

# Inflammation II

Outcomes of inflammation with  
clinical examples

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# Acute Inflammation: Sequelae

## RESOLUTION

(see Acute Inflammation 1 lecture)

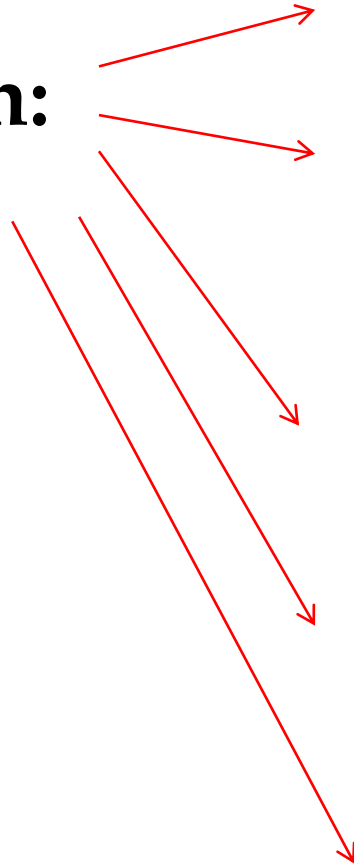
## SUPPURATION

+/- abscess formation  
+/- discharge of pus

## CHRONIC INFLAMMATION

## ORGANISATION AND REPAIR

## FIBROSIS



# SUPPURATION

## *Definitions*

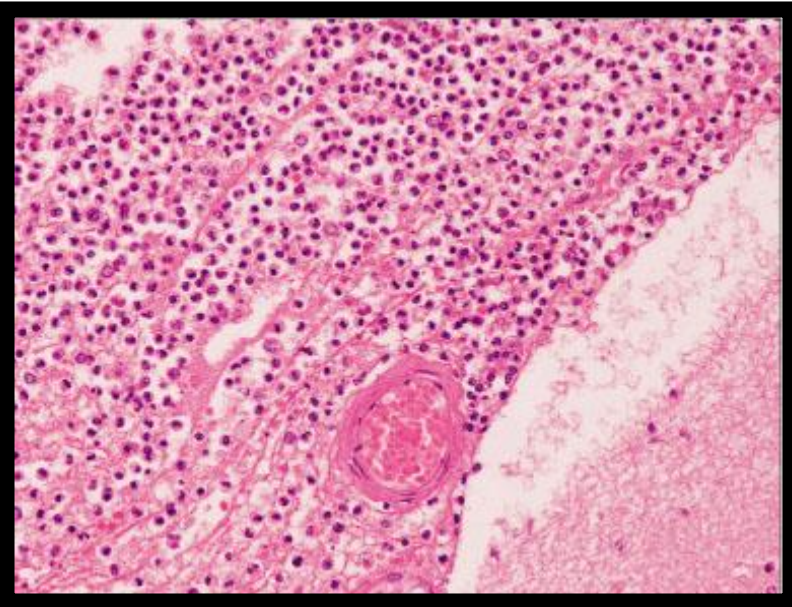
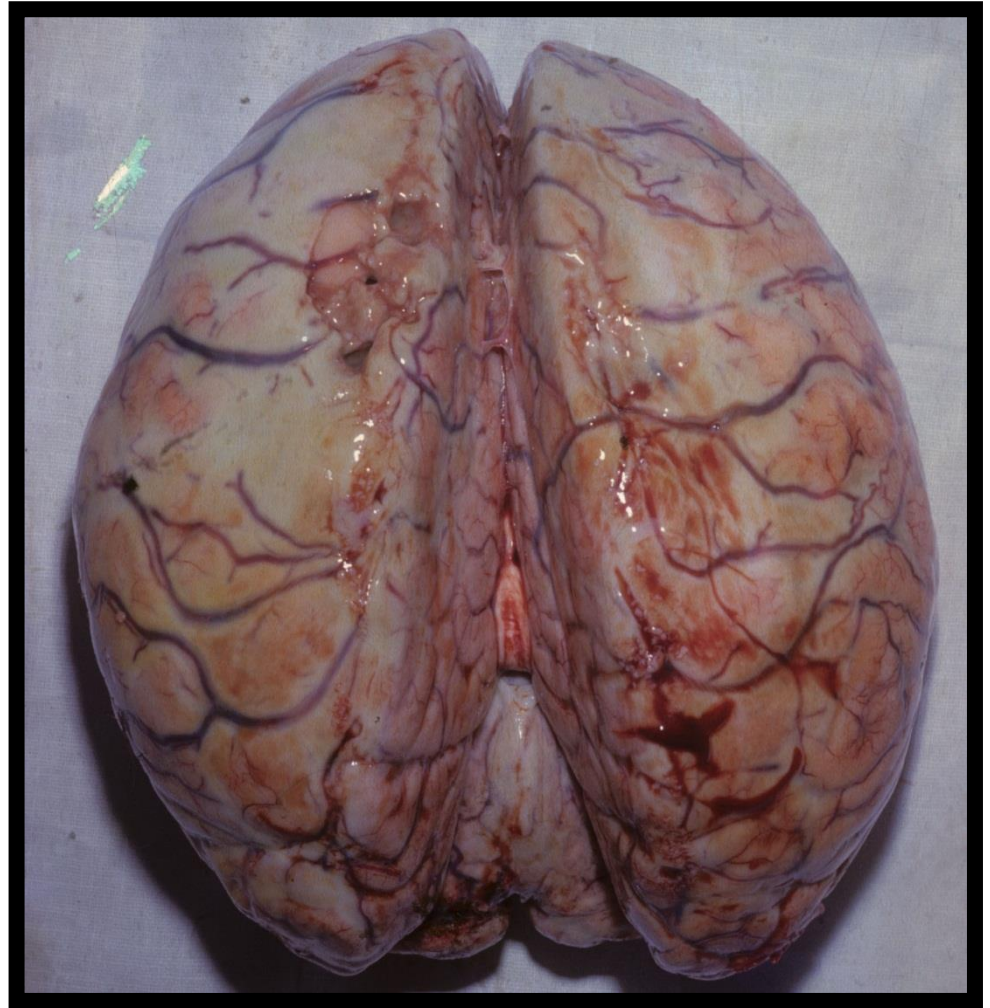
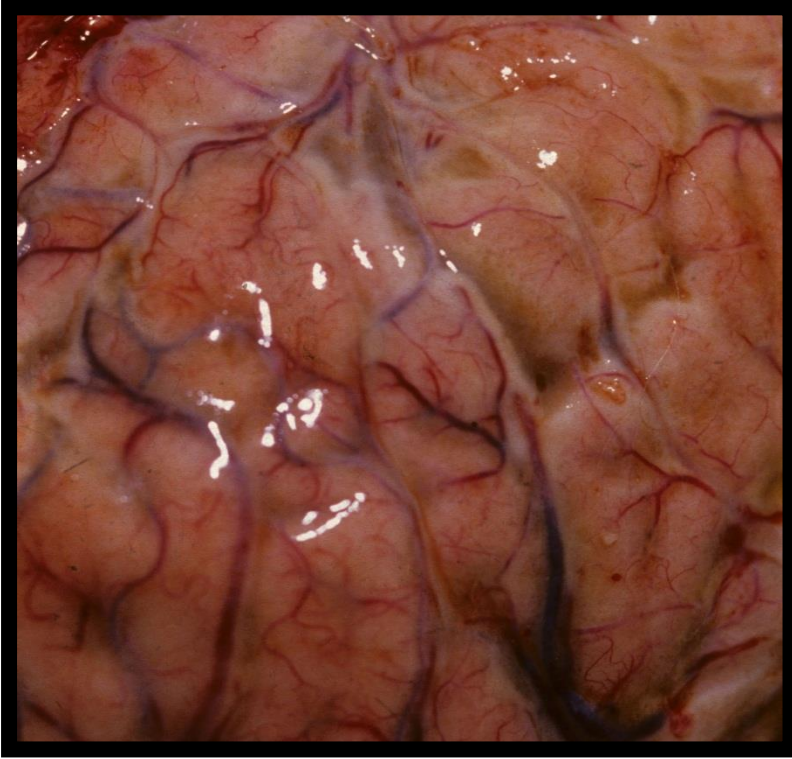
**Suppuration:** the formation of pus.

