MORPHOLOGIC PATTERNS AND OUTCOMES OF ACUTE INFLAMMATION

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Learning objectives:

- By the end of this lecture student should be able to;
- Enumerate special morphologic patterns of inflammation
- Describe those patterns
- Discuss outcomes of acute inflammation
- Describe defects of inflammation

- The morphologic hallmarks of acute inflammatory reactions are;
- Dilation of small blood vessels
- Accumulation of leukocytes and proteins
- Fluid in the extravascular tissue
- However, special morphologic patterns are often superimposed on these general features.

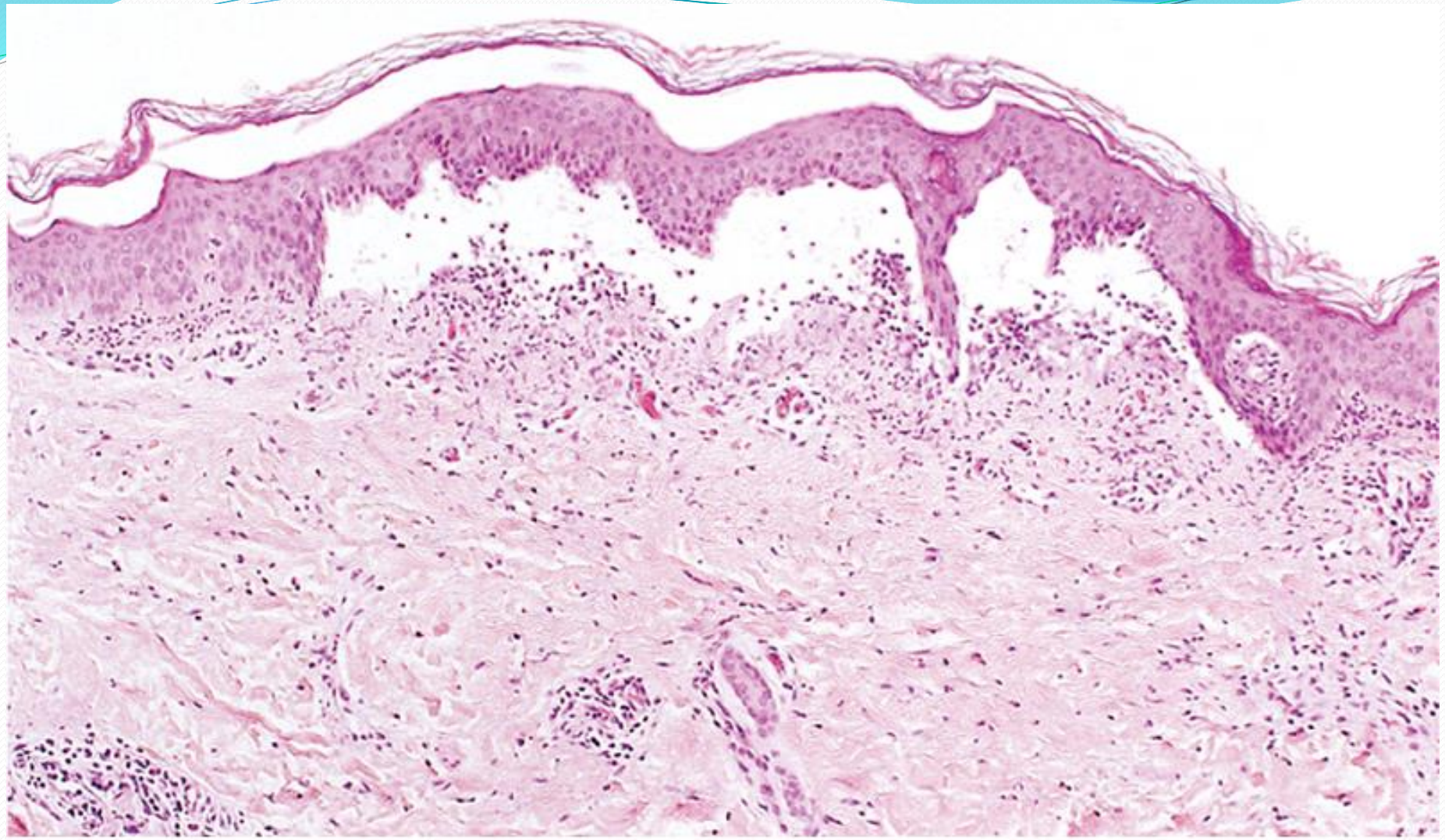
- Special circumstances and specialized tissues.
- Morphologic patterns of acute inflammation are;

1. Serous inflammation
2. Fibrinous inflammation
3. Suppurative (purulent) inflammation and abscess

Serous inflammation:

- There is outpouring of a watery, protein-poor fluid.
- Fluid is derived either from blood due to increased vascular permeability in inflammation.
- Or from the secretions of epithelial cells lining the peritoneal, pleural, and pericardial cavities (local irritation).
- The skin blister resulting from a burn or viral infection is a good example of a serous inflammation.

