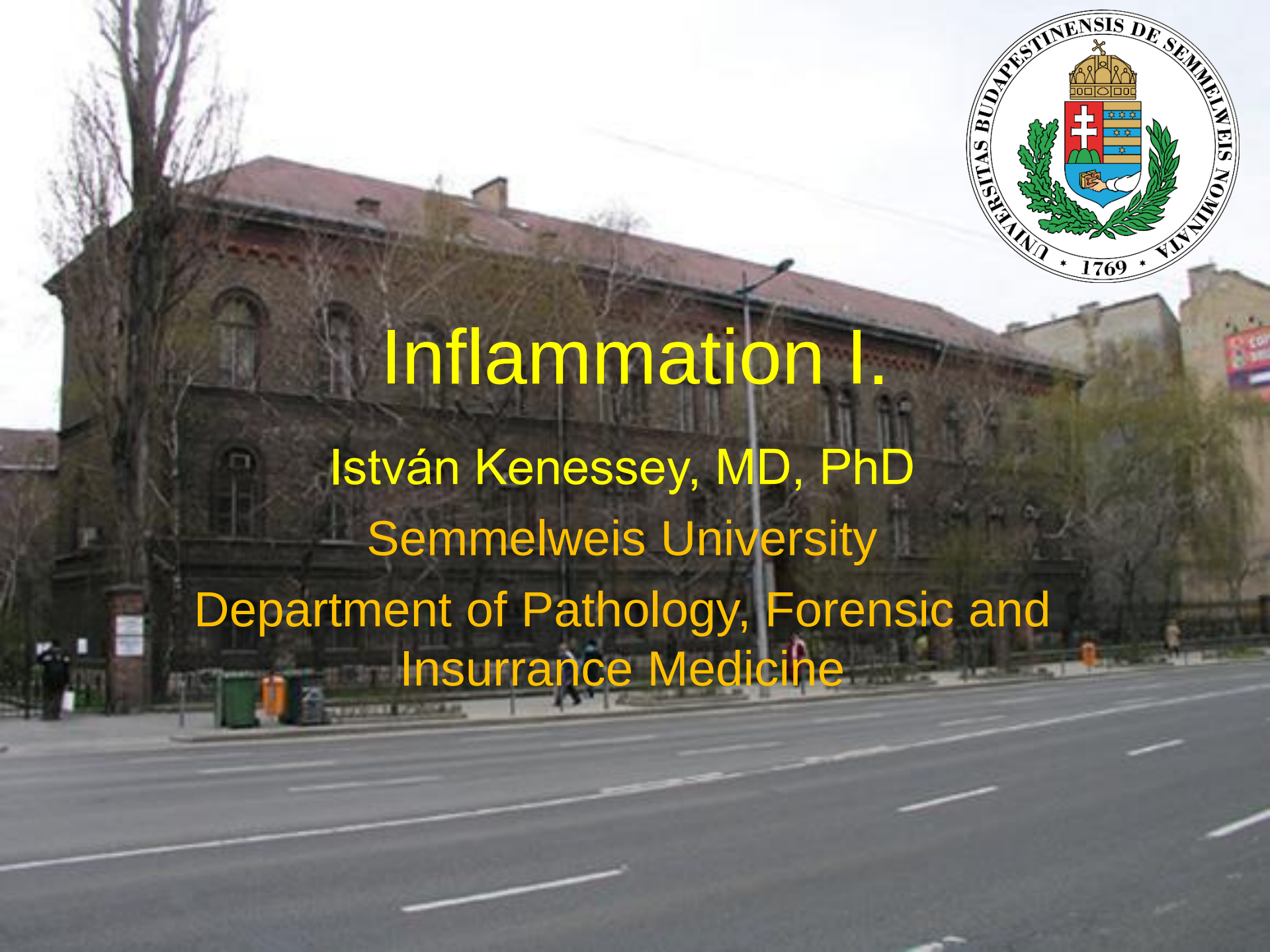
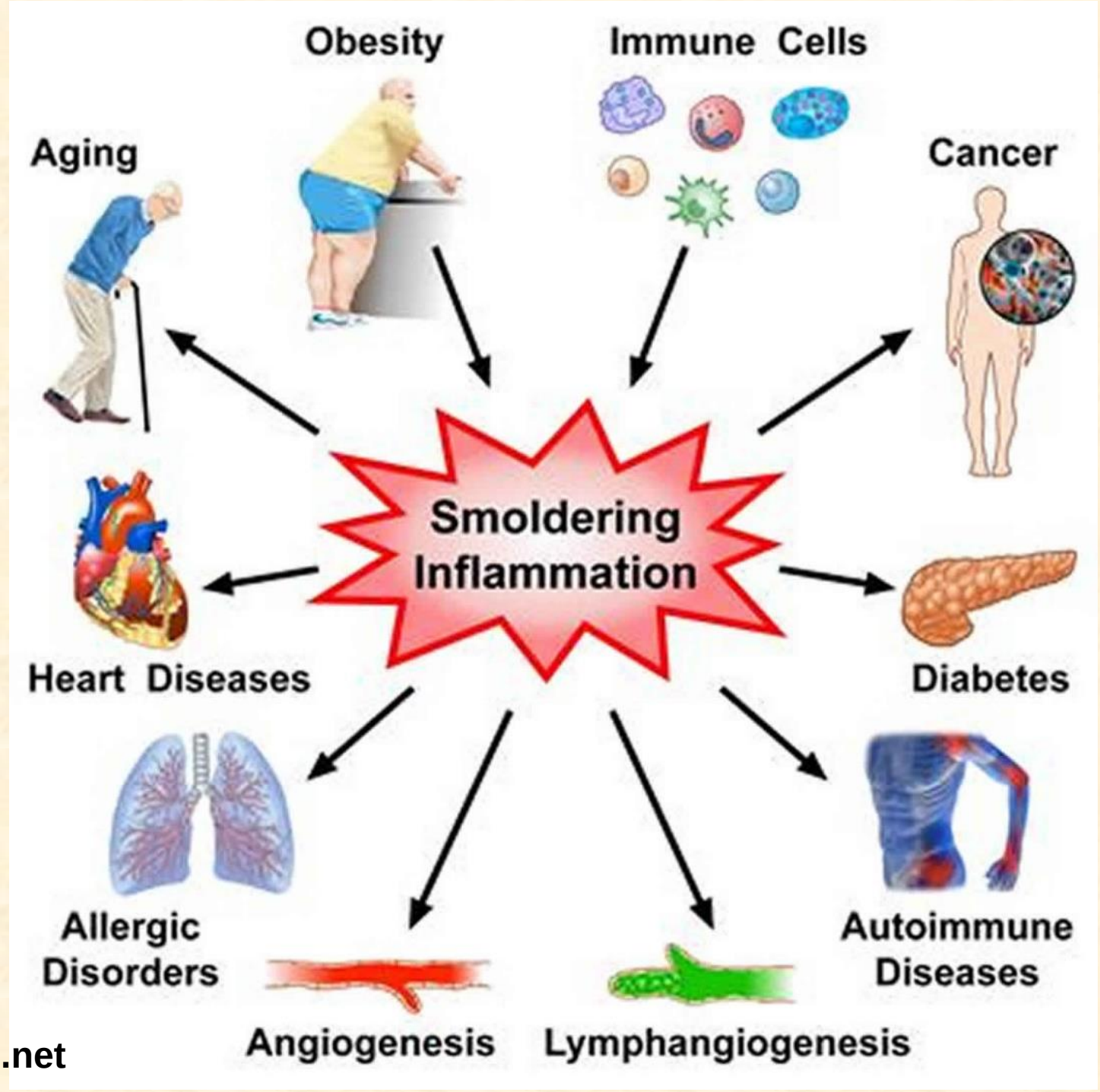


Inflammation I.

István Kenessey, MD, PhD

Semmelweis University

Department of Pathology, Forensic and Insurrance Medicine



Source:
healthjade.net

The Definition of Inflammation

The body's complex response against a harmful stimuli to eliminate inducing agent, and remove damaged cells and tissues.

Categorization by time

- Acute (~1 week) → exsudation
- Subacut
- Chronic → connective tissue

Exogenous causes of inflammation

- Mechanic

  trauma, surgery, foreign body

- Physical

  burn, freezing, radiation

- Chemical

  acids, alkalis, other

- Biological

  bacteria, viruses, fungi, worms, protozoa, insects, exogenous toxins, allergens

Endogenous causes of inflammation

- Activation of proteases / lack of inhibitors

  pancreatitis, DIC, ARDS
- Disorders of blood supply

  ischaemia, infarction
- Immunological processes

  autoimmune reactions, immunecomplexes
- Accumulation of endogenous materials

  kidney stone, bile stone, uraemia

The signs of acute inflammation

- Rubor (redness)
- Calor (heat)
- Dolor (pain)
- Tumor (swelling)
- Functio laesa (loss of function)

Celsus (~B.C. 25 – A.D. 50)

Galenus (A.D. 129 – A.D. 216)

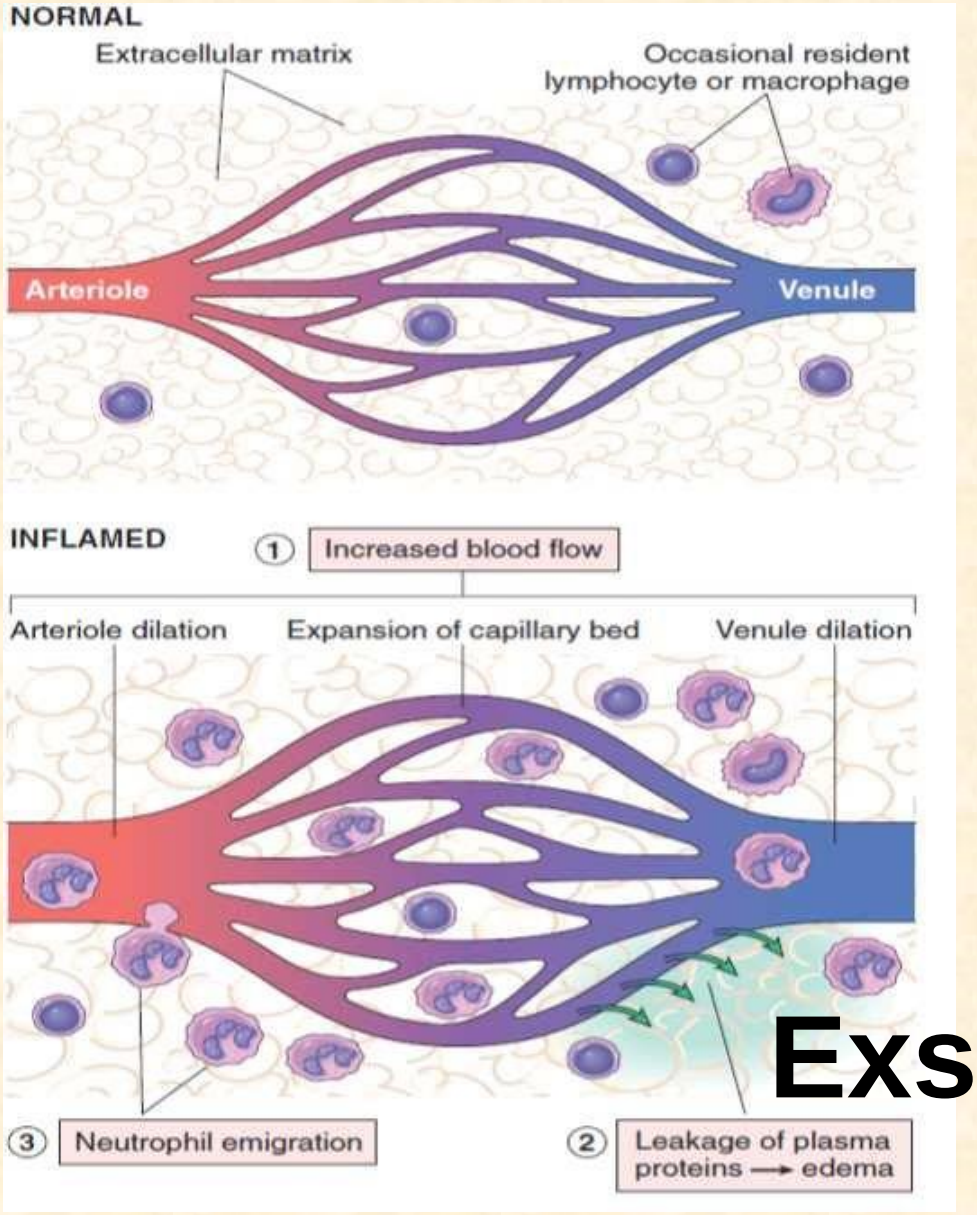
The phases of acute inflammation

Tissue damage

- Vascular phase
- Cellular phase

Reparation

Vascular phase



Exsudate

Source: **Robbins Basic Pathology**

Exsudate vs. transudate

- Both are liquid accumulation (edema) in the interstitial (extravascular) space
- **Transudate**: low protein content