**Pus:** an accumulation of dead and living neutrophils, dead and living bacteria ( when inflammation caused by **pyogenic bacteria**), protein (especially fibrin) and other particulate matter (eg cell fragments etc)

**Abcess:** a pus-filled cavity

**Empyema:** an accumulation of pus in a naturally occurring body cavity

*This type of exudate is referred to as '**suppurative**' or '**purulent**'*



**Suppurative meningitis**  
*Note creamy yellow exudate around vessels in subarachnoid space*  
*Micro: Exudate predominantly neutrophils, fibrin, bacteria*

# Evolution of an abscess

Starts as an acute **inflammatory exudate with many neutrophils**. Proteins (mainly fibrin), bacteria and polymorphs aggregate to form a mass

**Tissue death (necrosis)** ensues

New capillaries and fibroblasts develop at edge of accumulated material = '**granulation tissue**' (process of **organisation**)

Fibroblasts start to lay down scar tissue (collagen)

Pus resorbed (if small amount) or can burst onto ('point') to external surface (**sinus**) or adjacent body cavity (**fistula**) and be discharged in this way

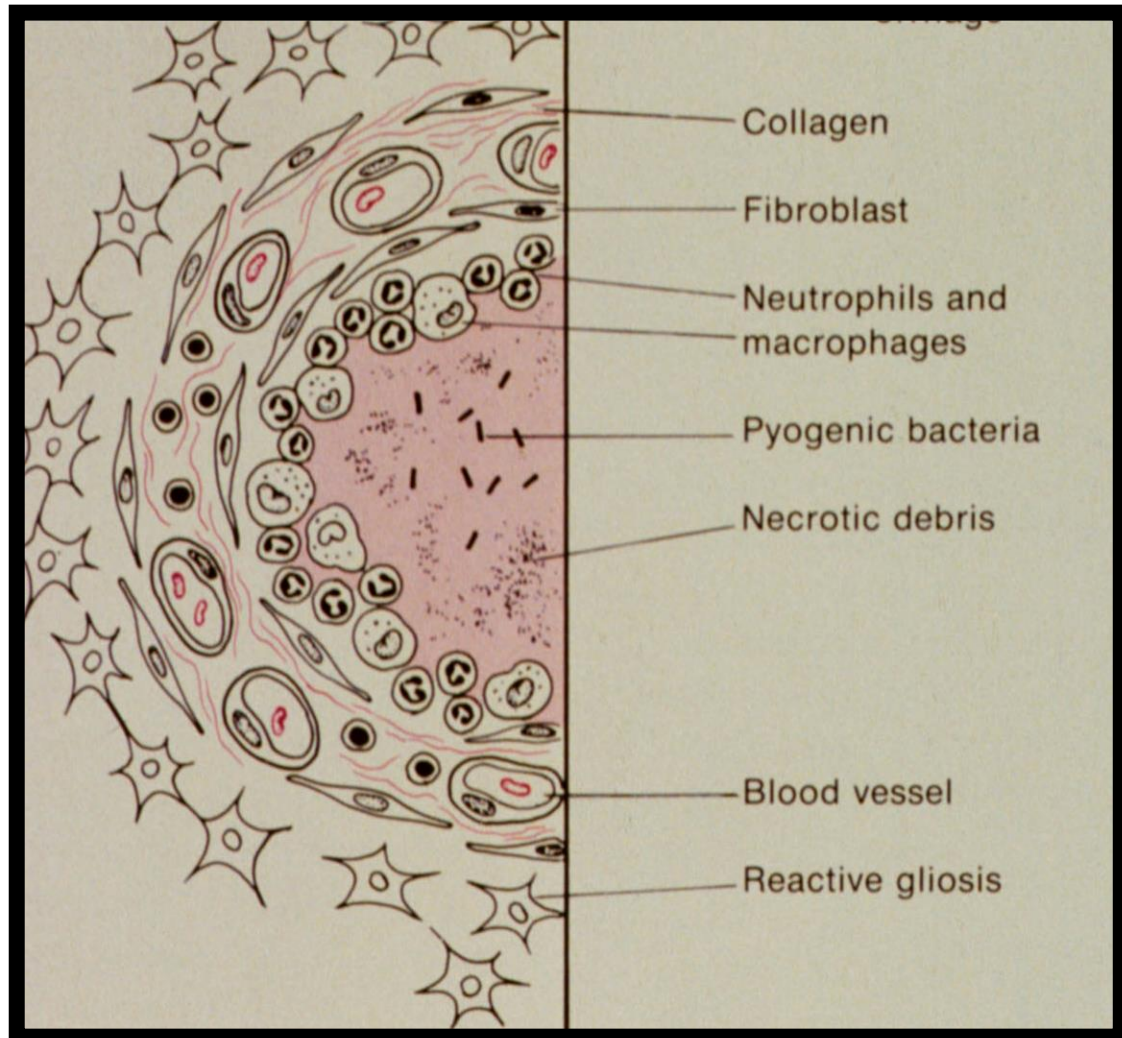
Collagen deposition proceeds to formation of mature scar







Brain abscess  
Pus filled cavity with  
peripheral organisation





# Wall of abscess

Note: suppurative exudate and surrounding organisation



**EXAMPLES of ACUTE  
INFLAMMATORY REACTION IN  
THE LUNG caused by PYOGENIC  
BACTERIA**

***(Bacterial Pneumonia)***

- **Lobar pneumonia**
- **Bronchopneumonia**





**LOBAR  
PNEUMONIA  
*S. Pneumoniae***

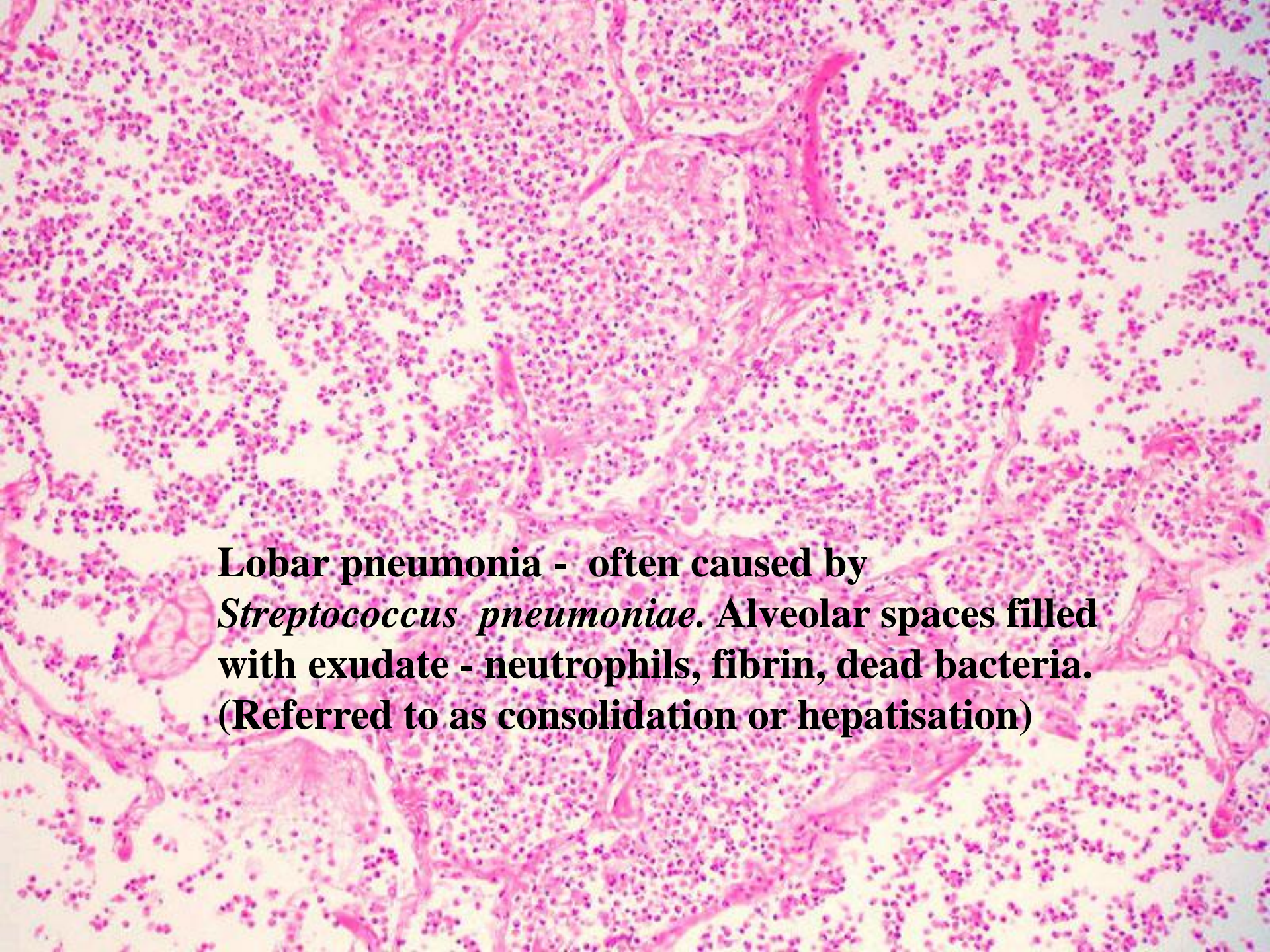
**Note consolidation  
(hepatisation) of  
lower lobe**