- Fluid in a serous cavity is called an **effusion** (peritoneal, pericardial and pleural cavities).
- They are named after their cavities as
- Pericardial effusion
- Pleural effusion
- Peritoneal effusion more commonly named as Ascites (from the Greek *askítes* meaning "baglike).
- Biologic purpose is immediate dilution of injurious agent.

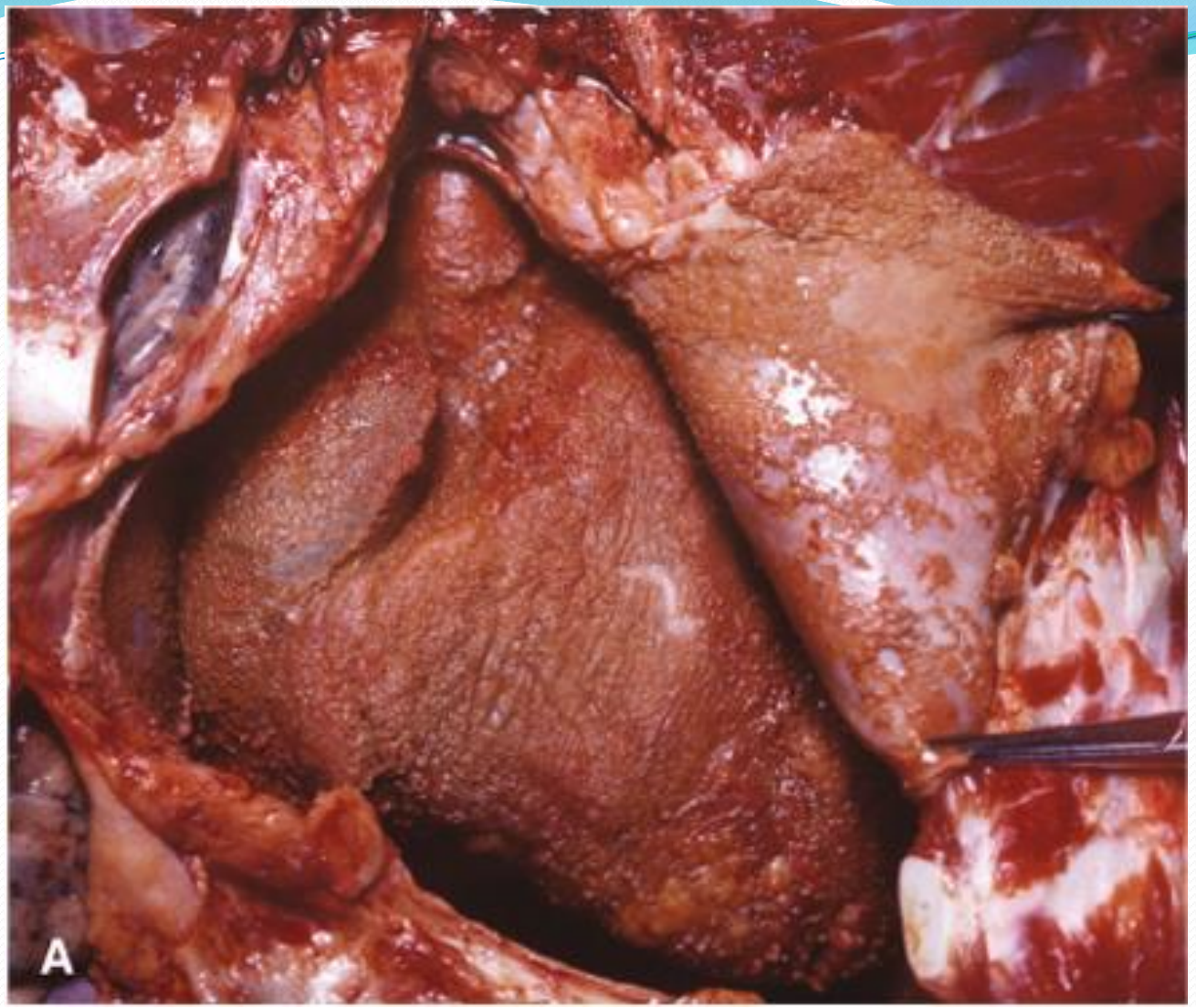


Serous inflammation. Low-power view of a cross-section of a skin blister showing the epidermis separated from the dermis by a focal collection of serous effusion.

