



INFLAMMATION

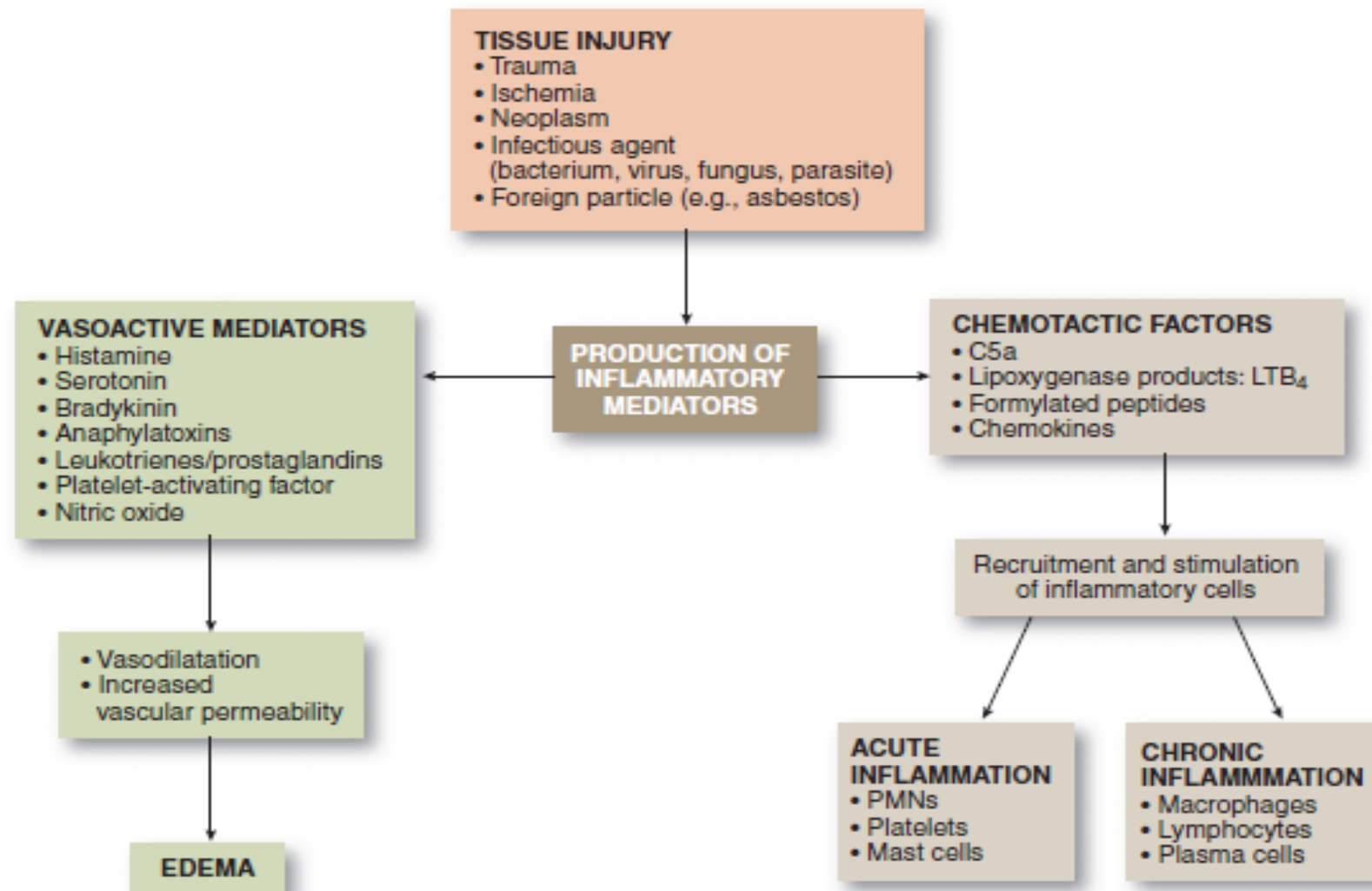
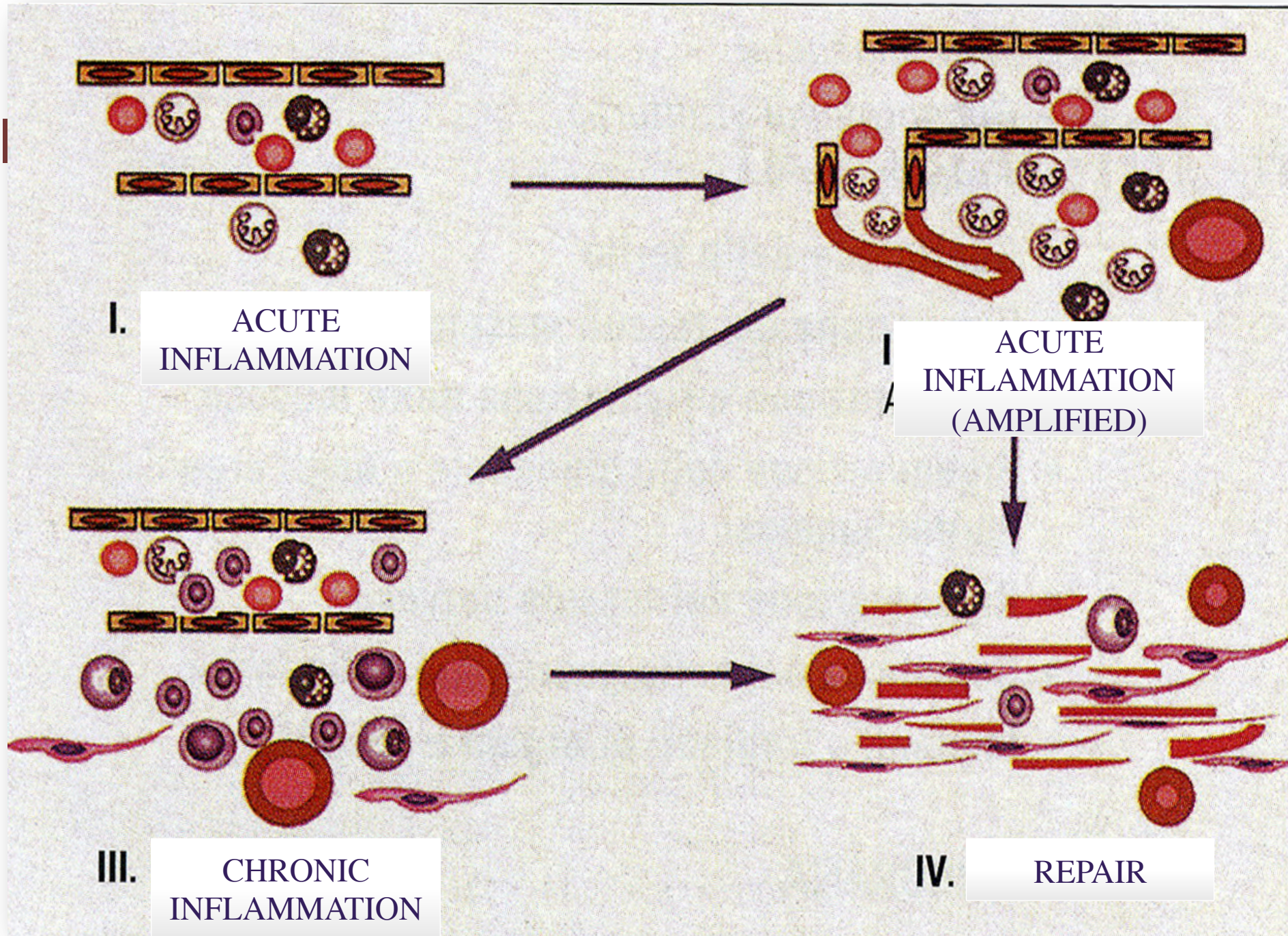
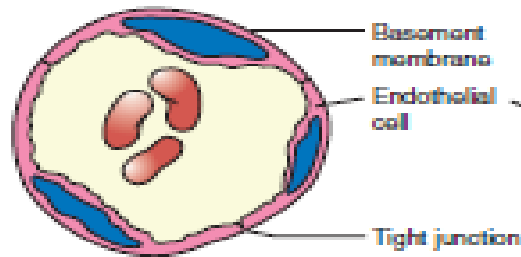


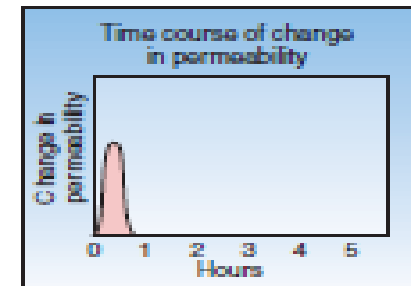
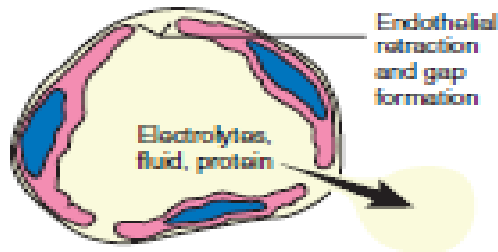
FIGURE 2-5. Mediators of the inflammatory response. Tissue injury stimulates the production of inflammatory mediators in plasma, which are released in the circulation. Additional factors are generated by tissue cells and inflammatory cells. These vasoactive and chemotactic mediators promote edema and recruit inflammatory cells to the site of injury. LTB₄, leukotriene B₄; PMNs, polymorphonuclear neutrophils.



A NORMAL VENULE



B VASOACTIVE MEDIATOR-INDUCED INJURY



C DIRECT INJURY TO ENDOTHELIUM

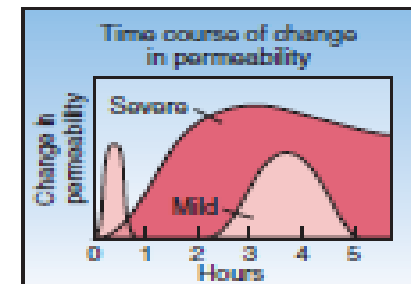
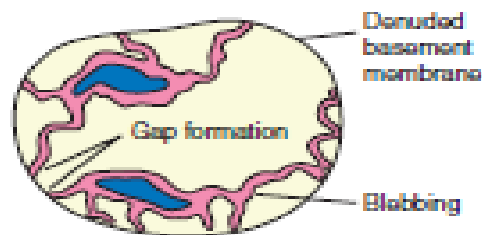


FIGURE 2-3. Responses of the microvasculature to Injury. **A.** The wall of the normal venule is sealed by tight junctions between adjacent endothelial cells. **B.** During mild vasoactive mediator-induced injury, the endothelial cells separate and permit the passage of the fluid constituents of the blood. **C.** With severe direct injury, the endothelial cells form blebs (*b*) and separate from the underlying basement membrane. Areas of denuded basement membrane (arrows) allow a prolonged escape of fluid elements from the microvasculature.

Clinico-pathologic correlations

CALOR

DOLOR

RUBOR

TUMOR

FUNCTIO
LAESA

FEBBRE

ANORESSIA

ASTENIA

LEUCOCITOSI

AUMENTO
DELLA VES

INFLAMMATION

Inflammations aims at:

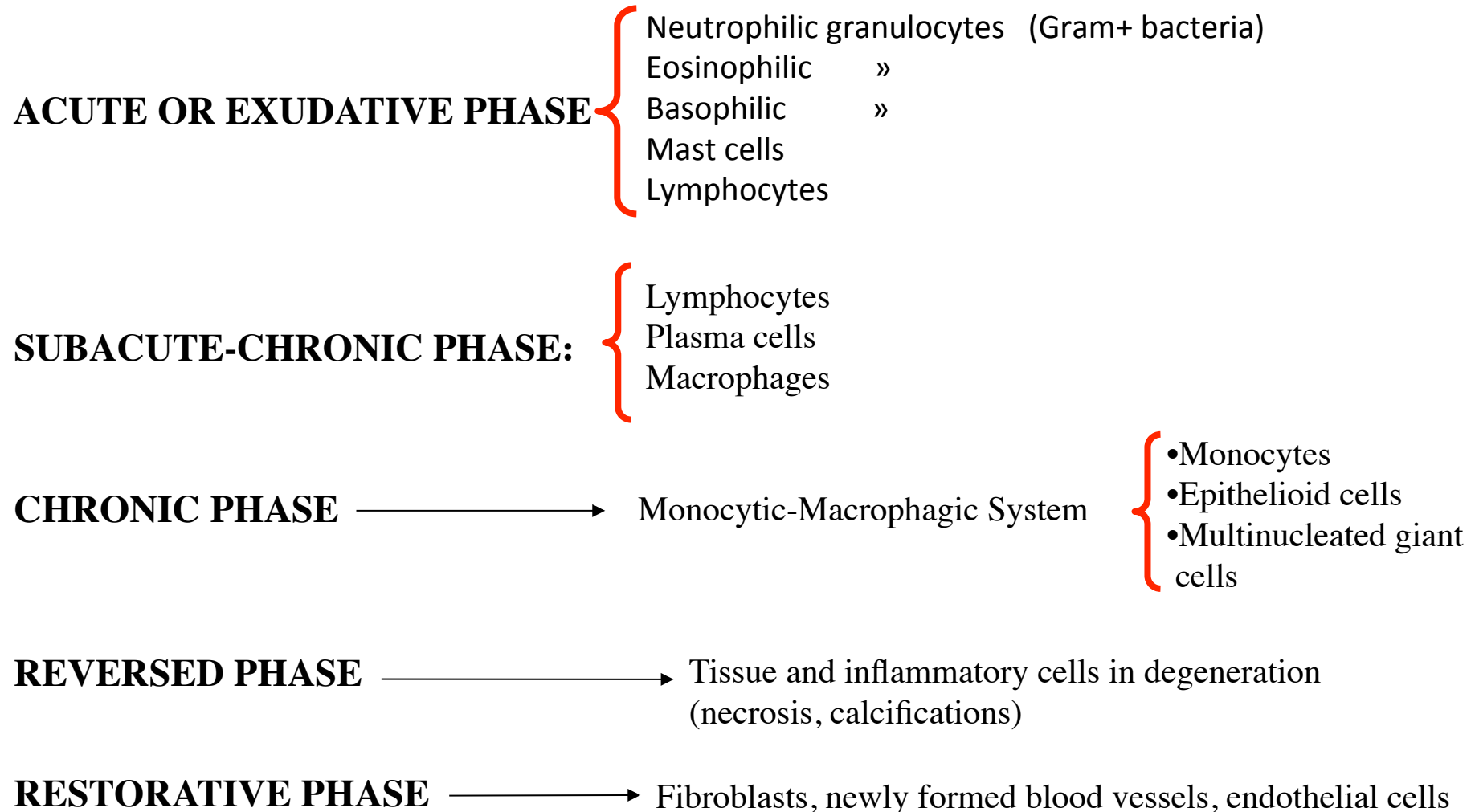
- destroying or limiting harmful agents,
- initiating reparation processes,
- bringing damaged tissues back to their original shape and function.

