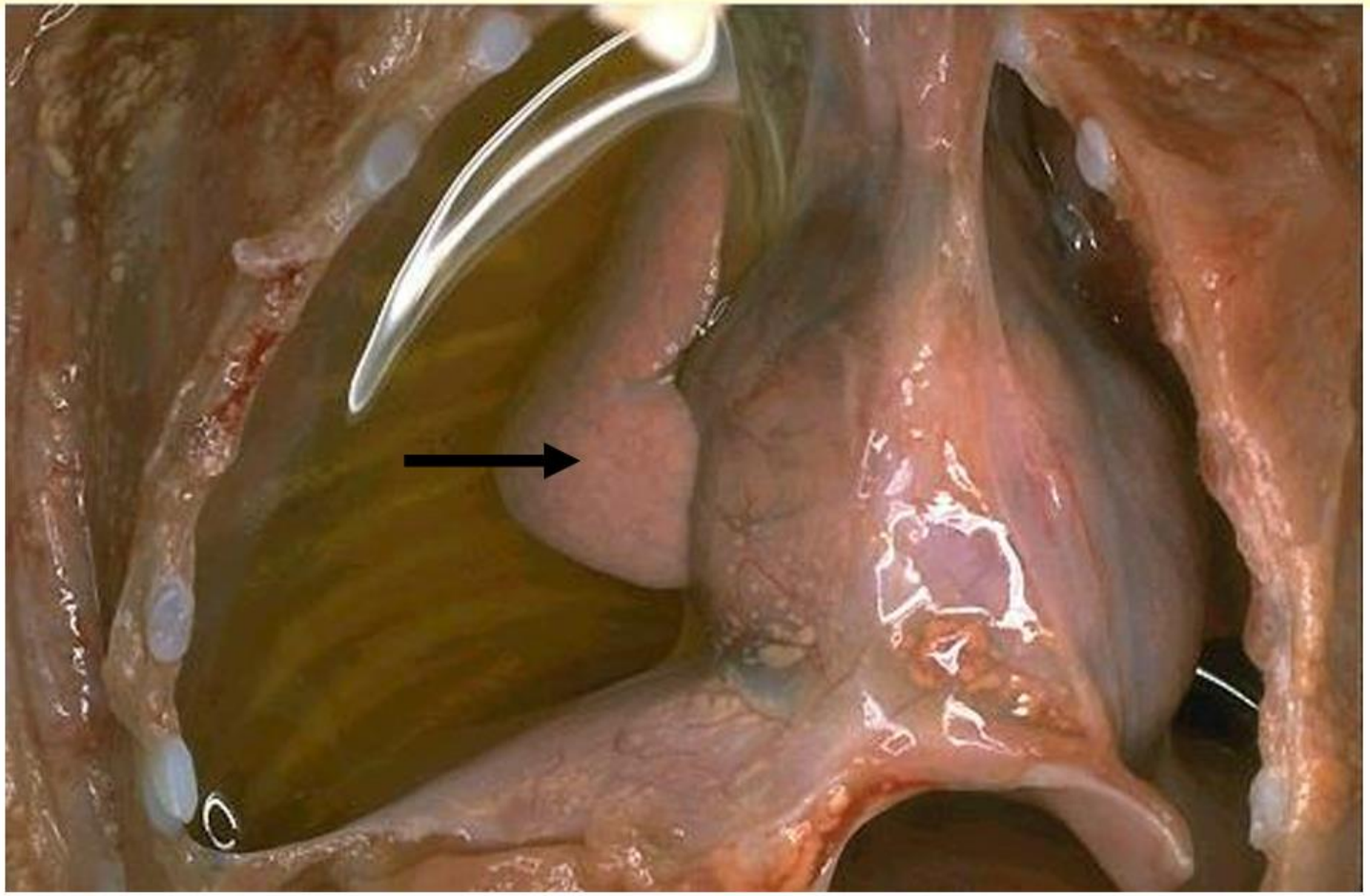
is marked by the exudation of cell poor fluid into spaces created by cell injury or into body cavities lined by the peritoneum, pleura, or pericardium. accumulation of fluid in these cavities is called an effusion.

(Effusions also occur in non-inflammatory conditions, such as reduced blood outflow in heart failure or reduced plasma protein levels in some kidney and liver diseases.)

Is characterized by the outpouring of a thin fluid that is derived from either the plasma or the secretions of mesothelial cells lining the peritoneal, pleural, and pericardial cavities.

meaning of **SEROUS** is of, relating to, or resembling serum; especially : of thin watery constitution. that are typically pale yellow or transparent and

Serous inflammation (serous pleural effusion)



Excessive accumulation of clear, thin fluid within pleural cavity. It is transparent but note the reflection of light in the upper part of the photograph and lung collapse (arrow) due to pressure induced by the fluid.


Medscape
www.medscape.com
Source: Dermatol Nurs © 2003 Jannetti Publications, Inc.

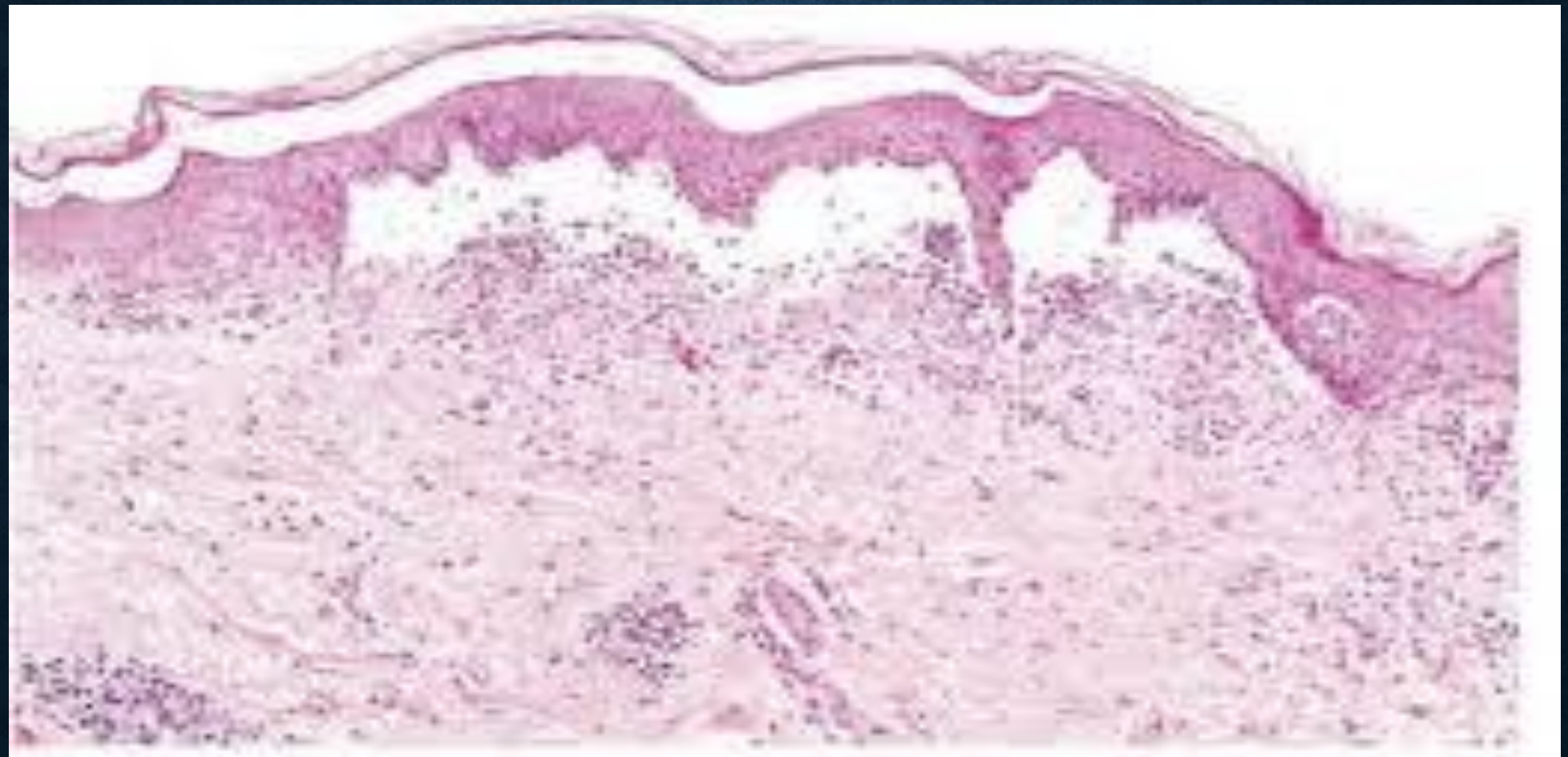


Fig. 3.12 Serous inflammation. Low-power view of a cross section of a skin blister showing the epidermis separated from the dermis by a focal collection of serous effusion.

2. Fibrinous Inflammation:

A fibrinous exudate develops when the vascular leaks are large or there is a local procoagulant stimulus (e.g., caused by cancer cells). A fibrinous exudate is characteristic of inflammation in the lining of body cavities, such as the meninges, pericardium. Histologically, fibrin appears as an eosinophilic meshwork of threads or sometimes as an amorphous coagulum. If the fibrin is not removed, over time it may stimulate the ingrowth of fibroblasts and blood vessels and thus lead to scarring. Conversion of the fibrinous exudate to scar tissue is called organization.