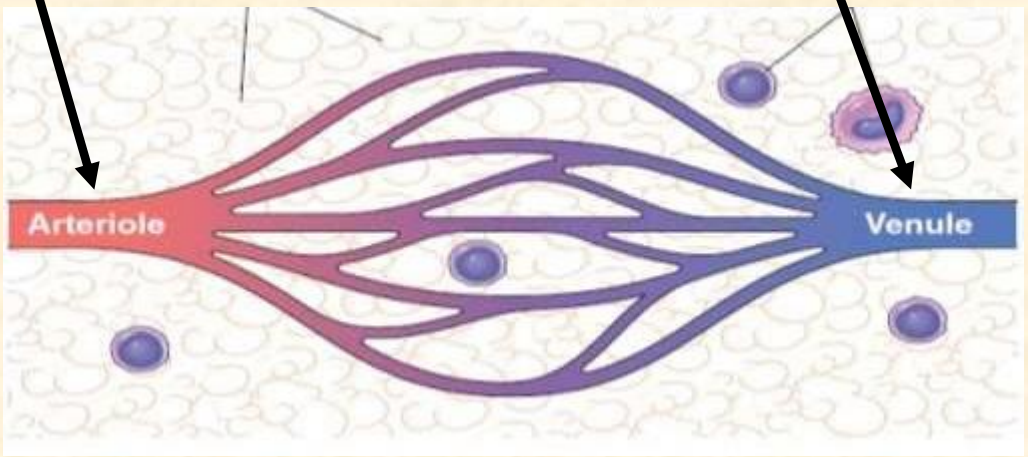
cause: increased hydrostatic pressure (passive hyperaemia)

- **Exsudate**: protein rich

cause: inflammation (active hyperaemia + increased permeability)

Rivalta test:

Denaturation due to acetic acid

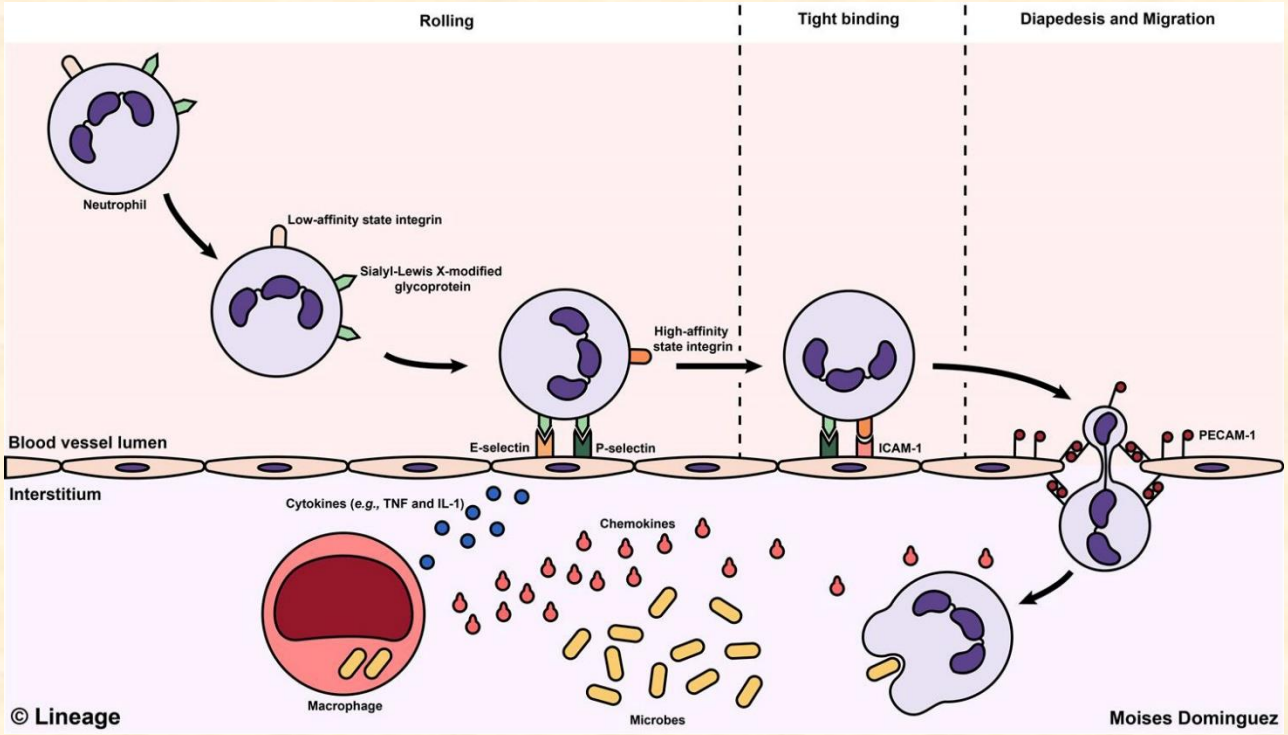


Vascular phase – increased permeability

- Contraction of endothelial cells (histamine, bradykinin, TNF, IL-1)
- Alteration of cell adhesion structures
- Direct endothelial damage
- Leukocyte-dependent endothelial damage
- Increased transcytosis (vesicular transport)
- Leaking from the new vessels

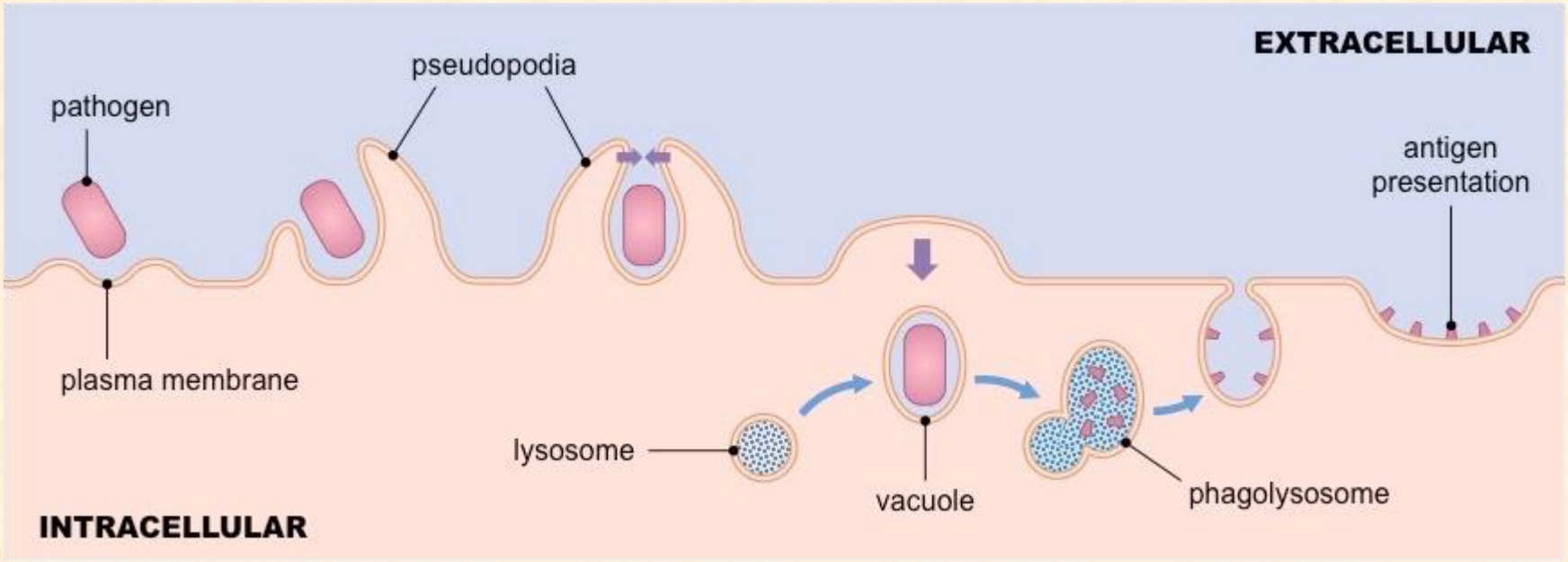
Cellular phase – Migration of leukocytes

- Margination (due to hemodynamics)
- Rolling (selectins)
- Docking (adhesion molecules – integrins)
- Migration (PECAM-1 – CD31)
- Chemotaxis-directed (bacterial proteins, cytokines, complement, arachidonic acid lipoxygenase metabolites)



Cellular phase – Activation of leukocytes

- Phagocytosis
- Lysosomal digestion (degradation)
- Synthesis and release of mediators (degranulation)



Source: **BioNinja**

The inflammatory cells

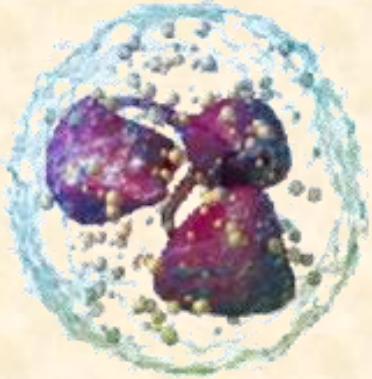
T cells

B cells

Eosinophils

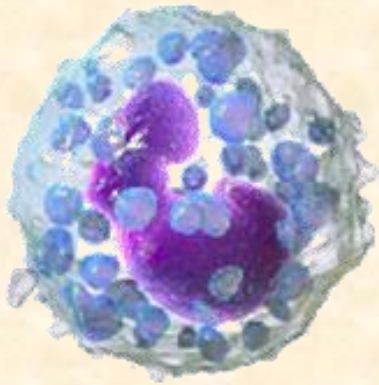
Polymorphonuclear cells (granulocytes)

Neutrophils



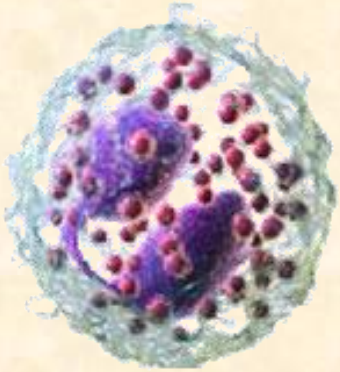
40-70% of all leukocytes
„Innate” immune system
Chemotexis-directed
Phagocytosis
Lysosomal free agents
Degranulation