Depending on etiology:

- Acute inflammation may evolve into chronic over time.
- The acute phase may be inapparent and inflammation seems chronic since the beginning

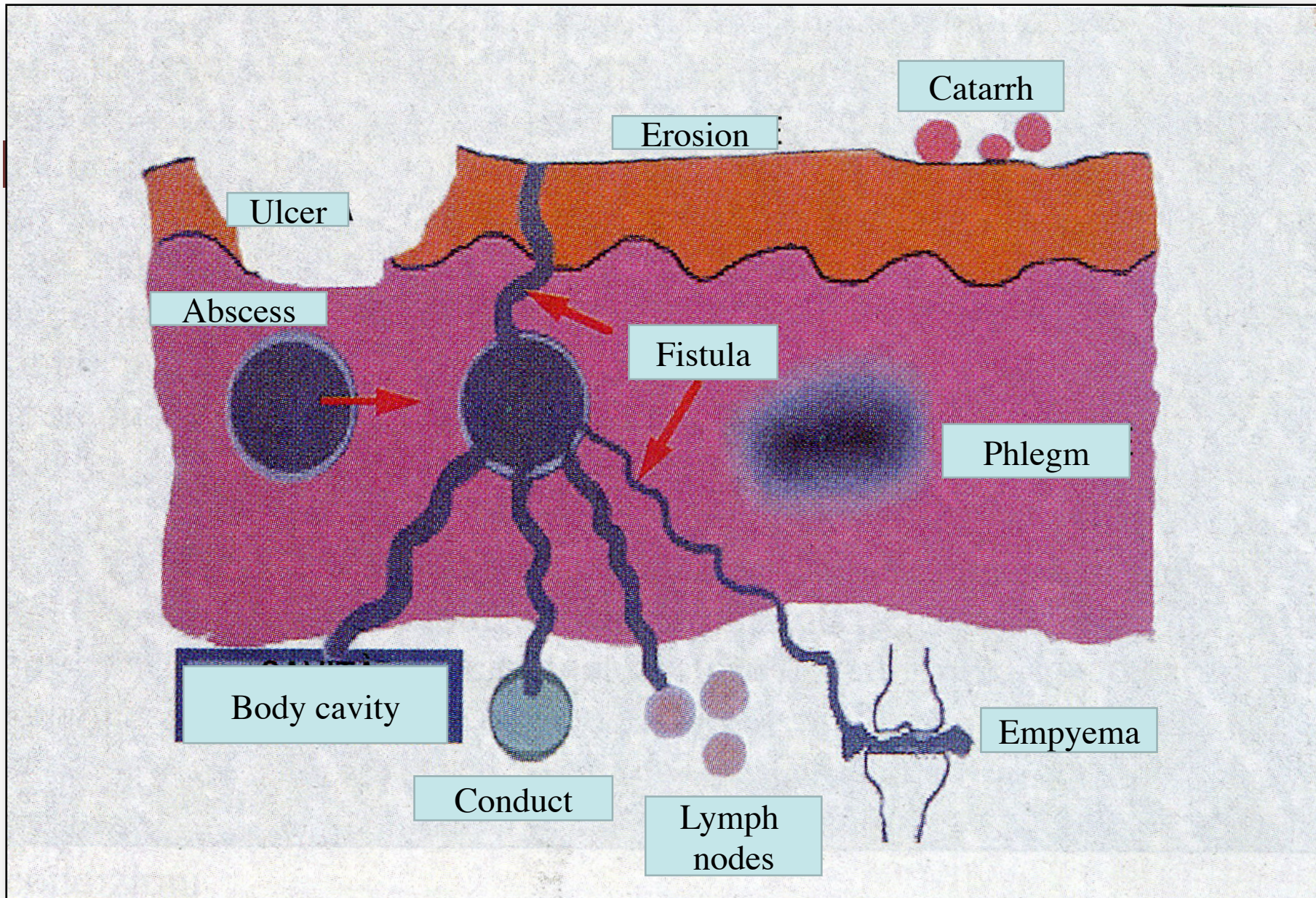
Inflammatory cells

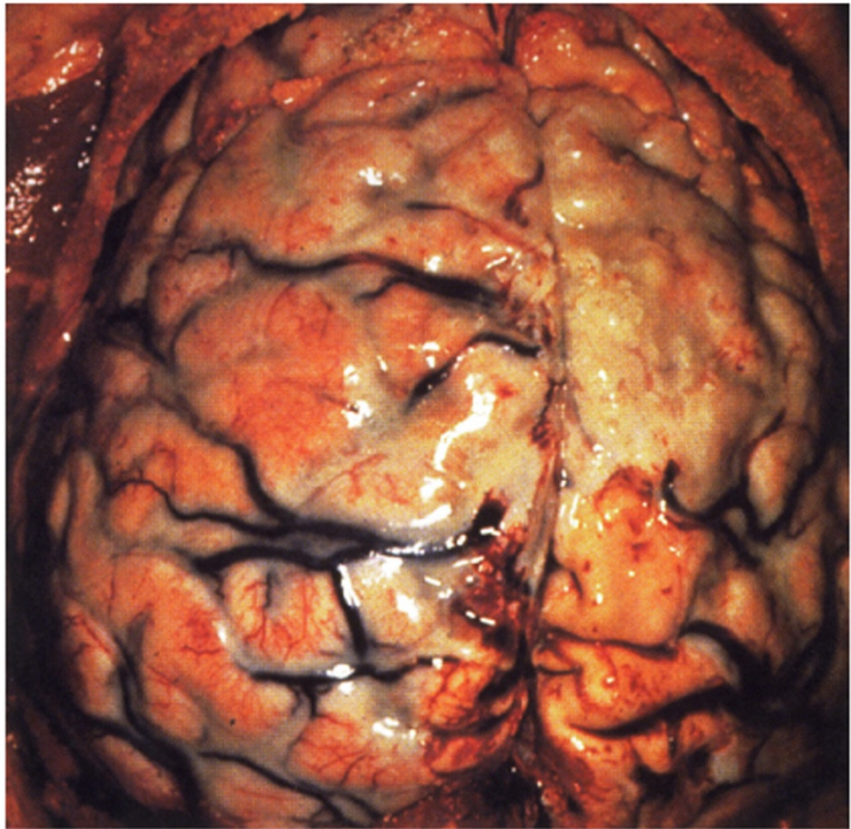
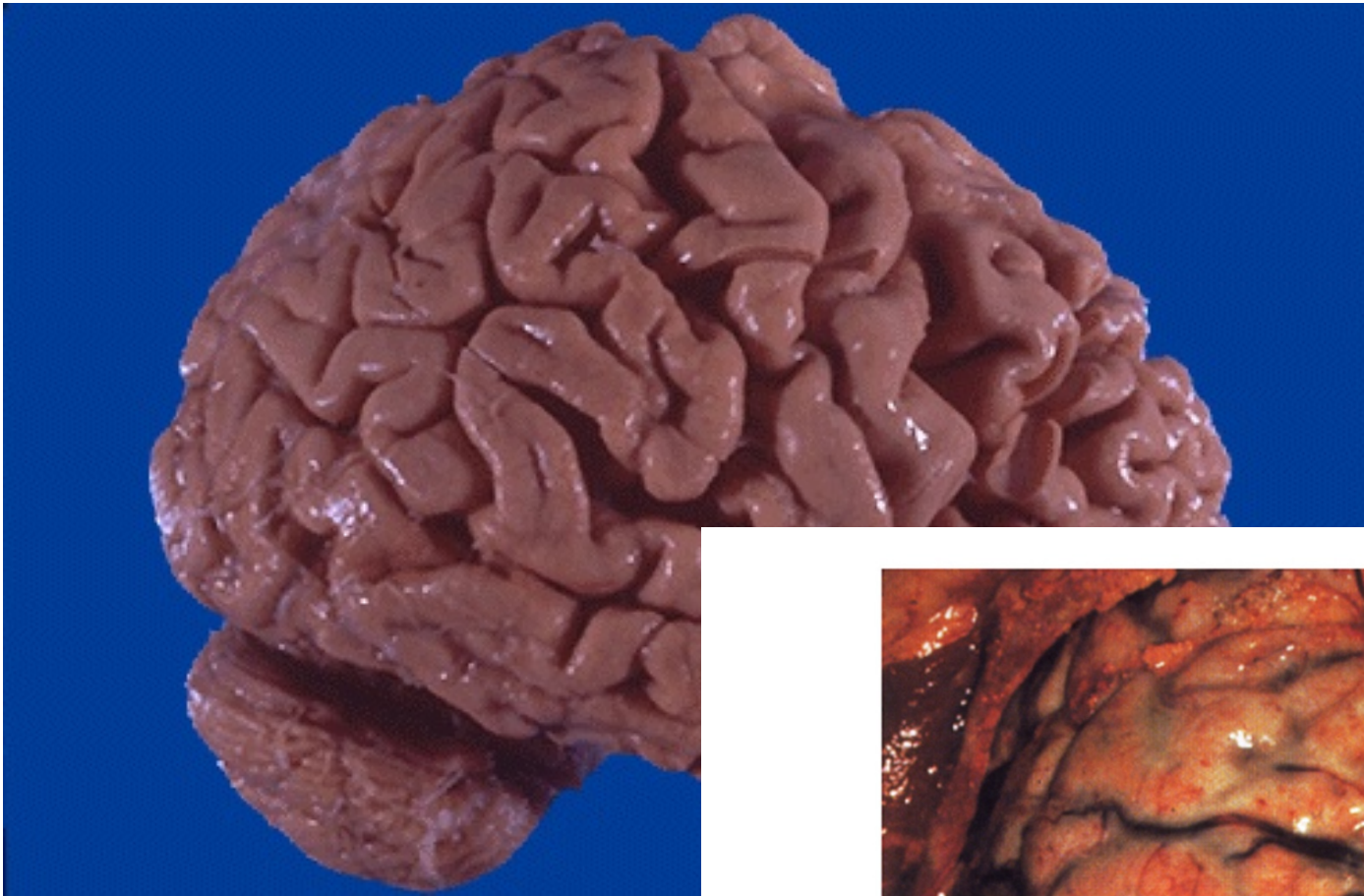
The cellular elements of the exudate and how they accumulate allow us to define the **phase**, **type** and sometimes the **etiology of the inflammatory process (specific inflammations)**.

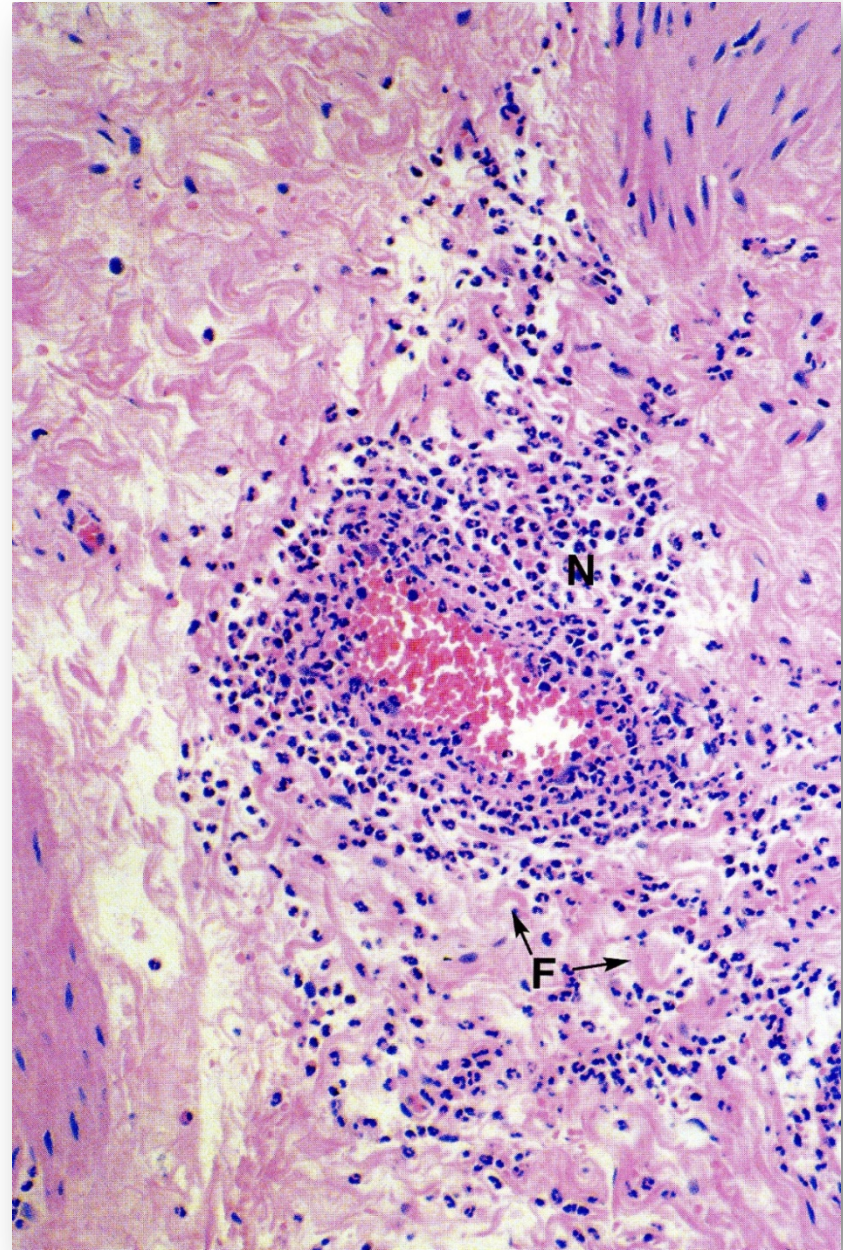
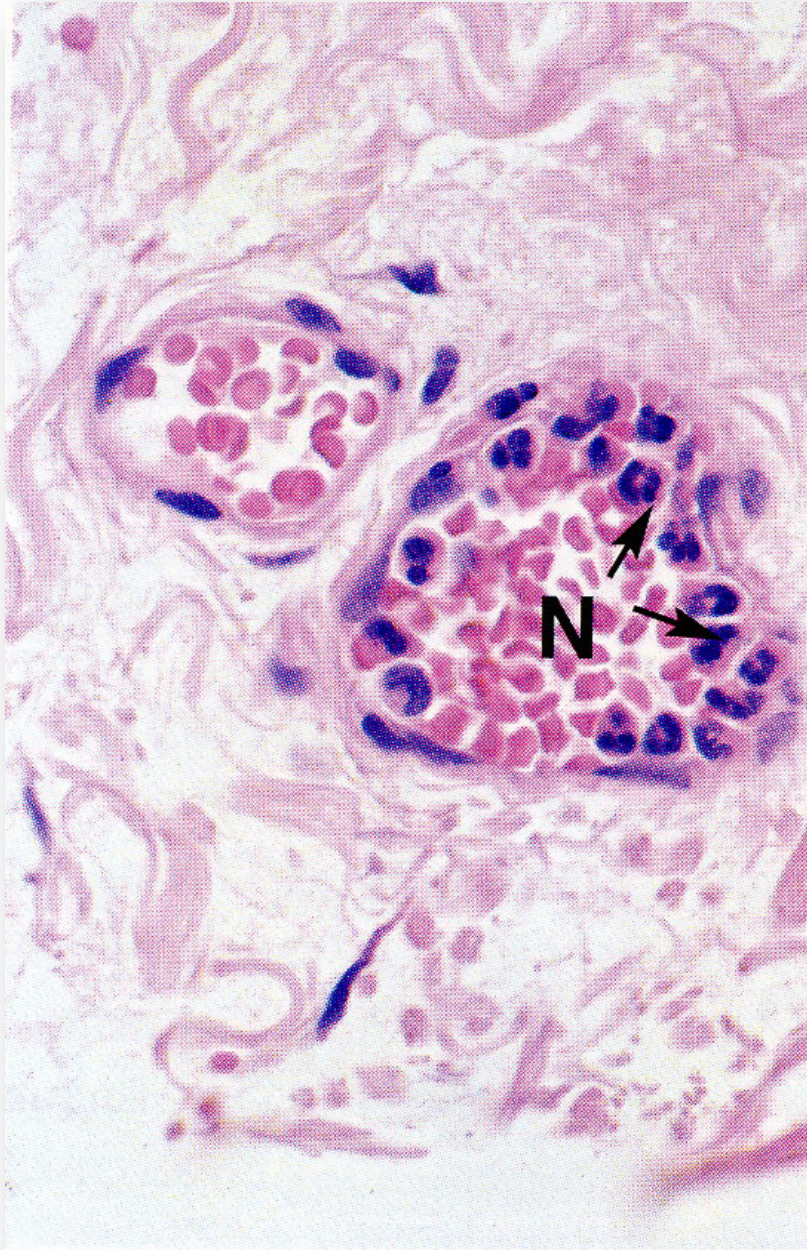