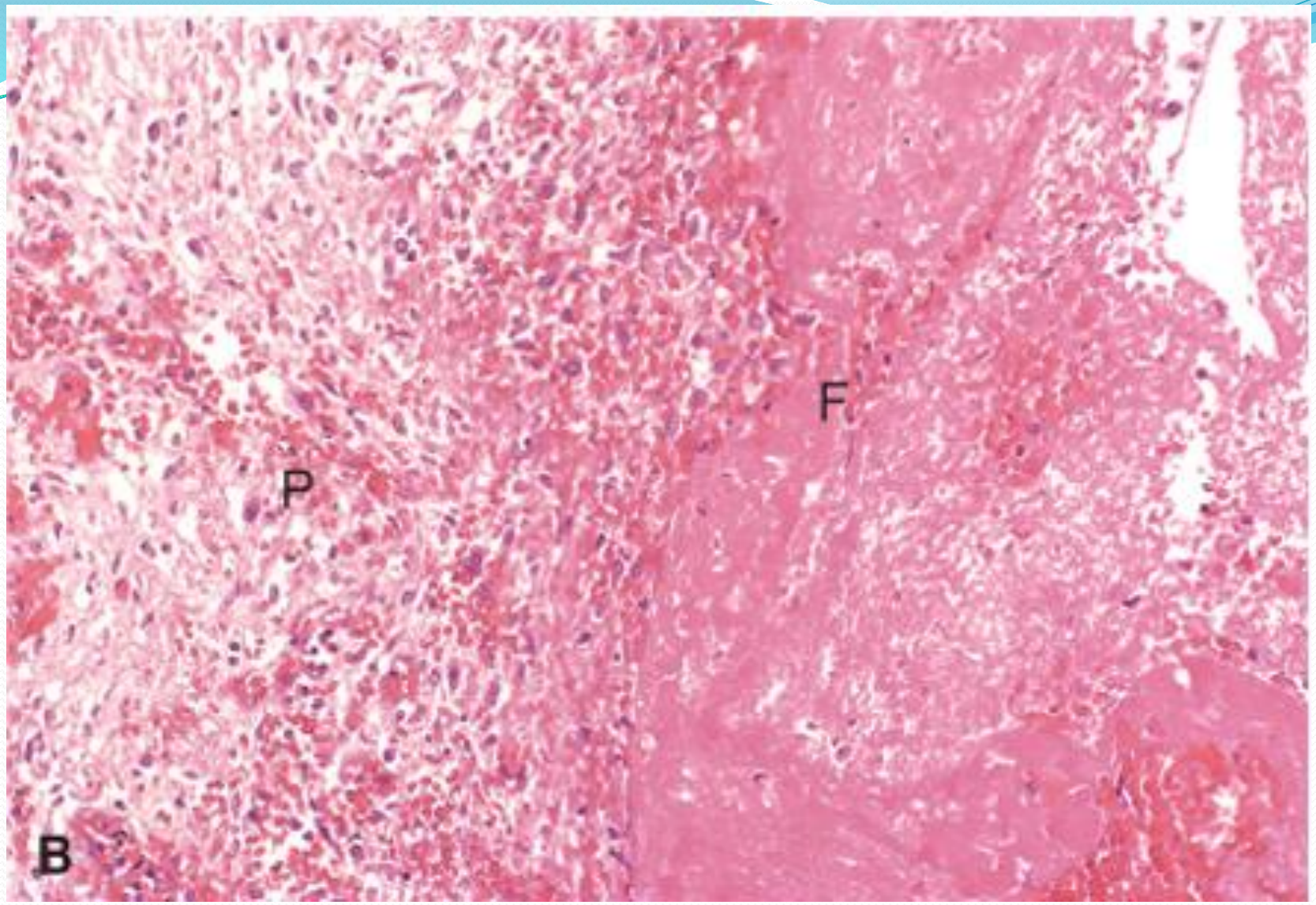
Fibrinous inflammation

- It occurs as a consequence of more severe injury, resulting in greater vascular permeability that allows large molecules (such as fibrinogen) to pass the endothelium.
- Fibrinogen converted into fibrin in extracellular spaces.
- Microscopically the accumulated fibrin appears as a pinkish network of threads.

