Histopathology: acute inflammation

These presentations are to help you identify, and to test yourself on identifying, basic histopathological features. They do not contain the additional factual information that you need to learn about these topics, or necessarily all the images from resource sessions.

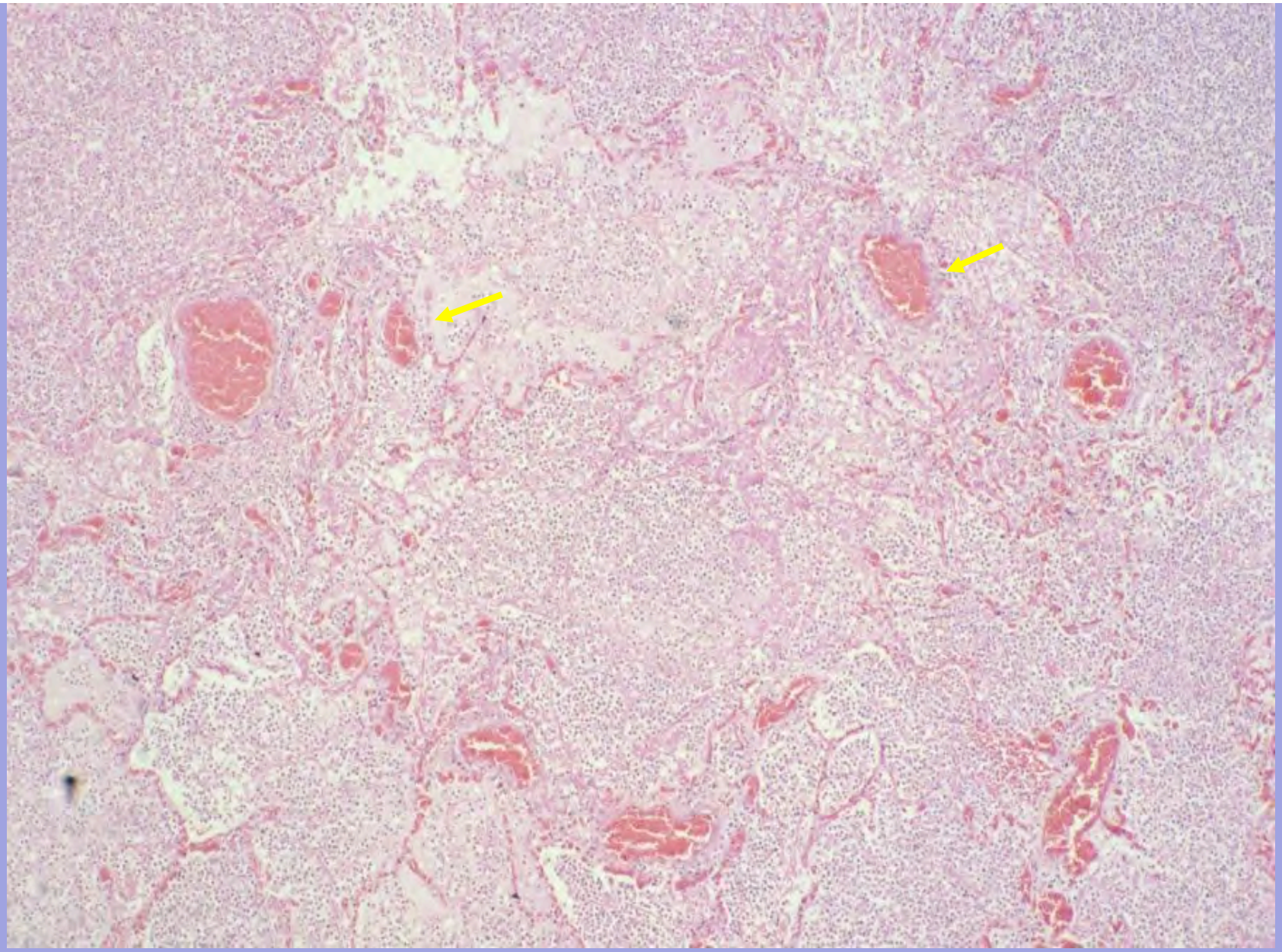
This presentation contains images of basic histopathological features of acute inflammation and examples of some relevant diseases.

Before viewing this presentation you are advised to review relevant histology, relevant sections on acute inflammation in a pathology textbook, relevant lecture notes and relevant sections of a histopathology atlas.