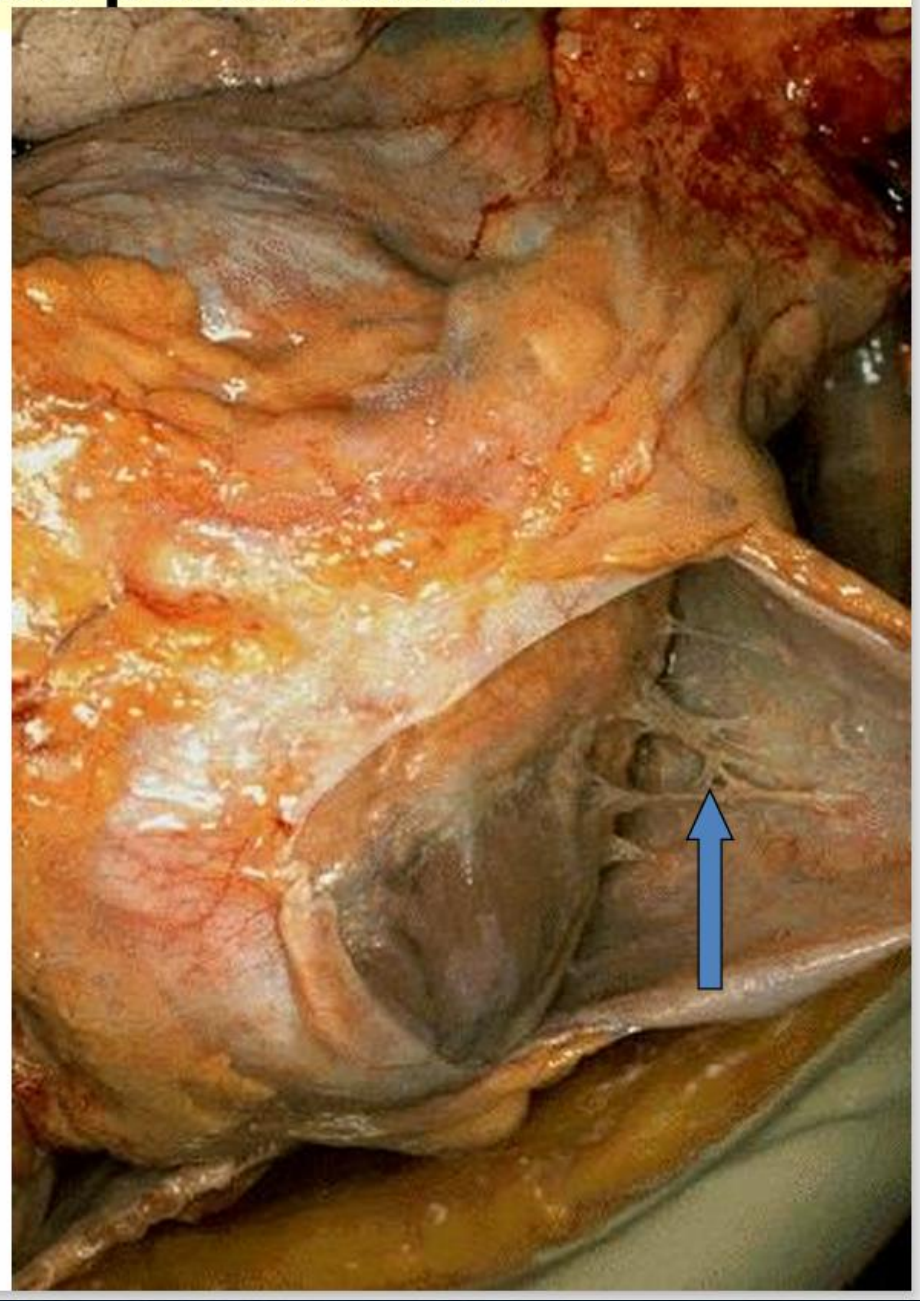
Serous: a transudate with mainly edema fluid and few cells.

Serosanguinous: an effusion with red blood cells.

Fibrinous (serofibrinous): fibrin strands are derived from a protein-rich exudate.

Fibrinous exudate-pericardium

- there is a lot of fibrin.
- the visceral and parietal surfaces become stuck together (by fibrin).
- Separation of the two layers imparts rough irregular appearance (the so called bread and butter).



The typical "bread and butter" appearance of fibrinous pericarditis.

Fibrinous inflammation

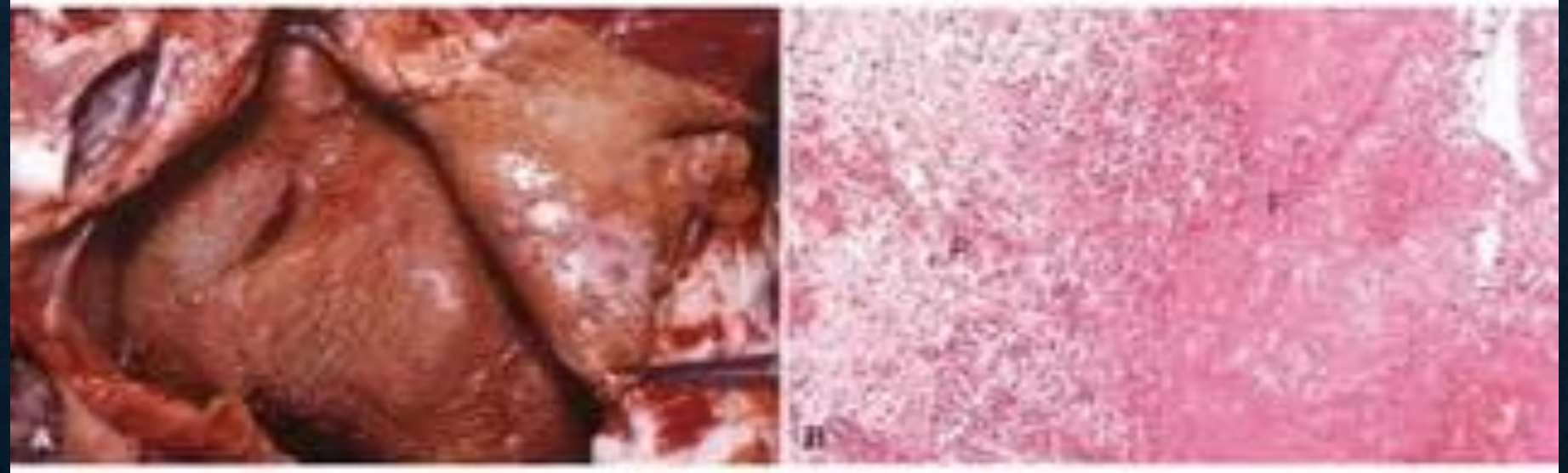


FIGURE 2-19 Fibrinous pericarditis. **A**, Deposits of fibrin on the pericardium. **B**, A pink meshwork of fibrin exudate (F) overlies the pericardial surface (P).

v3-CSBRP-May-2012

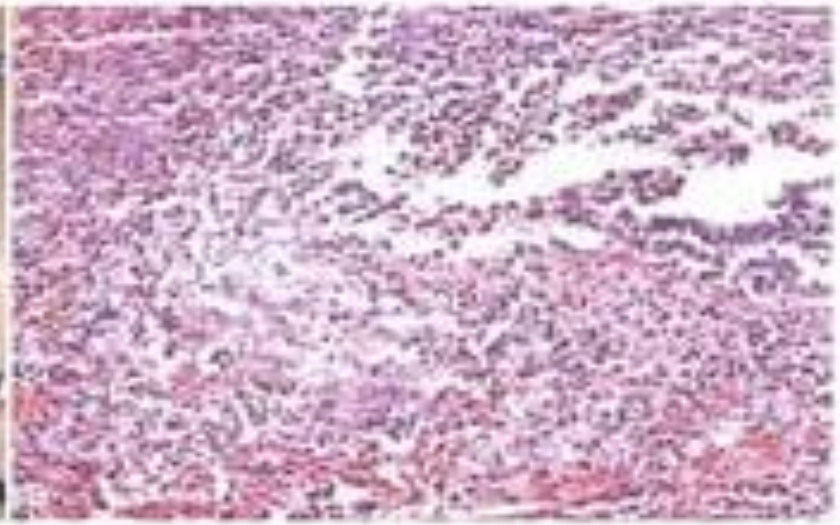
Purulent (Suppurative) Purulent inflammation is characterized by the production of pus, an exudate consisting of neutrophils, the liquefied debris of necrotic cells, and edema fluid.
The most frequent cause of purulent (also called suppurative) inflammation is infection with bacteria that cause liquefactive tissue necrosis, such as staphylococci

Abscesses are localized collections of pus caused by suppuration buried in a tissue, an organ, or a confined space.

Appendix: acute suppurative inflammation

Upper half of excised appendix. Lt: fibrino-purulent serosal exudate Rt: lumen filled with pus

Suppurative or purulent inflammation is characterized by the production of large amounts of pus or purulent exudate consisting of neutrophils, necrotic cells, and edema fluid.