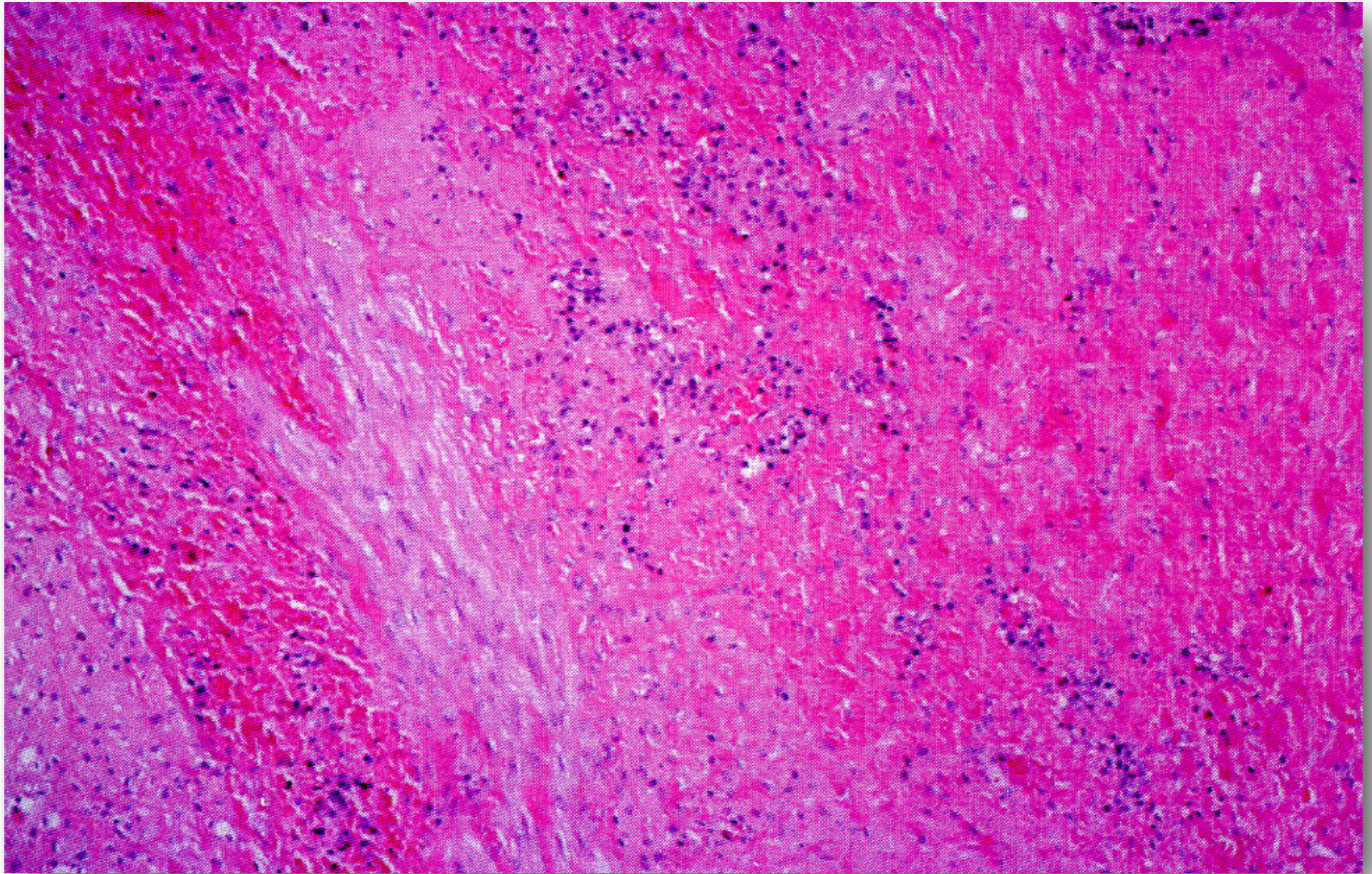
Exudate characteristics

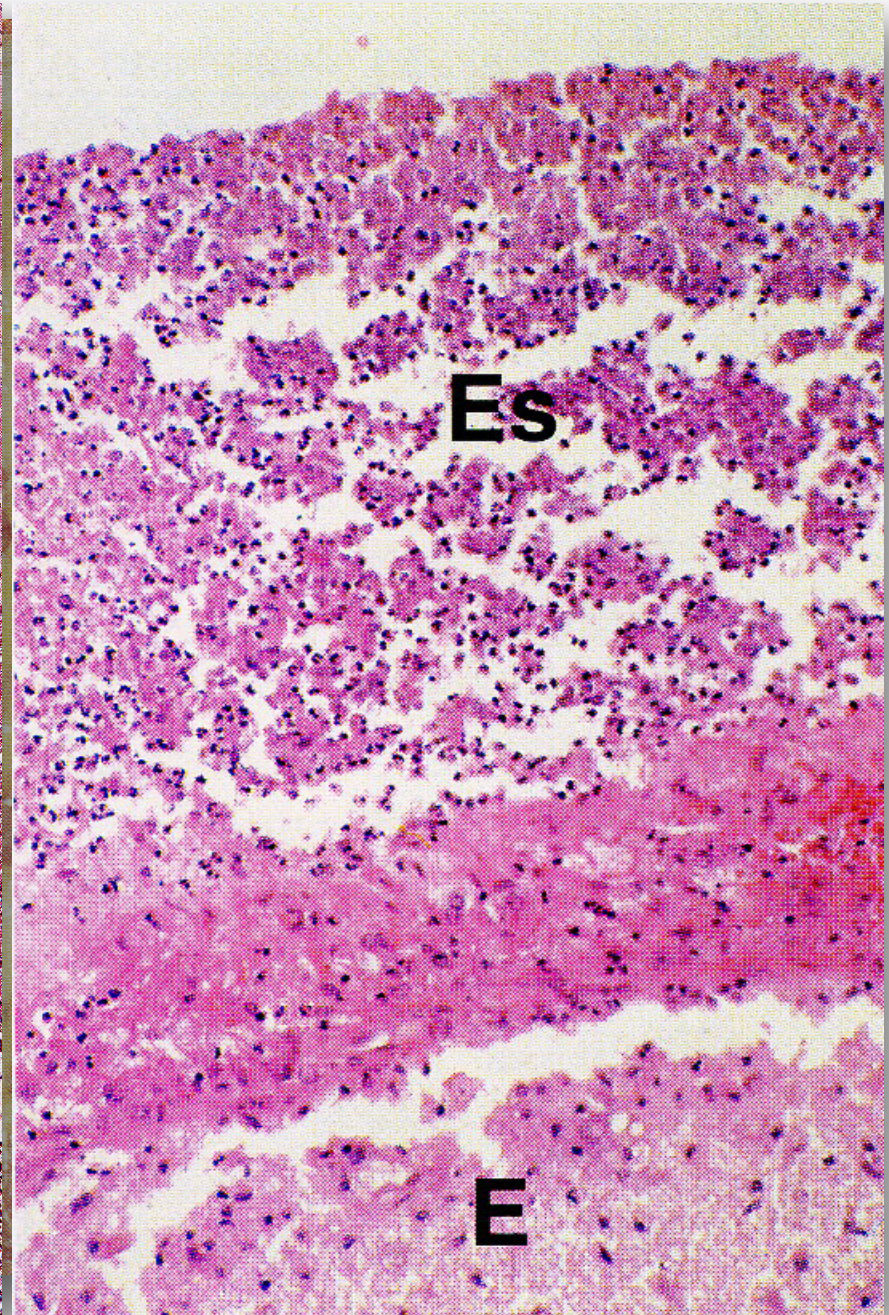
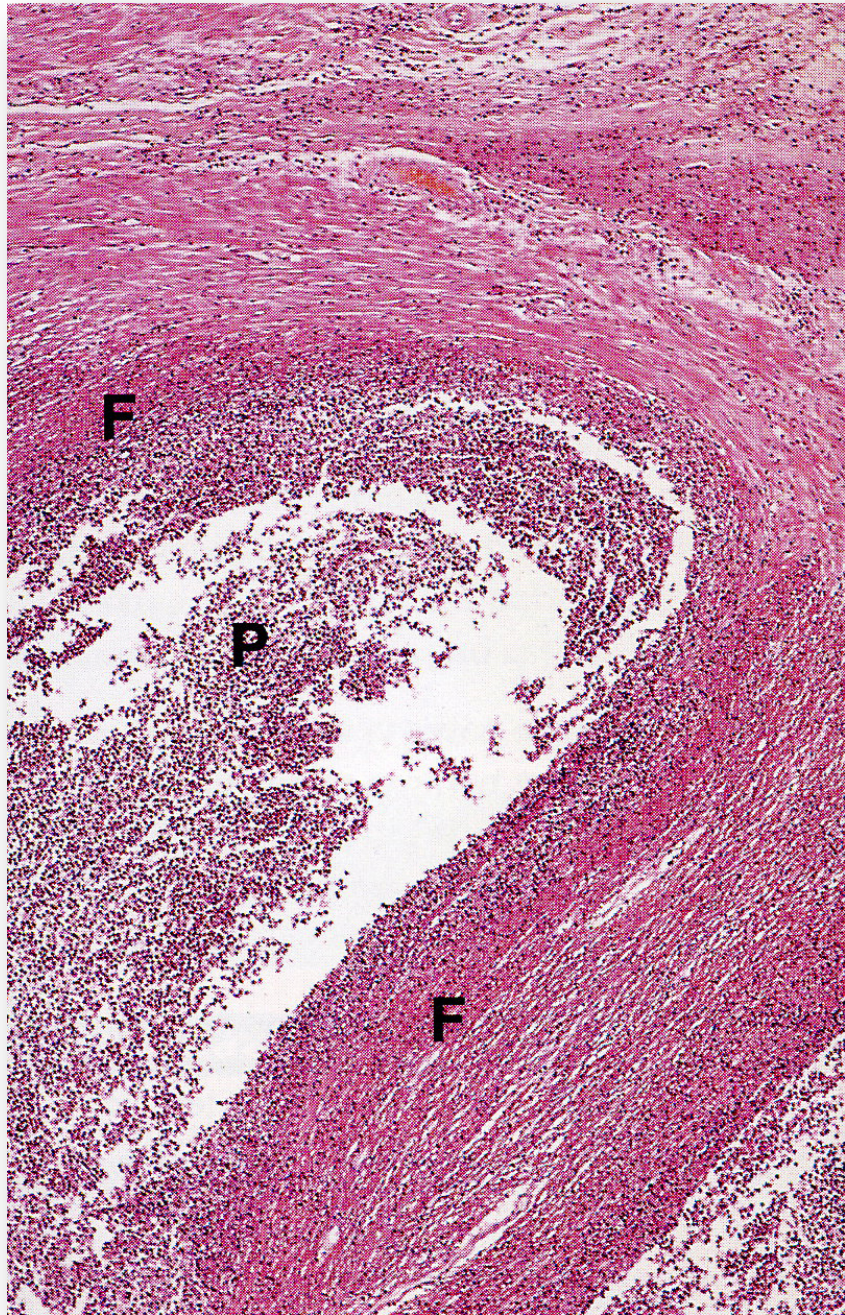
TYPE	PLACE	Pathological framing
Erythematous	Skin Mucosae	Reddening
Serous/ Catarrhal	Skin and mucosae Secreting mucosae (bronchi, intestine)	Vesicle, blister Catarrh (phlegm)
Fibrinous & Sero-fibrinous	Serous Parenchyma	Pleuritis Pericarditis Peritonitis
Suppurative	Serous	Pseudomembranes with no necrosis Pseudomembranes + tissue necrosis
	Mucosae	
	Parenchyma	Bronchopneumonia, hepatitis, nephritis
	Ubiquitous	Pustole, foruncle , abscess , phlegm , empyema.
Hemorrhagic (fibrino-hemorrhagic) Necrotizing Putrid-Gangrenous		Pustole, foruncle, abscess , phlegm, empyema, gangrene .

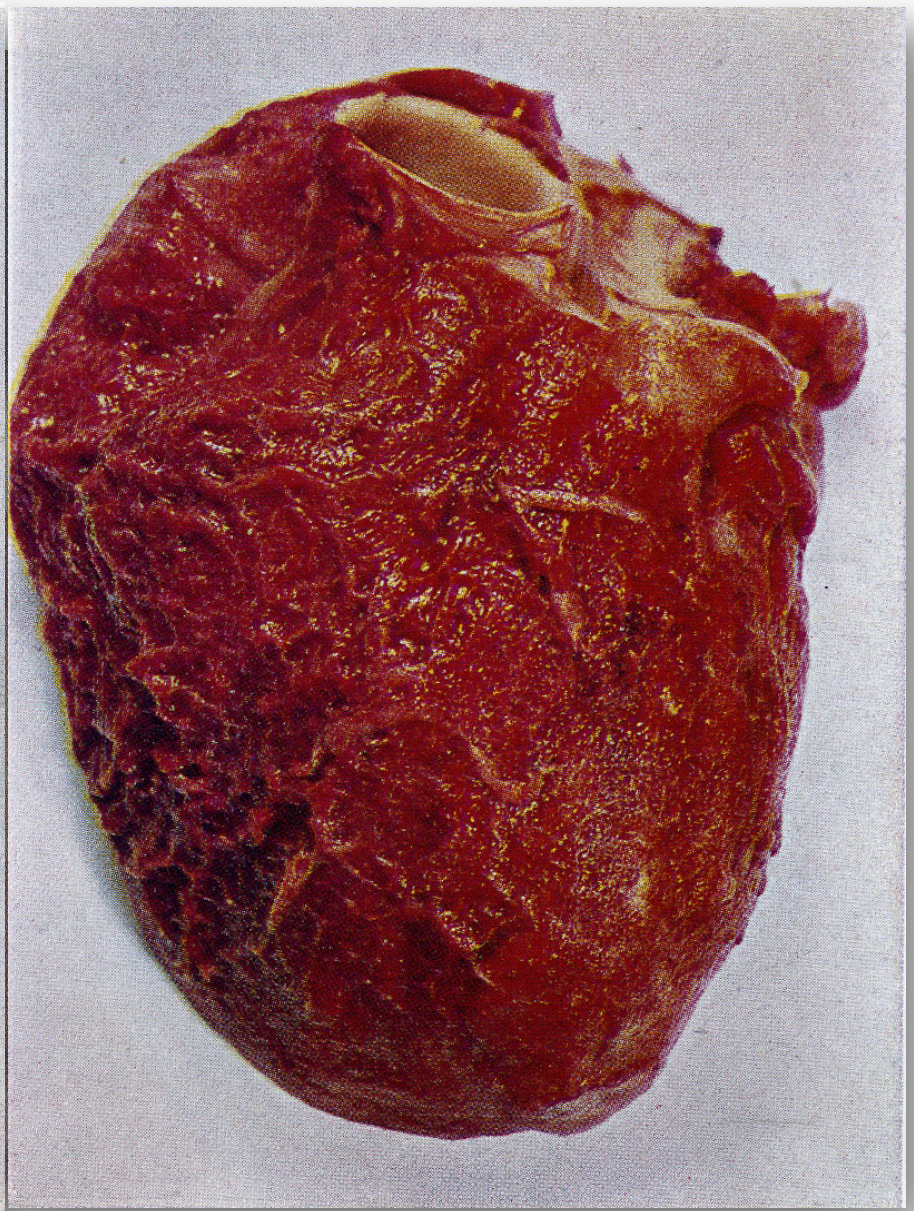


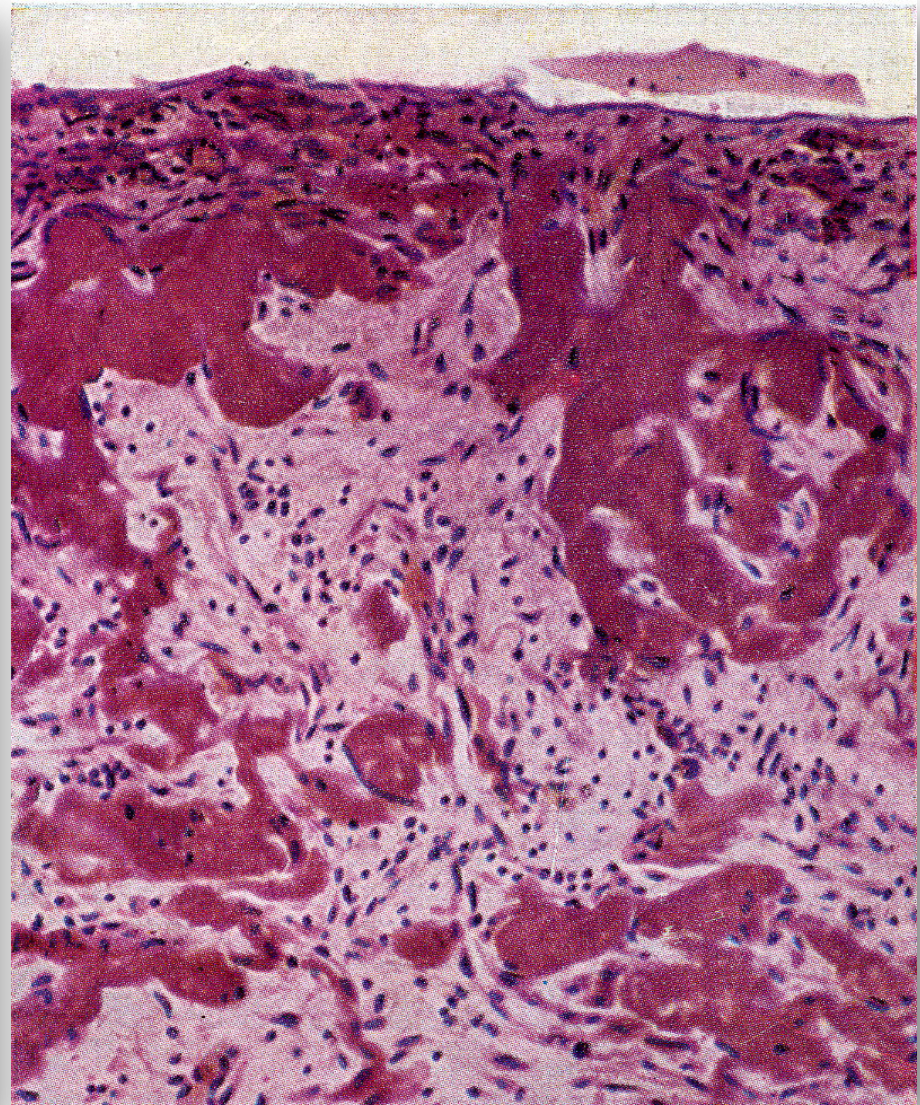
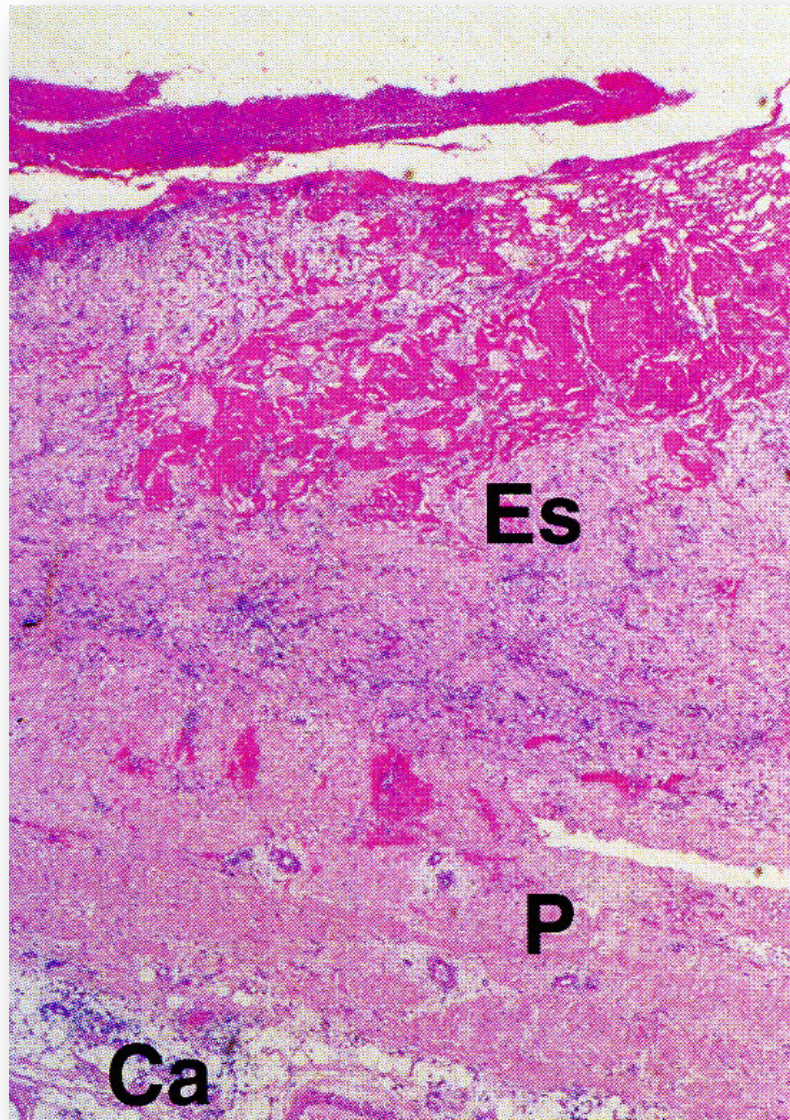