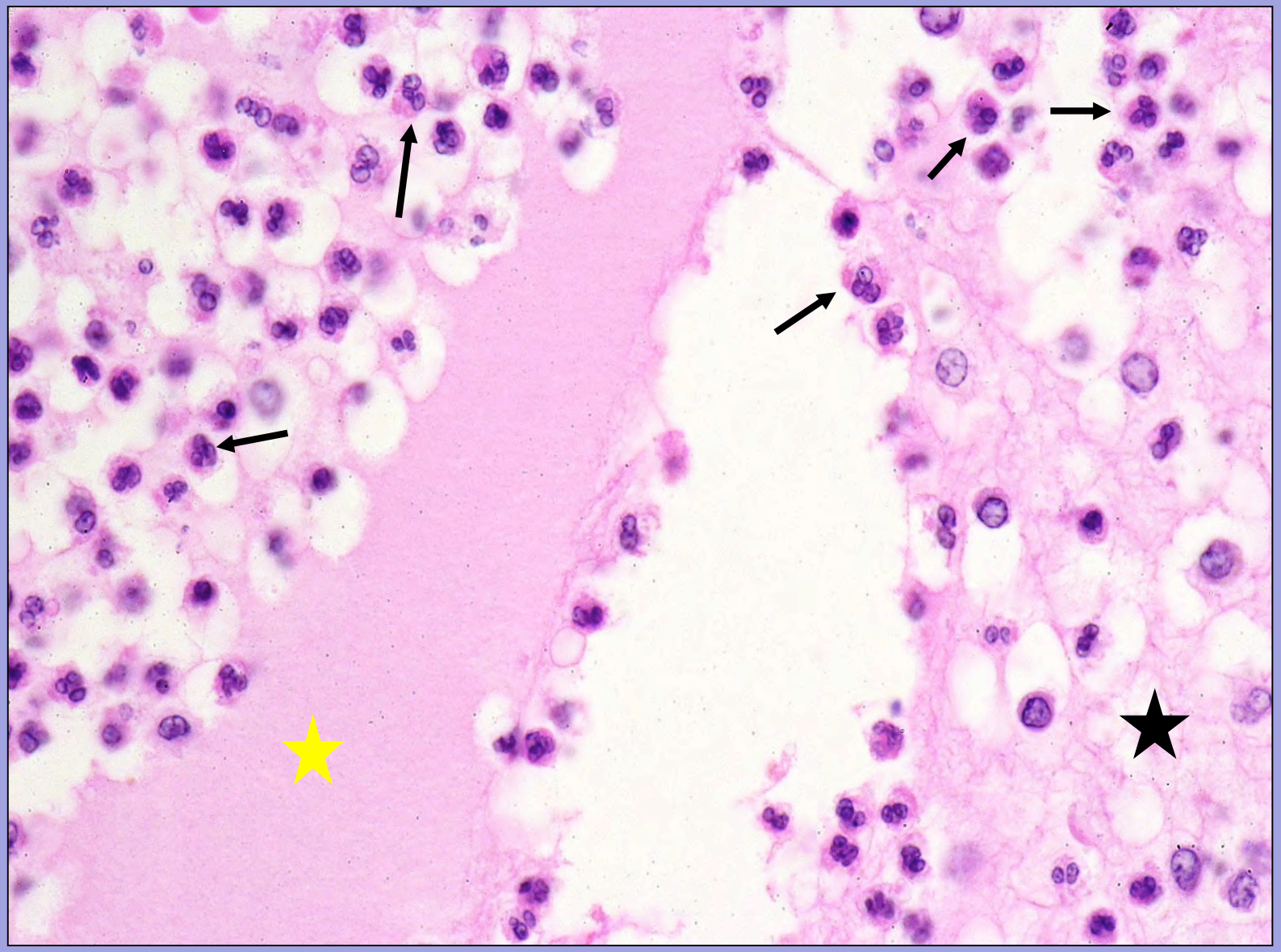
(The histopathology of acute inflammation is introduced in semester 1, year 1)

Histopathology: acute inflammation

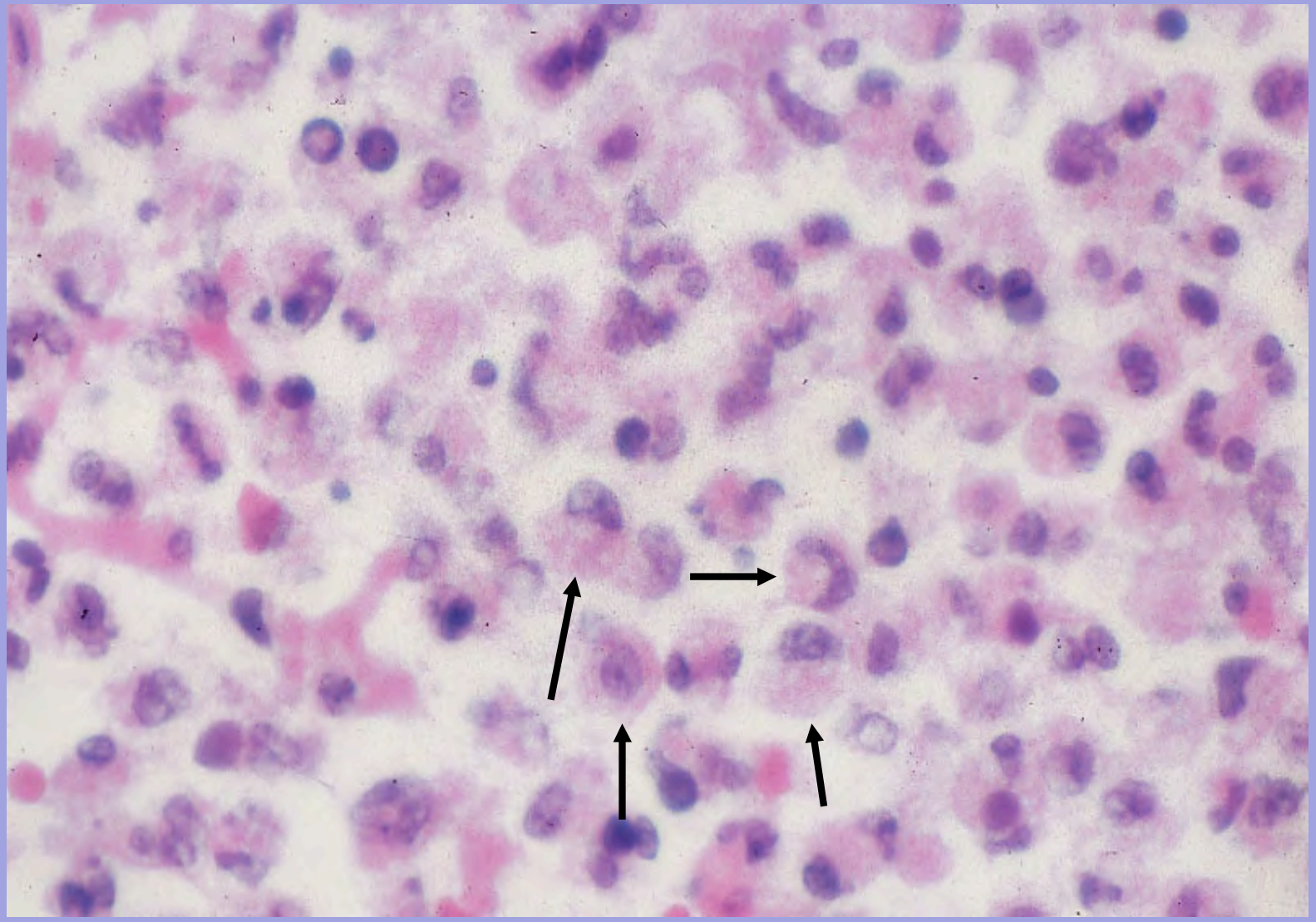
Acute inflammation is the body's initial non-specific response to an injury. There are many different causes. The response starts almost immediately and progresses over several days. Depending on the cause, severity, and the body's ability to respond, healing may then occur, chronic inflammation may develop or an abscess may form (with certain infectious agents).



