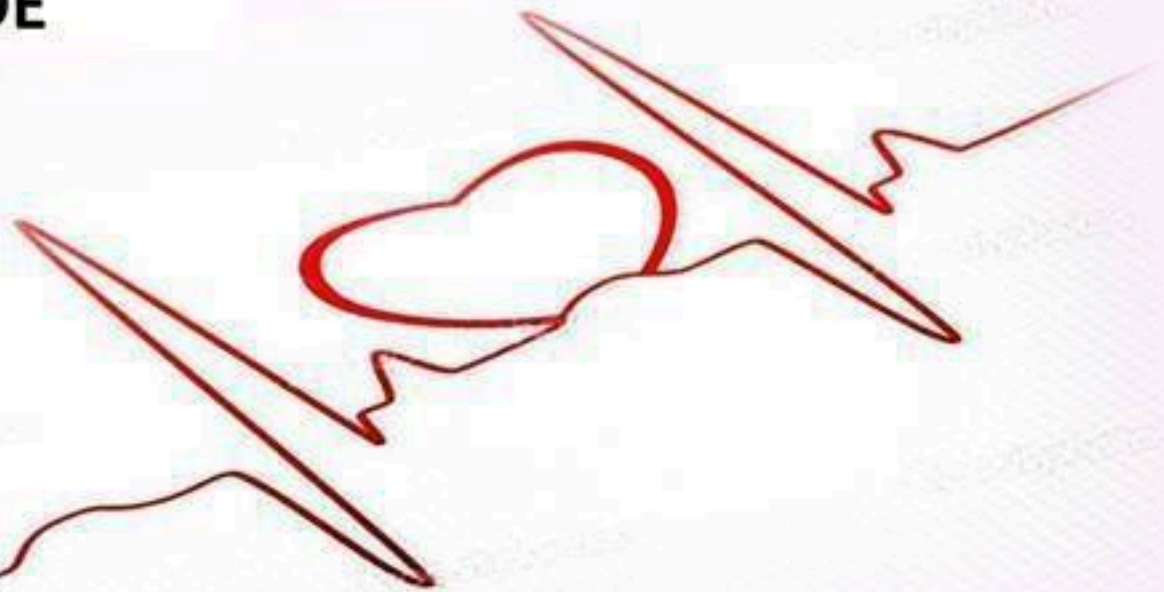


SHEET



SLIDE



Slide : 5- Inflammation



Doctor: Heyam Awad (1)