**Consolidation of  
entire lobe**





A high-magnification photomicrograph of lung tissue stained with hematoxylin and eosin (H&E). The image shows alveolar spaces that are densely packed with a pink-staining exudate, characteristic of consolidation in lobar pneumonia. The exudate contains numerous small, dark-staining nuclei, likely neutrophils, and some larger, more irregular structures that could be dead bacteria. The surrounding lung architecture, including alveolar walls and some capillaries, is visible but partially obscured by the dense infiltrate.

**Lobar pneumonia - often caused by *Streptococcus pneumoniae*. Alveolar spaces filled with exudate - neutrophils, fibrin, dead bacteria. (Referred to as consolidation or hepatisation)**

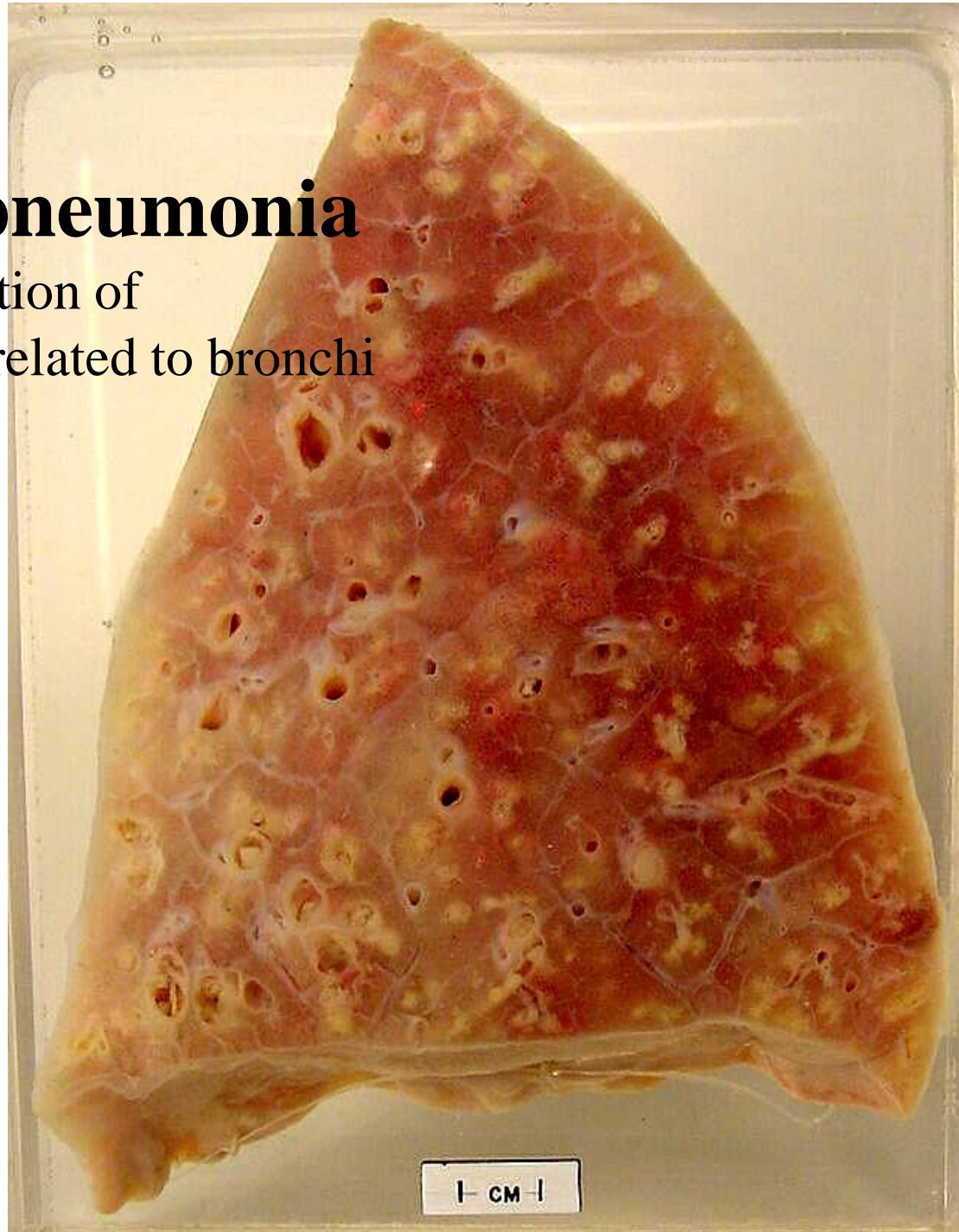
# Outcomes of pneumonia

- Resolution
- Abscess formation
- Empyema
- Fibrosis and scarring
- Septicaemia
- Death