- Characteristic of inflammation in the lining of body cavities, such as the meninges, pericardium, and pleura.



Deposits of fibrin on the pericardium



A pink meshwork of fibrin exudate (F) overlies the pericardial surface (P).

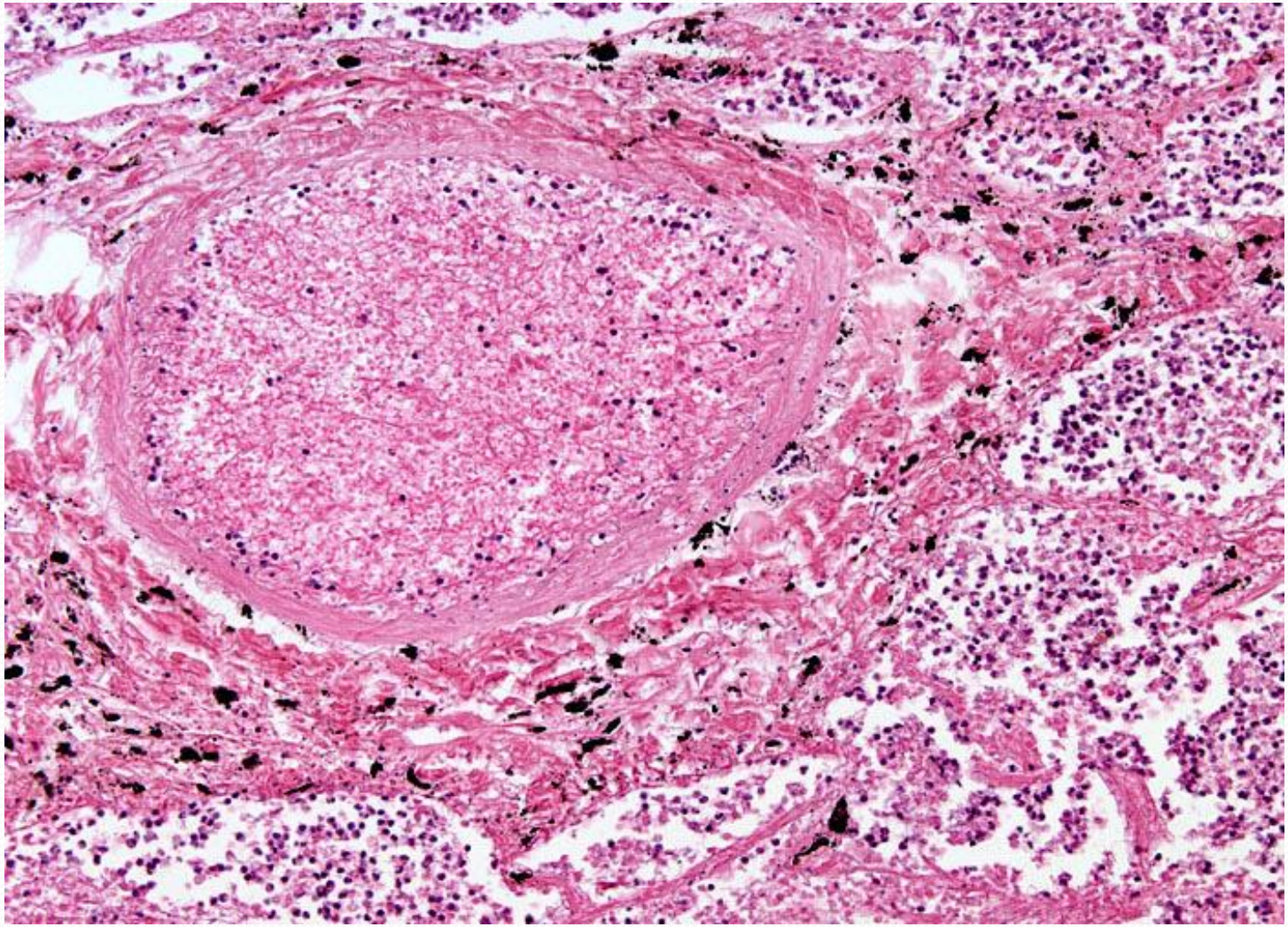
- Fibrinous exudates may be degraded by fibrinolysis resulting in restoration of the normal tissue structure.
- But failure to completely remove the fibrin results in the ingrowth of fibroblasts and blood vessels leading ultimately to scar formation.
- Fibrous scar tissue that fills and obliterates the pericardial or pleural space may restrict lung or heart function if fibrosis is extensive.