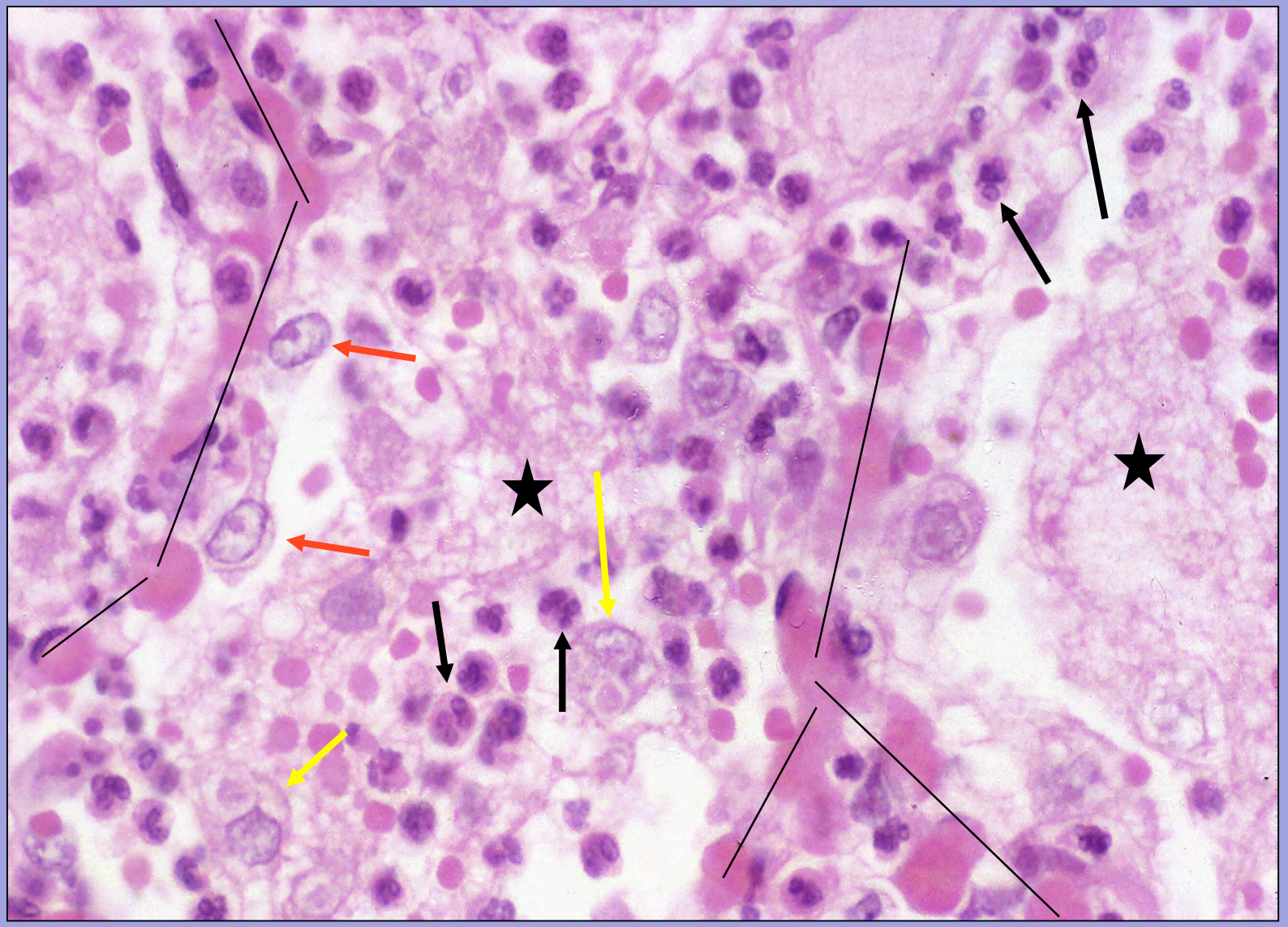
A characteristic feature of acute inflammation is dilation of small blood vessels, particularly venules, seen here (yellow arrows) at low power in lobar pneumonia. Note also the cellular exudate causing consolidation of alveolar spaces.