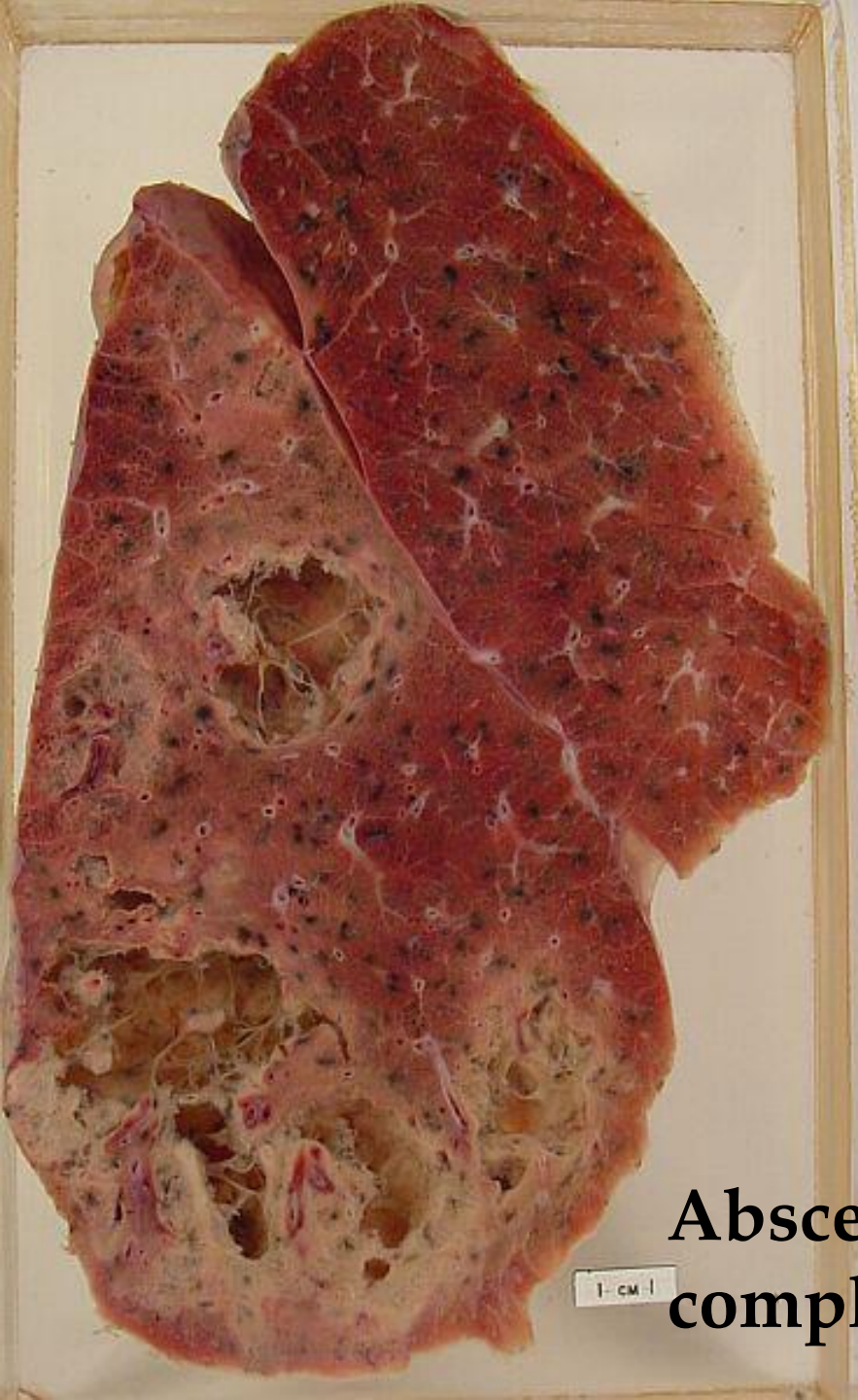


# Bronchopneumonia

Patchy distribution of consolidation, related to bronchi







**Abscess formation  
complicating pneumonia**

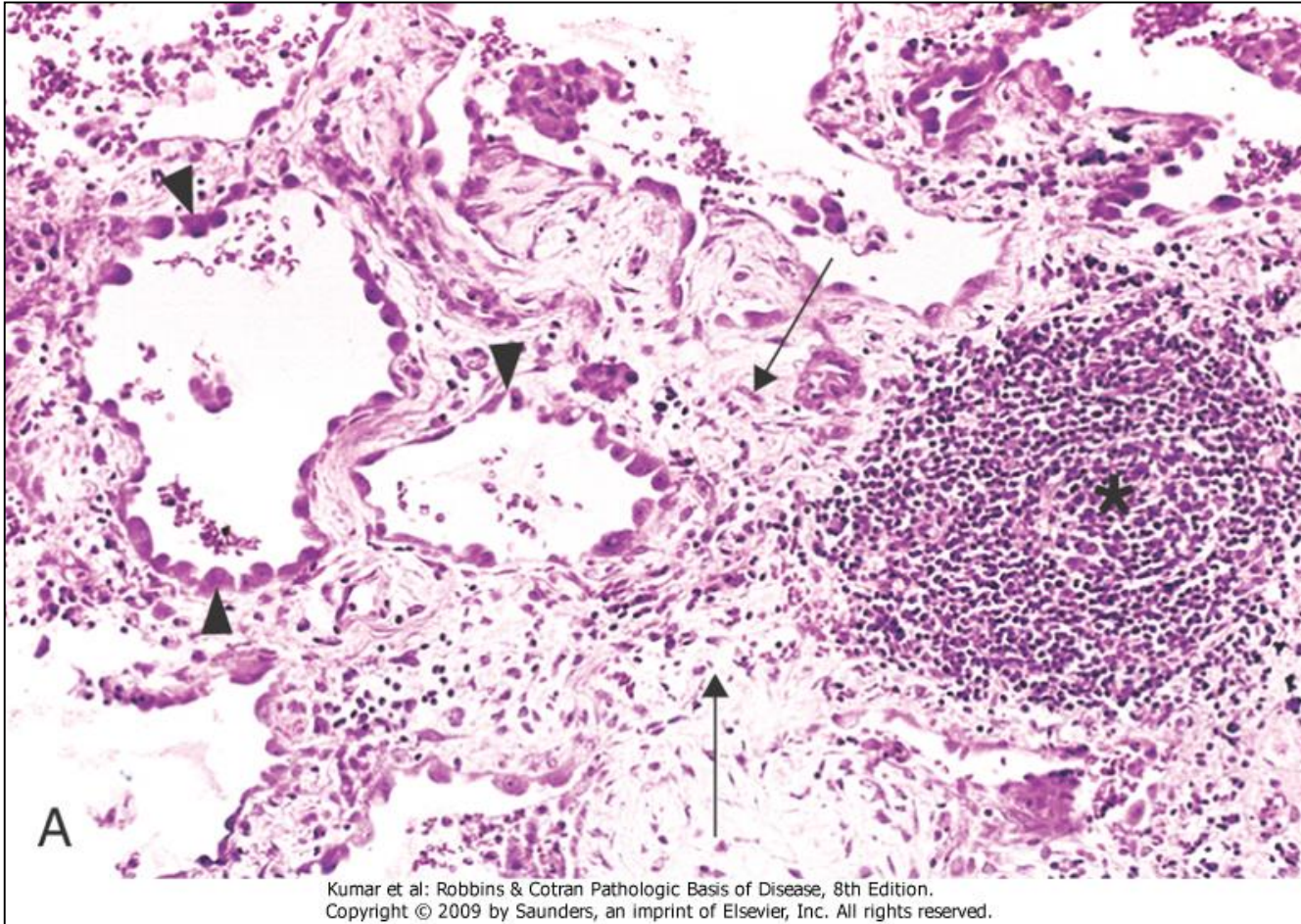
<https://healthed.hms.uwa.edu.au/pelc/search.php>

# CHRONIC INFLAMMATION

## *Description:*

- Inflammation enduring longer than acute inflammation
- May be primary but often results from acute inflammation when causative agent cannot be removed
- Polymorphs (neutrophils) largely replaced by lymphocytes, plasma cells (and macrophages)
- Macrophages often fuse to form giant cells
- Often proliferation of vascular endothelium and fibroblasts esp at periphery (= **organisation**)
- Fibrosis





Lung – chronic inflammation  
Note lymphocytic aggregate (\*), interstitial fibrosis  
(long arrows), Type 2 pneumocytes (blunt arrows)