Suppurative inflammation

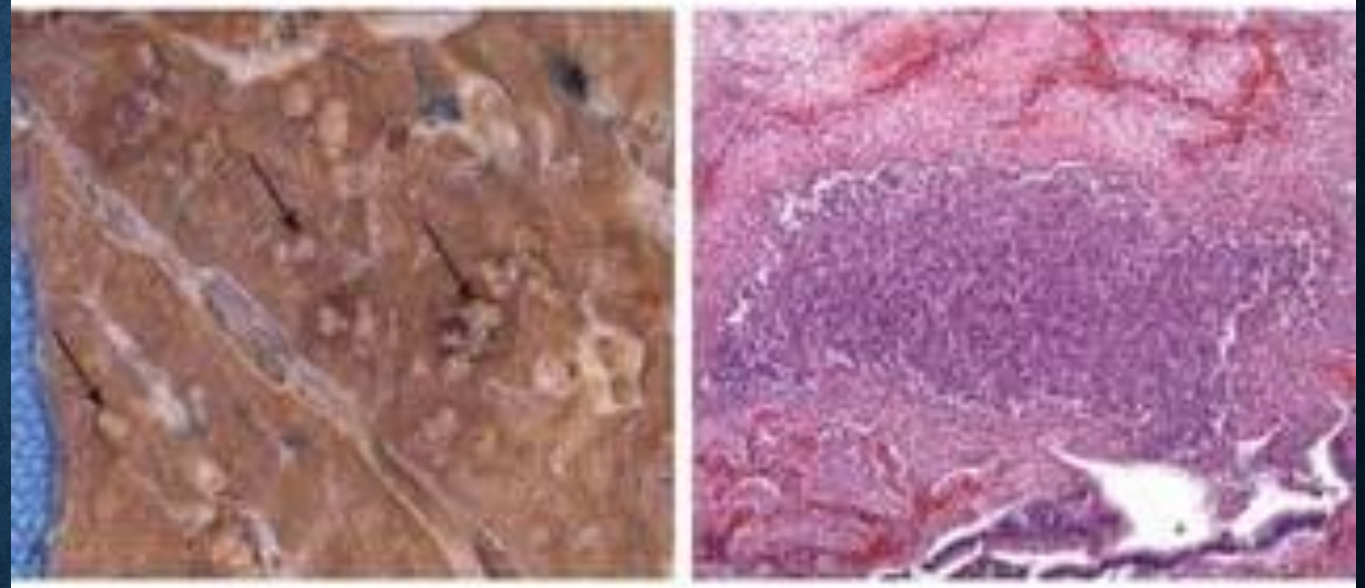
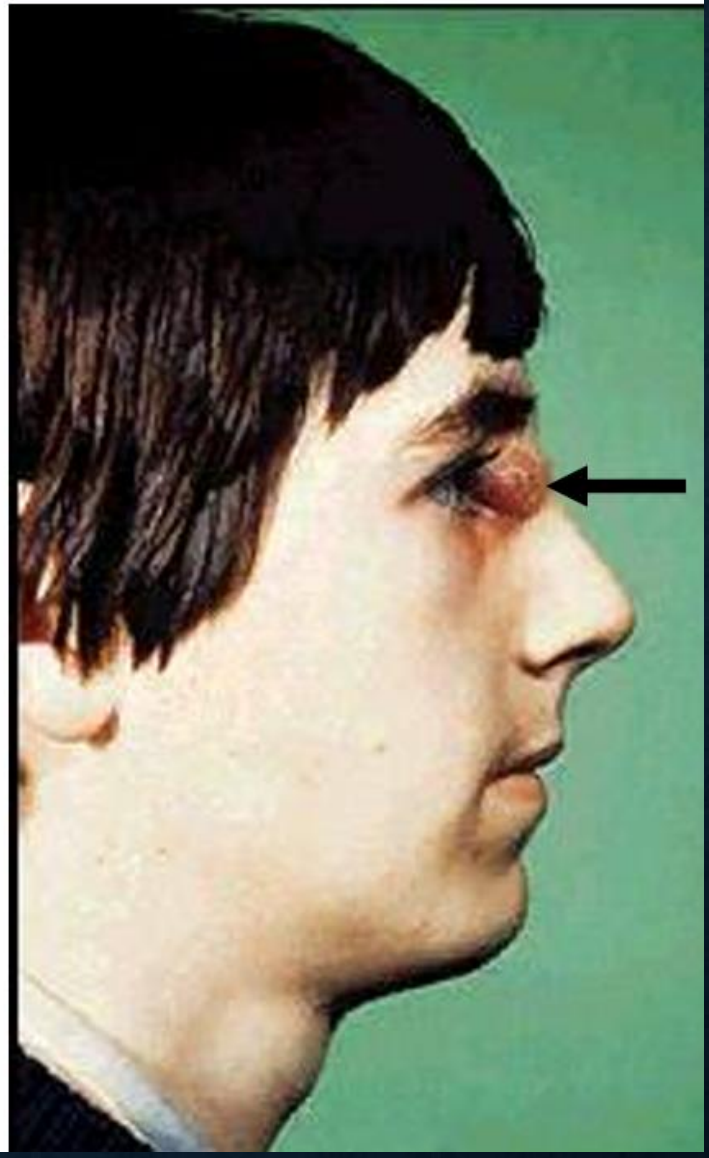


FIGURE 2-20 Purulent inflammation. **A**, Multiple bacterial abscesses in the lung, in a case of bronchopneumonia. **B**, The abscess contains neutrophils and cellular debris, and is surrounded by congested blood vessels.

Abscess (Furuncle) (boil)

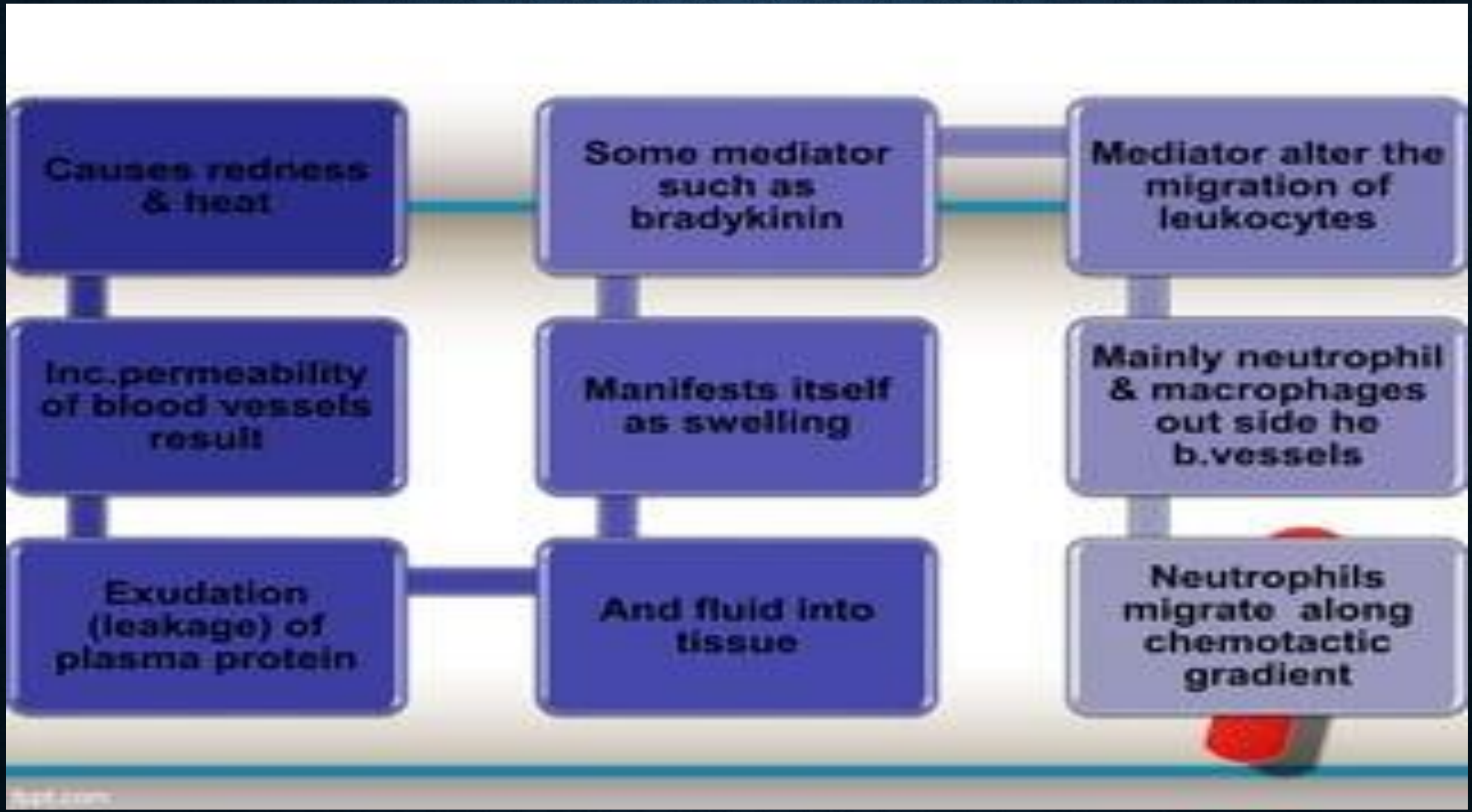
Abscess that involves the skin is called "Boil" or "furuncle".