Suppurative inflammation and Abscess

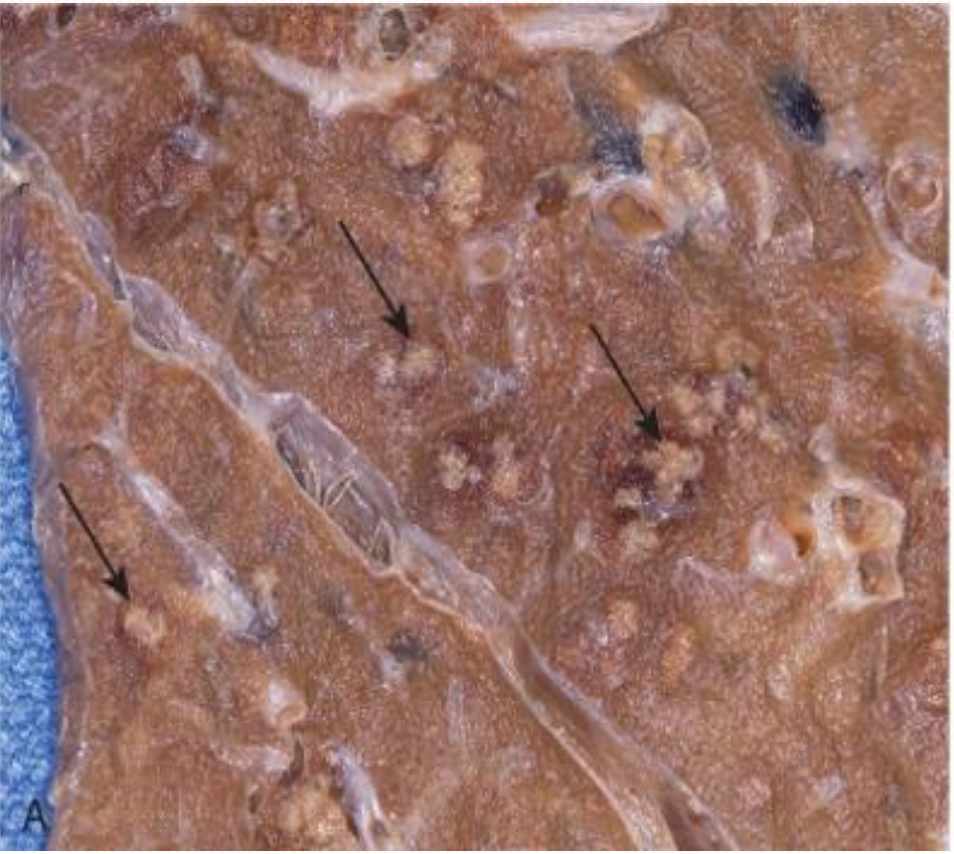
- Also called purulent inflammation.
- There is presence of large amounts of purulent exudate (pus)
- Purulent means related to pus
- Pus is a thick creamy liquid and composed of
- A large number of living or dead leukocytes (neutrophils/pus cells)
- Liquefied debris of necrotic tissue
- Living and dead bacteria
- Edema