Basophils



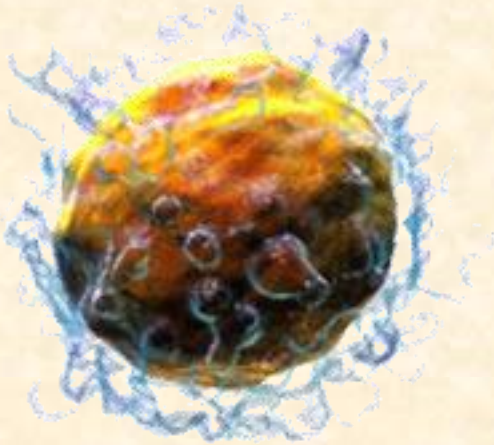
Allergic reactions
- Anaphylaxia
- Asthma
- Atopic dermatitis

Eosinophils

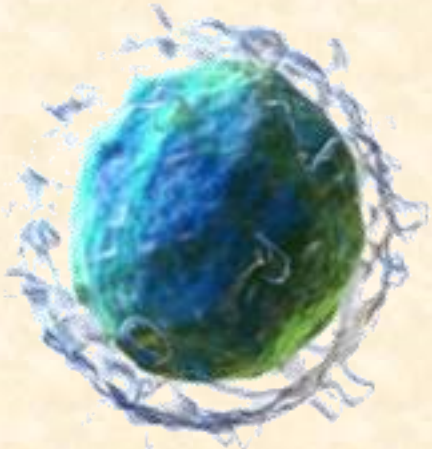


Anti-parasite response

Lymphocytes



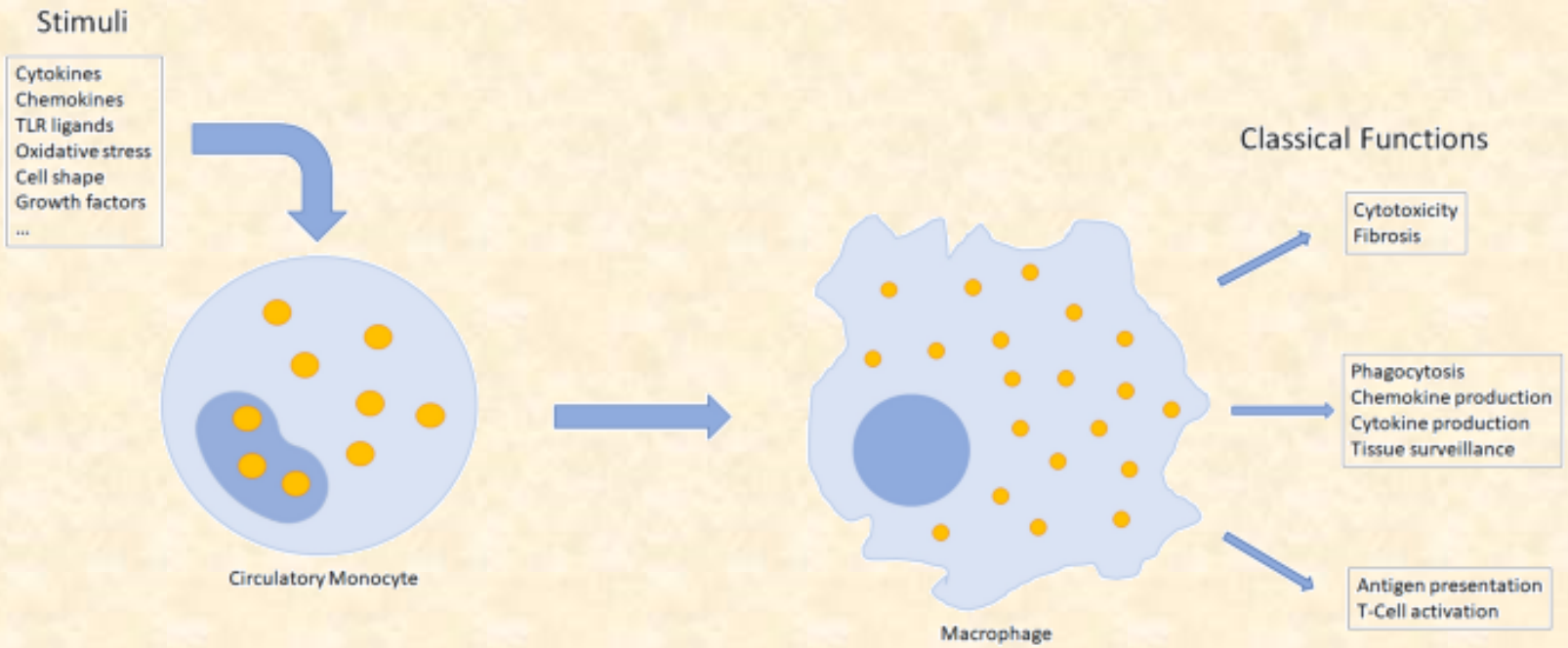
T cells



B cells

Specific immune response
Cellular and humoral immunity

Monocyte - macrophage



Nomenclature

„-itis” ending

E.g.

- Colon – colitis
- Trachea – tracheitis
- Pancreas – pancreatitis
- Hepar – hepatitis

BUT EXCEPTIONS!

- Pneumonia
- Oophoritis
- Typhlitis
- Colpitis
- etc..

Types of acute inflammation – categorization by exsudate

- Serous – inflammatio serosa
- Fibrinous – inflammatio fibrinosa
- Purulent (pustulous) – inflammatio purulenta
- Hemorrhagic– inflammatio haemorrhagica
- Gangrenous – inflammatio gangraenosa *seu* ichorosa

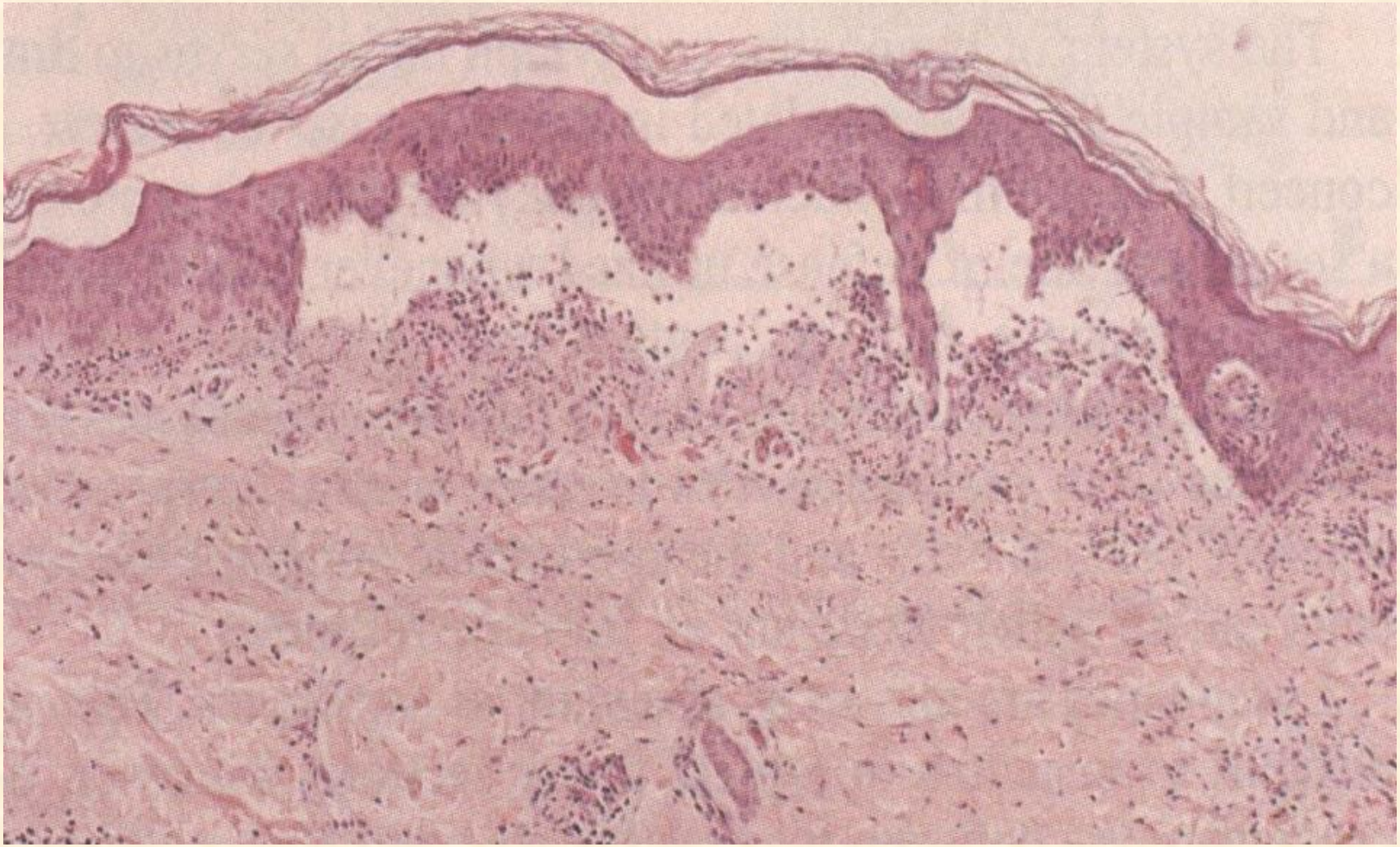
Serous inflammation

- The most simple types
- Relatively low amount of protein
- Dilute, watery, liquid

Examples:

- Burn / frost injury
- Viral infections (e.g. common cold, HSV)
- Allergy

Serous inflammation of the skin



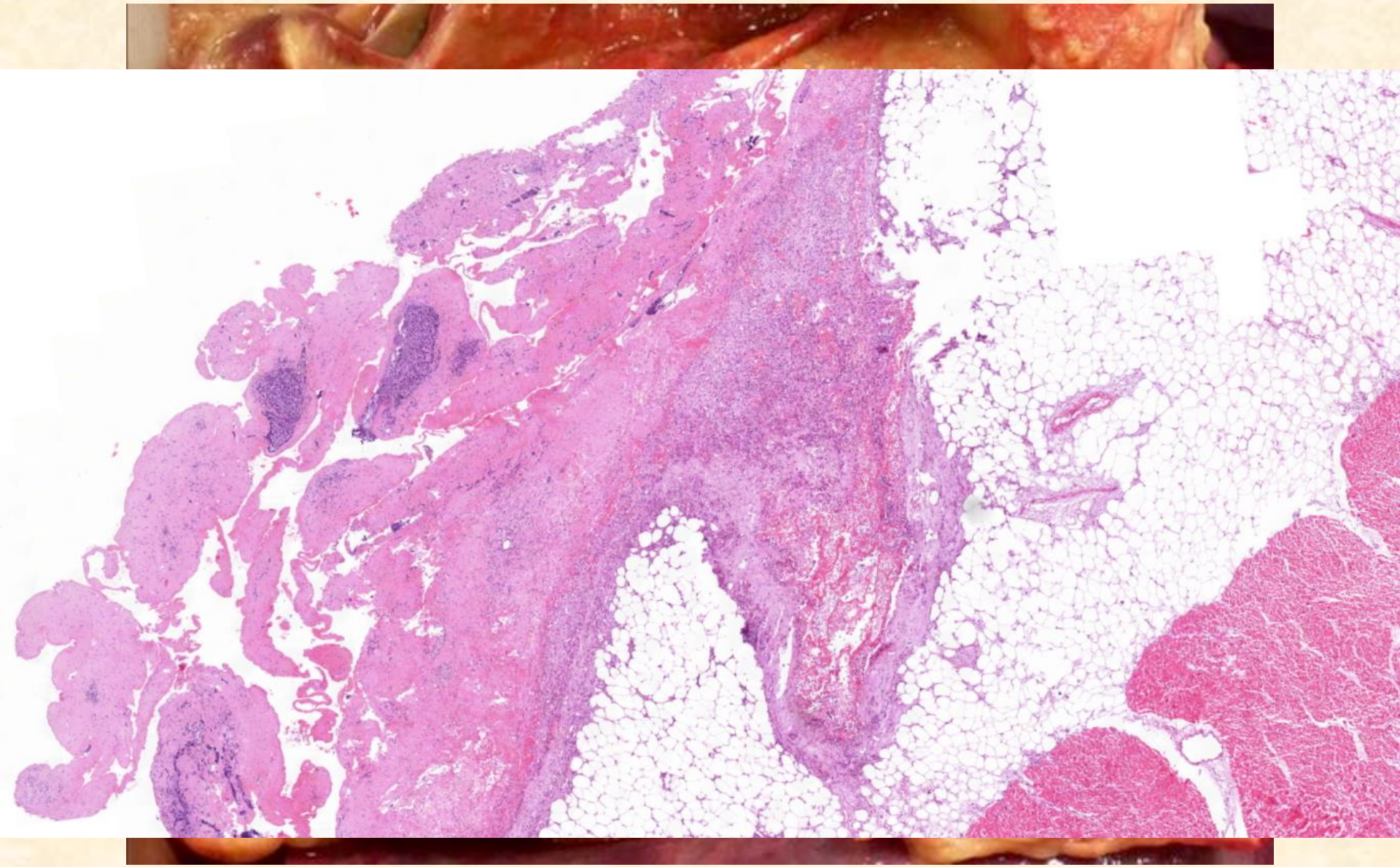
Fibrinous inflammation

- Due to the increased permeability of the vessels fibrinogen is also transferred
- Macroscopically greyish membrane
- Microscopically eosinophil fibrin mesh

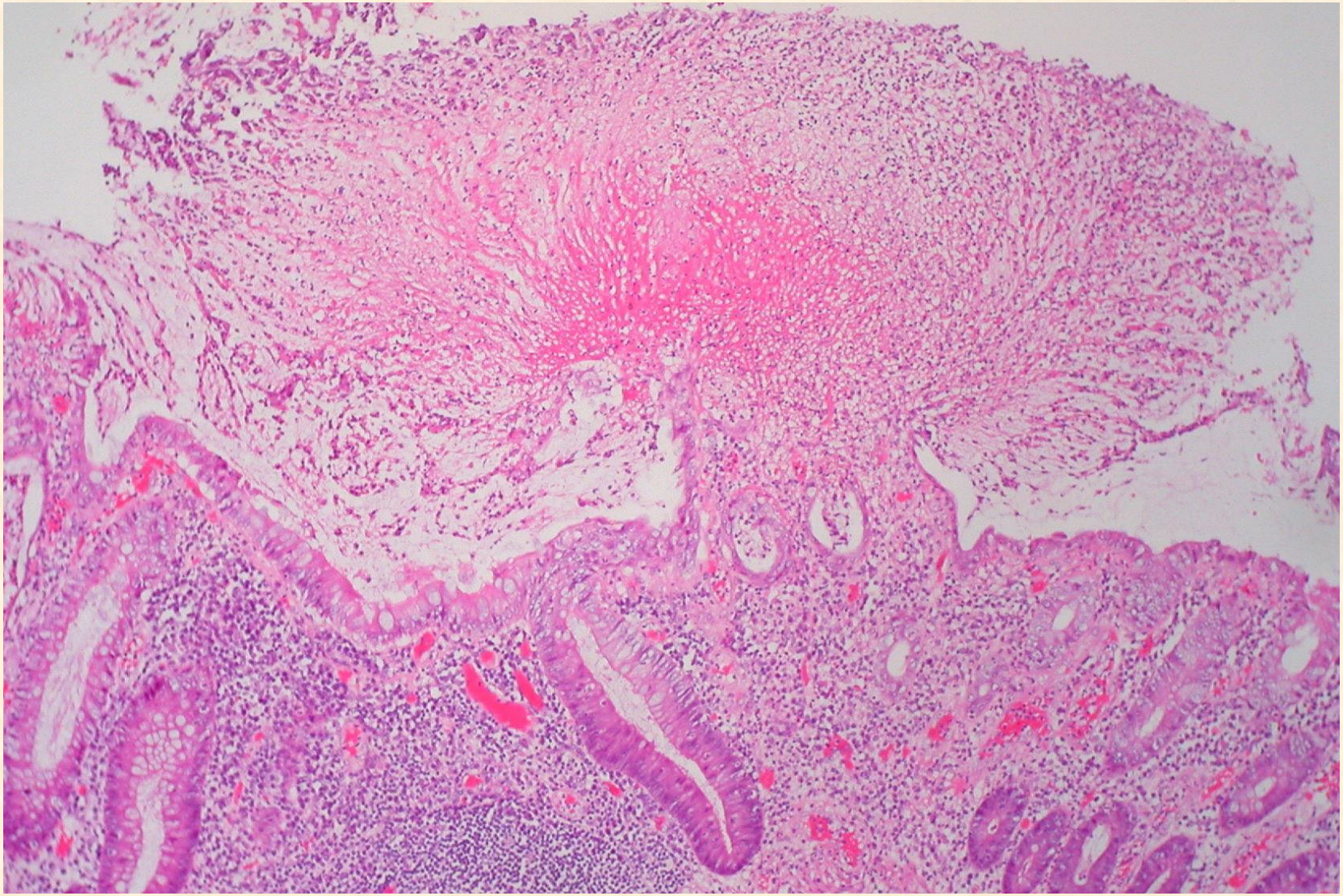
Examples:

- Inflammation of serous membranes
- Pseudomembranosous colitis
- Diphteria

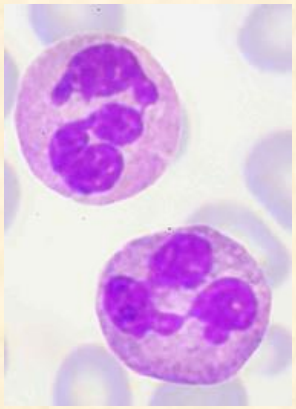
Fibrinous pericarditis



Pseudomembranosus colitis



Purulent (suppurative) inflammation

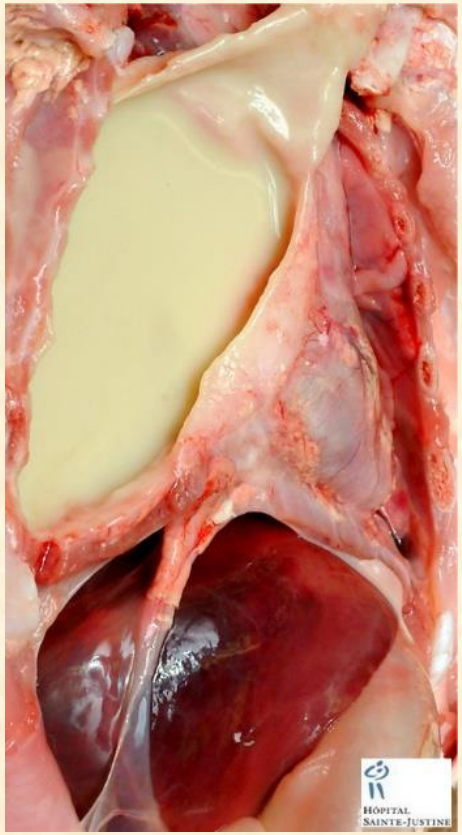


- Pus: neutrophil granulocytes + dead tissue
- Pyogenic bacteria (e.g. Staphilococcus)
- Yellowish, concentrated liquid

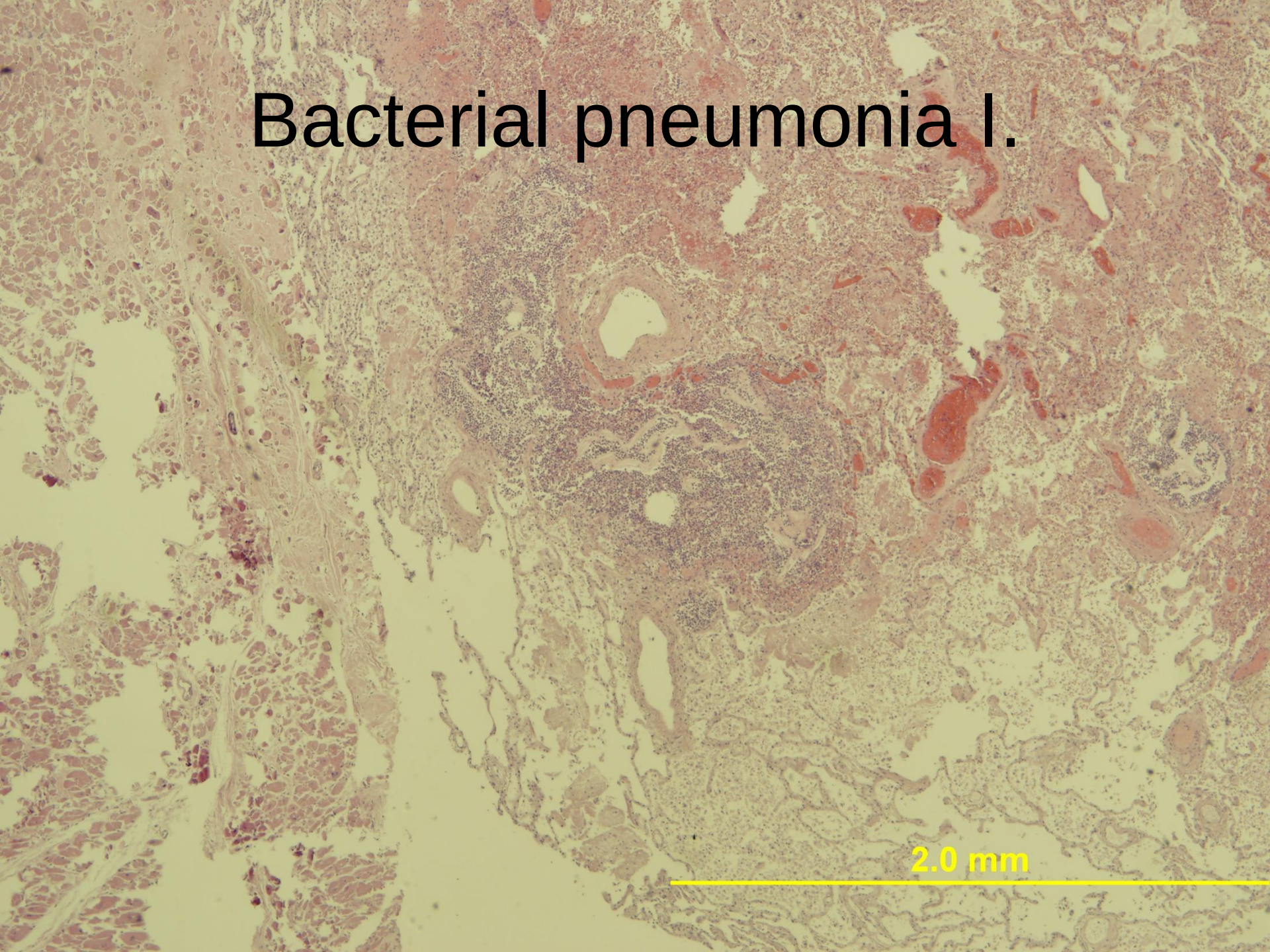
Special forms:

- Abscess
- Phlegmon
- Pustule
- Folliculitis
- Furuncle
- Carbuncle

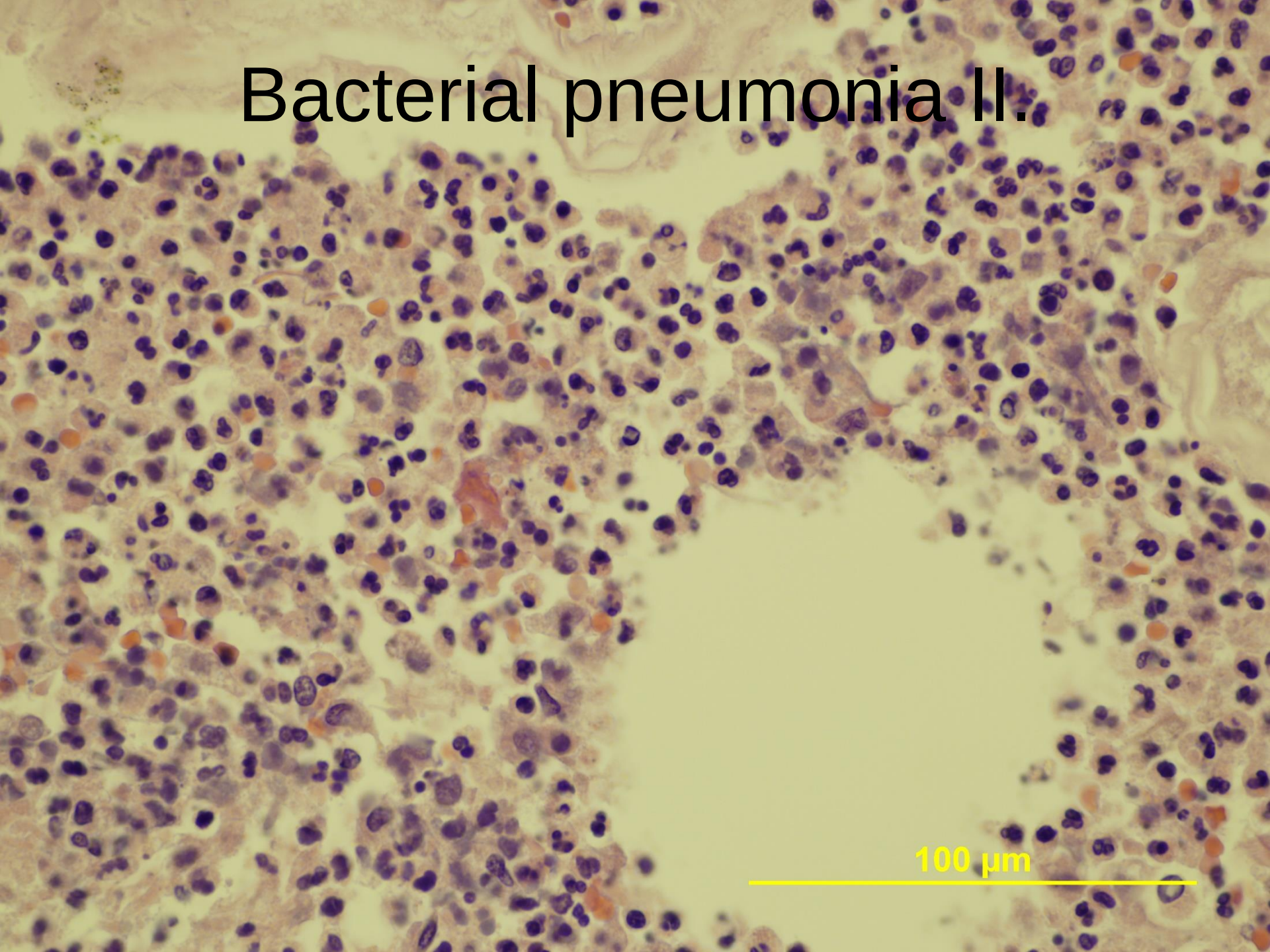
- Empyema
- Pyometra
- Pyonephros
- Pyoarthros
- Pyocephalus
- stb…



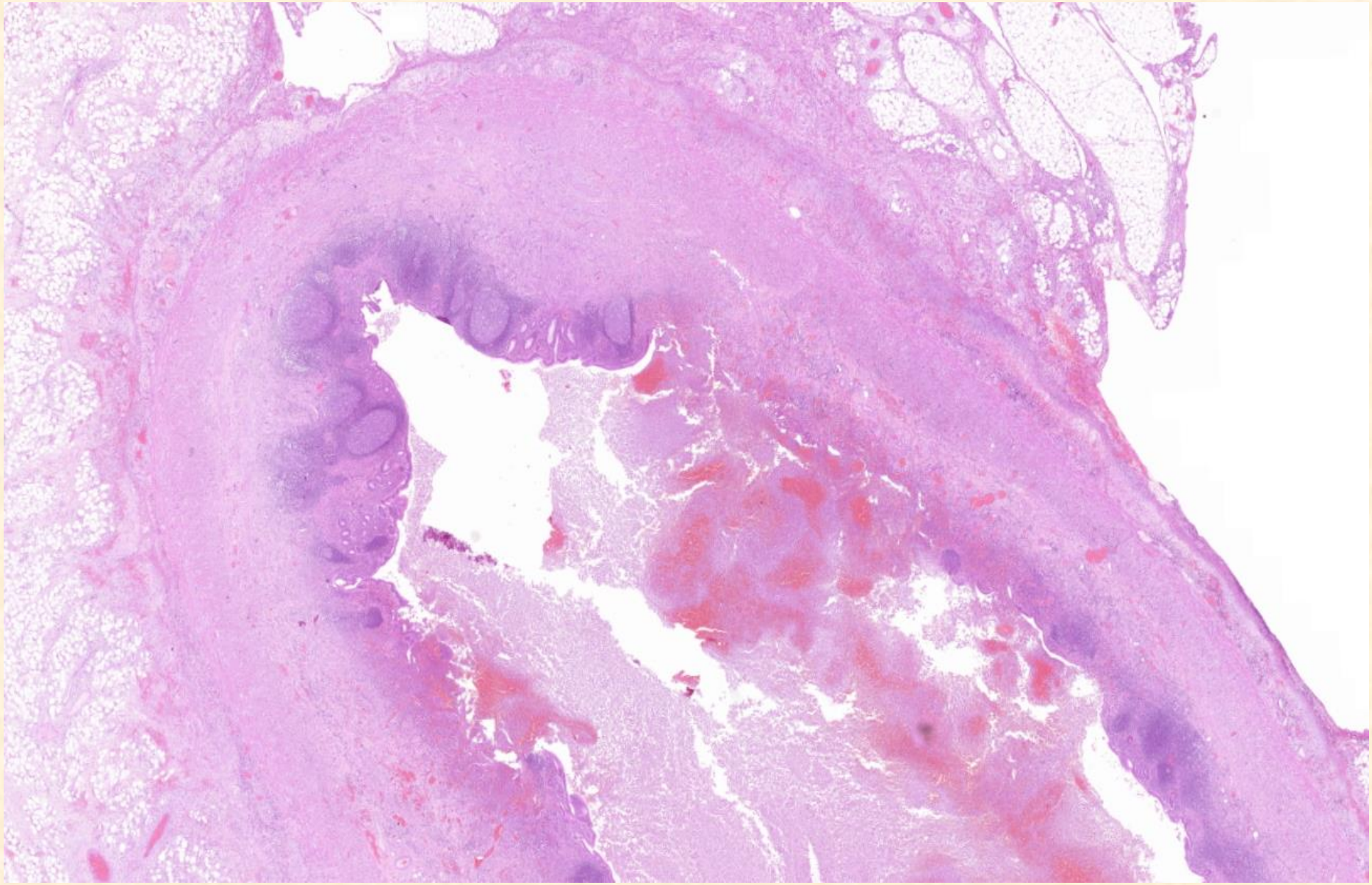
Bacterial pneumonia I.



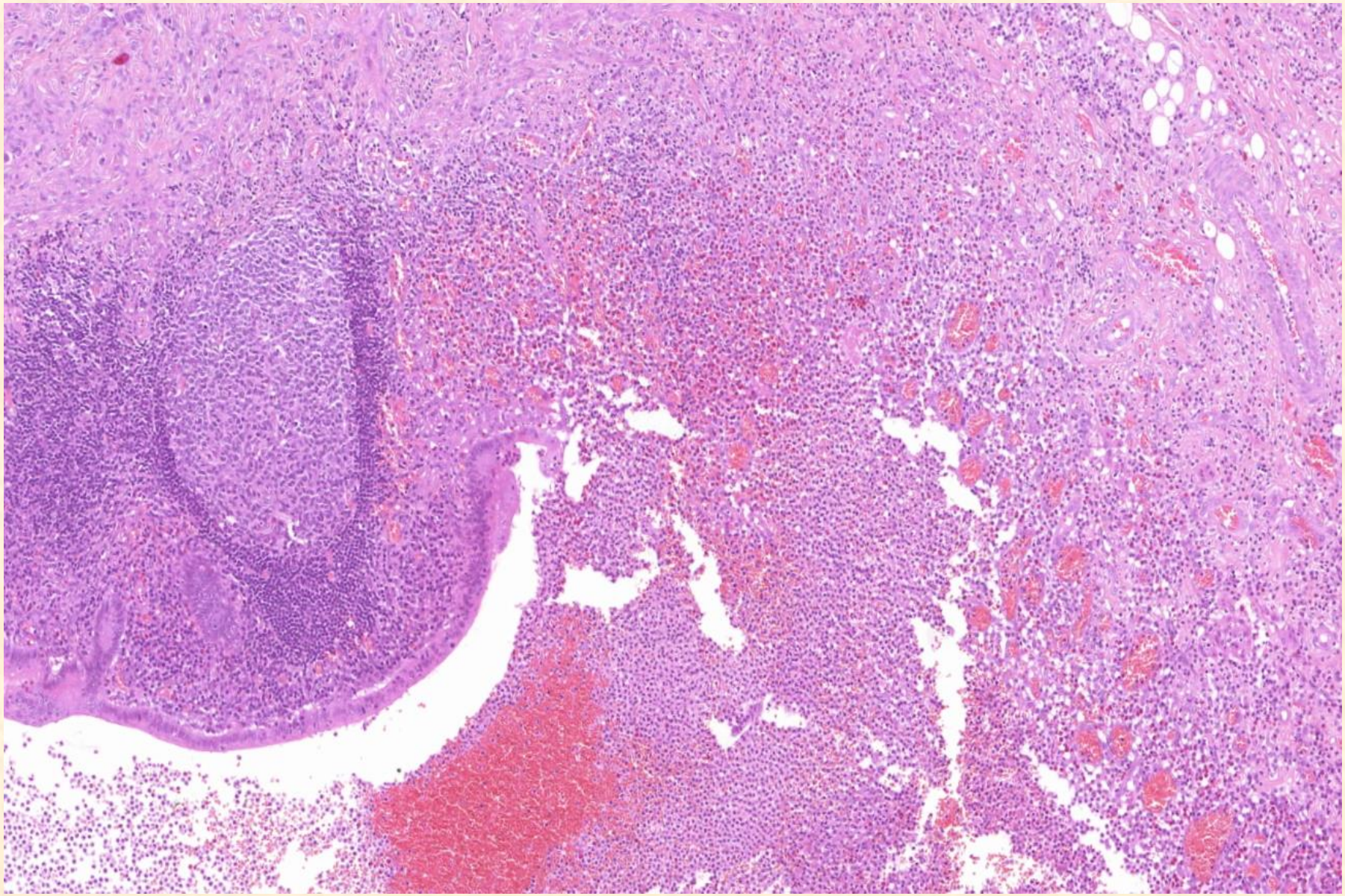
Bacterial pneumonia II.