# Example of chronic inflammatory reaction

## CHRONIC PEPTIC ULCER

Chronic ulcer occurring in an area of acid pepsin digestion

Commonly stomach duodenum oesophagus

Often associated with *Helicobacter pylori* infection

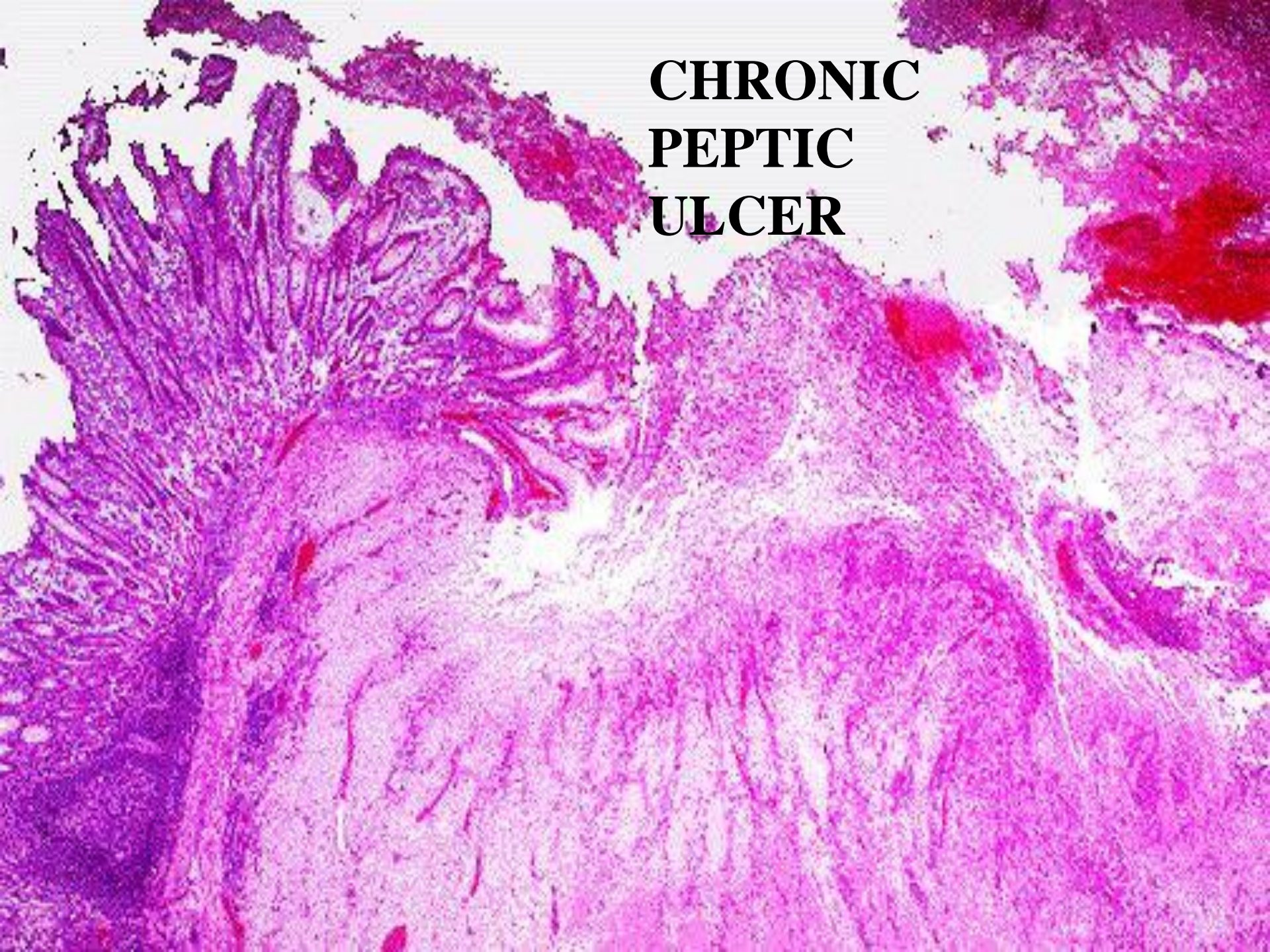


Chronic  
peptic ulcer of  
stomach (\*)





**CHRONIC  
PEPTIC  
ULCER**



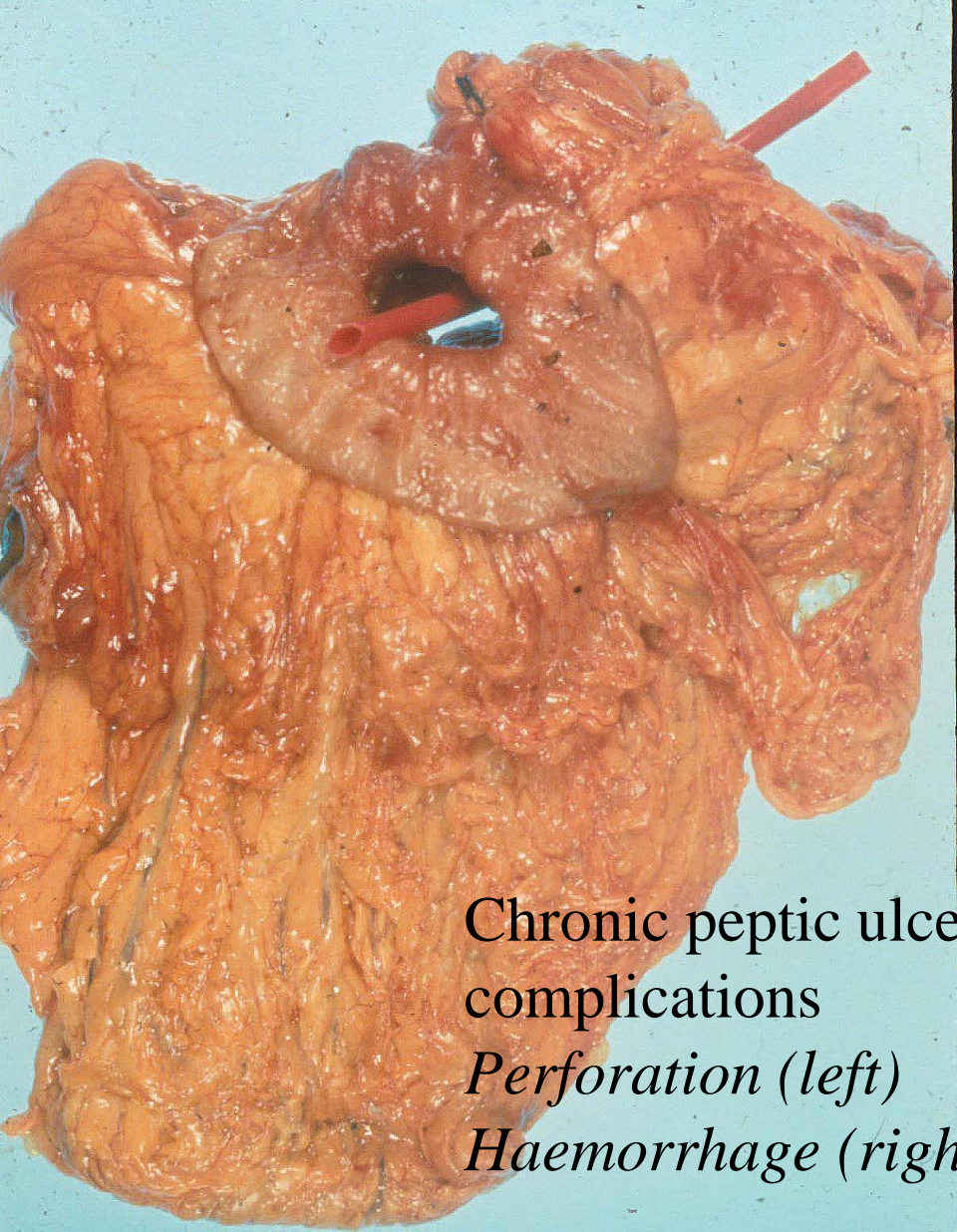


# OUTCOMES OF CHRONIC PEPTIC ULCER

- Resolution - rare without appropriate therapy
- Haemorrhage
- Fibrosis ( $\pm$  stenosis)
- Perforation
- Penetration ( $\pm$  fistula formation)
- Malignant transformation very rare







Chronic peptic ulcer  
complications  
*Perforation (left)*  
*Haemorrhage (right)*





# Example of chronic inflammatory disease

## **Tuberculosis**

*Mycobacterium tuberculosis*



## **Tuberculosis**

Tuberculous meningitis - above  
Tuberculosis of spine (Pott's  
disease) - right