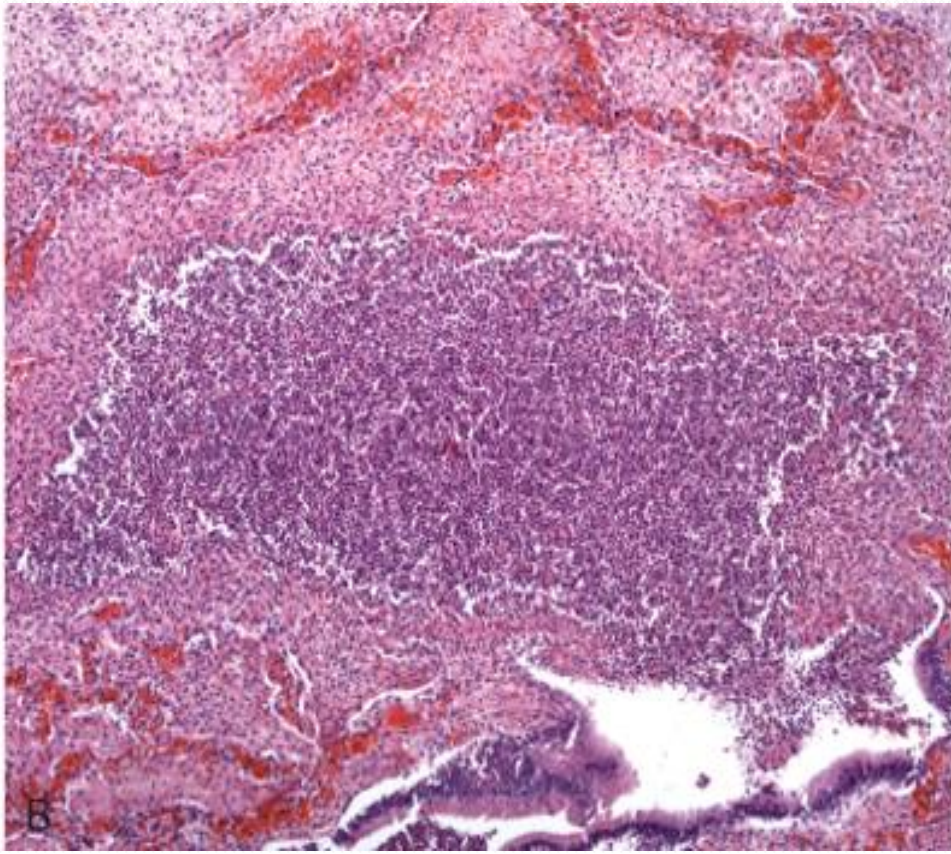
- Most common cause of purulent inflammation is bacterial infection.
- Organisms (e.g., staphylococci) which induce pus formation are called pyogenic organisms.
- Pyogenic means pus forming.
- A common example of an acute suppurative inflammation is acute appendicitis.

- **Abscesses** are focal collections of pus caused by pyogenic organisms buried into a tissue, organ or closed space.
- Seeding of organisms
- Abscesses have a central necrotic region surrounded by a layer of neutrophils which is again surrounded by dilated vessels and fibroblasts.
- As time passes the abscess may become completely replaced by connective tissue laid down by fibroblasts .



Pulmonary abscess





A, Multiple bacterial abscesses in the lung (*arrows*) in a case of bronchopneumonia. **B,** The abscess contains neutrophils and cellular debris, and is surrounded by congested blood vessels.