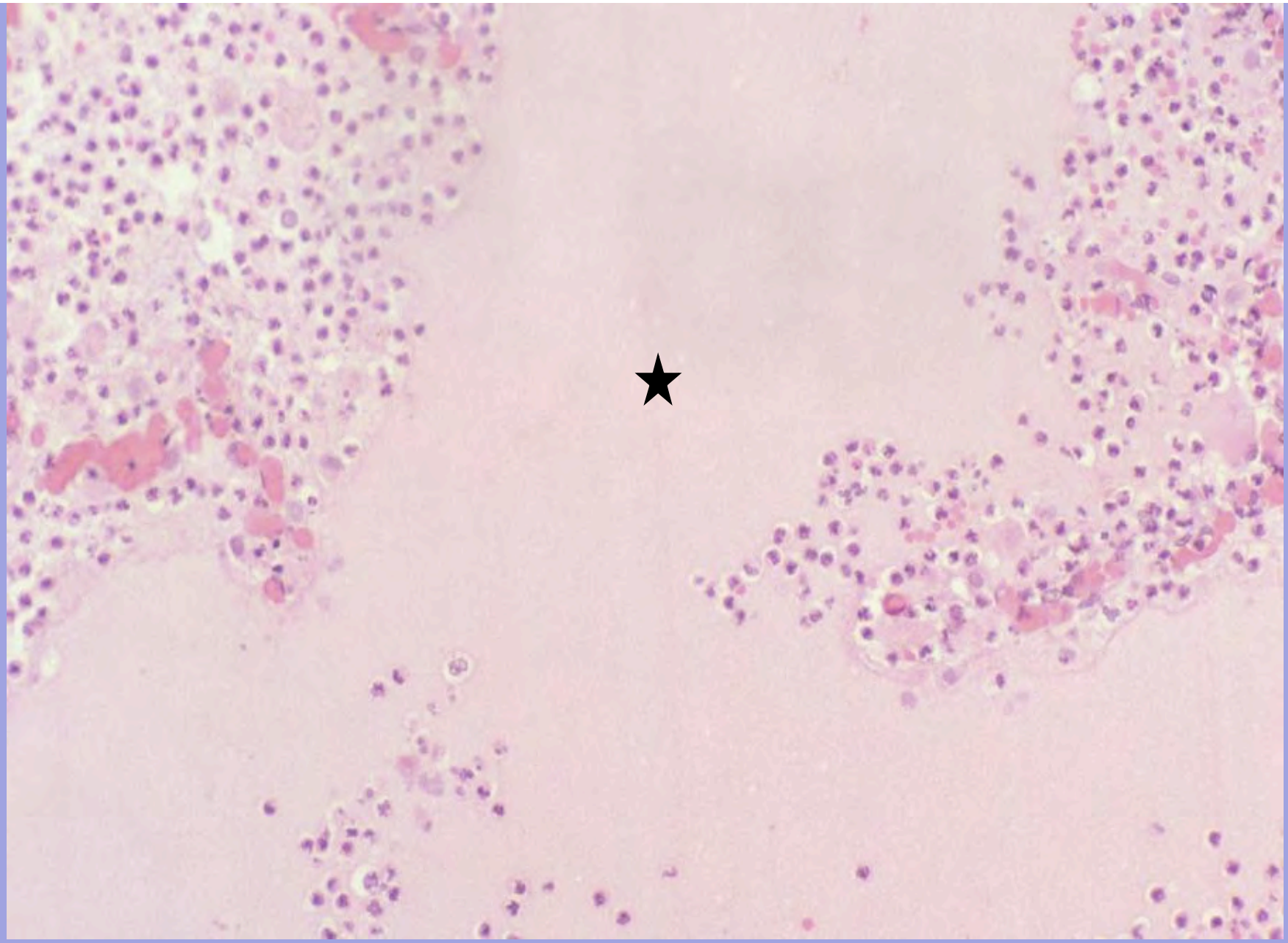
Acute inflammatory exudate, medium-high power. Neutrophils (black arrows) have multilobate nuclei. Also note homogenous oedema fluid (yellow star), eosinophilic due to its protein content, and the insoluble fibrin (black star) that is seen as an eosinophilic cobweb like matrix.



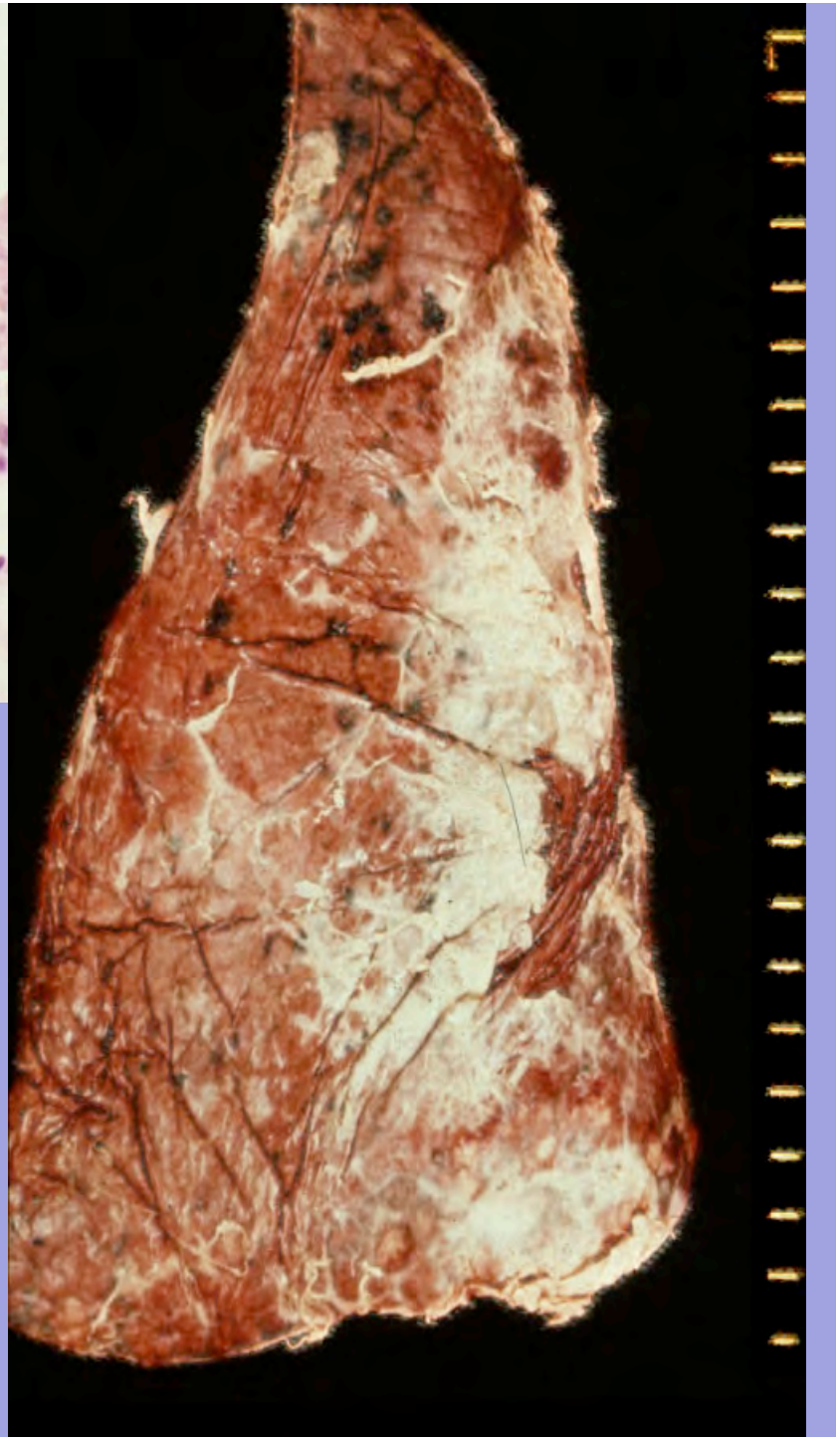
Macrophages (black arrows) generally have moderate amounts of cytoplasm and a kidney, oval or bean shaped nucleus, often at one side of the cell. In sections their size is similar to or larger than neutrophils.