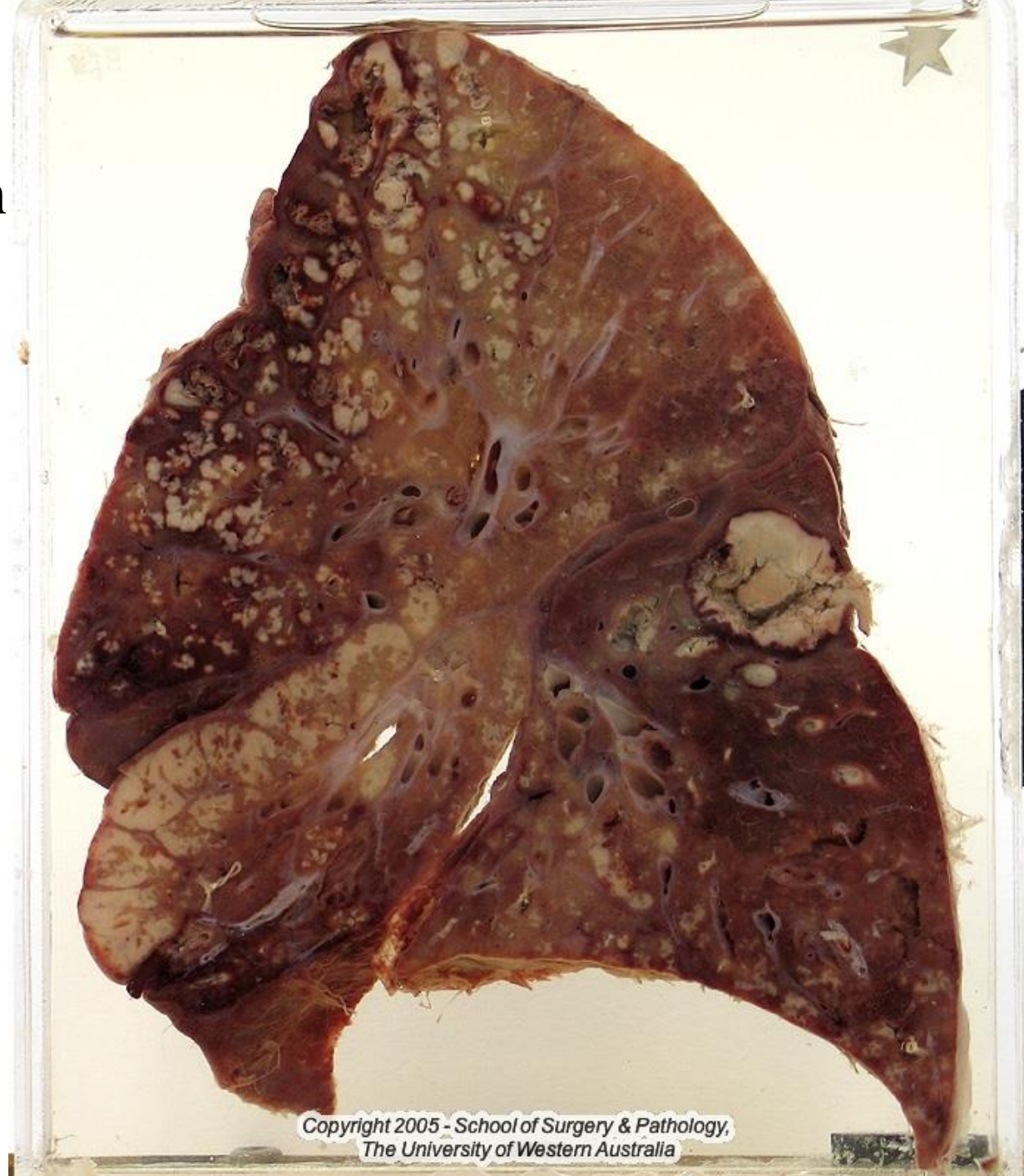


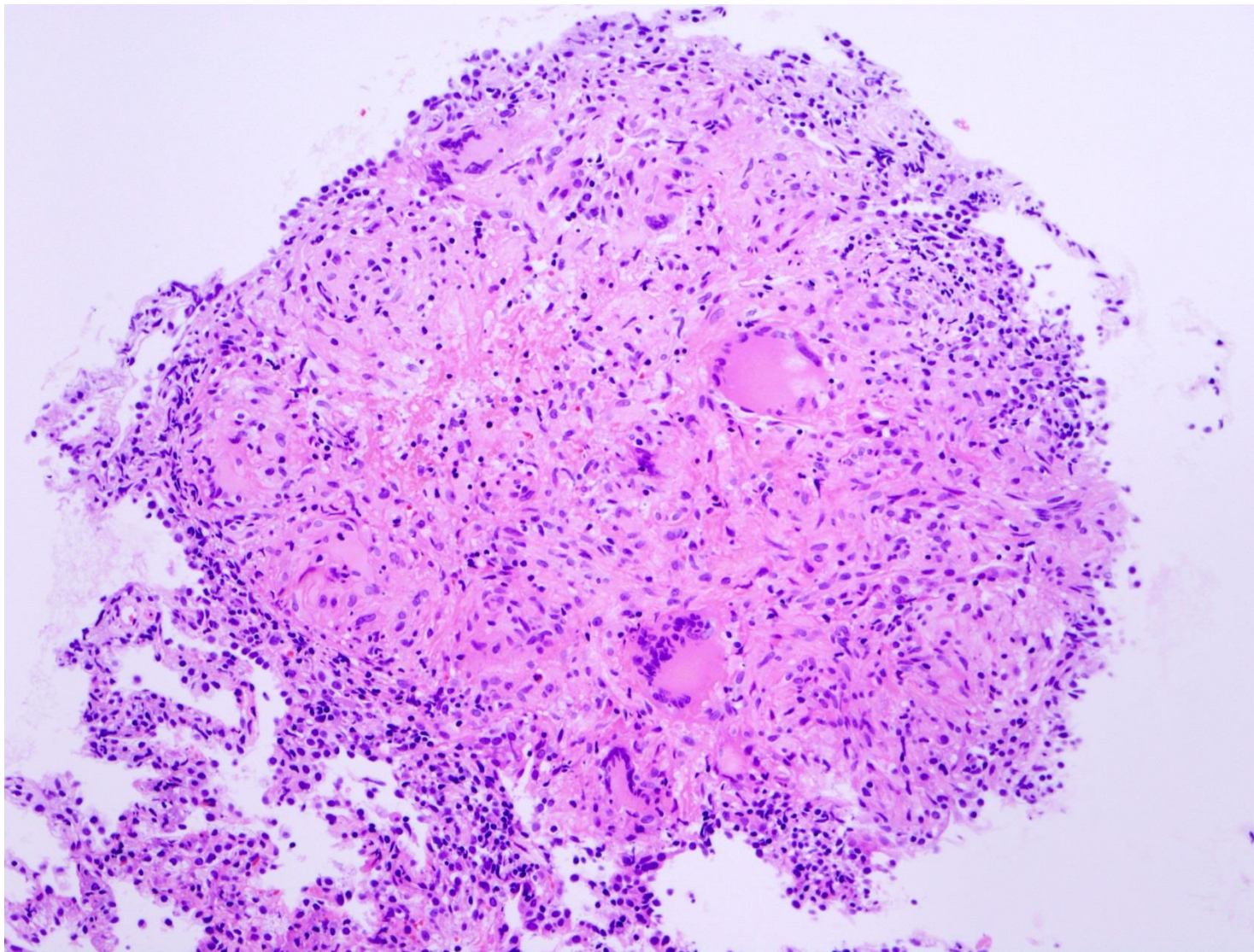
# Tuberculosis

Often associated with  
*'caseous' necrosis*

## LUNG

Caseous tuberculosis  
and tuberculous  
bronchopneumonia





## **Tuberculosis:**

Often associated with *granulomatous inflammation*

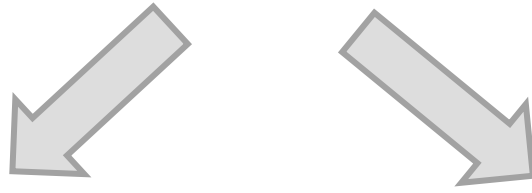
Note epithelioid macrophages and giant cells

**Outcomes of inflammation**

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**Healing and repair**



# Healing



## Resolution

- Removal of exudate
- Regeneration of tissue if possible
- Complete return to normal