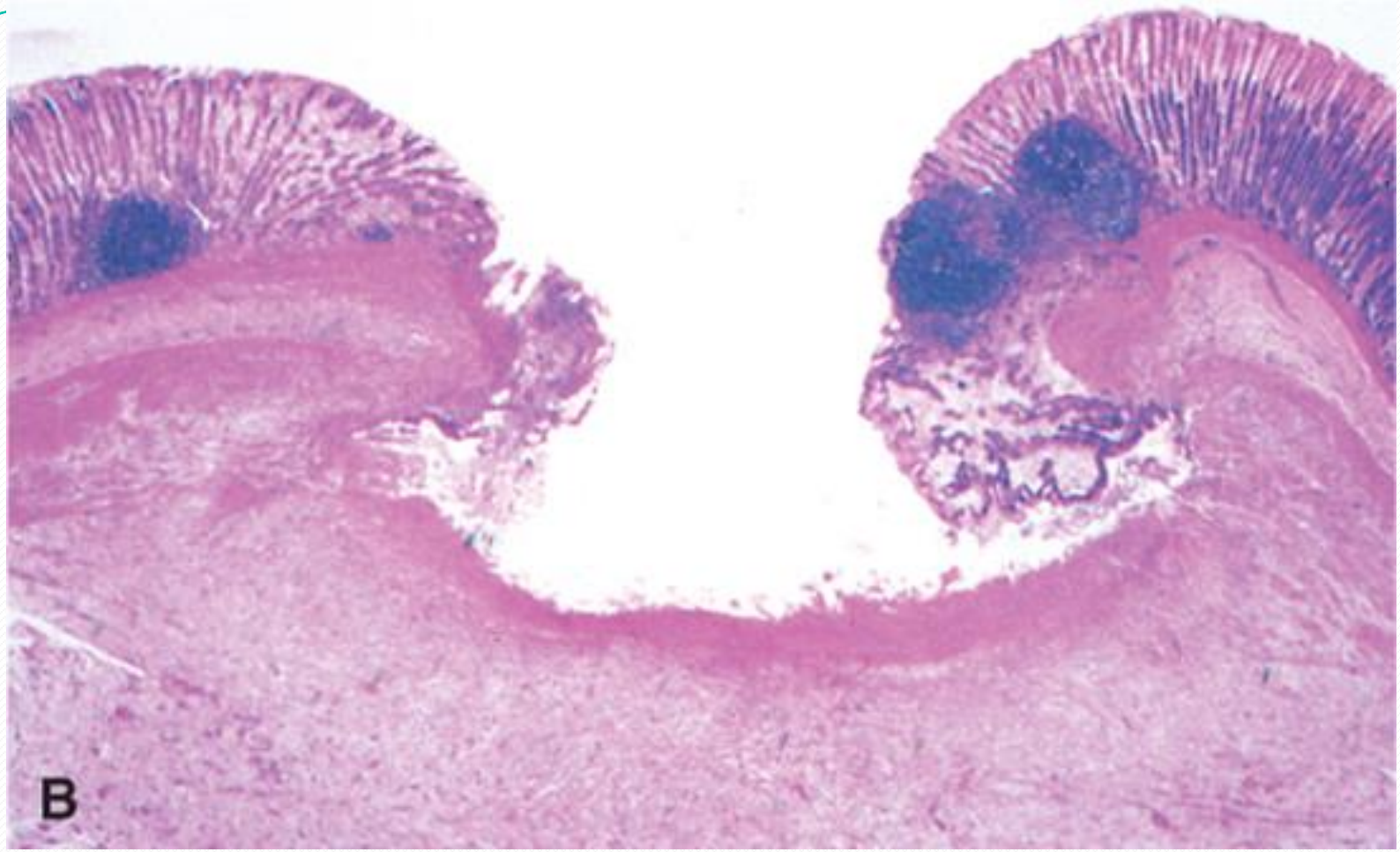
- An **ulcer** is a local defect in the surface of an organ or tissue that is produced by necrosis of cells and shedding of those cells.
- Ulceration can occur only when inflammation and tissue necrosis exist on or near a surface.
- It is most commonly seen in

1. The mucosa of the mouth, stomach, intestines and genitourinary tract.

2. The skin and subcutaneous tissue of the lower extremities in older persons who have circulatory disturbances that predispose them to ischemic necrosis.

- Best examples of ulcer are peptic ulcer of the stomach or duodenum.

A chronic duodenal ulcer



Low-power cross-section of a duodenal ulcer crater with an acute inflammatory exudate in the base.

Outcomes of Inflammation:

- Acute inflammatory reactions typically have one of three outcomes;

1. **Complete resolution**

- All inflammatory reactions, once have succeeded in eliminating the injurious agent, would end with restoration of tissue to normal.
- Is usual outcome when injury is limited, short-lived, and with little tissue destruction. Damaged parenchymal cells can regenerate.

- Involves removal of cellular debris and microbes by macrophages , resorption of edema by lymphatics followed by regeneration of damaged tissue.

2. **Healing by fibrosis or scarring:**

- Occurs after considerable tissue damage
- When injury involves tissues that are incapable of regeneration
- When there is abundant fibrin exudation in tissue or in serous cavities.

3. Progression to chronic inflammation

- Acute inflammation becomes chronic when acute response cannot be resolved either due to the persistence of injurious agent or some interference with the normal healing.