INFLAMMATION

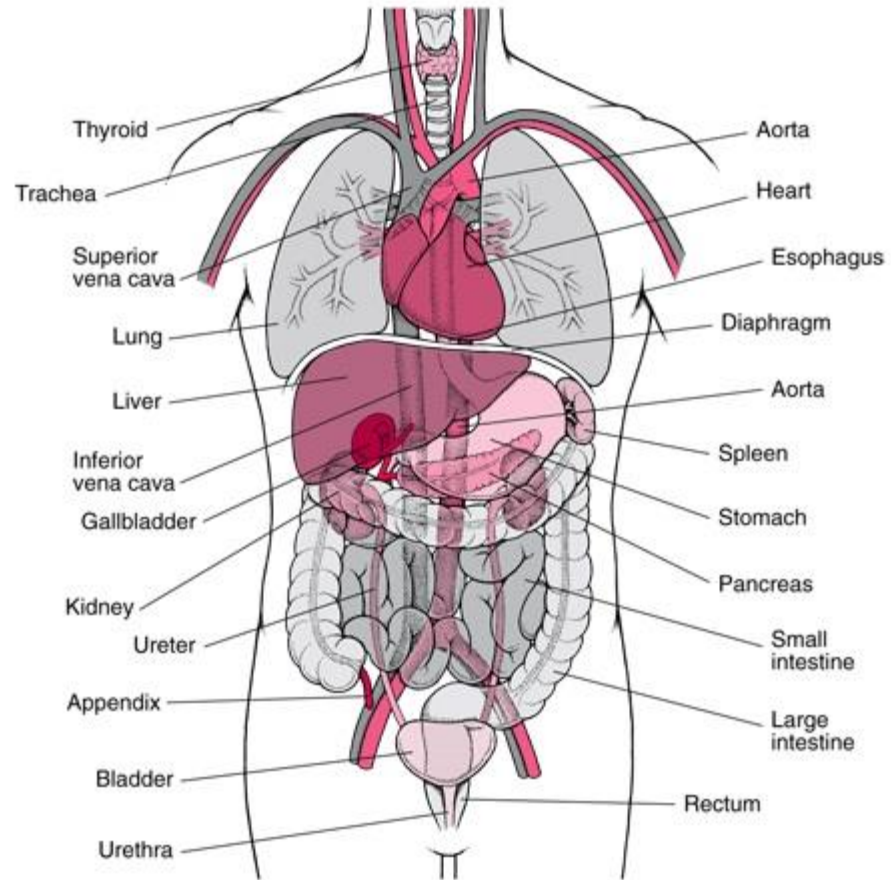
DR HEYAM AWAD

CONSULTANT HISTOPATHOLOGIST
MBBS, FRCPATH, JORDANIAN BOARD



WHAT IS INFLAMMATION

- EXAMPLES OF INFLAMMATION??????????



NOMENCLATURE

- ITIS..... INFLAMMATION.
- NOT ALL INFLAMMATORY CONDITIONS END WITH ITIS.

DEFINITION

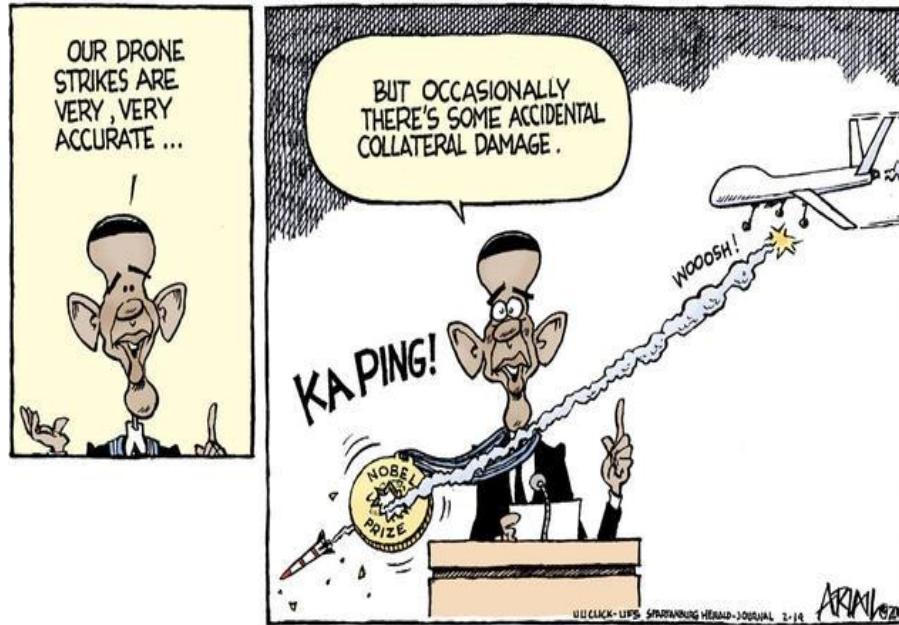
- PROTECTIVE REACTION IN VASCULARISED TISSUE TO ELIMINATE CAUSES OF CELL INJURY AND THE NECROTIC TISSUE RESULTING FROM THE INITIAL INSULT.

CAUSES

- ANYTHING THAT CAUSES CELL INJURY CAUSES INFLAMMATION!
- E.G : INFECTIONS, CHEMICAL AND PHYSICAL CAUSES.
- DIFFERENCE BETWEEN INFLAMMATION AND INFECTION.

- INFLAMMATION IS A PROTECTIVE MECHANISM NOT A DISEASE.
- SOOO....WHY DOES IT CAUSE PROBLEMS?

COLLATERAL DAMAGE!