## Repair

- Occurs when resolution impossible (severe, ongoing damage , or tissue cannot regenerate)
- Involves formation of granulation tissue (**organisation**)
- Maturation of granulation tissue to scar tissue (fibrosis)

# Organisation

## *Definition*

- The growth of new capillaries and fibroblasts into the damaged tissue together with migration of macrophages. Macrophages remove debris, fibroblasts lay down collagen.
- **New capillaries and fibroblasts = 'granulation tissue'** which matures to form fibrous tissue (collagen).
- Often occurs when exudation or damage is excessive and cannot be removed
- Is the process involved in **repair** (healing) of tissue (when resolution is not possible)



**Example of healing**  
**Healing of a skin wound**

# Healing of a skin wound

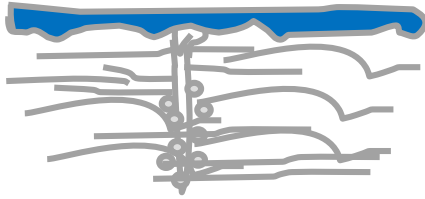
## Healing by primary intention

- Occurs in clean incised wounds with apposed edges (eg surgical wounds)
- Results in minimal scarring
- Occurs in shorter time (mainly healed in a week or two – stitches can be removed)
- Strengthening, devascularisation continues longer

## Healing by secondary intention

- Occurs in open wounds (loss of tissue, necrosis or infection)
- Often results in significant scarring (fibrosis)
- Process may continue for months or years





## Healing by primary intention

**Immediate:** small cavity fills with blood and fibrin

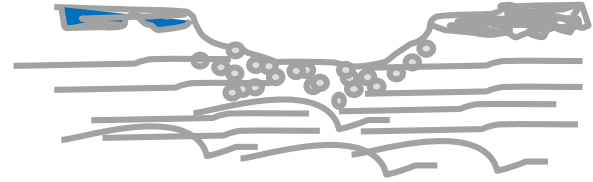
**2-3 hours:** minor inflammation

**2-3 days:** macrophage, fibroblast activity, new vessel formation (ie minimal granulation tissue)

**10-14 days:** re-epithelialisation complete, weak fibrous union

**Weeks:** good fibrous union continues strengthening for **months to years.**

Devascularisation. Minimal scarring



## Healing by secondary intention

**Immediate:** large cavity fills with blood and fibrin. Acute inflammation begins.

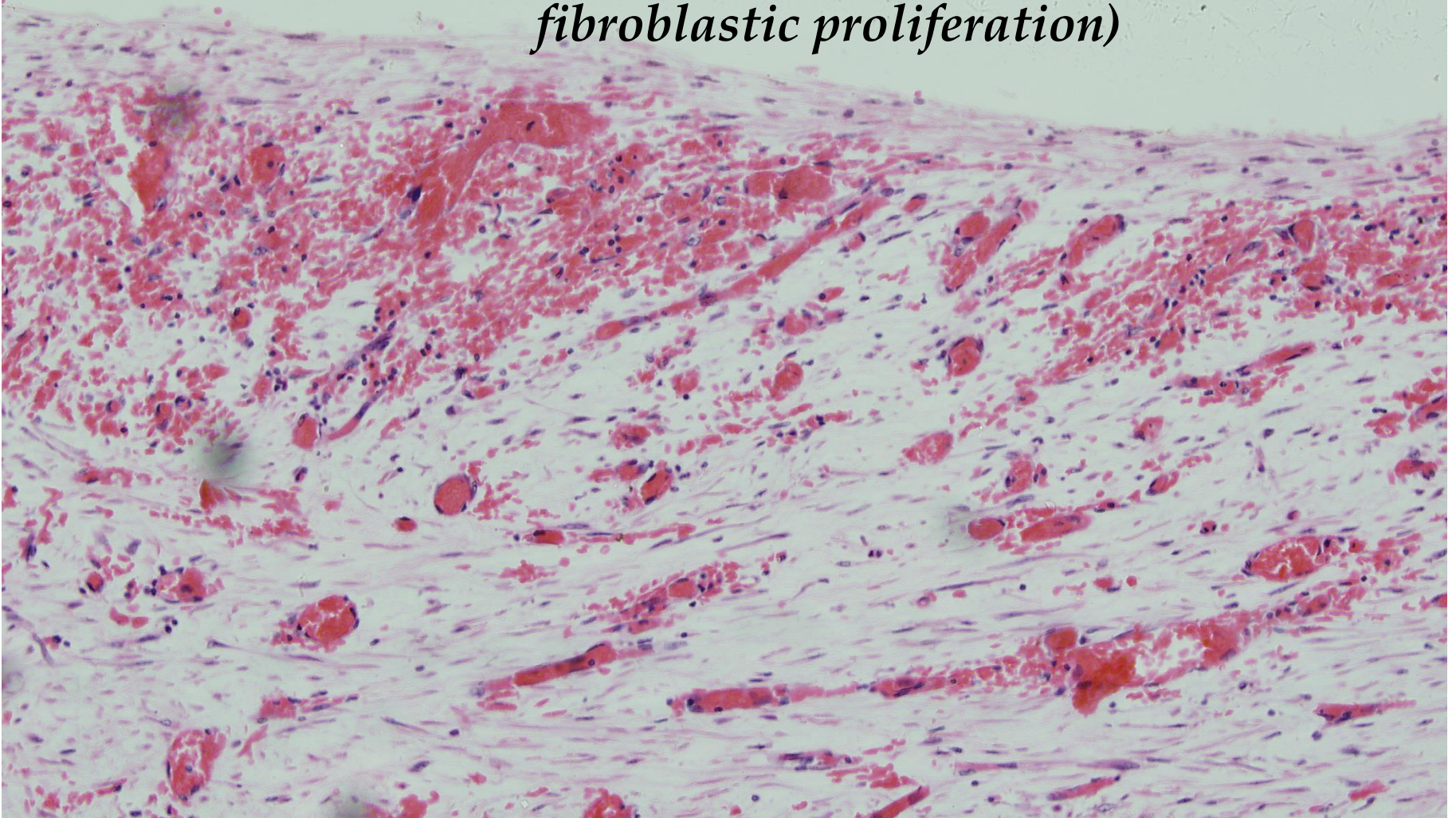
**Days:** epithelium begins to regenerate to cover lesion. Overlying exudate = **scab.**