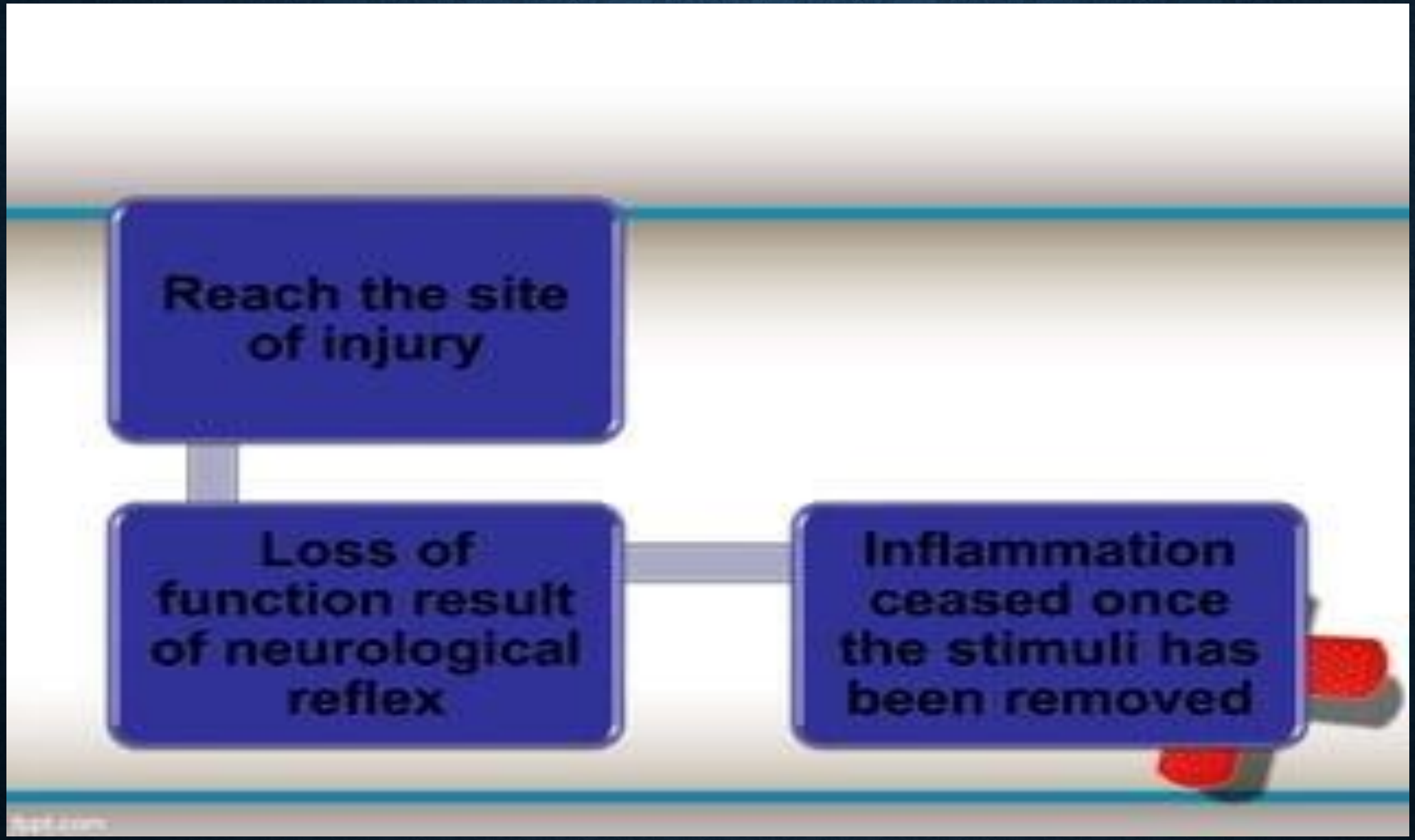


Ulcers :- An ulcer is a local defect, or excavation of the surface of an organ or tissue that is produced by the sloughing (shedding) of inflammatory necrotic tissue. Ulceration occurs only when tissue necrosis and resultant inflammation exist on or near a surface.

Chronic peptic ulcer stomach

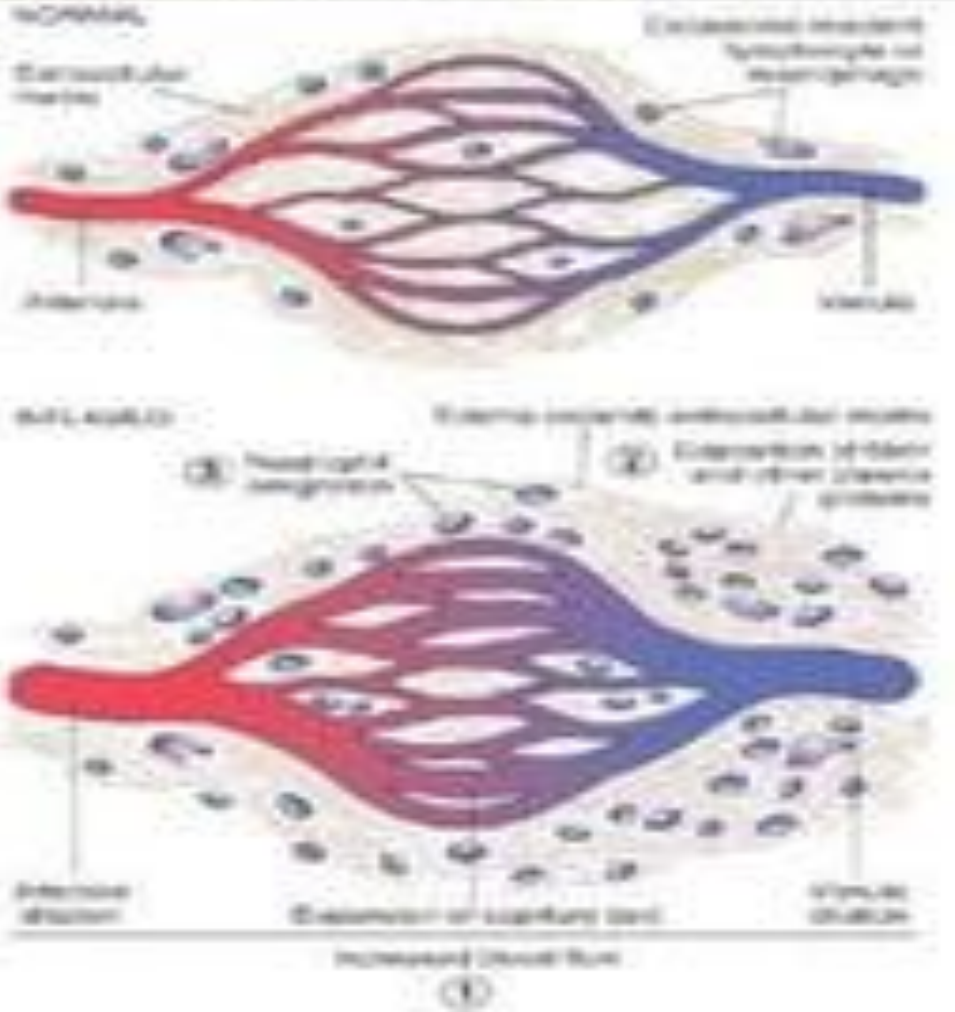
Sharply delimited chronic peptic ulcer with converging folds of mucosa in the upper half


Causes redness & heat
Some mediator such as bradykinin
Mediator alter the migration of leukocytes
Inc.permeability of blood vessels result
Manifests itself as swelling
Mainly neutrophil & macrophages out side he b.vessels
Exudation (leakage) of plasma protein
And fluid into tissue
Neutrophils migrate along chemotactic gradient


Reach the site of injury
Loss of function result of neurological reflex
Inflammation ceased once the stimuli has been removed

Vascular changes play an important role during acute inflammation (begin early after injury and depends upon the severity of the injury)

- **Vasodilation**, leads to increased blood flow causing redness and warmth (rubor and calor)
- **Increased Permeability**, leads to exudation of protein rich fluid into the extravascular space causing swelling (tumor)
- Loss of fluid from the vessels leads to **Concentration of red cells** resulting in decreased velocity and **stasis** of the blood flow
- **Leukocyte rolling, adhesion and migration** leads to the accumulation of inflammatory cells



Inflammation-16

SURGICAL WOUND INFLAMMATION:

RED, WARM & SWOLLEN (FLARE, FLUSH & WHEAL)
CALOR, RUBOR, DOLOR, TUMOR, LOSS OF FUNCTION.

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Inflammation-17

Mouth Aphthus ulcer

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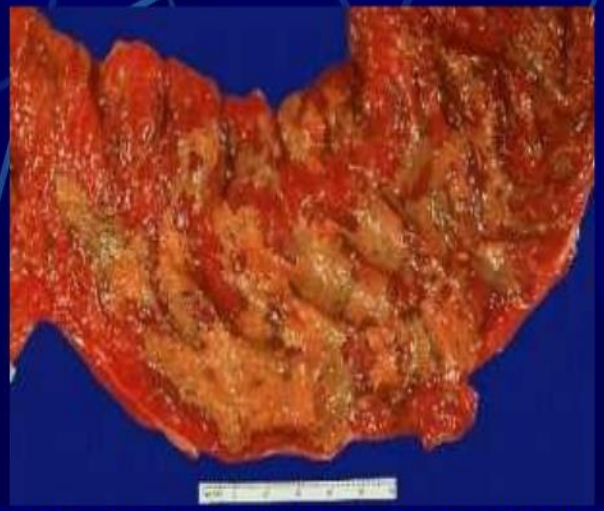
Inflammation-18

ACUTE ENTERITIS:



Red, Warm & Swollen (Flare, Flush & Wheal)
Calor, Rubor, Dolor, Tumor, Loss of function.