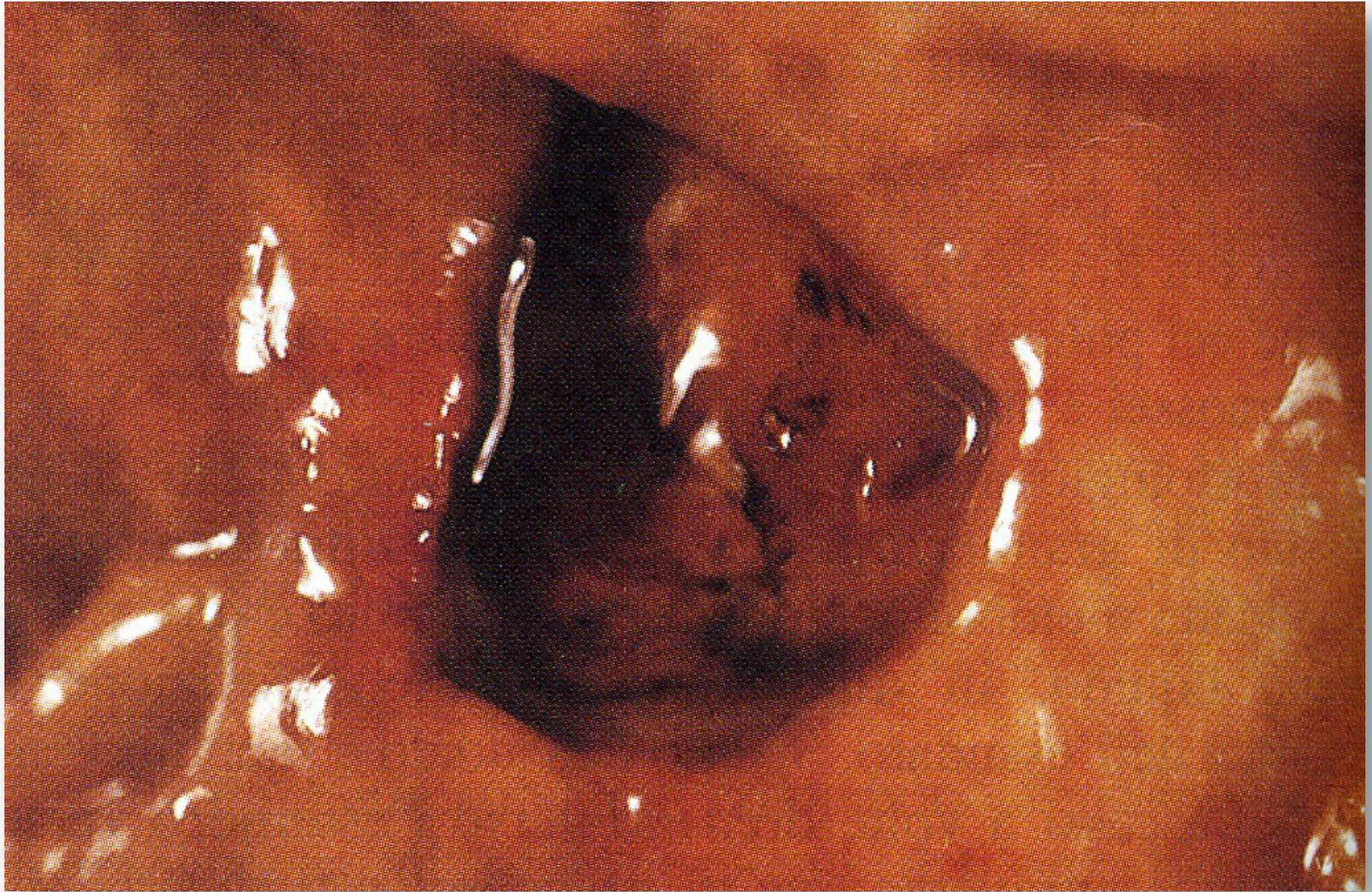
CHRONIC INFLAMMATION

Diffuse = no granuloma

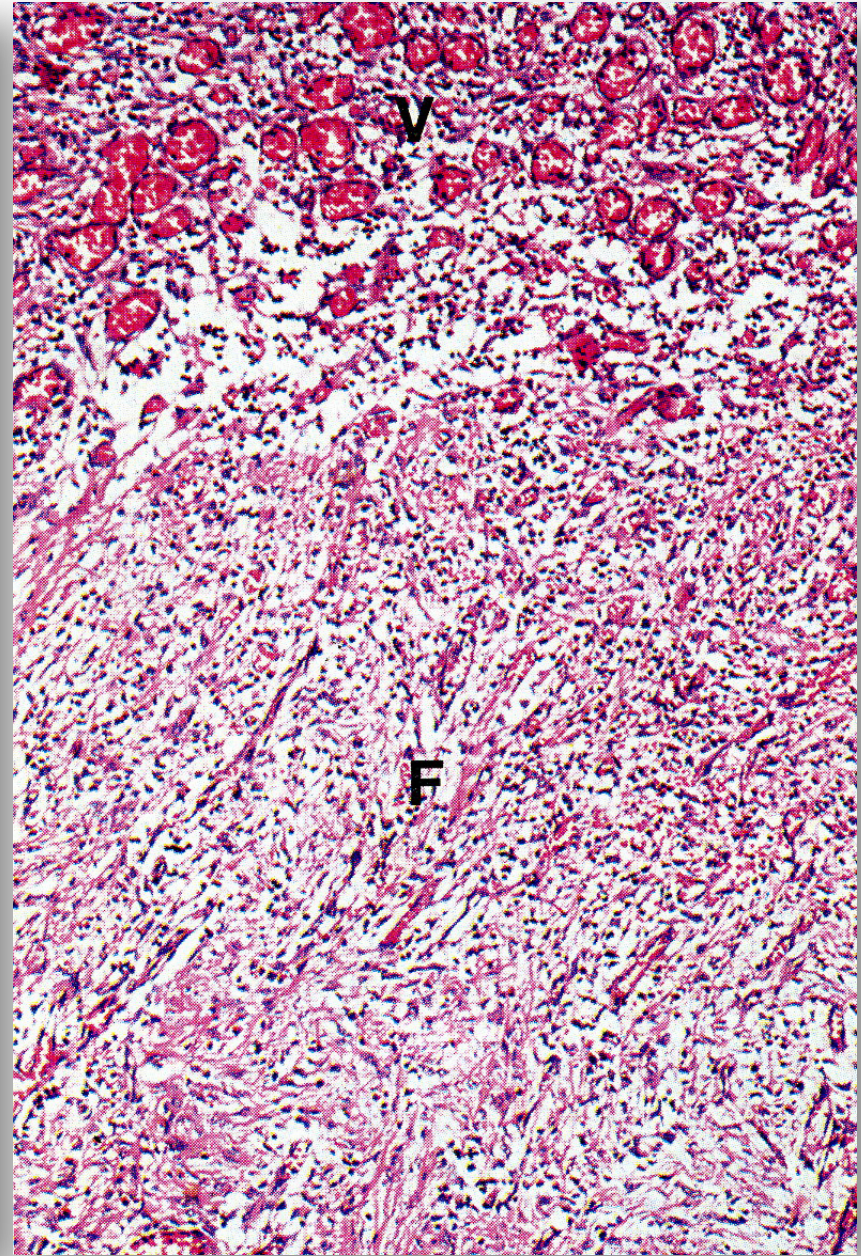
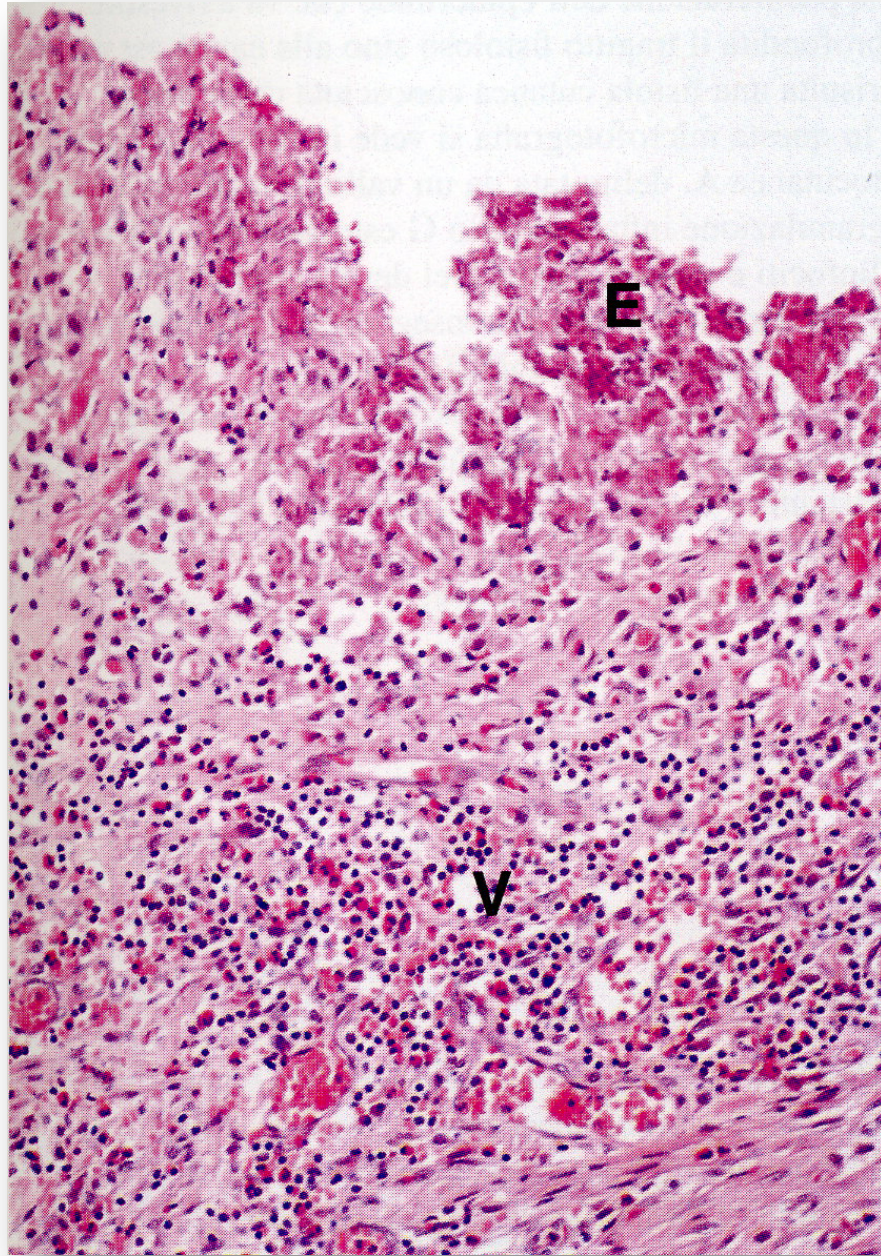
- Exudate formed by lymphocytes and plasma cells
Frequent lymphoid follicular aggregates
- Autoimmune diseases (ex.: Hashimoto thyroiditis, Lupus Erythematosus , Autoimmune Sialoadenitis)

Circumscribed = Granuloma

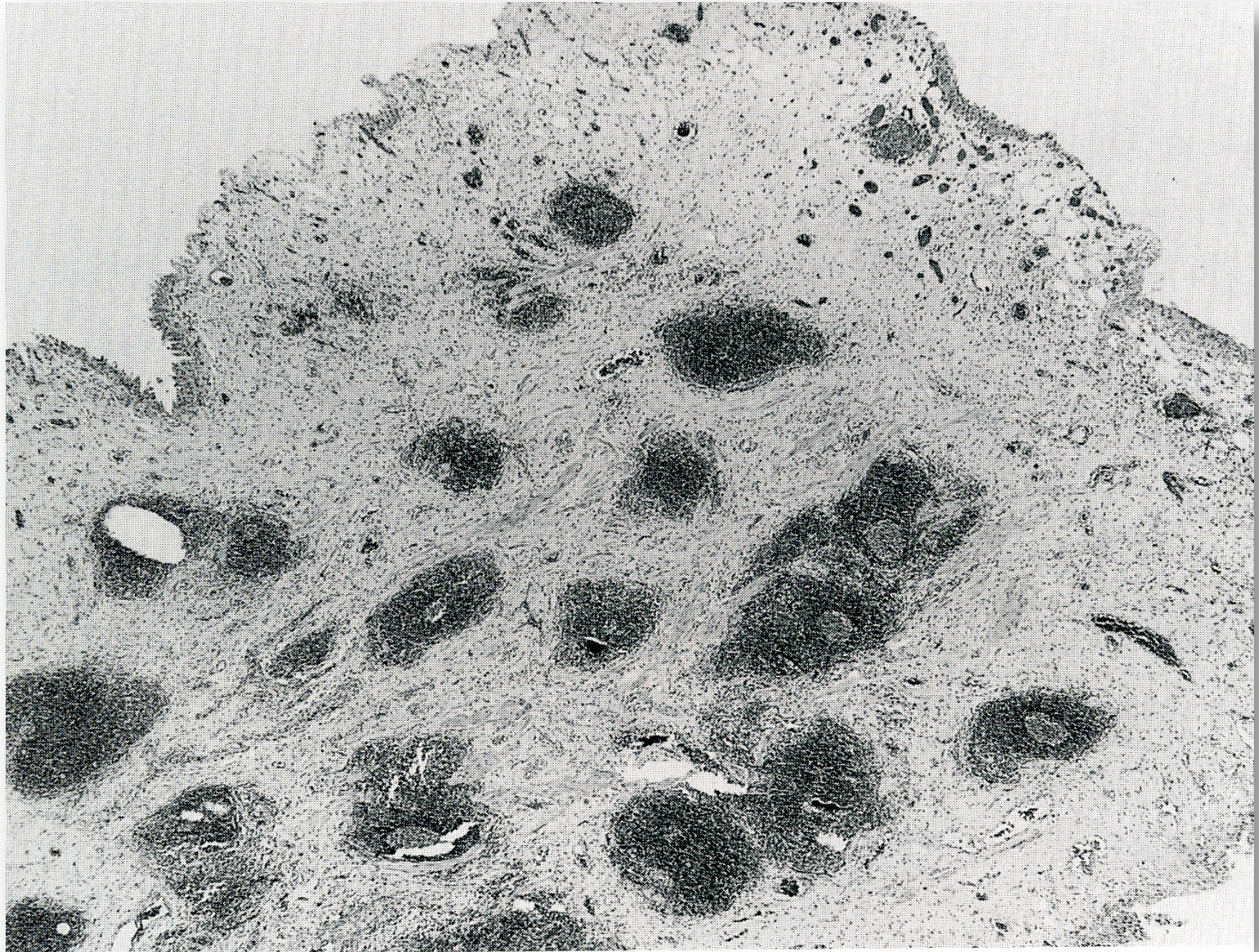
- Persistent and poorly reabsorbable antigens
- Monocytic-Macrophagic cells (macrophages, epithelioid cells, fibroblasts, newly formed vessels, lymphocytes, plasma cells)
- Demarcation of the necrotic focus
(gastric ulcer, infarction, organization, etc.)