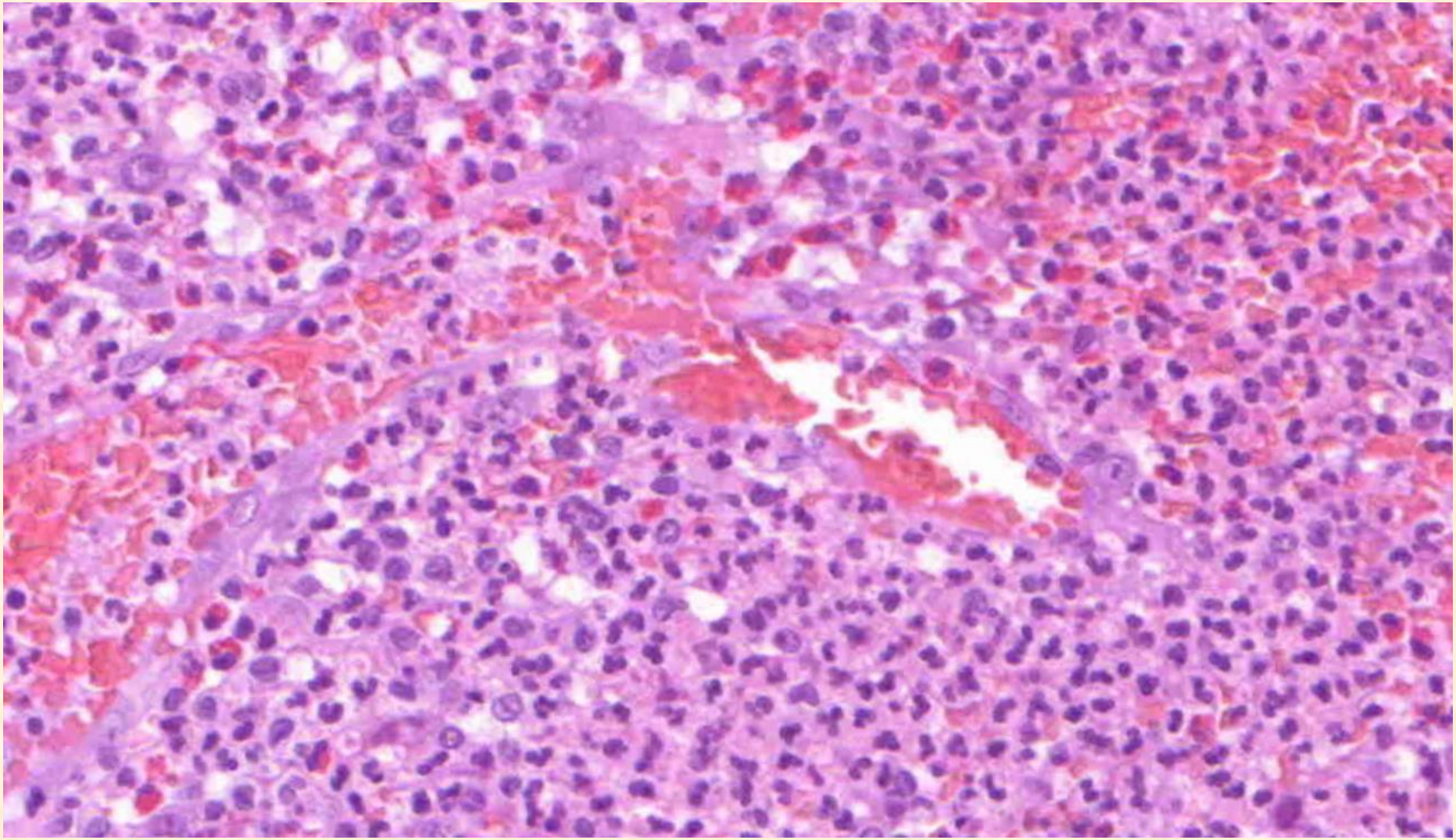
Acute appendicitis I.



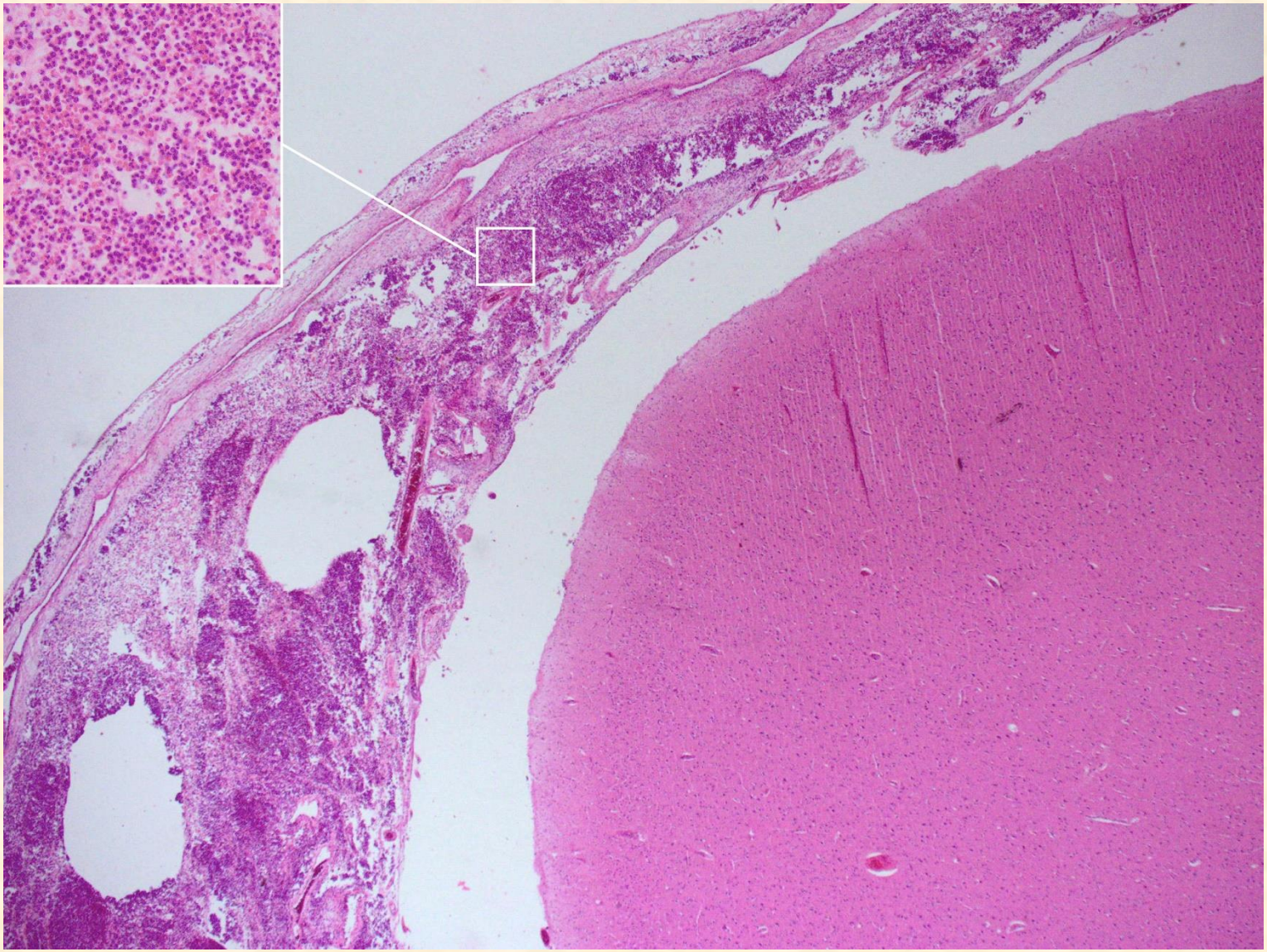
Acute appendicitis II.



Acute appendicitis III.