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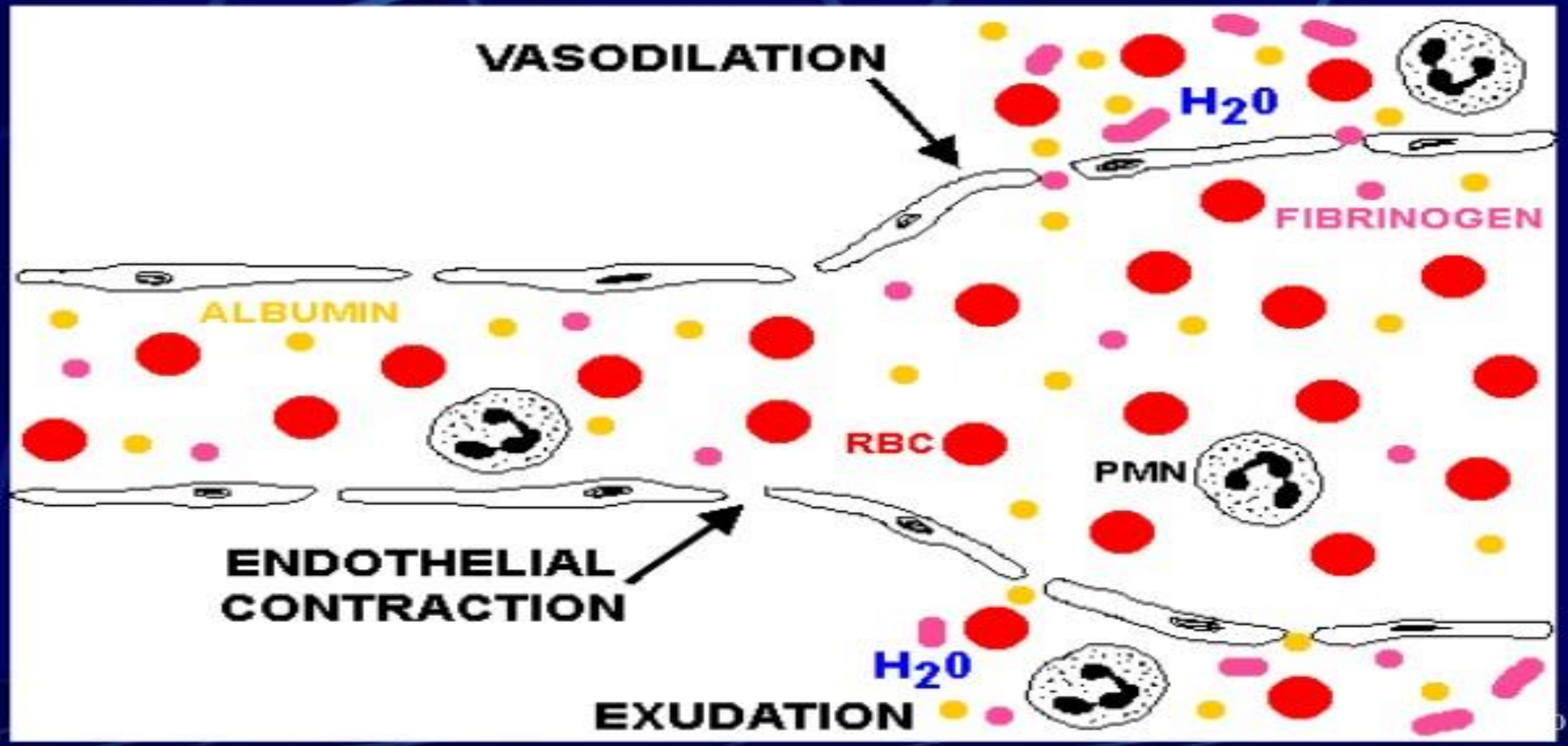
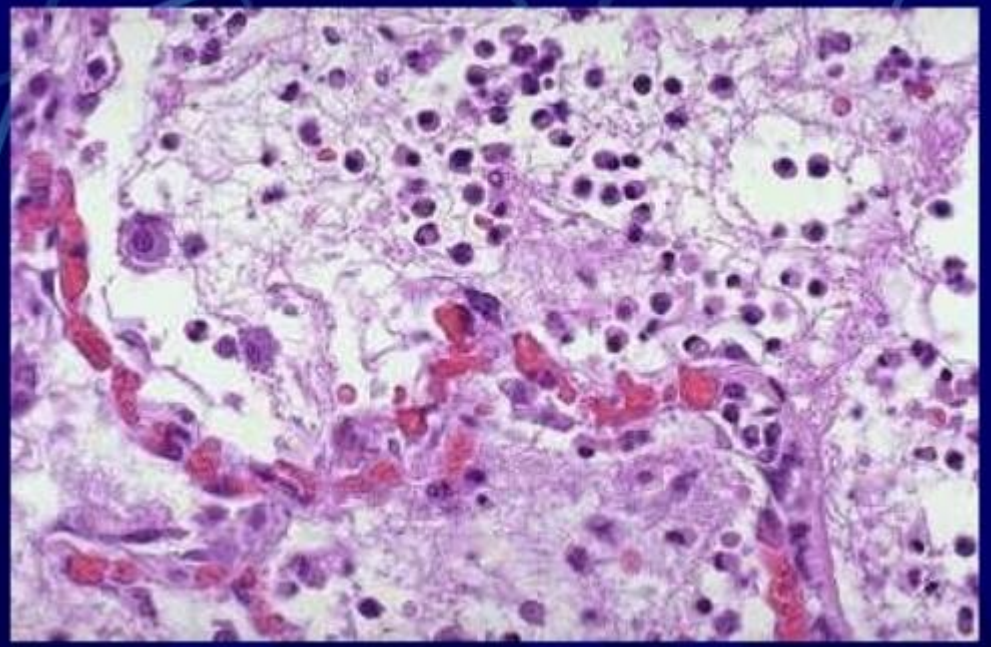
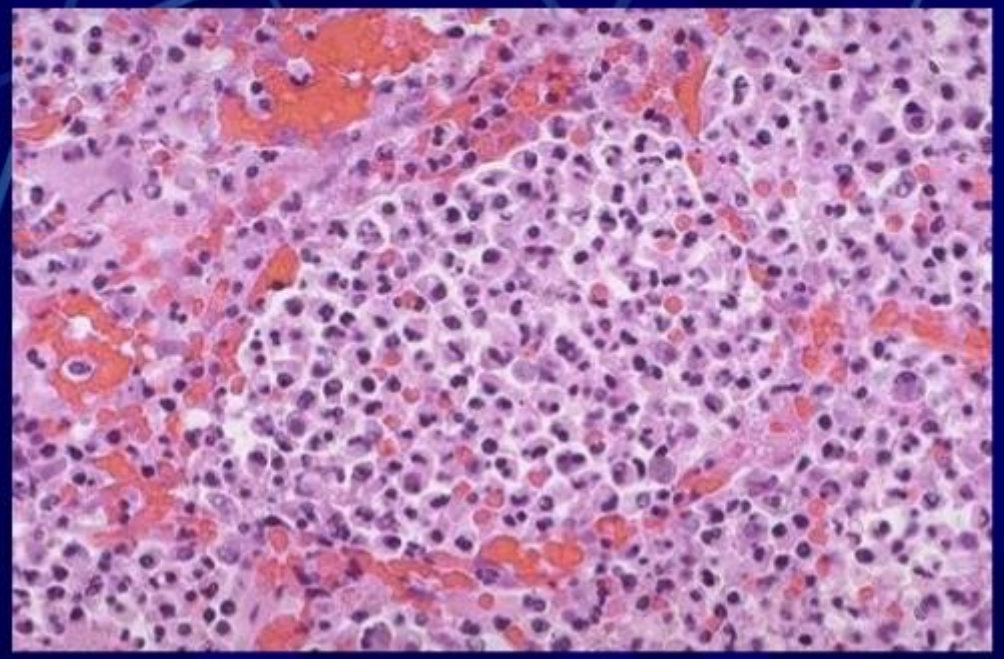
MECHANISM OF INFLAMMATION