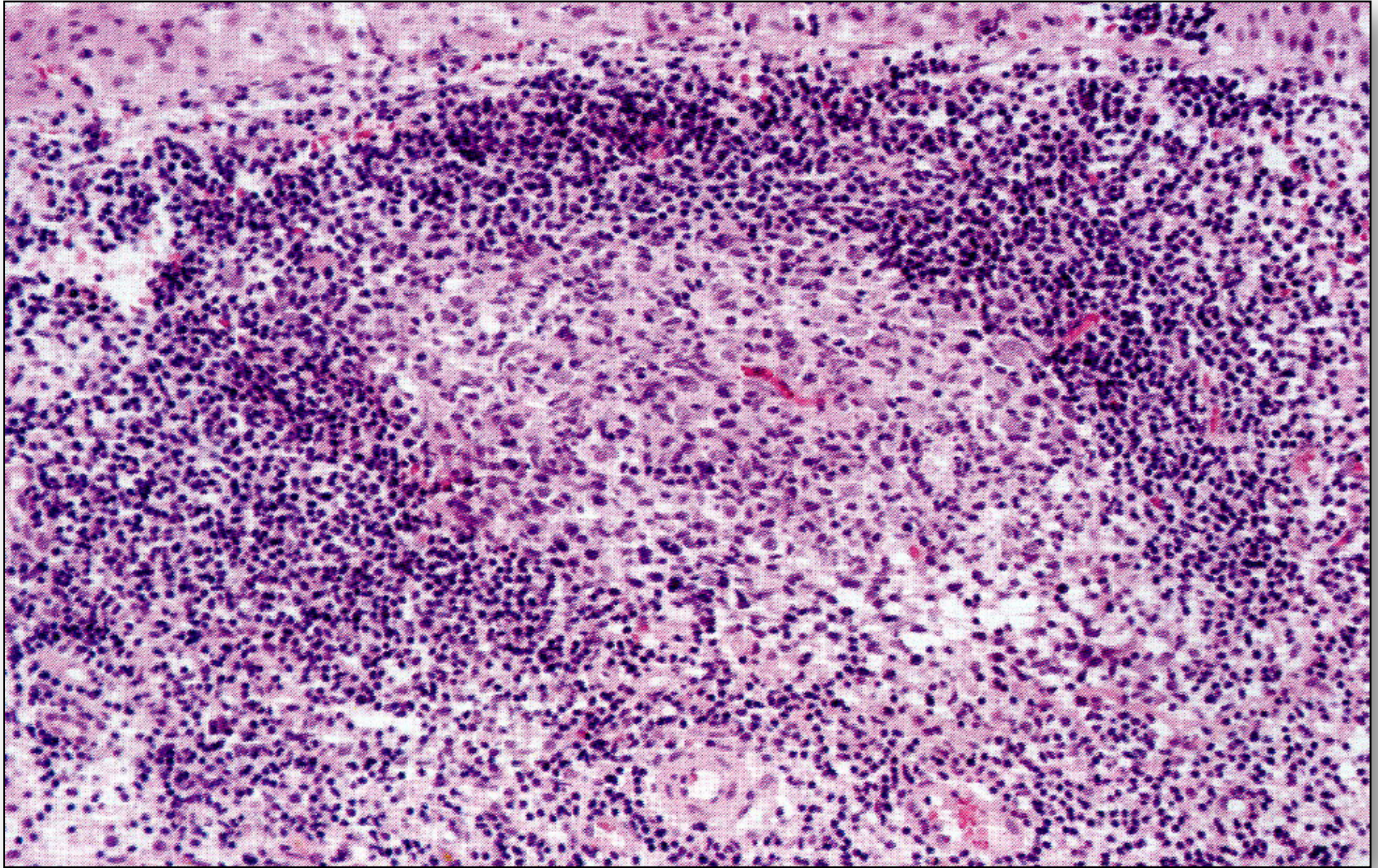






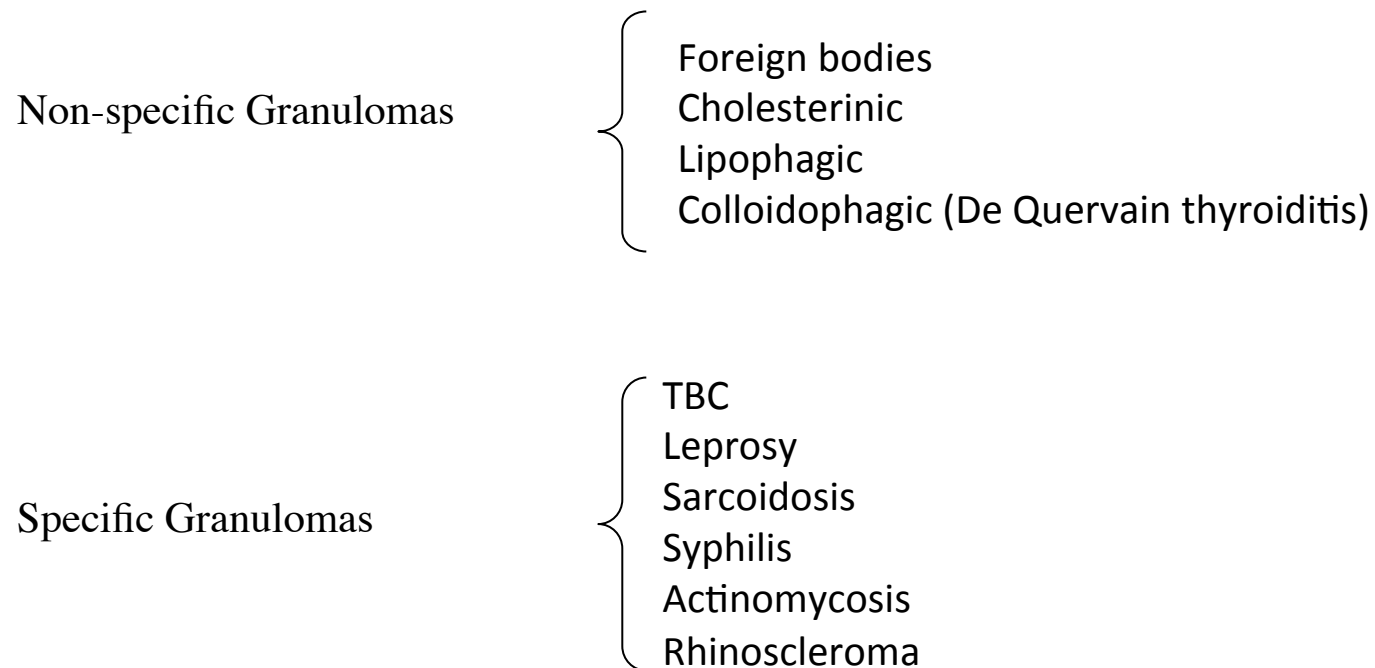


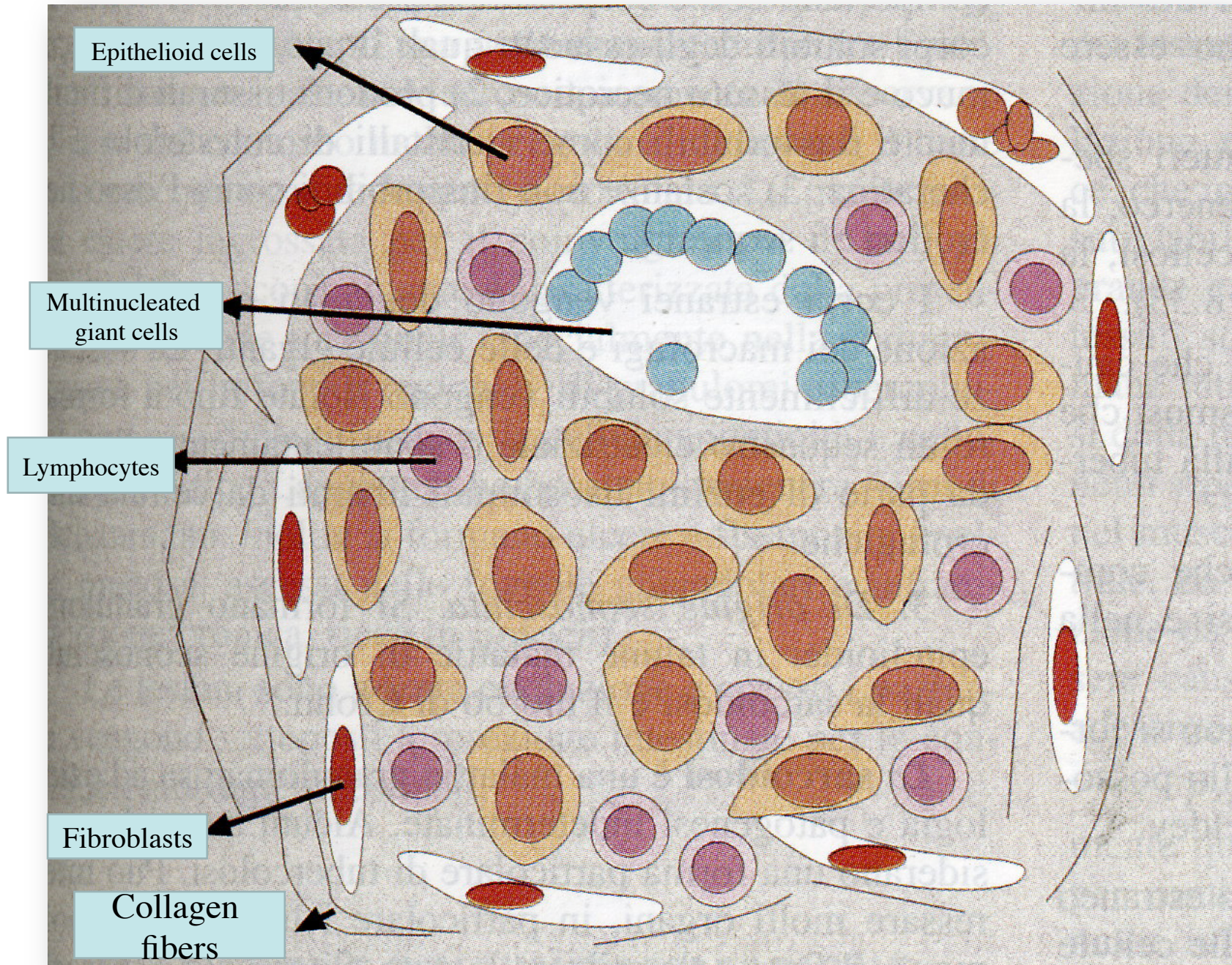




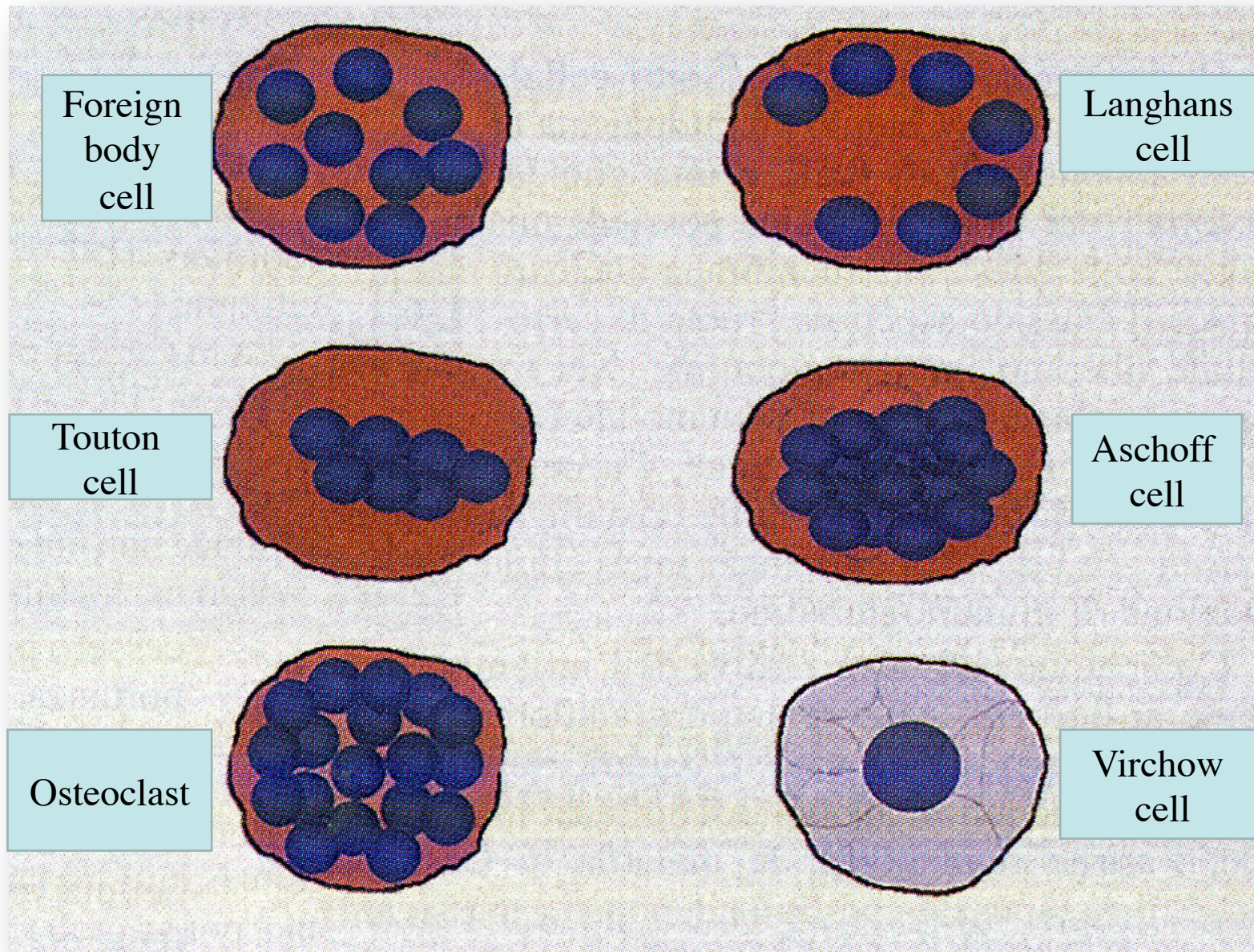
CHRONIC GRANULOMATOUS INFLAMMATION

GRANULOMA: nodular formation, tumor-shaped, constituted by antigens, tissue residues and inflammatory cells