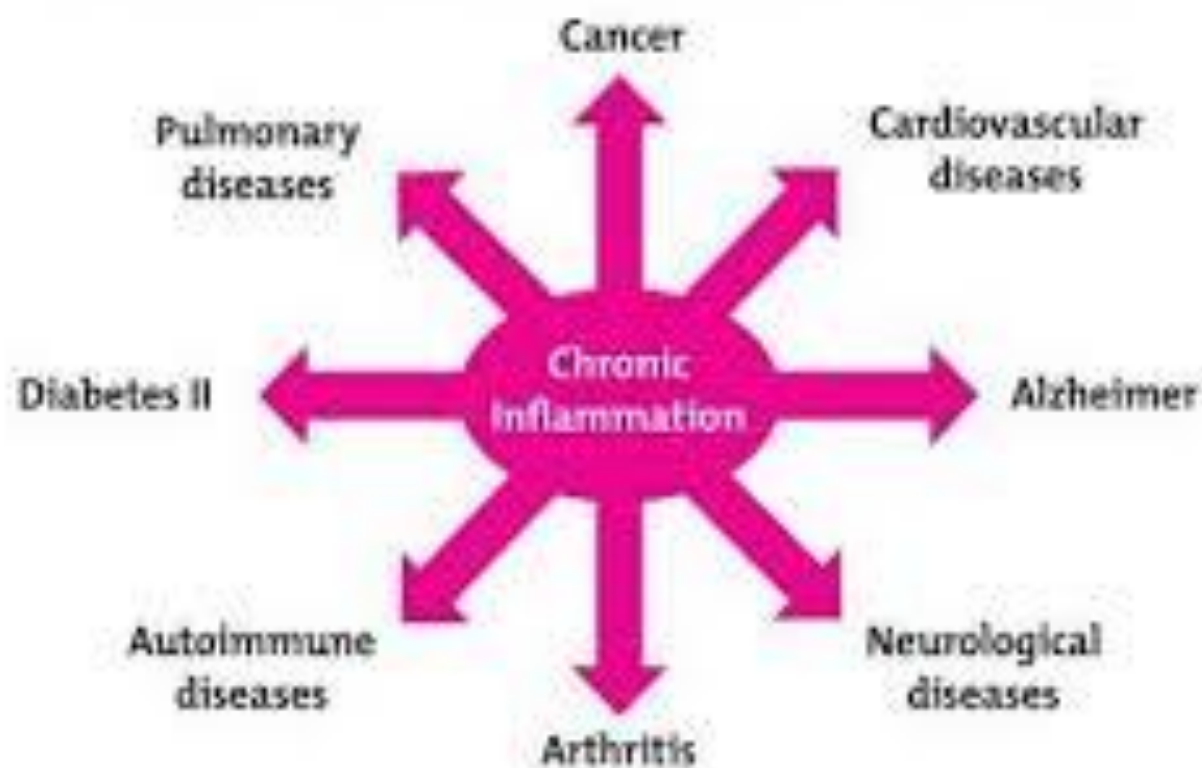
SIGNS AND SYMPTOMS



- RUBOR = REDNESS
- TUMOR = SWELLING
- DOLOR = PAIN
- CALOR = HOTNESS

LOSS OF FUNCTION.

Chronic Inflammation Can Lead To...



ACUTE INFLAMMATION

Innate Immunity

Stimulus



Immune helper cells do their job of healing



End stimulus/ Healing

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

Vasodilation

Prostaglandins E_2 , D_2 , $F_{2\alpha}$, I_2
Nitric Oxide

Increased Vascular Permeability

Histamine, Serotonin
Bradykinin
C3a and C5a (through liberating amines)
Leukotrienes C_4 , D_4 , E_4
PAF (AGEPC)
oxygen free radicals