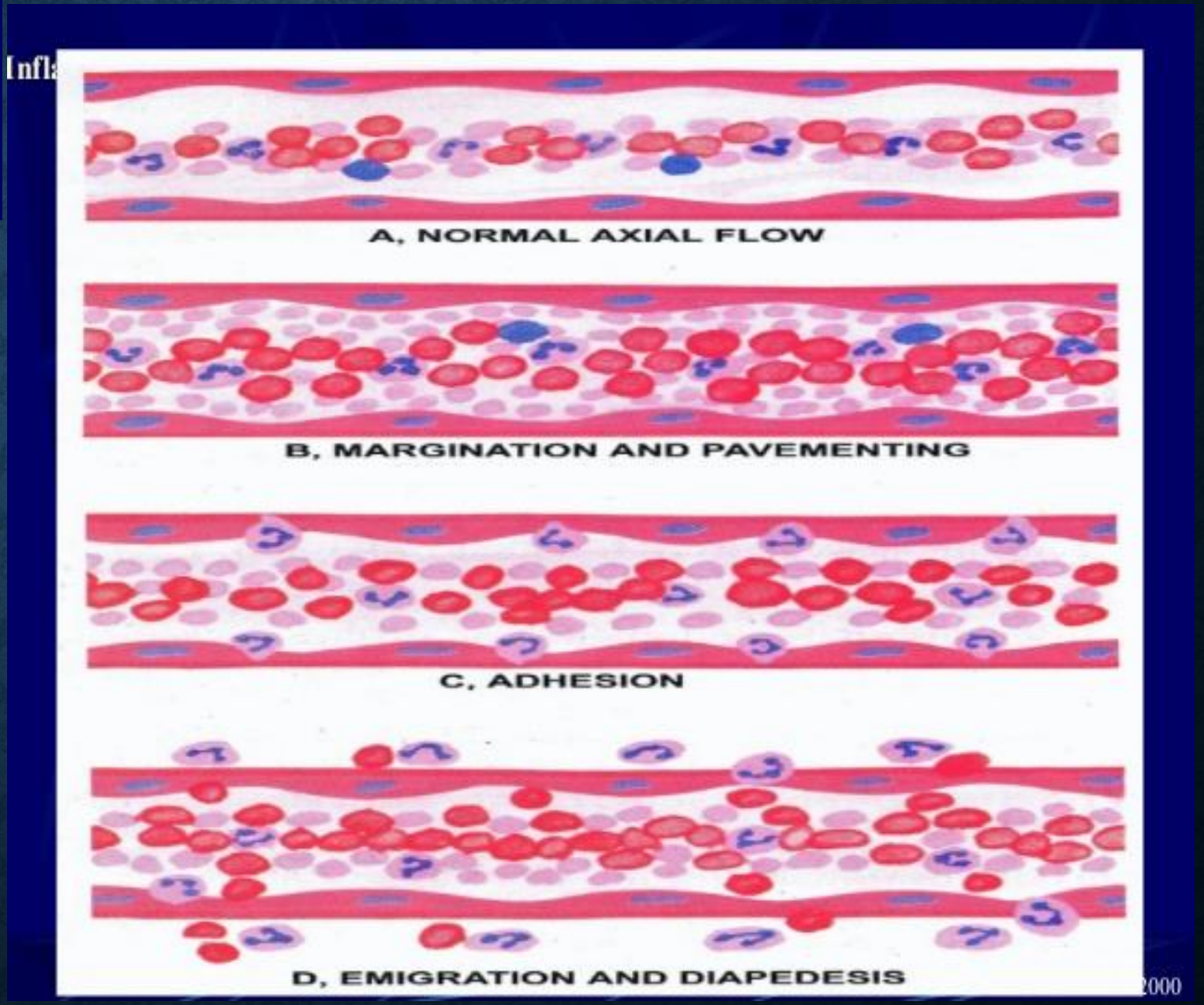
VASCULAR CHANGES



EXUDATION

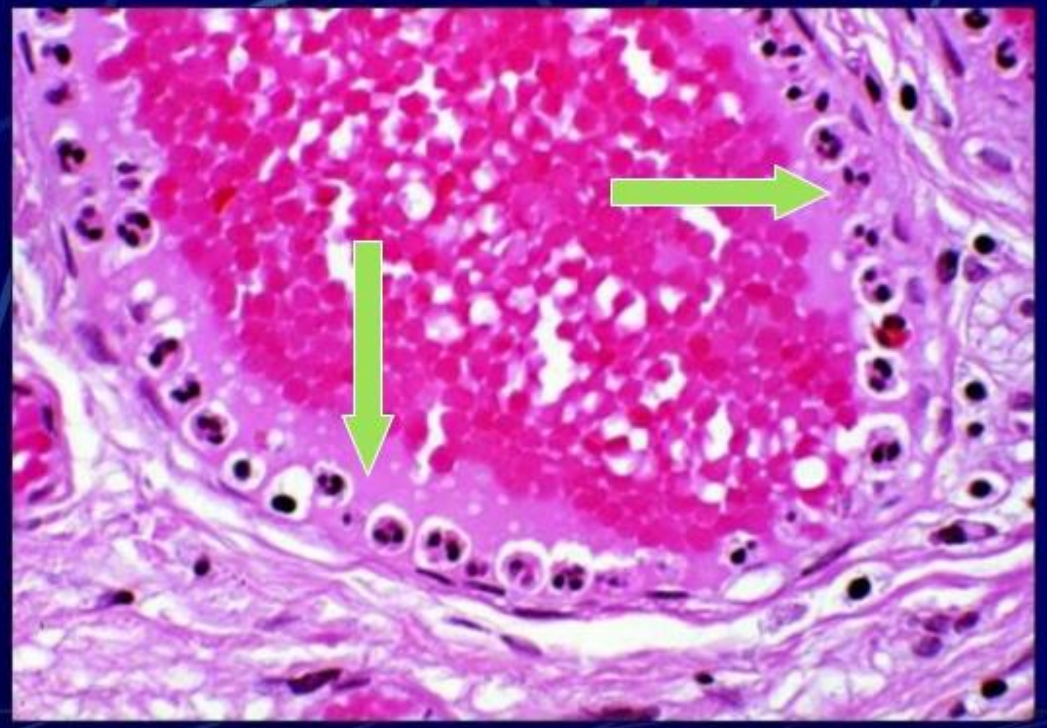


LEUKOCYTE CELLULAR EVENTS



Inflammation-31

NEUTROPHIL MARGINATION



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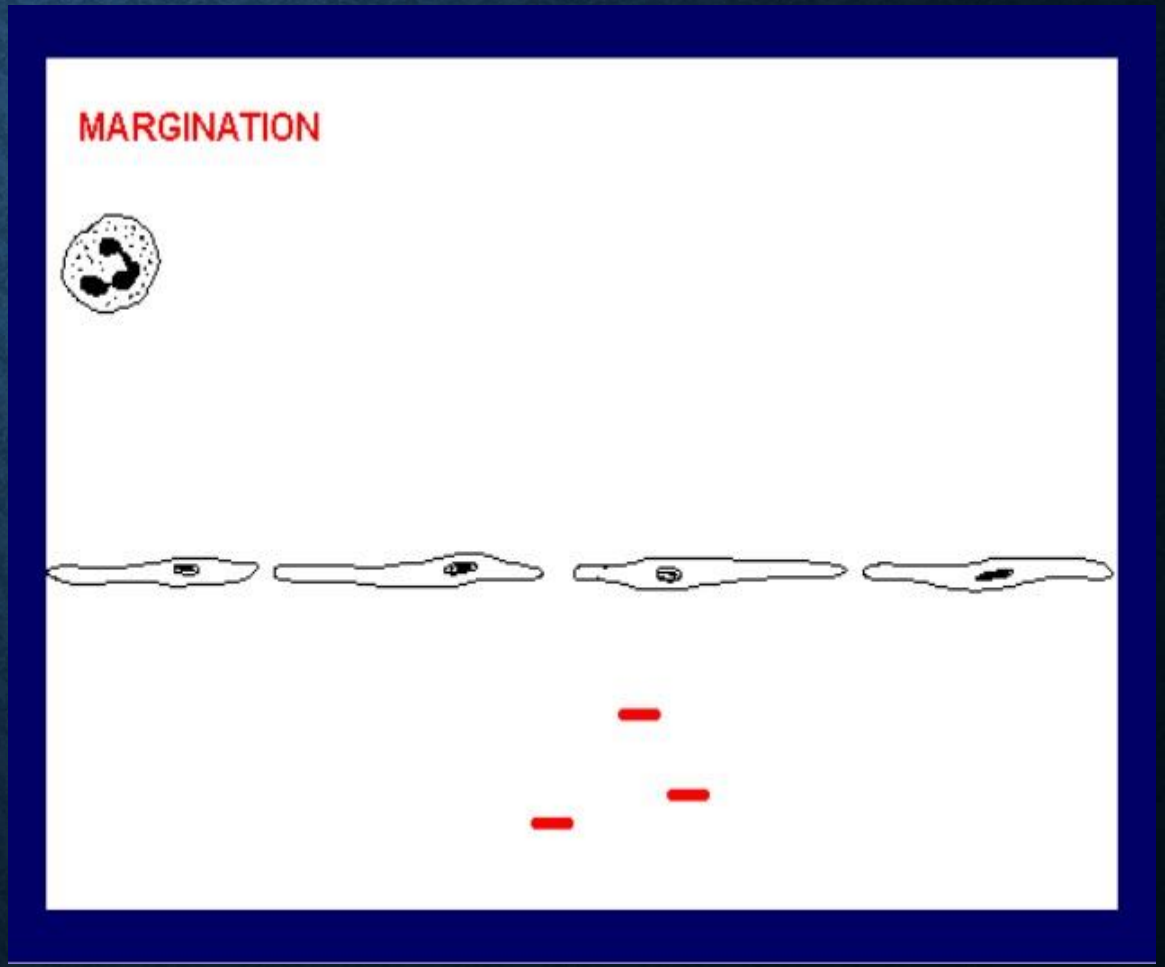
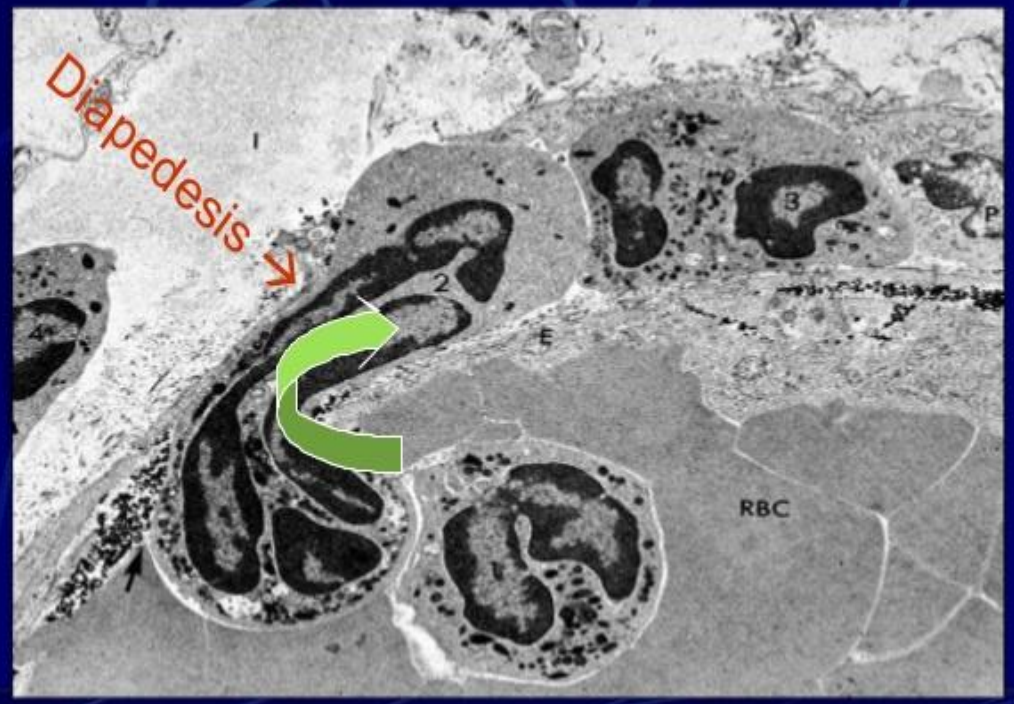
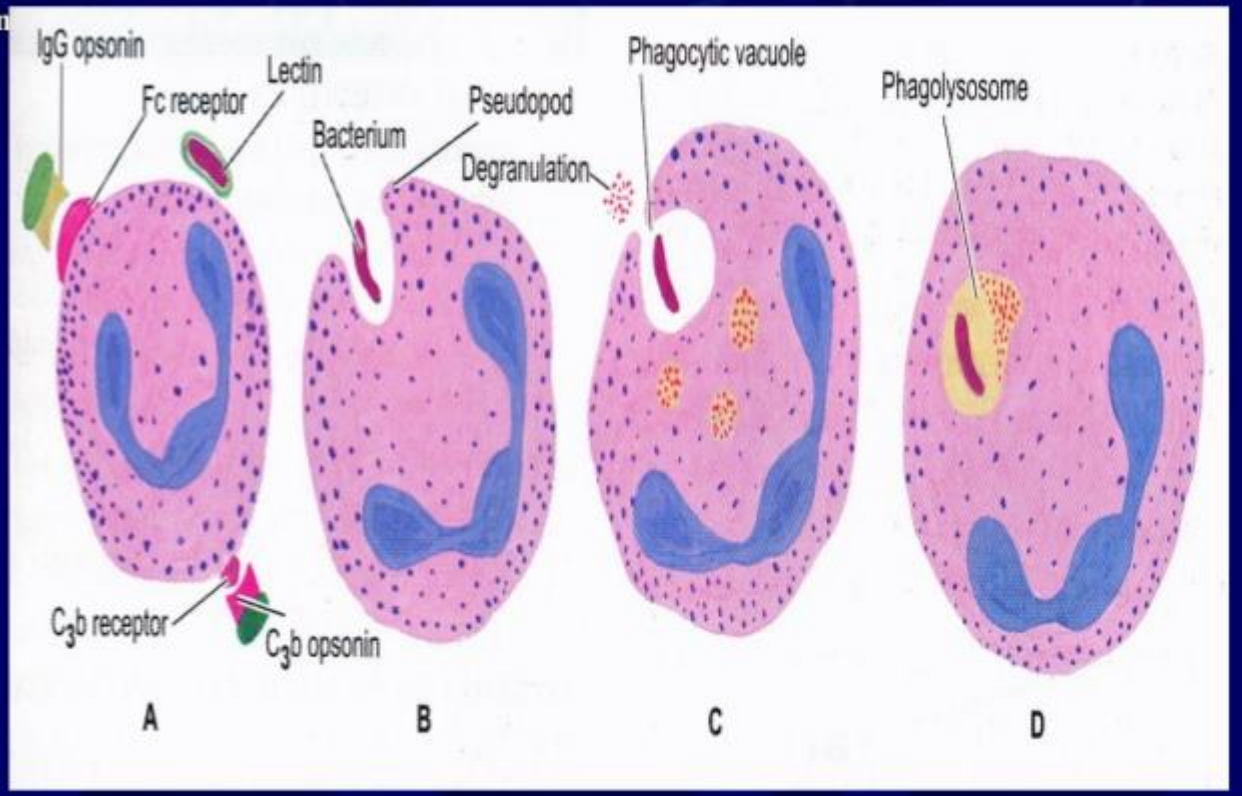