Contraction of wound

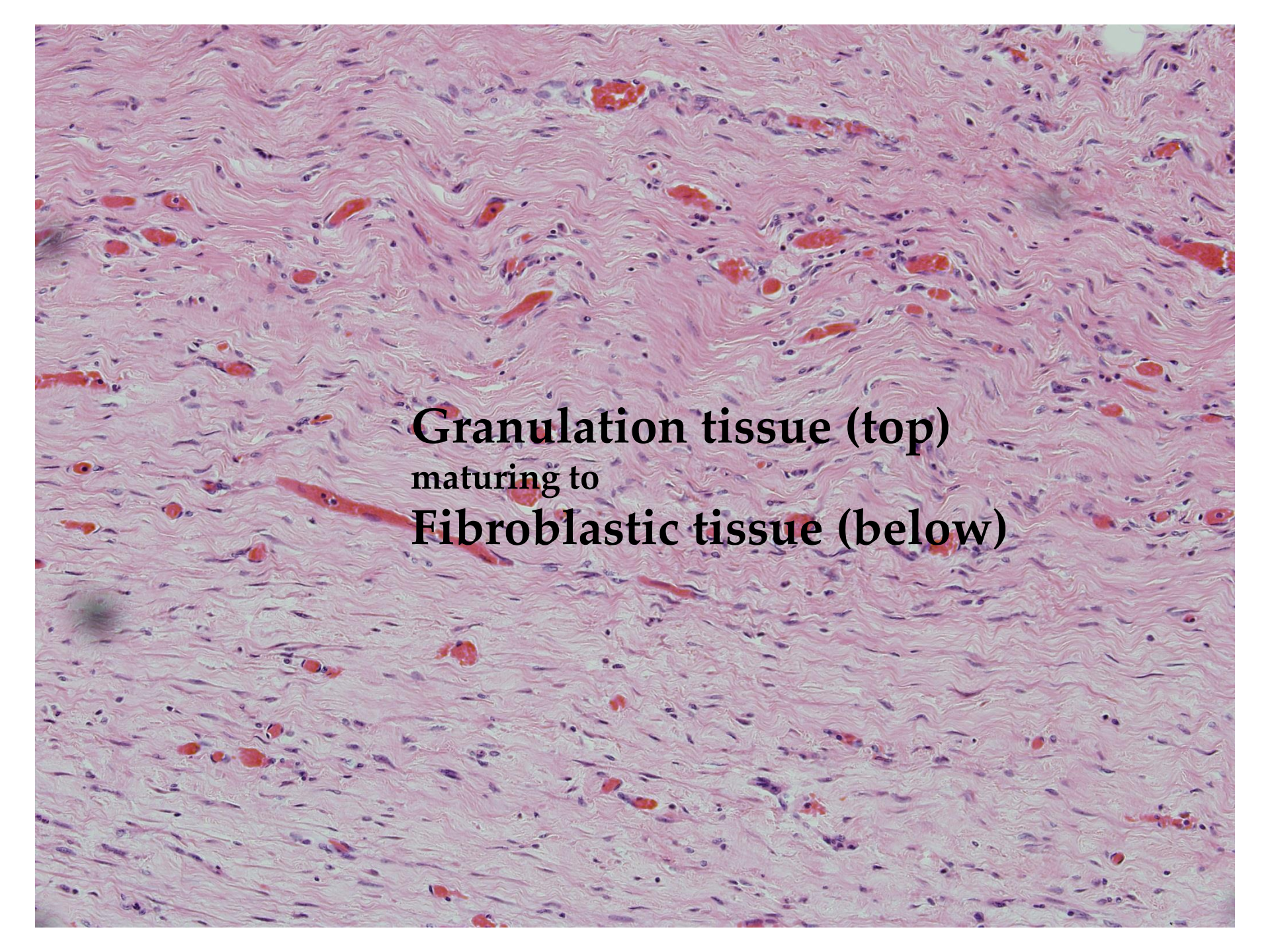
New capillary loops form, bring macrophages, neutrophils (prominent granulation tissue). Fibroblasts proliferate

**Weeks-months:** epithelium restored, collagen bundles thickened. Often extensive scarring

**Granulation tissue**  
*(New blood vessels and  
fibroblastic proliferation)*





A histological micrograph showing the transition from granulation tissue to fibroblastic tissue. The upper portion of the image displays granulation tissue, characterized by a dense population of small, dark-staining nuclei (likely inflammatory cells) and numerous small, red-staining granules. The lower portion shows fibroblastic tissue, characterized by a more organized, wavy pattern of pink-staining collagen fibers and fewer, more elongated nuclei. The overall appearance is that of a healing wound bed.

**Granulation tissue (top)**  
maturing to  
**Fibroblastic tissue (below)**





**Mature scar tissue  
(collagen)**



# FIBROSIS

**End result of organisation** in wound healing and chronic inflammation

## **The process:**

- Fibrocytes stimulated by polypeptides from surrounding damaged cells
- Become active fibroblasts. Commence protein synthesis
- Secretion of ground substance including fibrinonectins
- Secretion of procollagen
- Condensation to fine reticulin fibres
- Further condensation to mature collagen fibres
- Binding and weaving to form scar tissue
- Fibroblasts revert to fibrocytes



# Factors adversely affecting wound healing

## ***Local***

Poor blood supply

Infection

Excessive movement or irritation

Foreign material

## ***General***

Deficiency of Vitamin C, essential amino acids, zinc

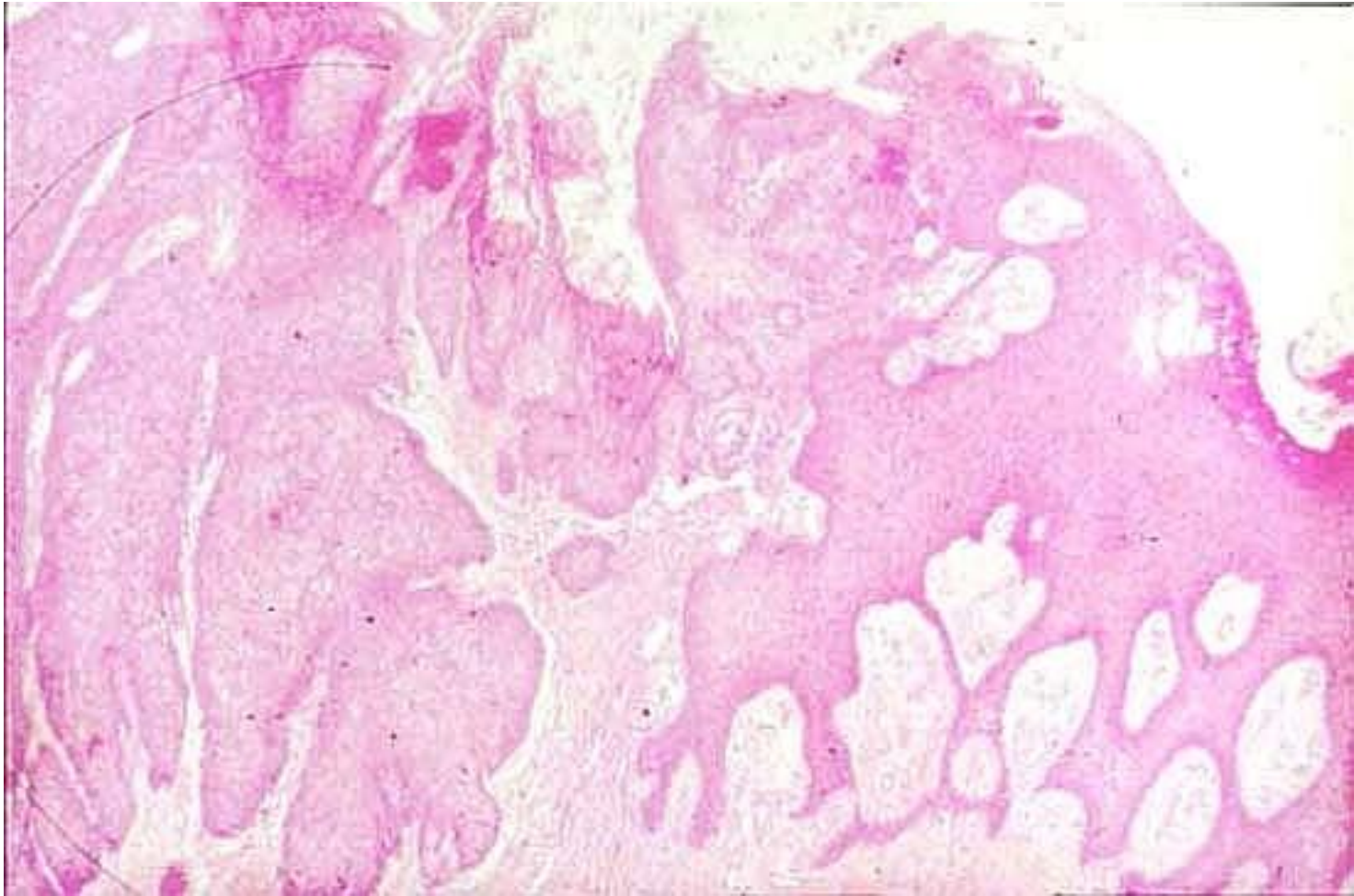
Excess adrenal corticosteroids

Intercurrent debilitating chronic disease



# COMPLICATIONS OF WOUND HEALING

- Infection
- Failure to heal
- Breakdown of wound
- Scarring/Stricture
- Keloid formation
- Pseudoepitheliomatous hyperplasia
- Malignancy



**Excessive epithelial proliferation  
(pseudoepitheliomatous hyperplasia)**

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**Complication of  
wound healing:  
excess collagen  
formation  
(keloid)**