Chemotaxis

C5a
Leukotriene B_4
IL-8
Bacterial products

Pain

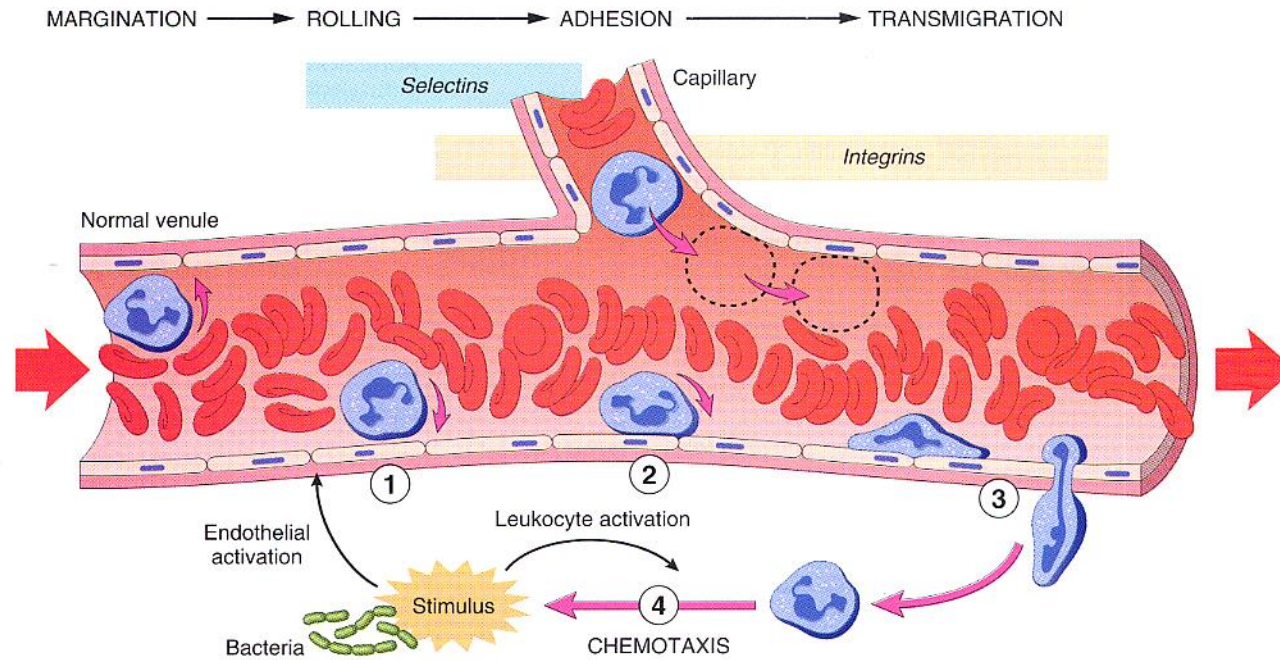
PGE_2
Bradykinin

Fever

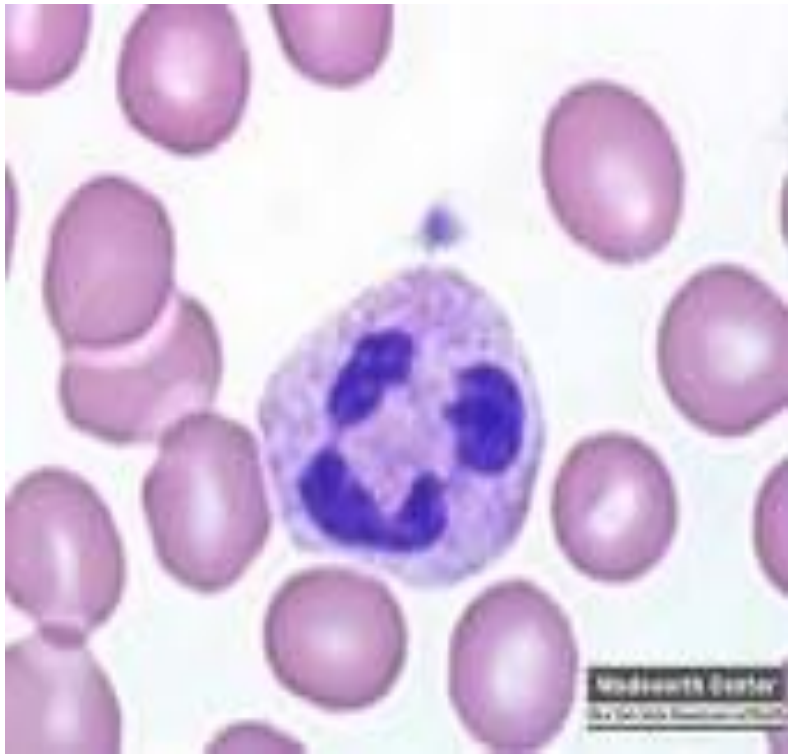
IL-1, IL-6, TNF
 PGE_2

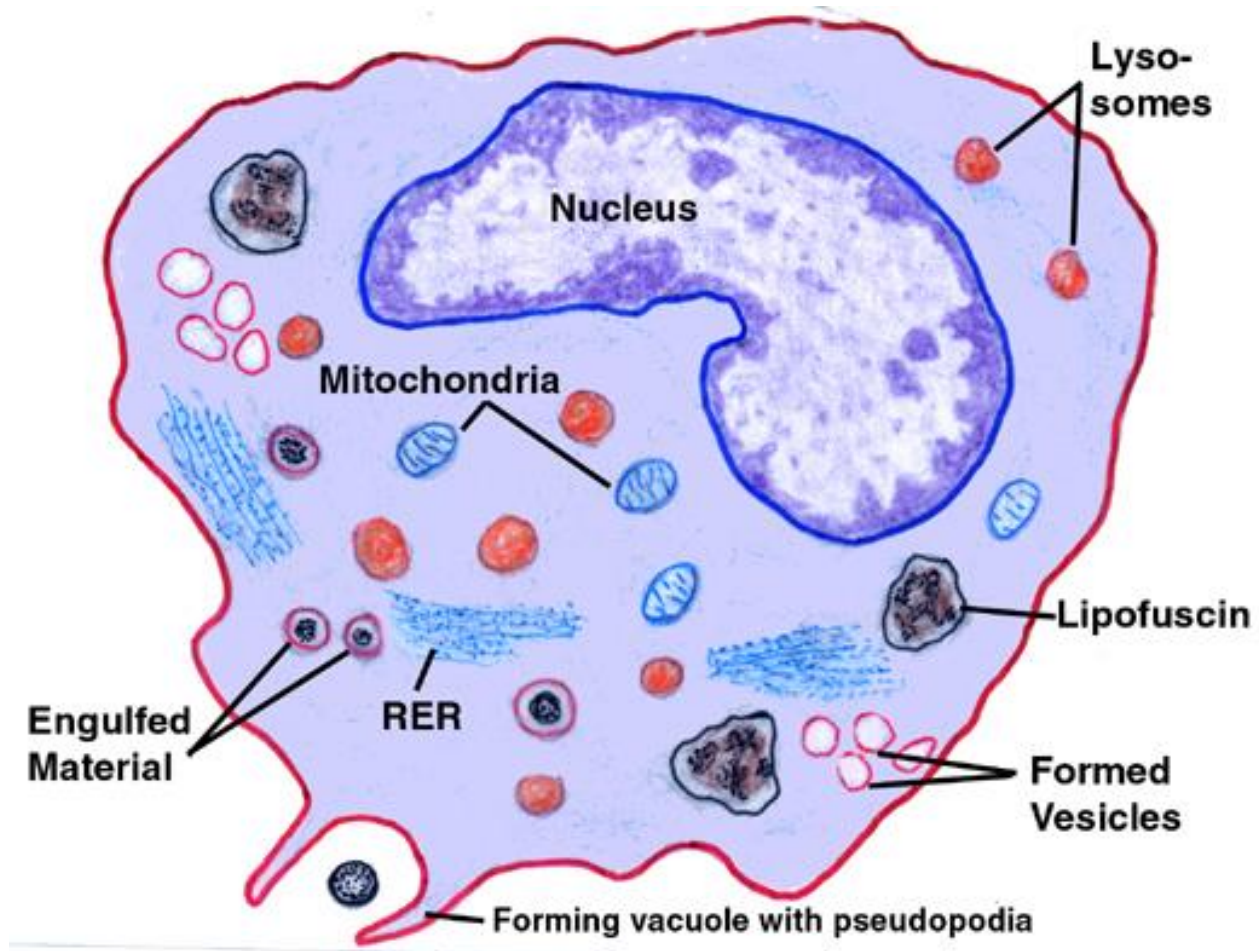
Tissue Damage

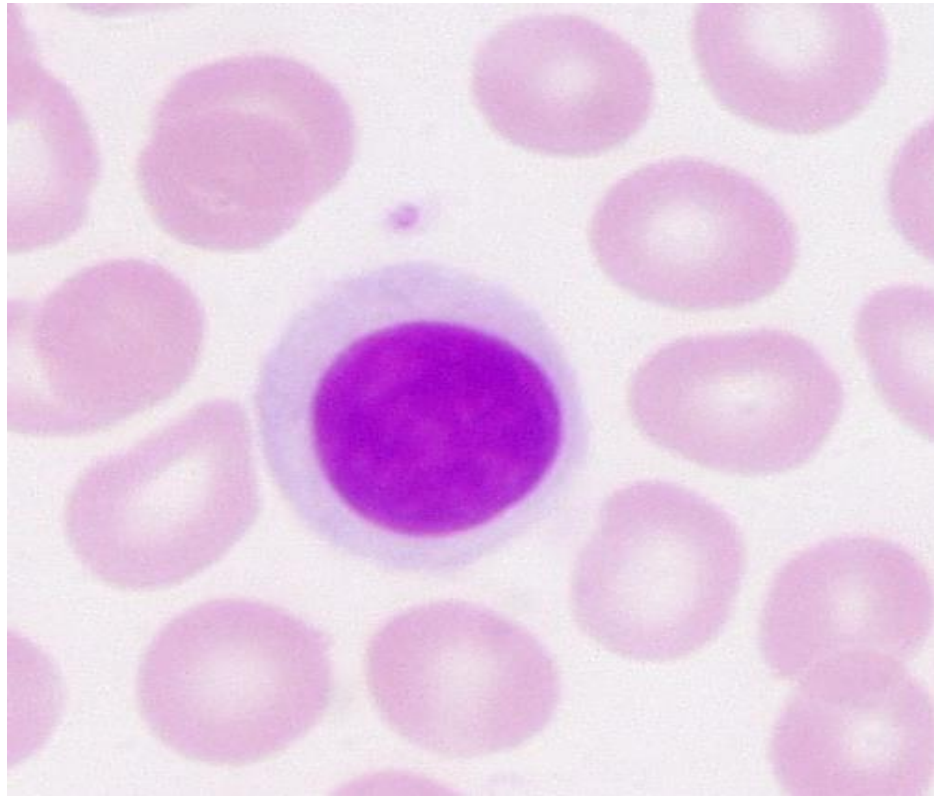
Neutrophil and macrophage lysosomal
enzymes
Oxygen derived free radicals
Nitric Oxide



Sequence of events in leukocyte emigration in inflammation. Laminar blood flow and the presence of red blood cells tend to push leukocytes against the venular wall, increasing their contact with endothelial cells (see the capillary branch at the top with cells entering the venule flow). The leukocytes (1) roll, (2) arrest and adhere to endothelium, (3) transmigrate through an intercellular junction and pierce the basement membrane, and (4) migrate toward chemoattractants released from a source of injury. The roles of selectins, activating agents, and integrins are also indicated.