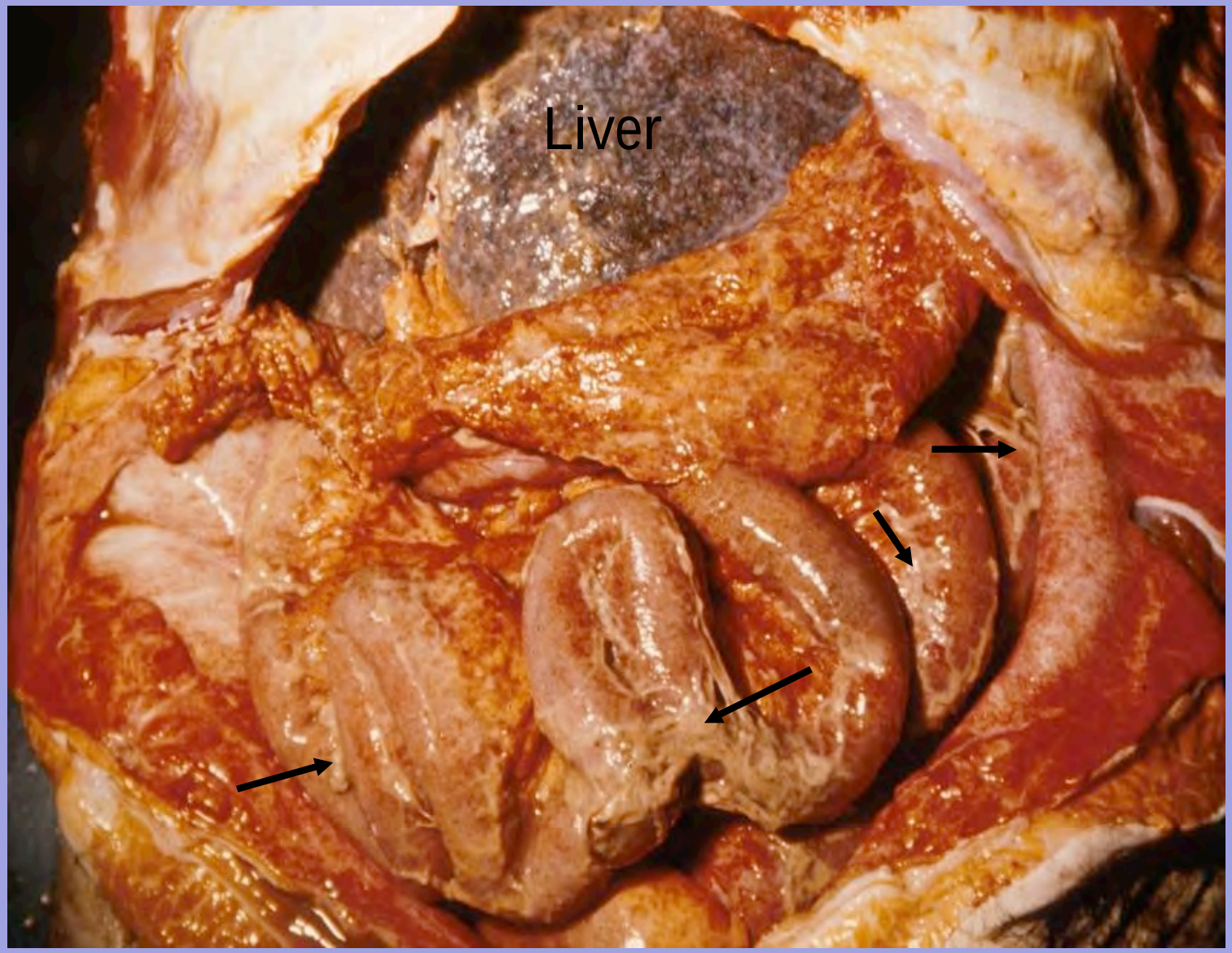
Lobar pneumonia, high power. Exudate filling alveolar spaces: neutrophils (black arrows), fibrin (black stars), macrophages (yellow arrows) and extravasated red blood cells. Note the relative sizes of cells. Neutrophils have a larger diameter than red blood cells (and lymphocytes - not seen here) and macrophages are often larger than neutrophils. The black lines mark the alveolar walls, indicated also by the capillaries and epithelial cells (red arrows).