EMIGRATION OF LEUCOCYTES



Shashi-Mar 2000



ENGULFMENT

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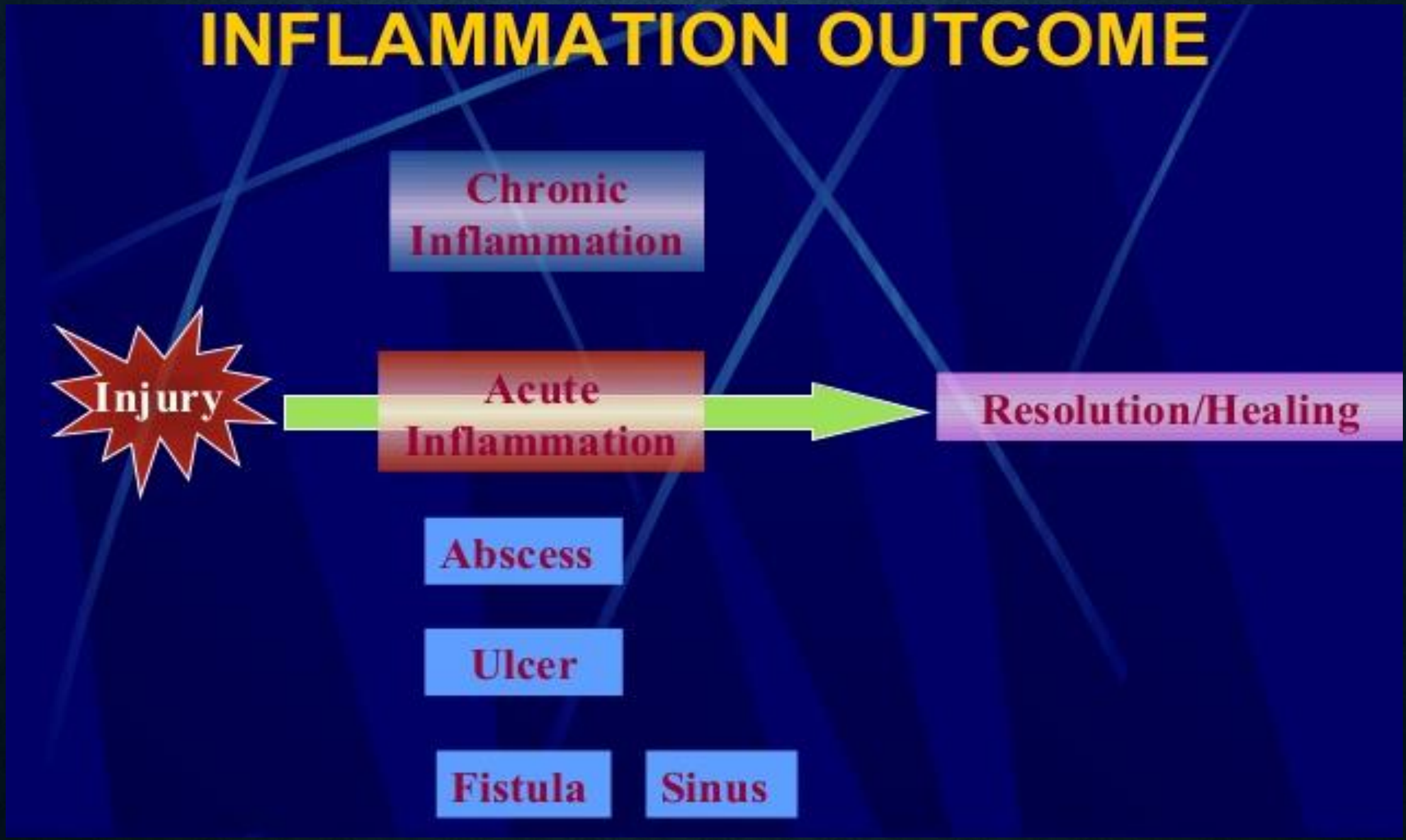
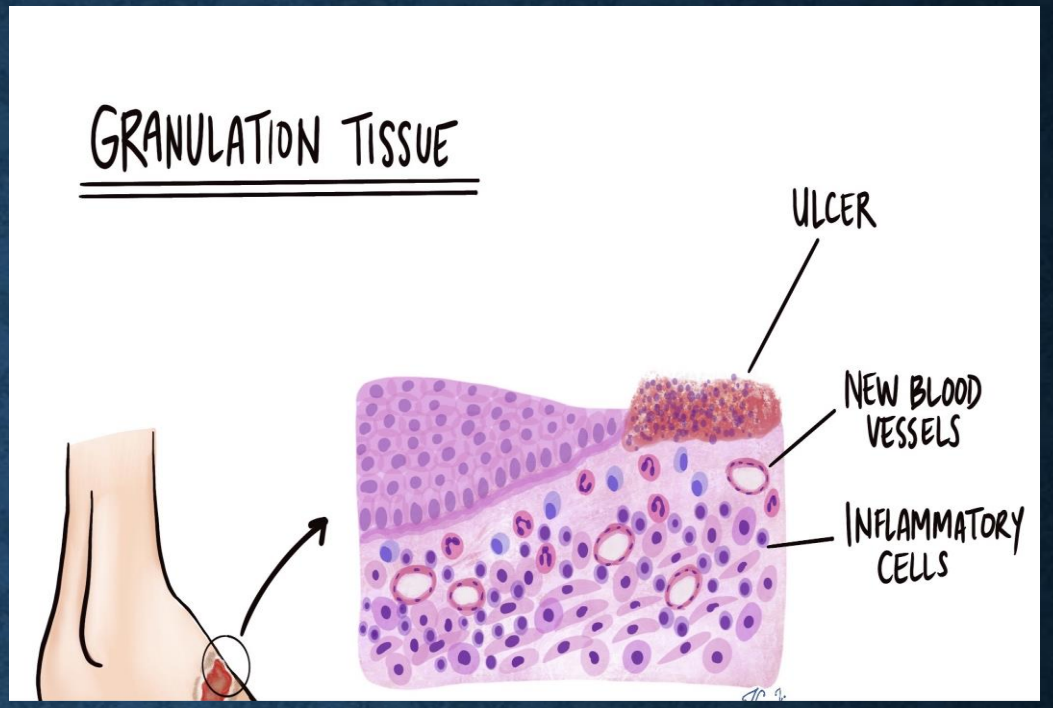
INFLAMMATION OUTCOME