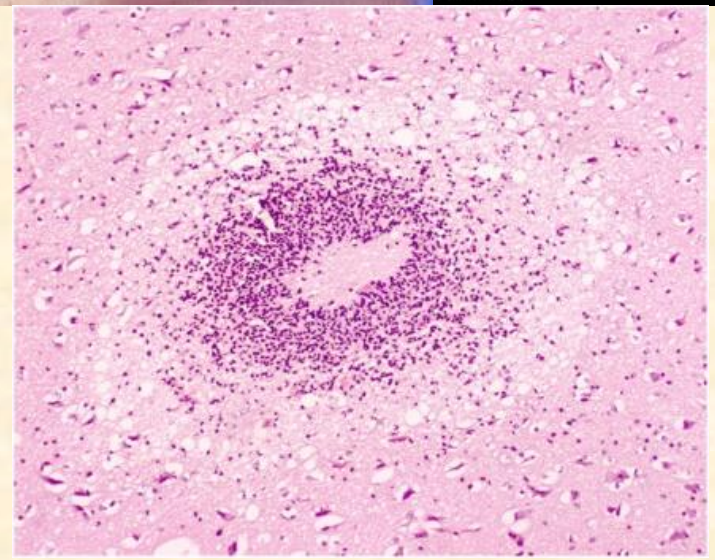
Purulent meningititis




www.husammedical.com

Pyelonephritis apostematosa

Brain abscess



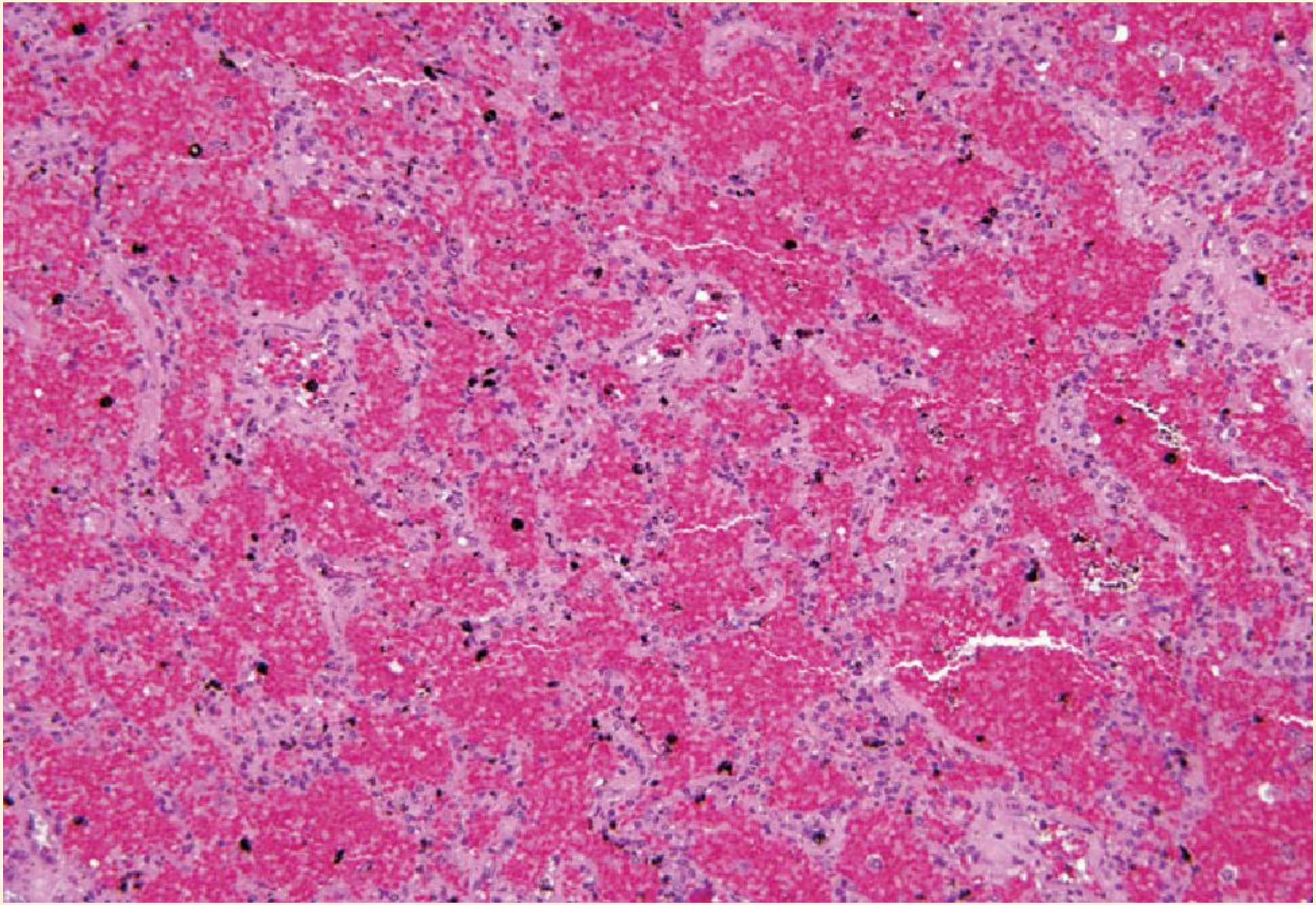
Hemorrhagic inflammation

- Damage of the small vessel's wall (toxin)
- Macroscopically massive blleding
- Microscopically erithrocytes

Example:

- Bacterial: pestis, anthrax
- Viral: smallpox, influenza
- Hemorrhagic pancreatitis

Hemorrhagic inflammation in lung



Source: **Tauenberger et al.**

Gangrenous inflammation

- The body is unable for effective defense
- Cause might be general weakness
- Atherosclerosis, diabetes mellitus

Examples:

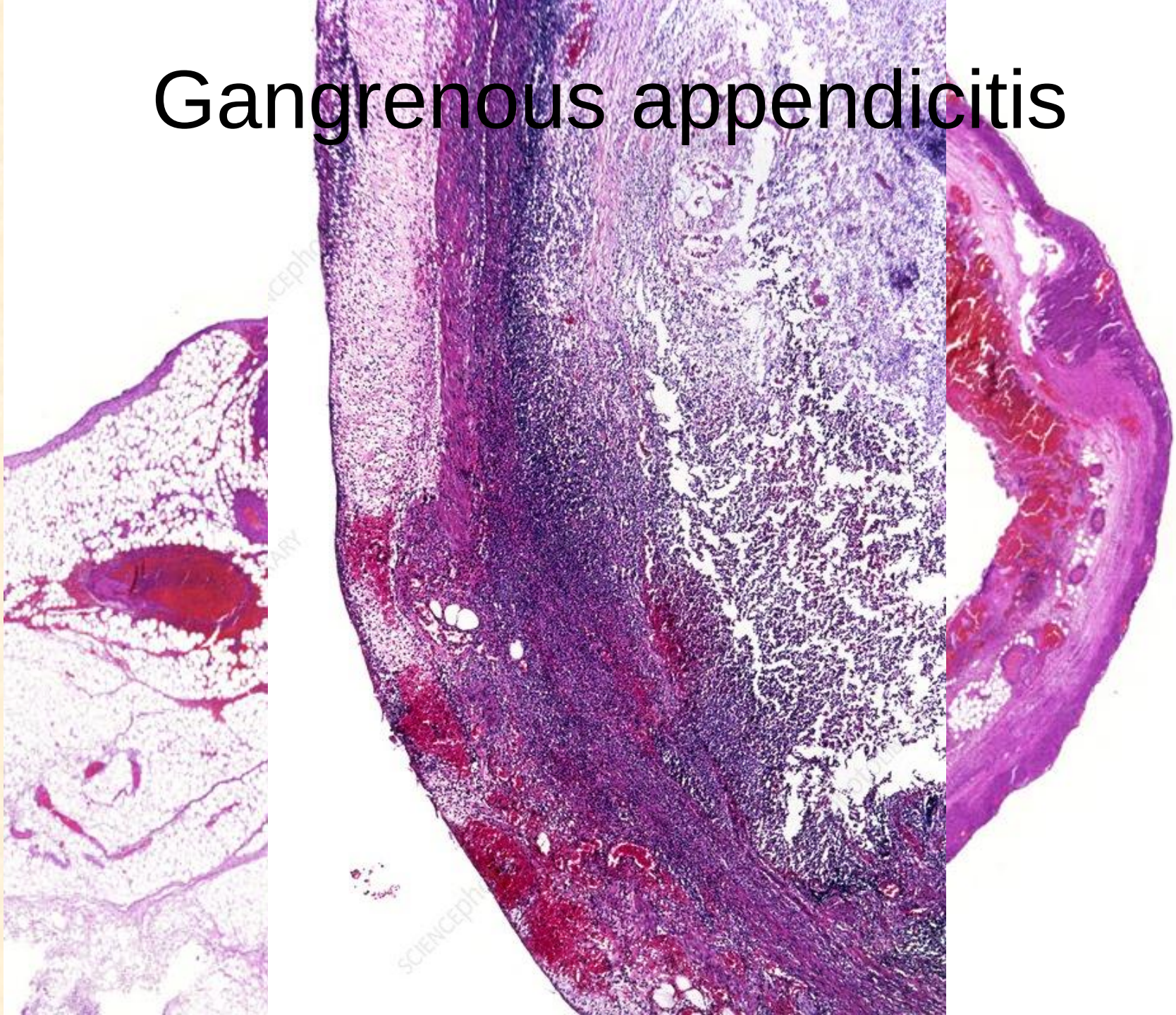
- Diabetic feet
- Gangraenous appendicitis
- Noma

Formái:

- Dry gangrene (gangraena sicca)
- Wet gangrene (gangraena humida)
- Gas gangrene (gangraena emphysematosa)

Diabetic feet

Gangrenous appendicitis



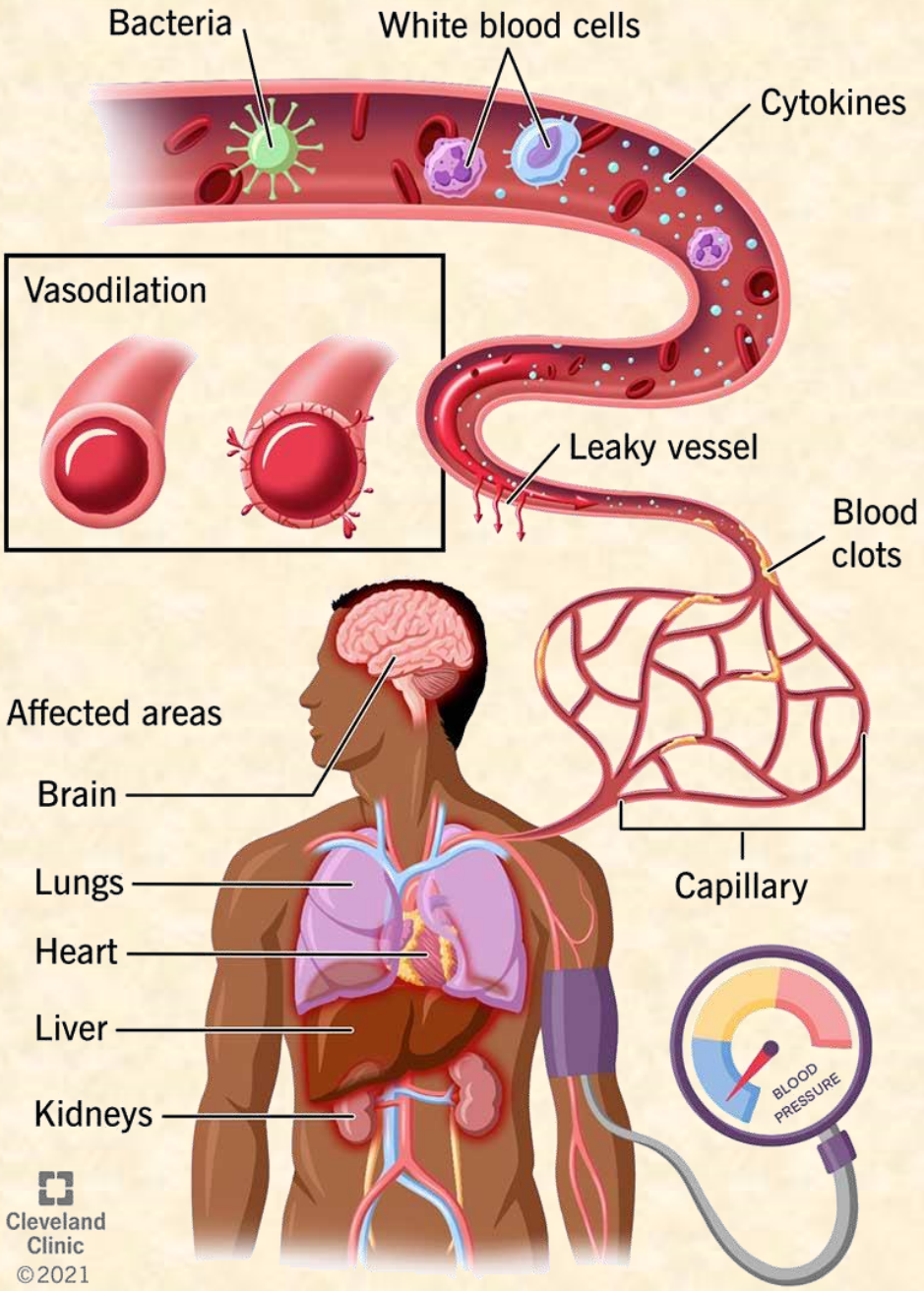
Sepsis

Bacteremia + systemic reaction

- High amount of pathogens in the blood flow
- Cytokine response *(TNF, IL-12, IL-1)*
- Fever
- Higher level of acute phase protein in the blood *(CRP, procalcitonin)*
- Leukocytosis
- Septic shock

<u>Clinical diagnosis</u>: inflammatory focus + systemic signs

Septic Shock



Subacute inflammation

- Transition between acute and chronic
- Weeks, 1-2 months
- Decreased exsudate
- Decreased edema
- Mixed cellular infiltration: eosinophil, lymphocyte
- Reparation processes