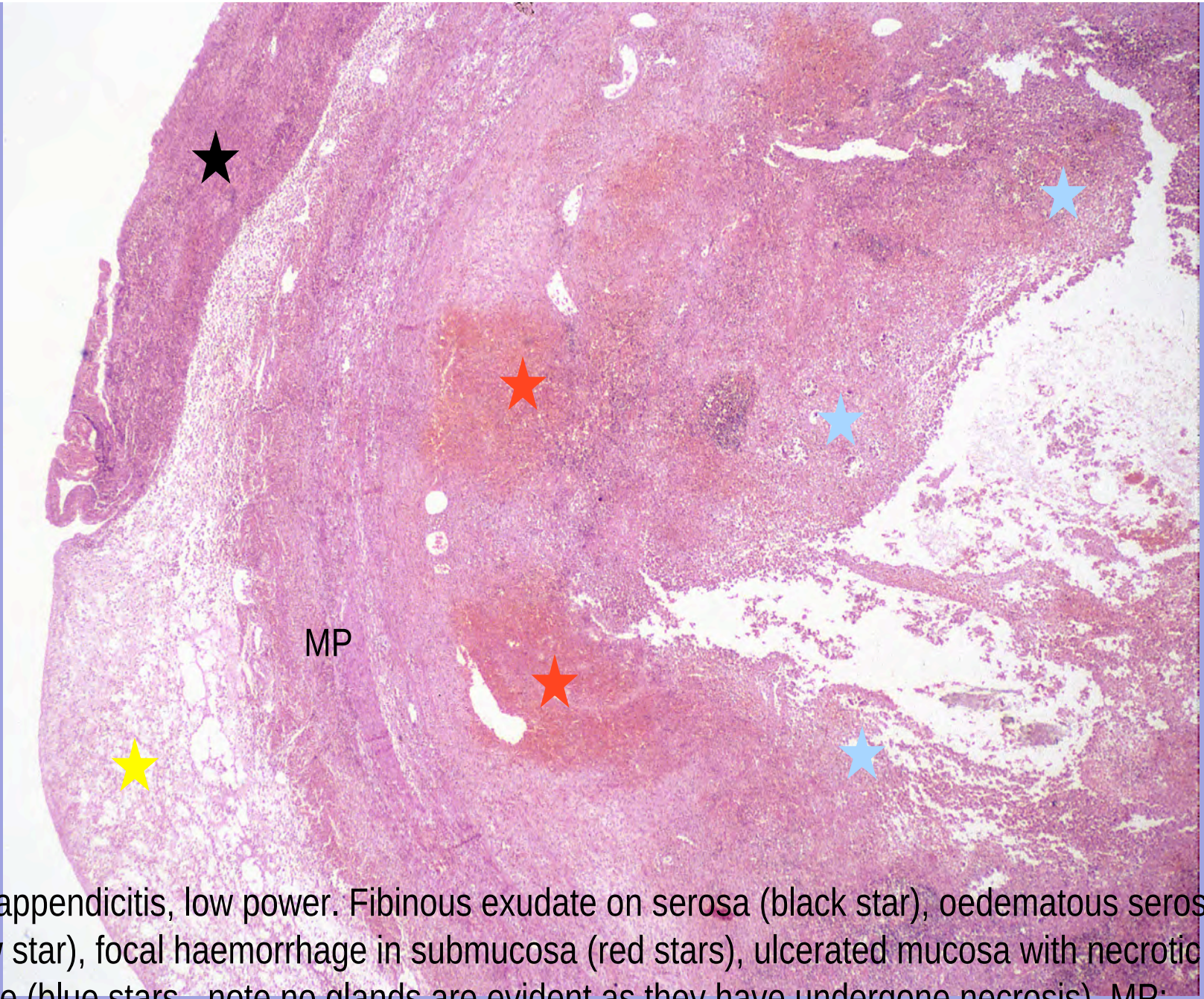
Lobar pneumonia, medium-low power. Exudate, here predominantly oedematous, filling alveolar spaces. Oedema fluid (black star) is eosinophilic due to its protein content.