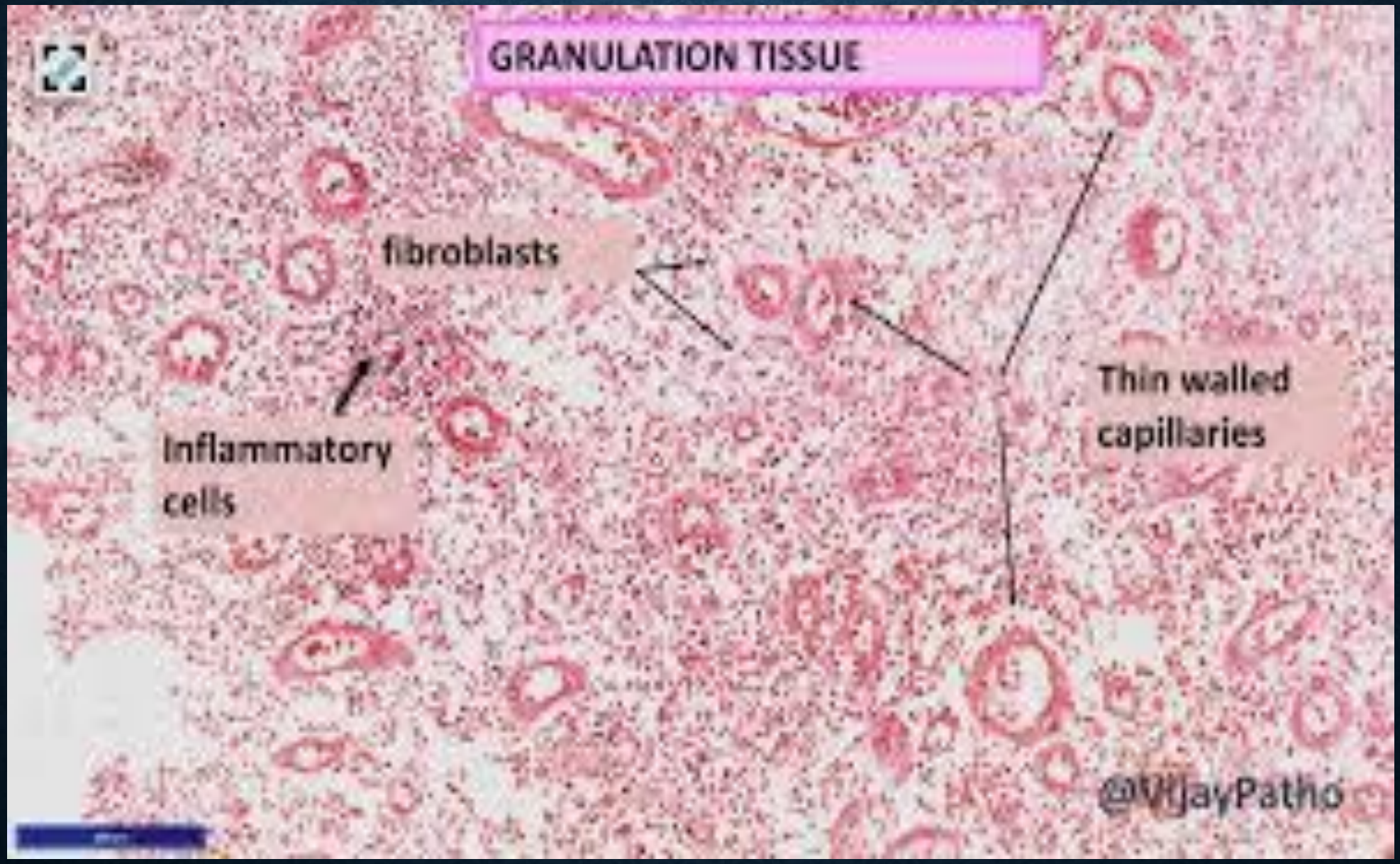
SYMPTOMS OF A SINUS INFECTION

- Green/yellow mucus drips from nose down back of throat
- A blocked or stuffy nose
- Reduced sense of smell
- A shooting pain in nose and forehead when head tilted
- Pain/tenderness around eyes, forehead or cheeks
- Toothache
- A high temperature (38C or higher)
- Headache
- Tiredness

LloydsPharmacy



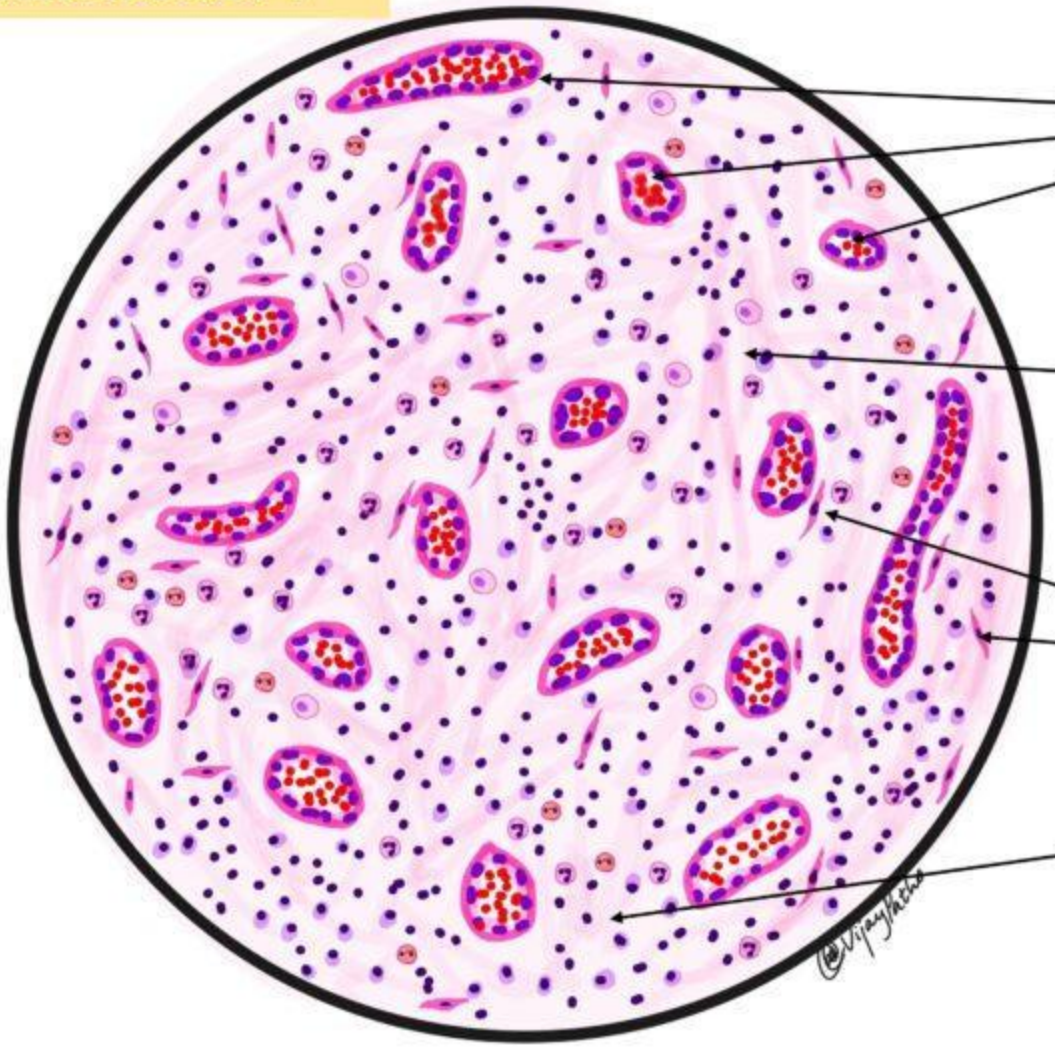
Without a microscope, the tissue usually looks red or pink and it often feels soft to the touch. When examined under a microscope, the tissue is made up of many small blood vessels surrounded by a combination of cells from the immune system.


GRANULATION TISSUE
fibroblasts
Inflammatory cells
Thin walled capillaries
@VijayPatho

GRANULATION TISSUE

Pink soft granular appearance on the surface of healing wounds

MICROSCOPY



Proliferating capillaries

Mixed inflammatory cells

Lymphocytes, plasma cells, neutrophils, eosinophils and macrophages

Fibroblasts with collagen production

Edematous stroma

Normal human red blood cells have an average life span of about 120 days in the circulation after which they are engulfed by macrophages. This is an extremely efficient process as macrophages phagocytose about 5 million erythrocytes every second without any significant release of hemoglobin in the circulation

Formed elements of blood.	Life Span
* RBC	— 120 days.
* WBC	— 13 - 20 days.
• Neutrophils	— 2 - 5 days.
• Eosinophils	— 7 - 12 days.
• Basophils	— 12 - 15 days.
• Monocytes	— 2 - 5 days.
• Lymphocytes	— ½ - 1 days.
* Platelets	— 10 days.

TB is an infectious disease that most often affects the lungs and is caused by a type of bacteria. It spreads through the air when infected people cough, sneeze or spit. Tuberculosis is preventable and curable.

TB is caused by bacteria (Mycobacterium tuberculosis) and it most often affects the lungs.
TB is spread through the air when people with lung TB cough, sneeze or spit.
A person needs to inhale only a few germs to become infected.

•

Medical history.

•Physical examination.

•Test for TB infection (TB blood test or TB skin test)

•Chest x-ray.

•Laboratory tests to see if TB germs are present (sputum smear and culture)

•Laboratory tests for drug resistance

Oral TB is an uncommon form, and often presents as a painless ulceration with undermined edges on palate, lips, buccal mucosa, or tongue, and is usually associated with palpable cervical lymph nodes.