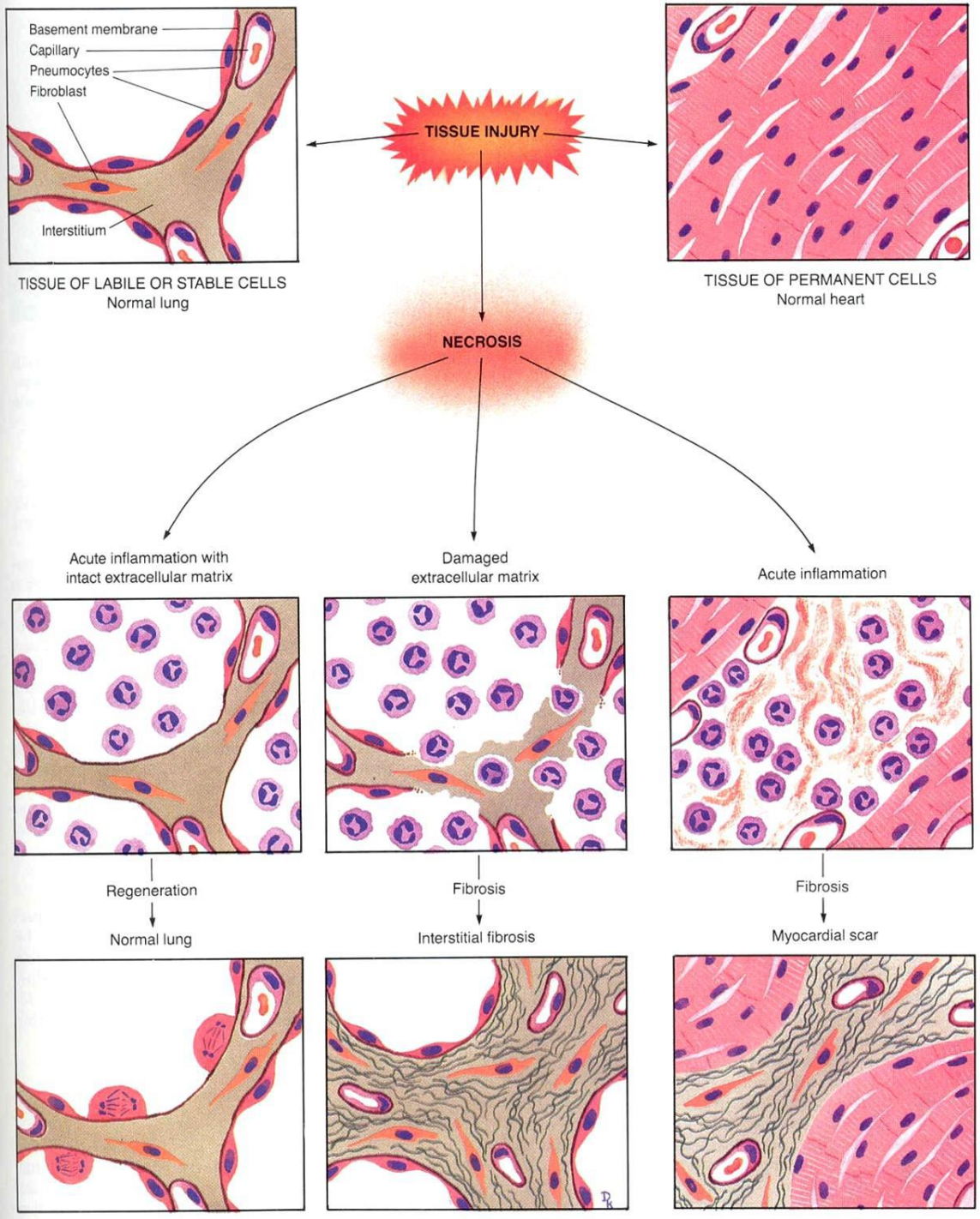
Regeneration, wound healing

Replacement of damaged tissue

- by identical cells
- by connective tissue (fibrosis, scars)

Proliferatory capacity:

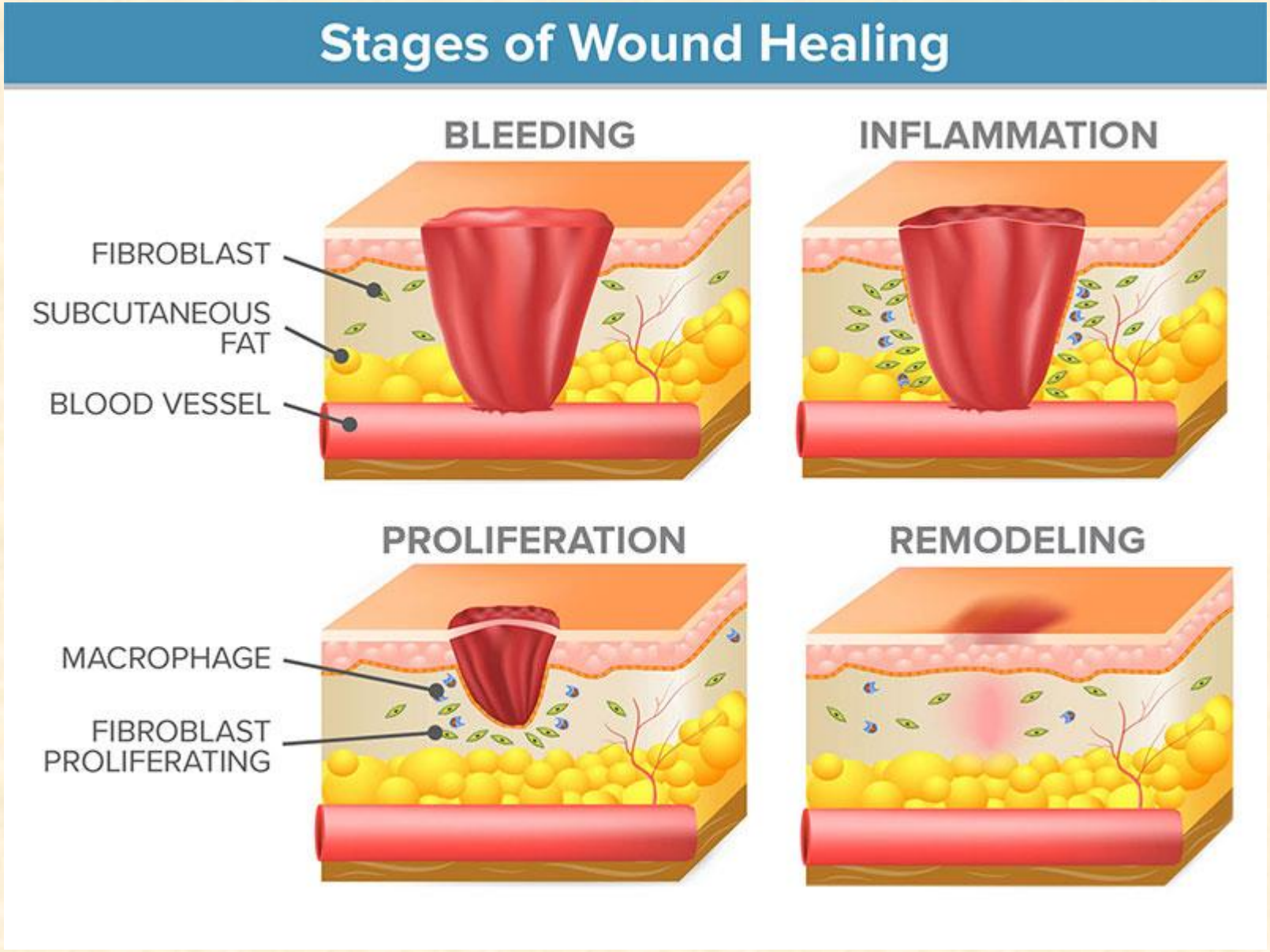
- Constantly dividing tissues
  (bone marrow, epidermis, mucosa)
- Stable tissues
  (connective tissue, endothel, smooth muscle, liver, kidney, pancreas)
- Permanent tissues
  (neural tissue, heart muscle)


Basement membrane
Capillary
Pneumocytes
Fibroblast
Interstitium
TISSUE INJURY
TISSUE OF LABILE OR STABLE CELLS
Normal lung
TISSUE OF PERMANENT CELLS
Normal heart
NECROSIS
Acute inflammation with
intact extracellular matrix
Damaged
extracellular matrix
Acute inflammation
Regeneration
Fibrosis
Fibrosis
Normal lung
Interstitial fibrosis
Myocardial scar

Steps of regeneration

Starts within 24 hours

- Angiogenesis
  (from existing vessels, and inducing bone marrow stem cells)
- Fibroblastic migration and proliferation
- ECM deposition – scar formation
  growth factors
- Maturation and remodeling of connective tissue (synthesis – degradation)



Source: **Wasatch Photonics**

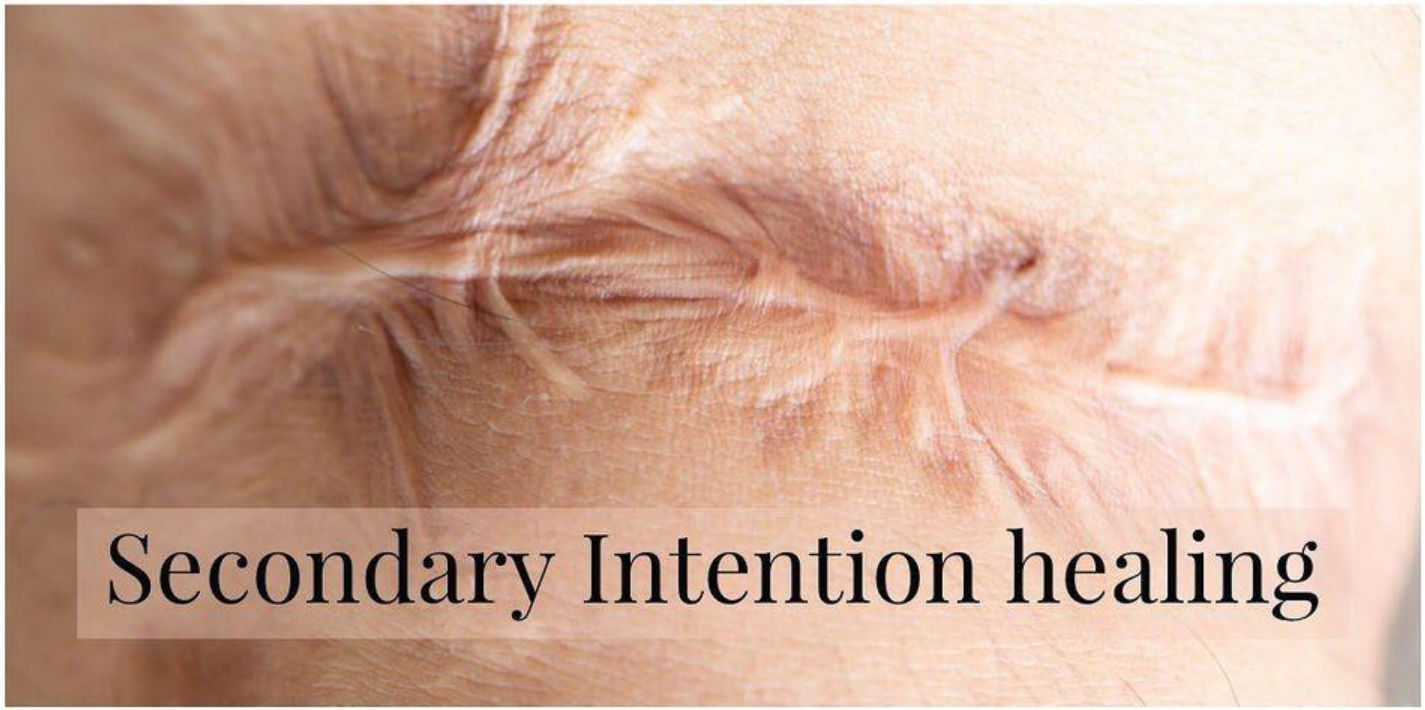
Primary wound healing

- Clear, non-infected surgical incision
- Minimal amount of scar tissue
- 0-24: fibrinous secretion + neutrophils
- Days: granulation tissue:
  collagen + vessels + macrophages
- Weeks: epithelial proliferation and scar

Secondary wound healing

- Extensive tissue damaged, irregular or infected wound margins
- Fibrinous secretion
- More expansive inflammatory reaction
- Larger amount of granulation tissue
- Scar tissue + contraction


Primary Intention healing


Secondary Intention healing

Thank you for your attention