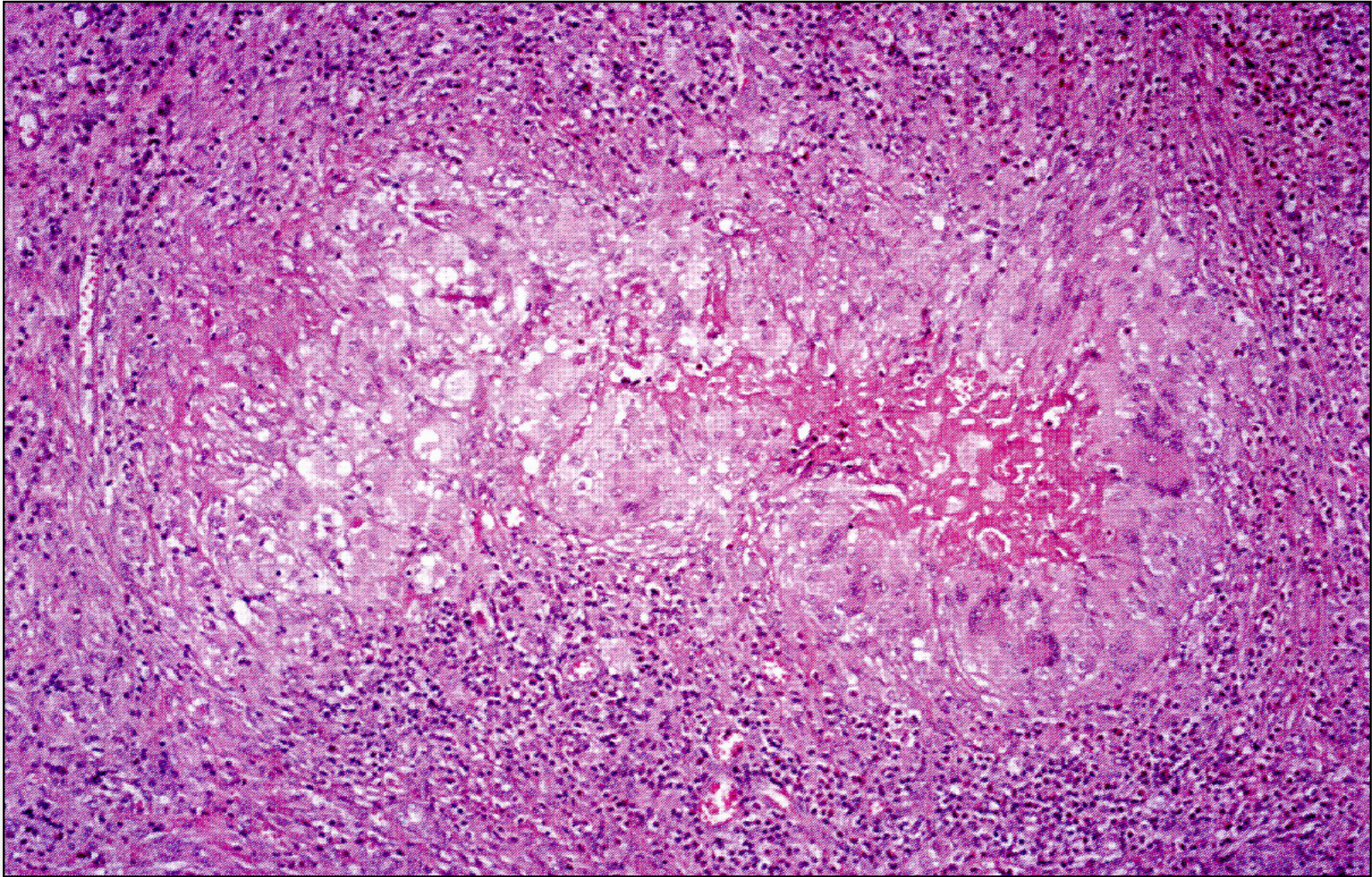


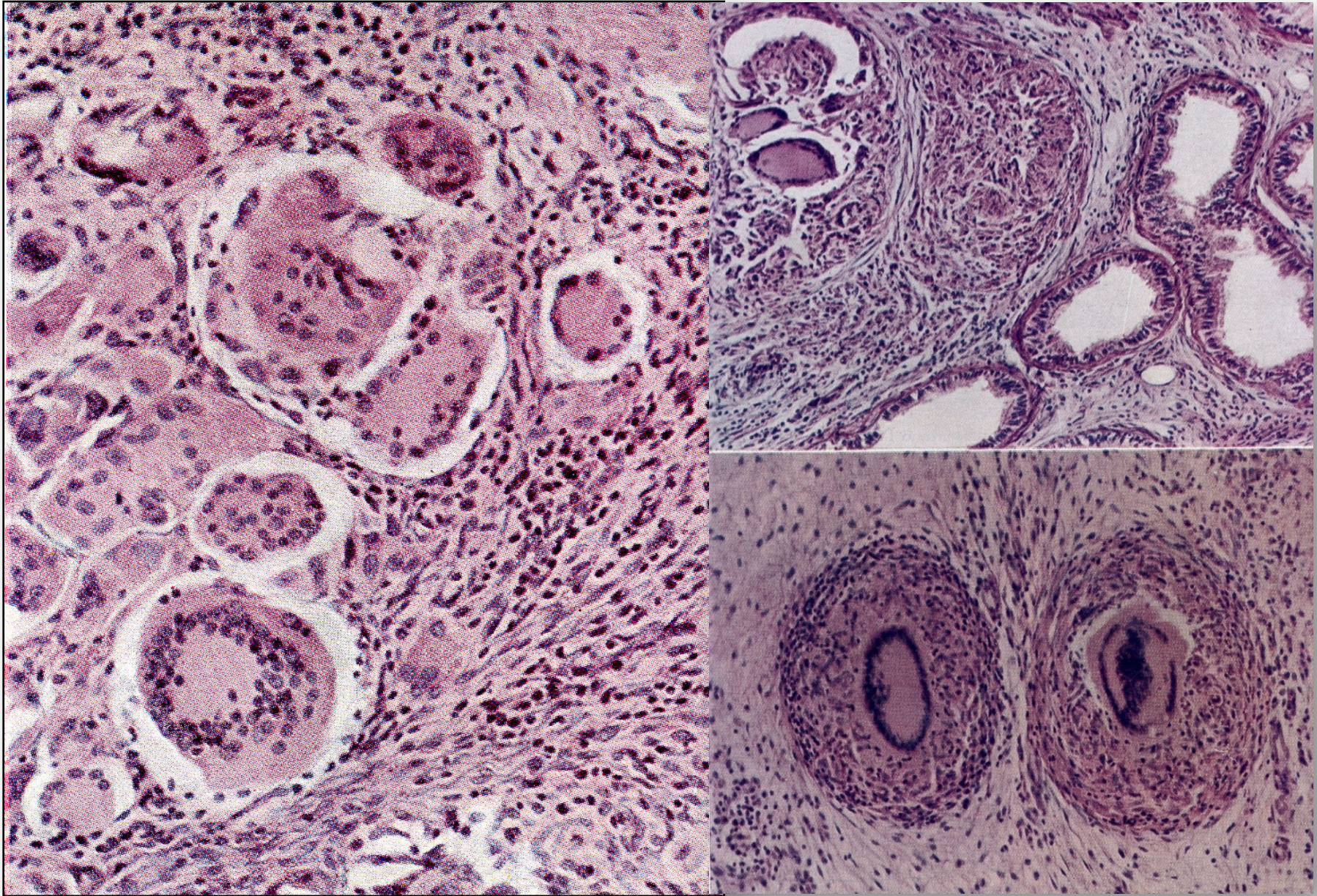
GIANT CELLS

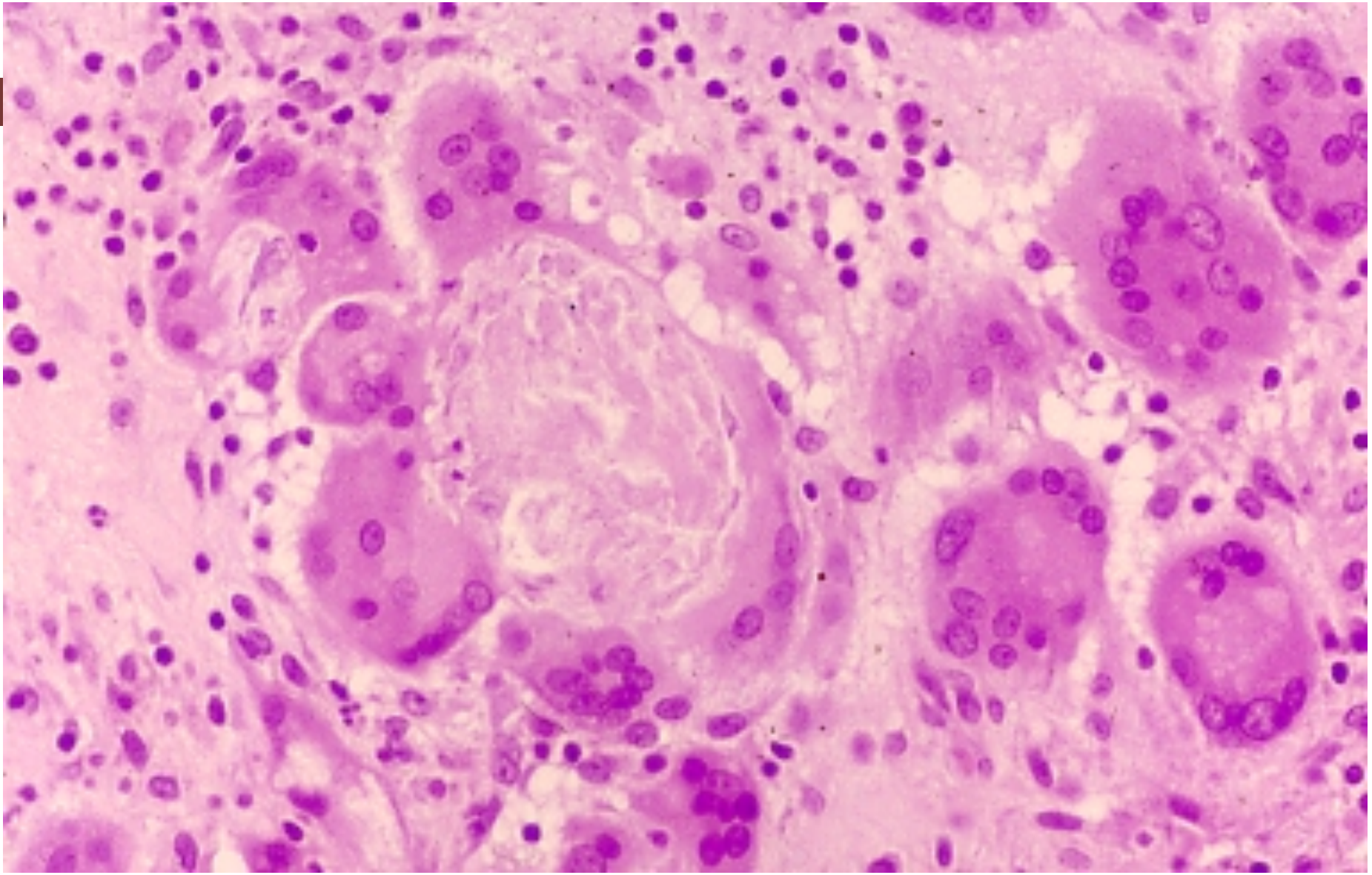


Foreign body granuloma

- Giant cells with nuclei positioned centrally, scattered or opposite to the foreign material
- Epithelioid macrophages
- “foreign bodies” (intra/extracellular)
 - sutures
 - lipids (cholesterine and neutral lipids) + calcification
 - hook or thorns
 - wood, metal, glass
 - plant material
 - beryllium, silicon
- Neutrophilic and eosinophilic granulocytes
- Fibroblastic capsular wall

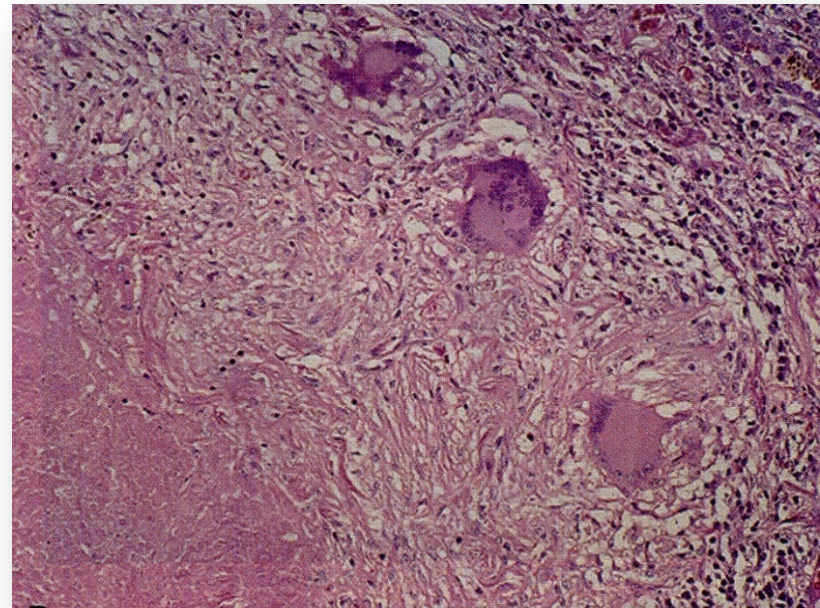
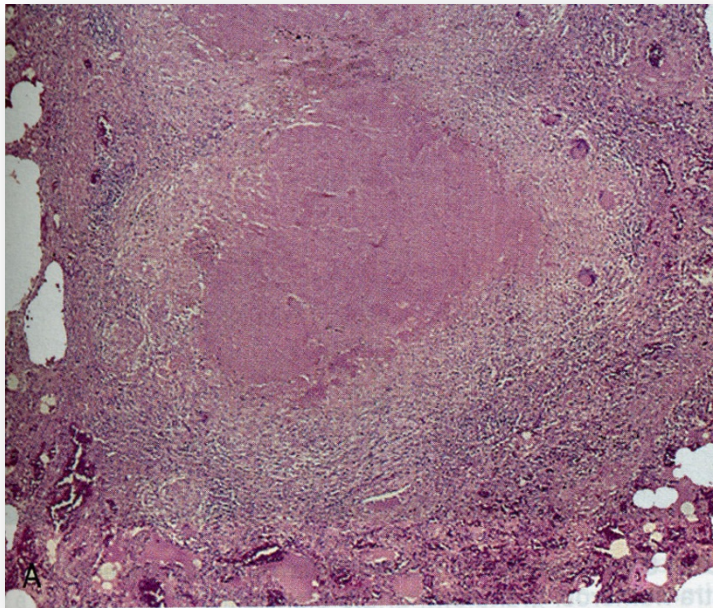
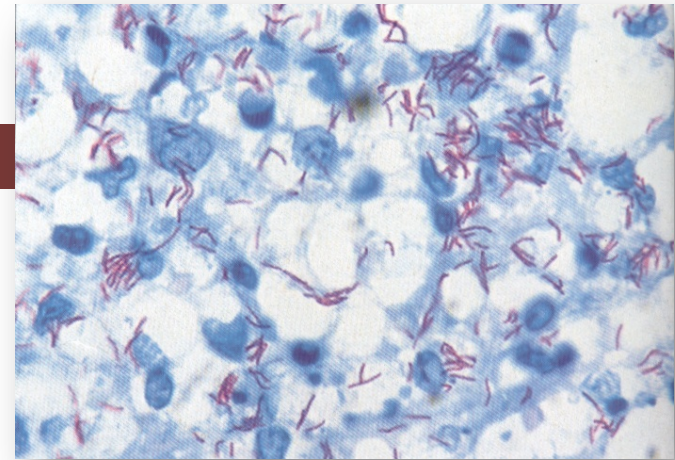






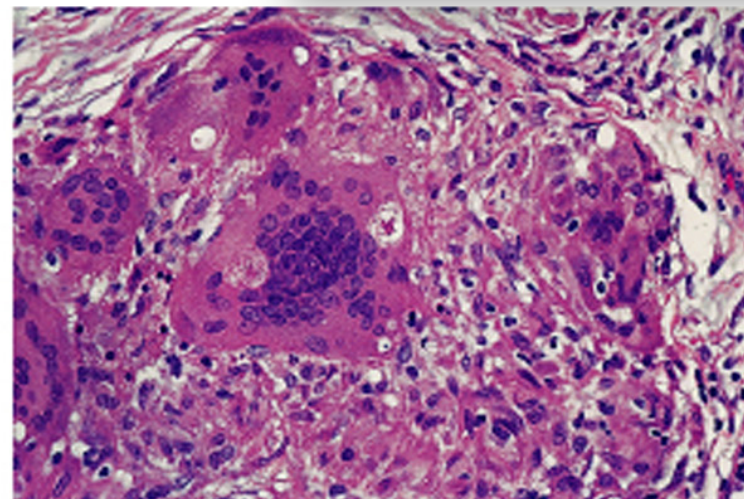
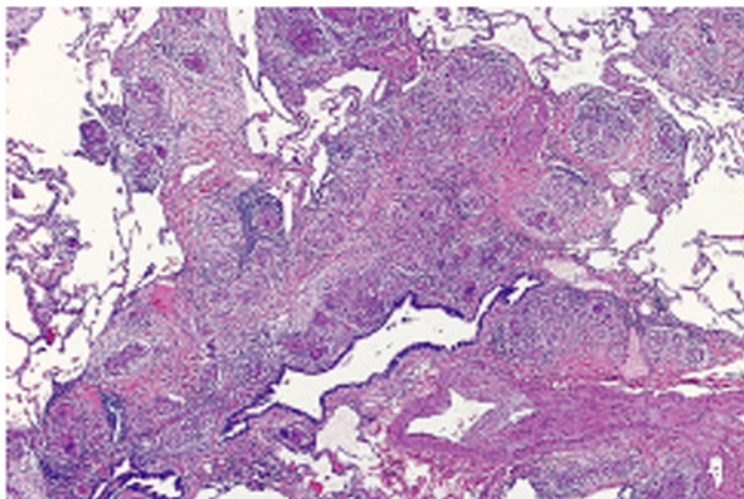
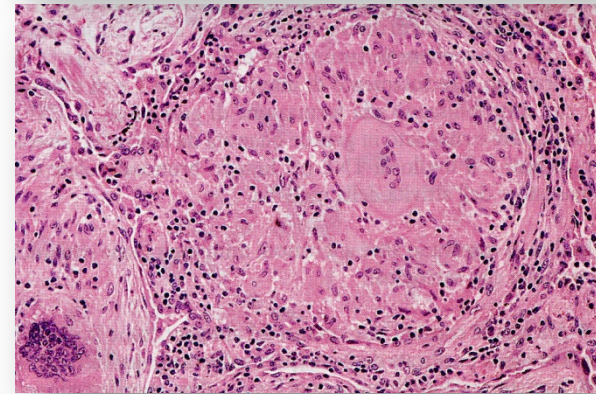
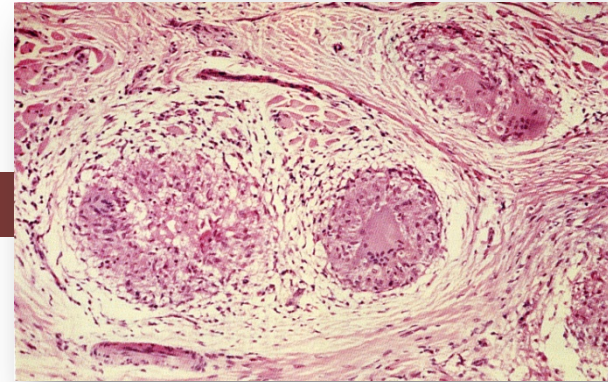
Tuberculous Granuloma (tubercle)

- “Caseous”, homogeneous necrosis
- Langhans type giant cells
- Epithelioid macrophages containing bacilli Z/N
- Lymphocytes



Sarcoid Granuloma

- Lack of necrosis
- Epithelioid macrophages
- Lymphocytes
- Asteroid and Schaumann bodies
- Net demarcation , non-confluent



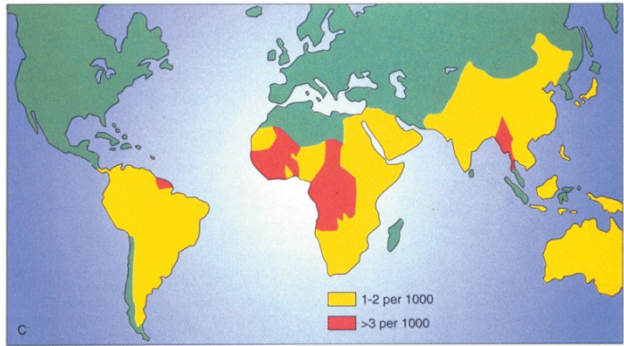
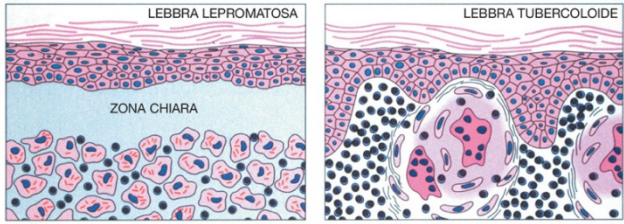
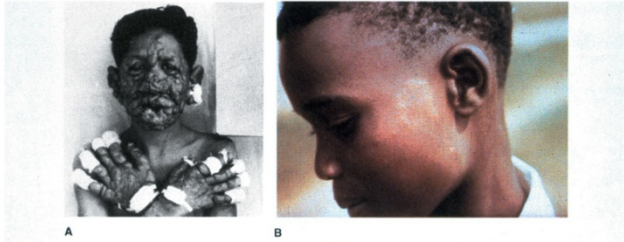
CHRONIC GRANULOMATOUS INFLAMMATION

Leprous Granuloma

- Scarce or absent necrosis
- Macrophages containing Wade/Fite+ bacilli
- Dispersed lipids
- Rich lymphocytic ring
- Perineural location