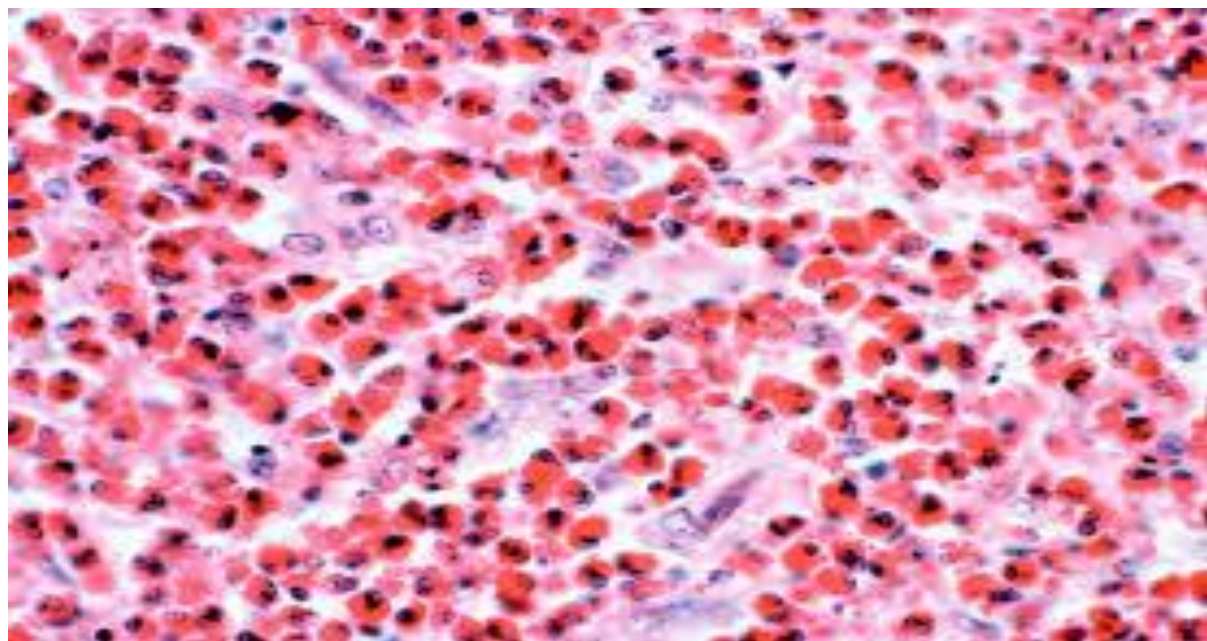




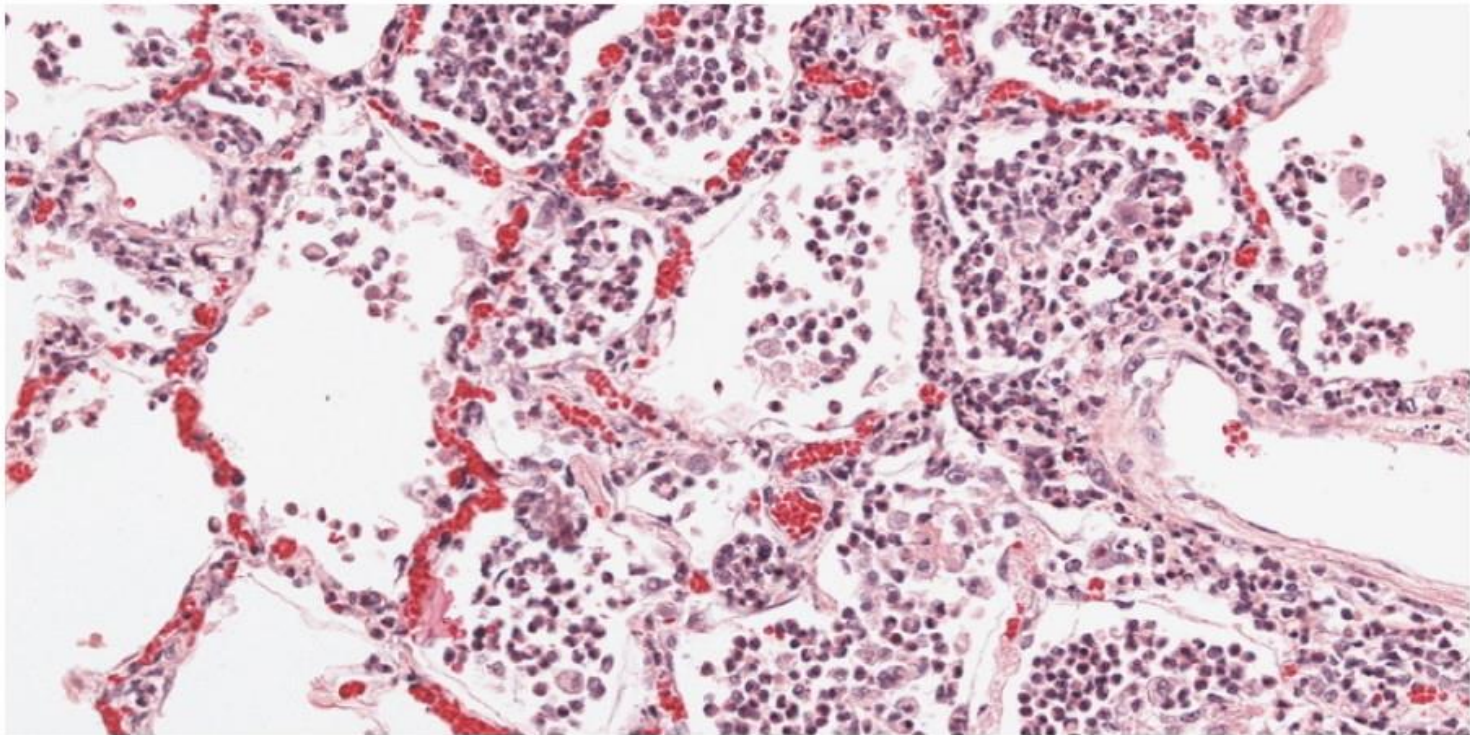


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By
Ed Reschke



Acute inflammation



from Robbins & Cotran's *Pathological Basis of Disease* 8th ed. Kumar V et al. (eds). Saunders Elsevier. Philadelphia (2010)