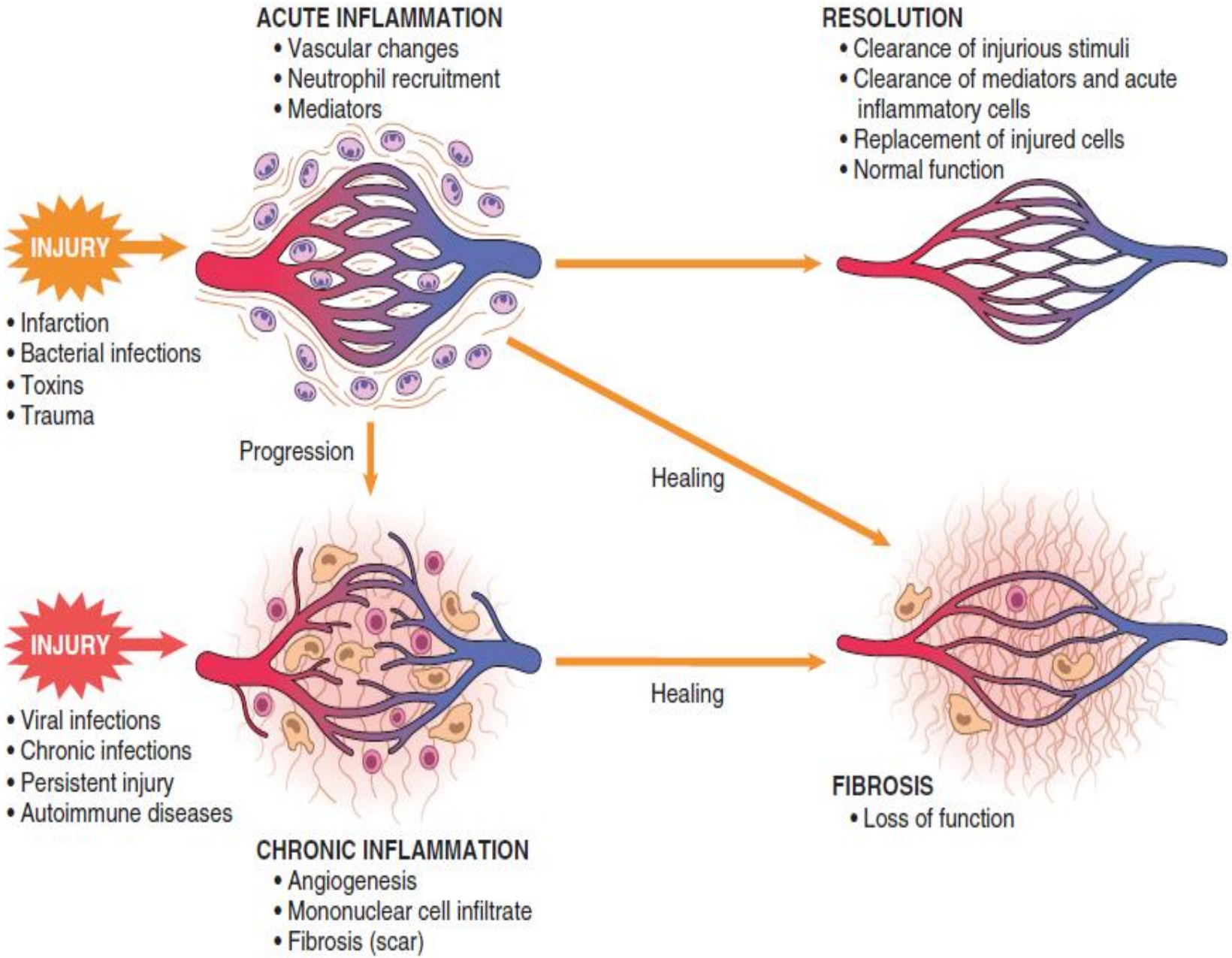


Figure 3.17 Outcomes of acute inflammation: resolution, healing by fibrosis, or chronic inflammation. The components of the various reactions and their functional outcomes are listed.

Defects of inflammation:

- Activated leukocytes cause injury to the normal tissue under different circumstances.

1. Normally they cause certain level of damage to adjacent normal tissues as a collateral damage.

- When inflammatory response is prolonged e.g. tuberculosis or certain viral infections this collateral damage becomes the main pathology

instead of offending agent.

- Similarly inflammatory response is counterproductive when misdirected against self antigens as in autoimmune disorders.
- Host response may be misdirected against harmless substances as occurs in allergic or hypersensitivity conditions.

- Lysosomes release their contents to kill microorganisms.
- These contents along with killed organisms are also discharged outside the cell in a controlled manner.
- When leukocytes encounter substances which they cannot phagocytose like immune complexes present on glomerular basement membrane, they empty lysosomal contents in external environment (frustrated phagocytosis).

- There may be genetically acquired defects of leukocyte functions.

1. **Chediak–Higashi syndrome:** disorder of lysosomal granules; failure of fusion of lysosomes with phagosomes to form phagolysosomes
2. **Chronic granulomatous disease of childhood:** X-linked/autosomal recessive disease characterized by absence of NADPH oxidase
3. **Myeloperoxidase deficiency:** absent MPO–H_2O_2 halide system

THANK YOU