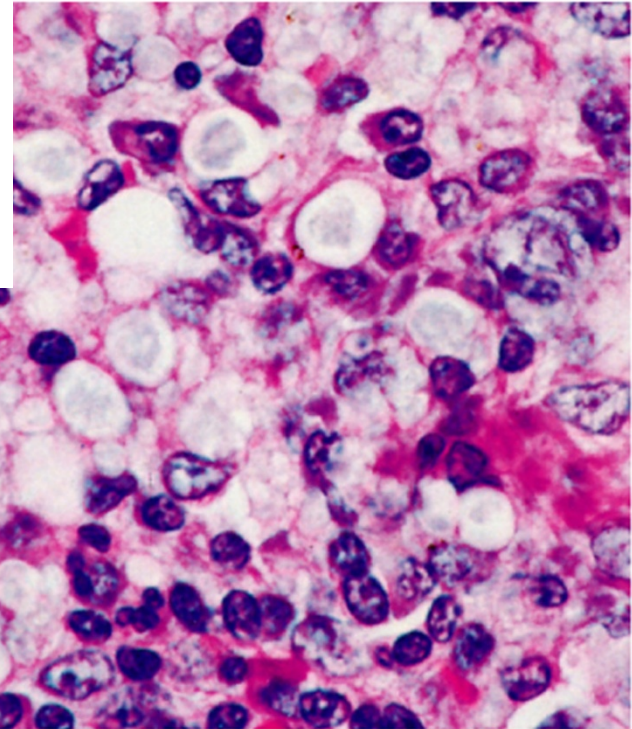
Syphilitic Granuloma or “gumma”

- Gummy necrosis (cellular residues)
- Epithelioid macrophages (scarce)
- Plasma cells (+++)
- Lymphocytes and fibroblasts
- Perivascular location



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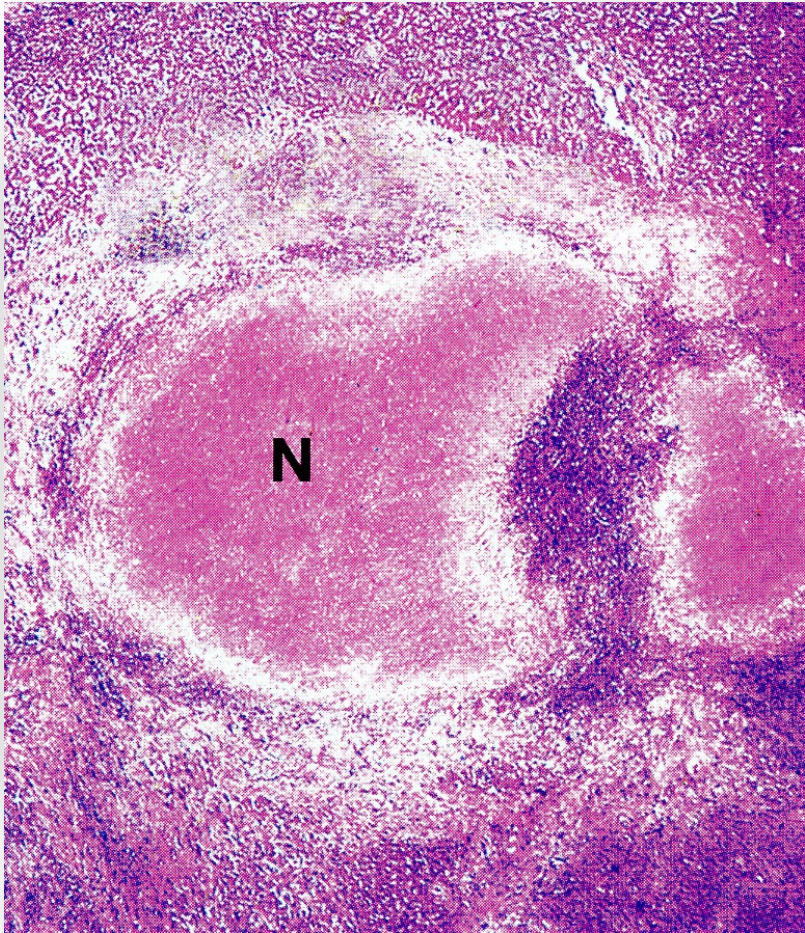
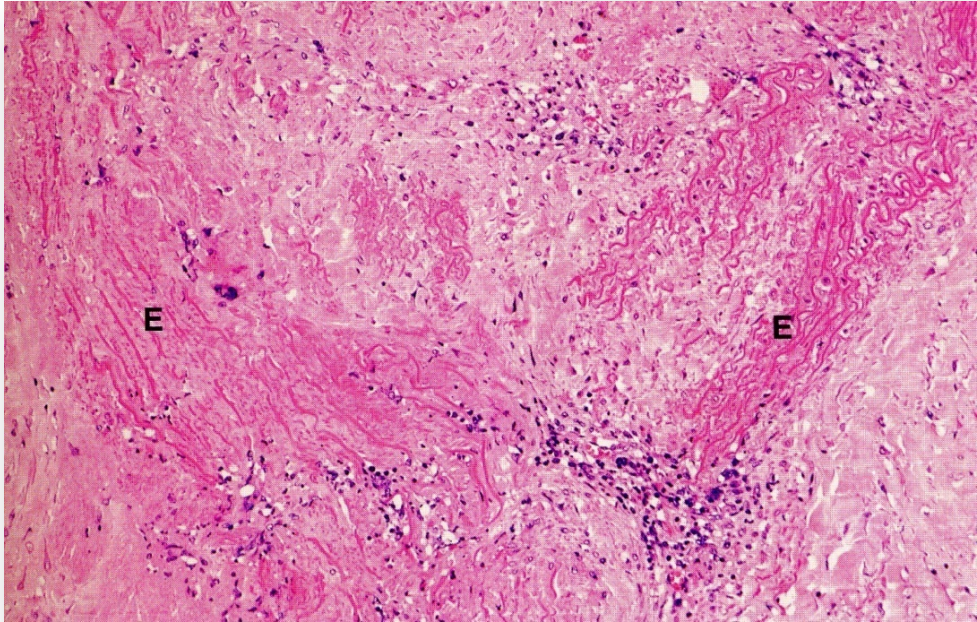


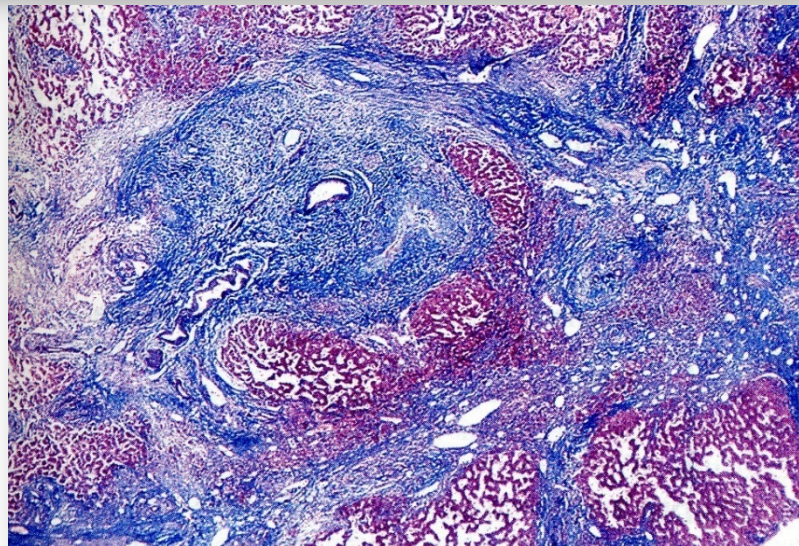
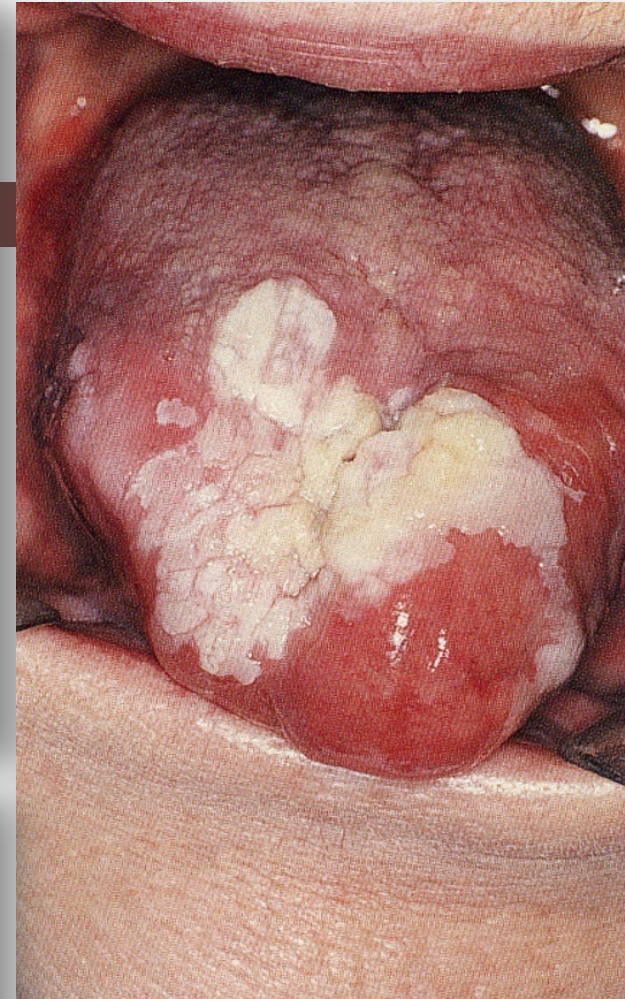
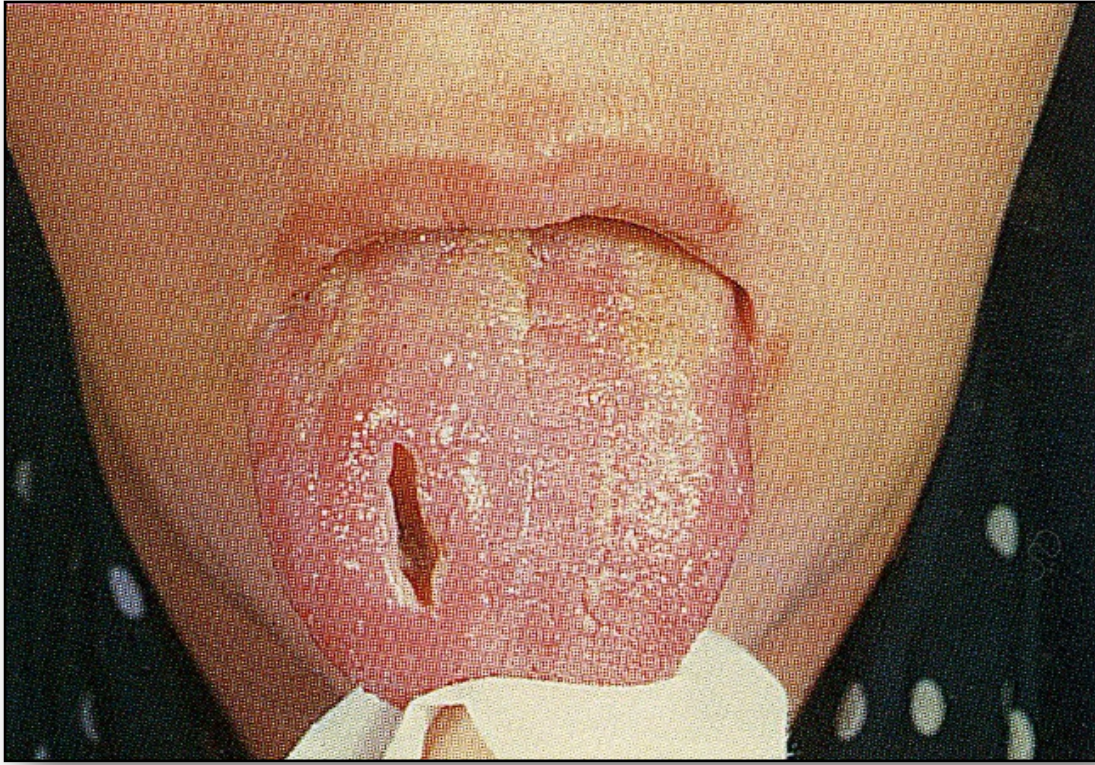


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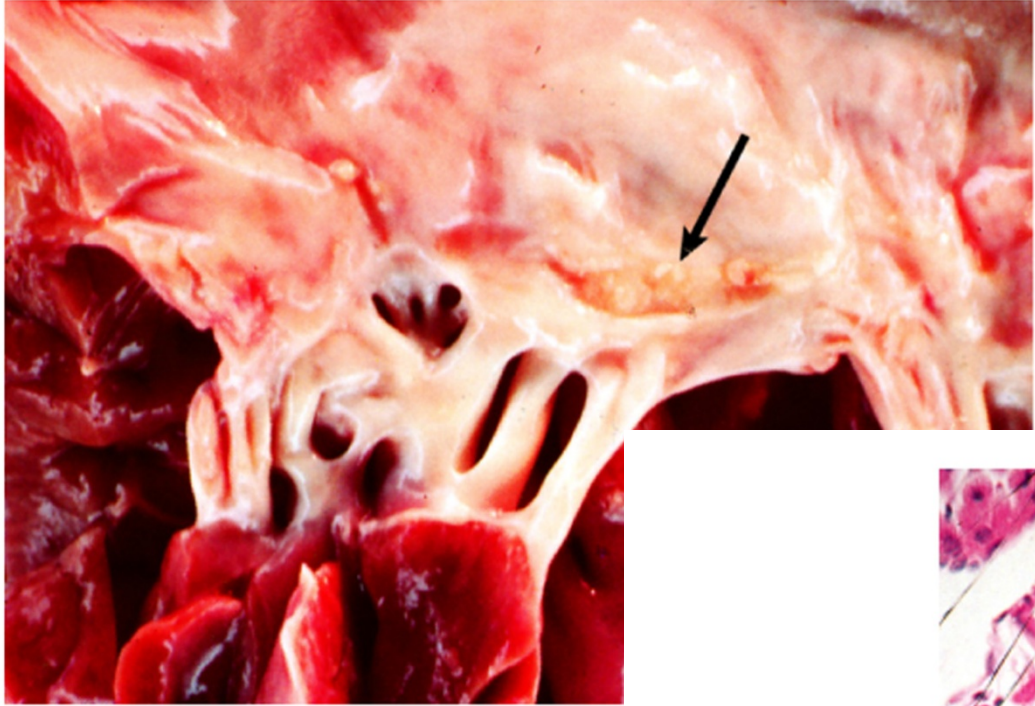
CHRONIC GRANULOMATOUS INFLAMMATION

Rheumatic Aschoff nodules

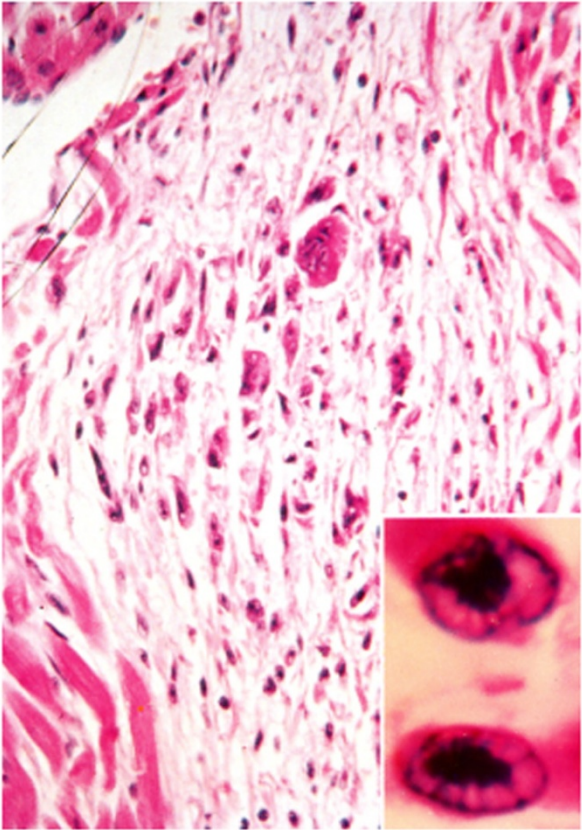
- Fibrinoid necrosis
- Fence-forming histiocytic wall
- Lymphocytes (rare)