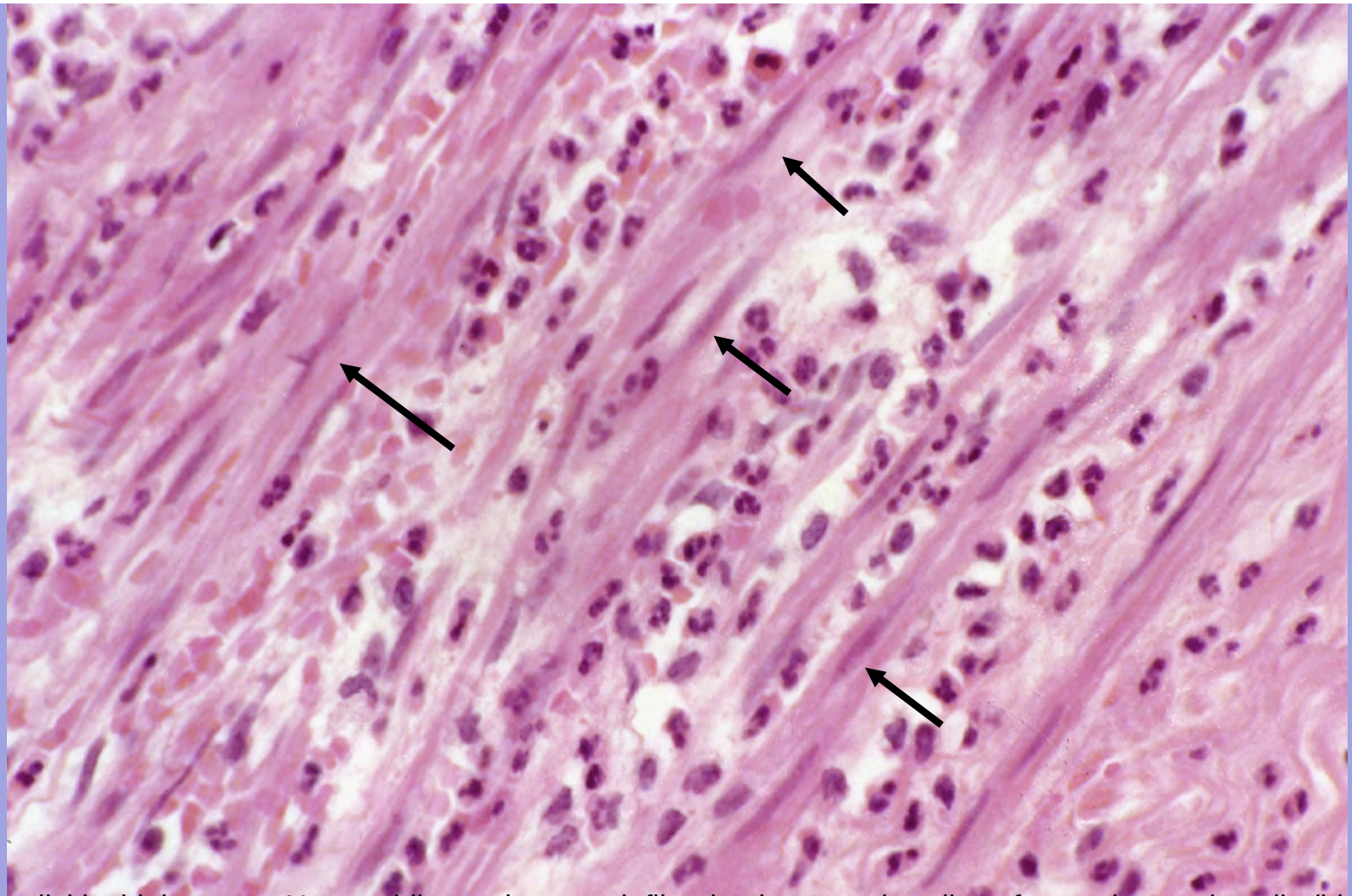
Acute inflammation of a serosal surface leads to a predominantly fibrinous exudate (sometimes fibrinopurulent depending on the cause).
Macroscopic (right): pale whispy fibrinous exudate on pleura of lung.
Microscopic (top): eosinophilic material, here with intermixed neutrophils, on a serosal surface.
Fibrinous serosal exudates heal by organization.



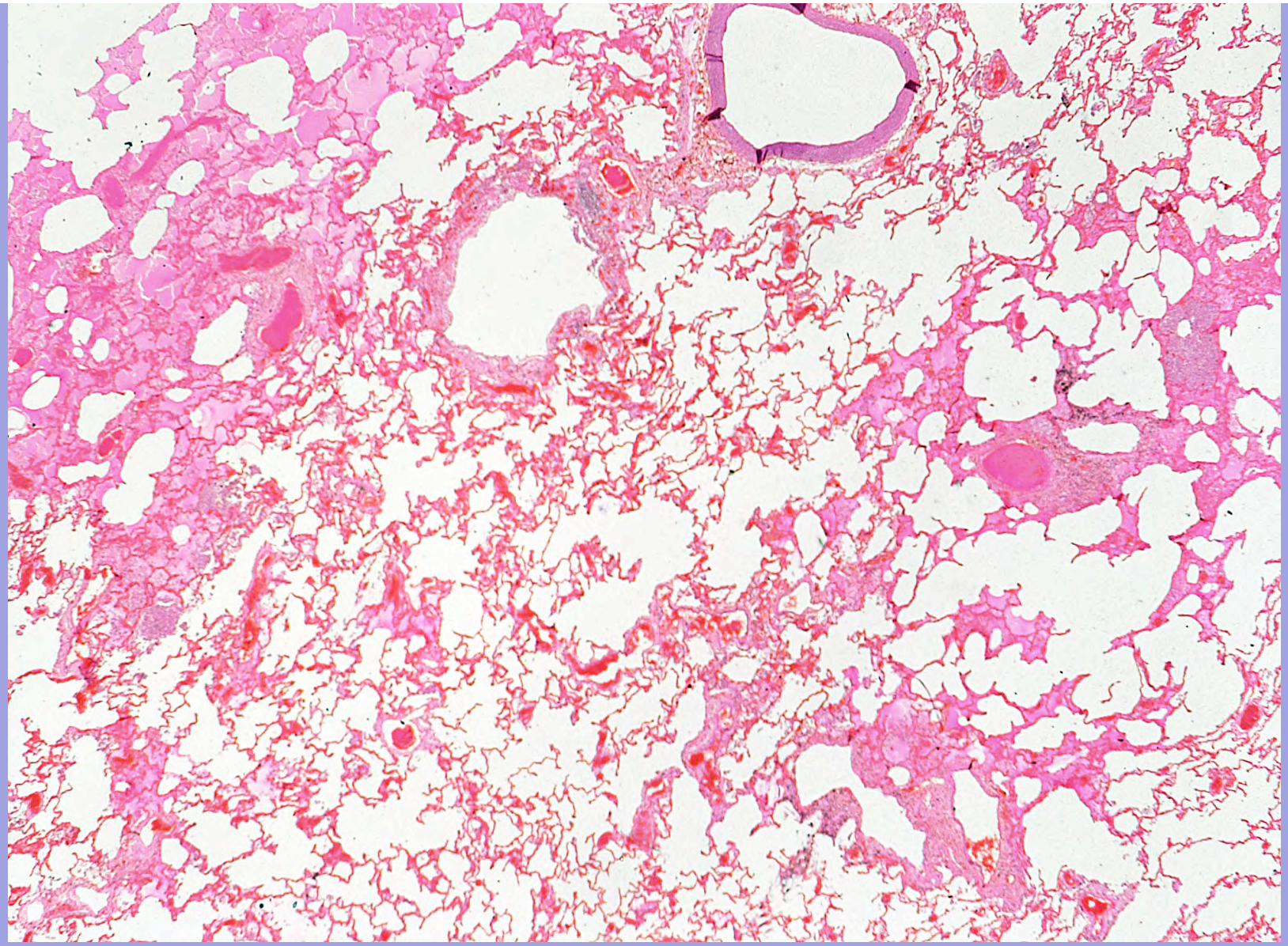
Generalised acute peritonitis. Note generalised erythema and fibrinopurulent exudate (black arrows) on serosal surface (visceral peritoneum) of bowel and parietal peritoneum. Generalised acute peritonitis develops following perforation of a hollow viscus with secondary bacterial infection of the peritoneal cavity.