Crohn's disease Granuloma

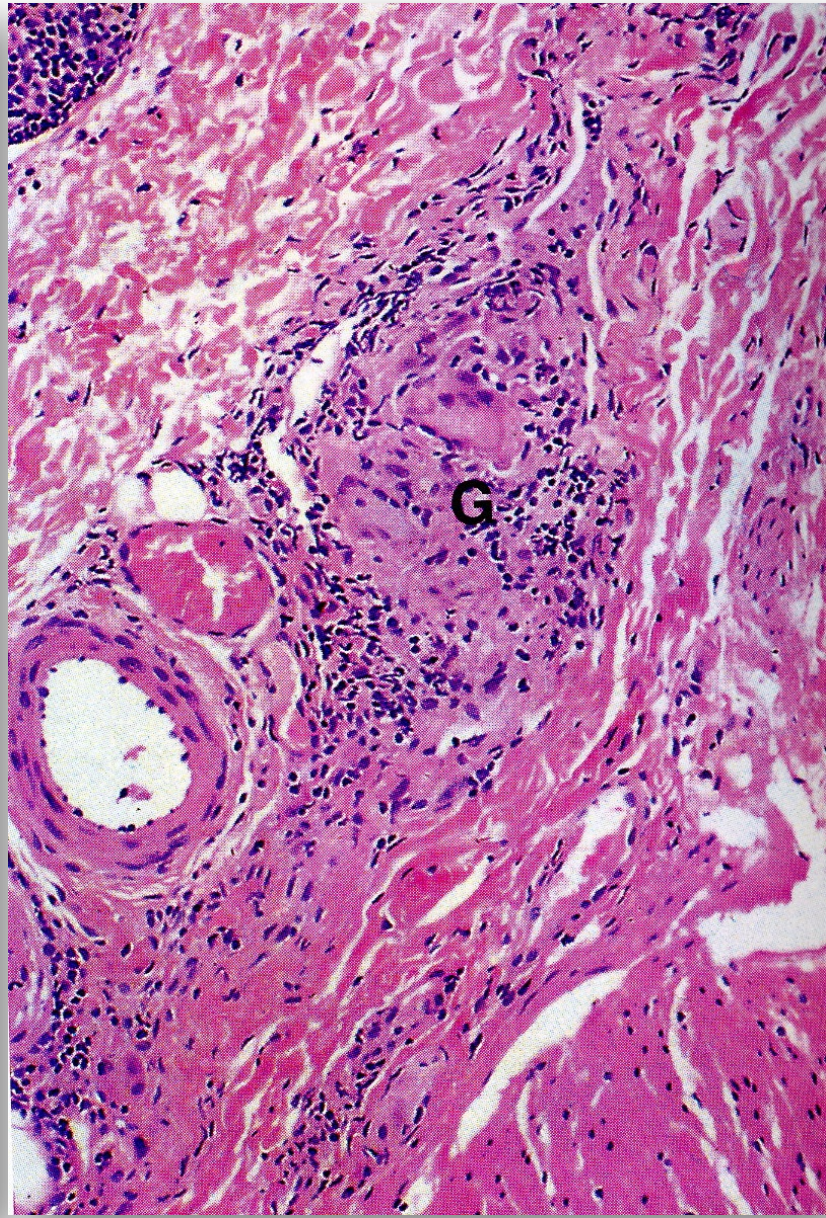
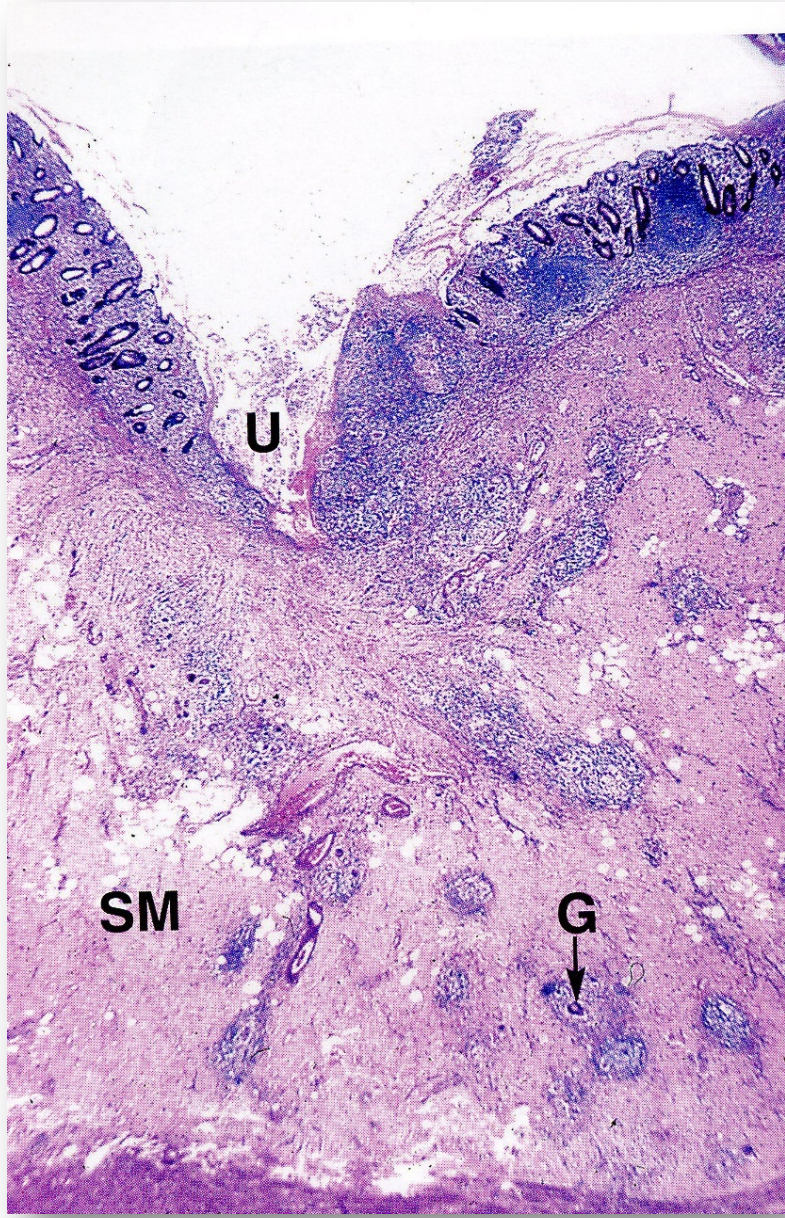
- Diffuse (transmural) inflammation
- Epithelioid cells (scarce)
- Rare giant cells
- Lymphocytes



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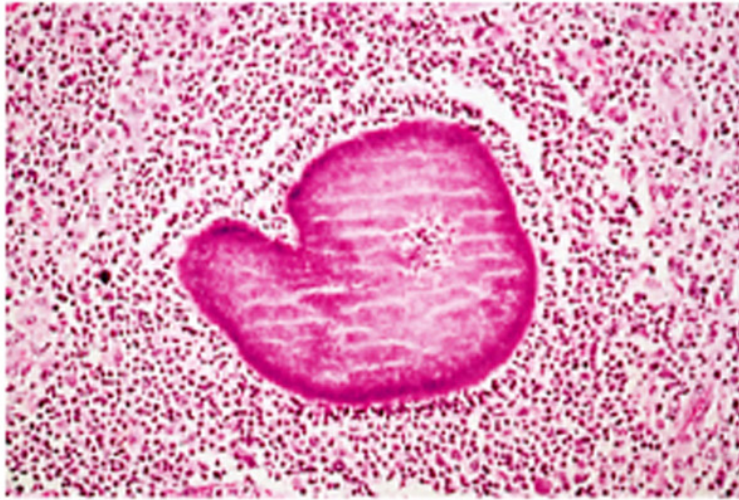
CHRONIC GRANULOMATOUS INFLAMMATION

Actinomycotic Granuloma

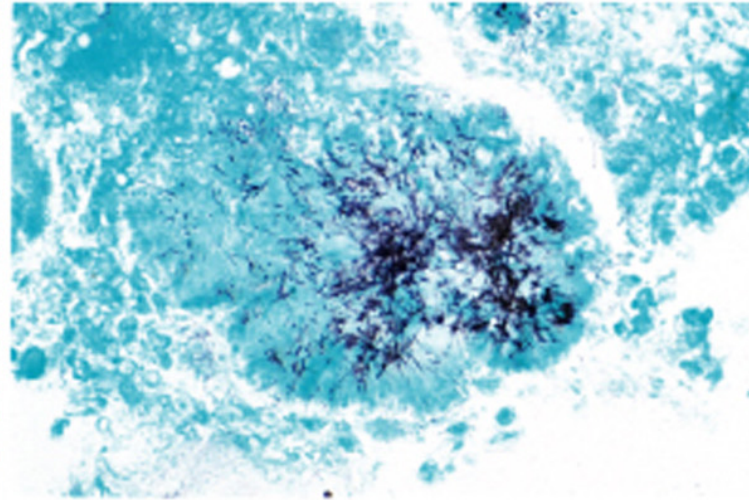
- Druse PAS+
- Granulocytic wall
- Macrophages

Lymphogranuloma venereum

- Central abscessed necrosis
- Palisading Histiocytes
- Lymphocytes and plasma cells



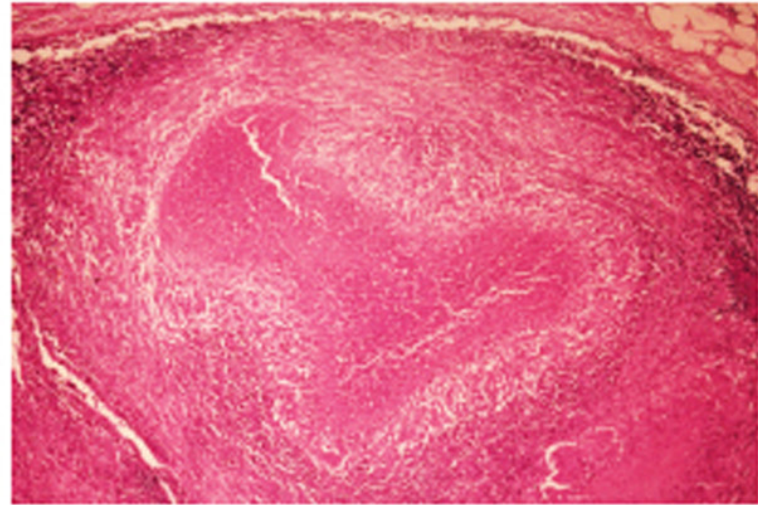
A



B



A



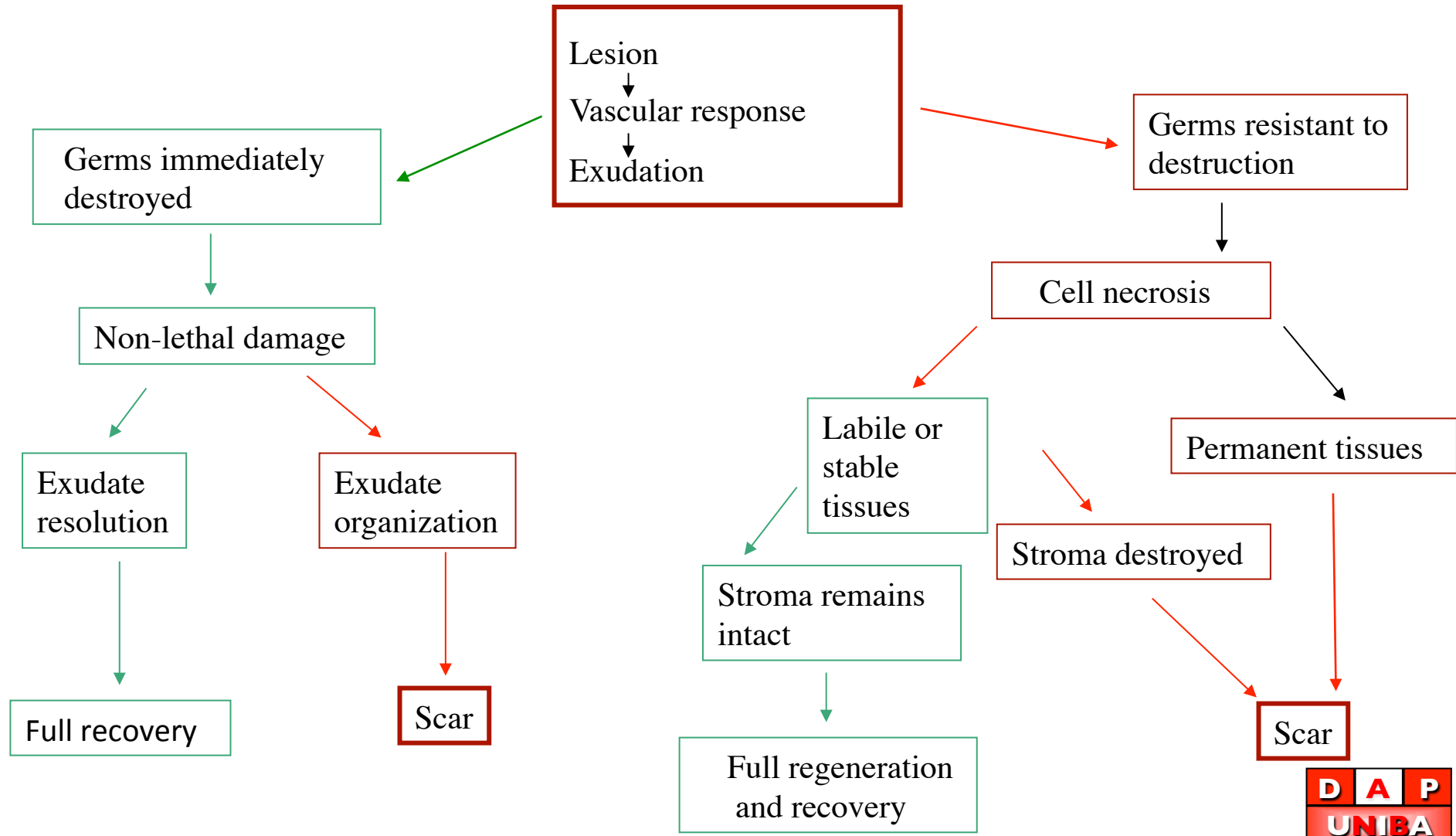
B

CHRONIC GRANULOMATOUS INFLAMMATION

Rhinoscleroma

- Globular macrophages with foamy cytoplasm (Mikulicz cells), containing bacilli (*Klebsiella Rhinoscleromatis*)
- Plasma cells with Russell bodies (cells with yellowish globules)

Inflammation: reparative phase



SCAR

1 – Elimination of tissue debris (macrophages)

2 – Organization process

Endothelial cell proliferation
Fibroblasts proliferation
+
PMN, lymphocytes, plasma cells etc.
↓
Inflammatory granulation tissues

3 - Fibrosis with sclerosis

Loss of tissue

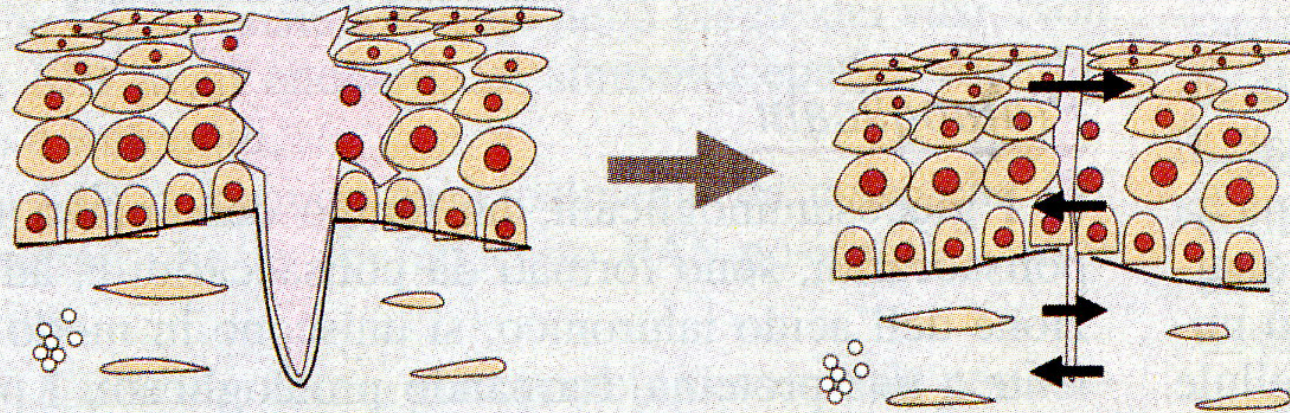
Vascularization
Extent of necrosis
Tissue type
Etc.

Factors affecting the repair process

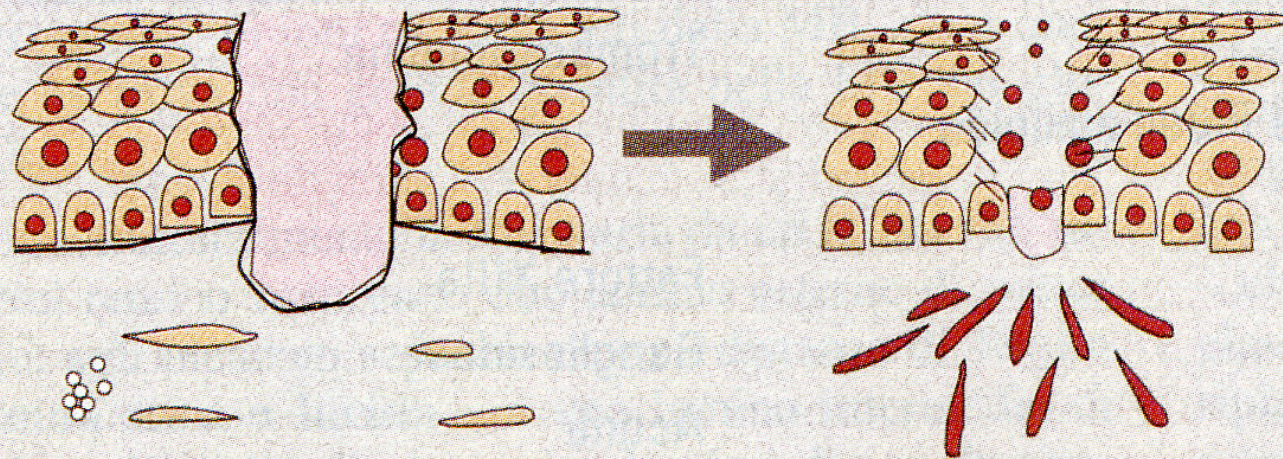
Local

General

Age
Nutritional state
State of immunity
Disendocrine states



Healing by primary intention



Healing by secondary intention