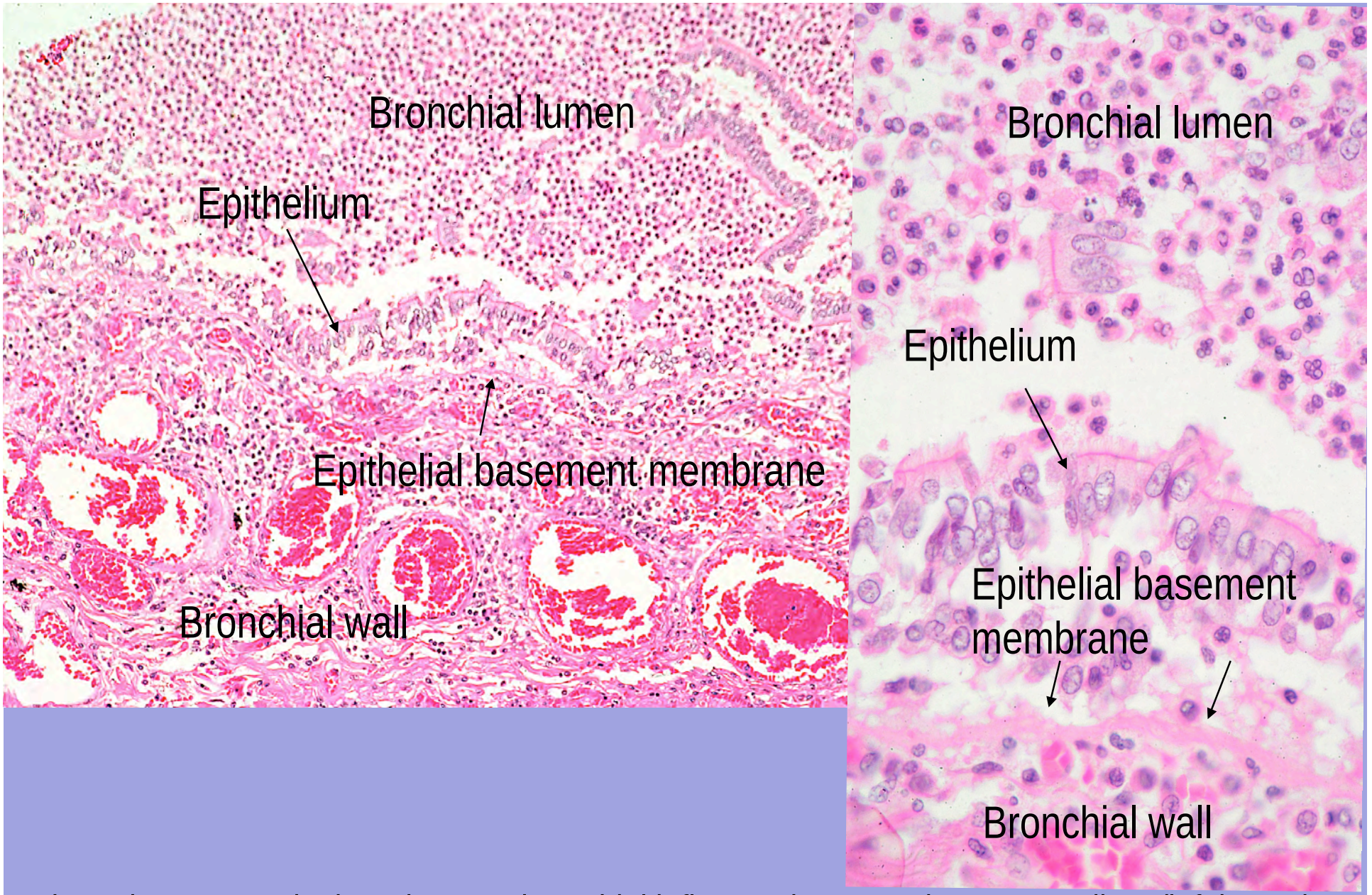
Acute appendicitis, low power. Fibinous exudate on serosa (black star), oedematous serosa (yellow star), focal haemorrhage in submucosa (red stars), ulcerated mucosa with necrotic exudate (blue stars - note no glands are evident as they have undergone necrosis). MP: muscularis propria. Make sure that you can identify the layers.



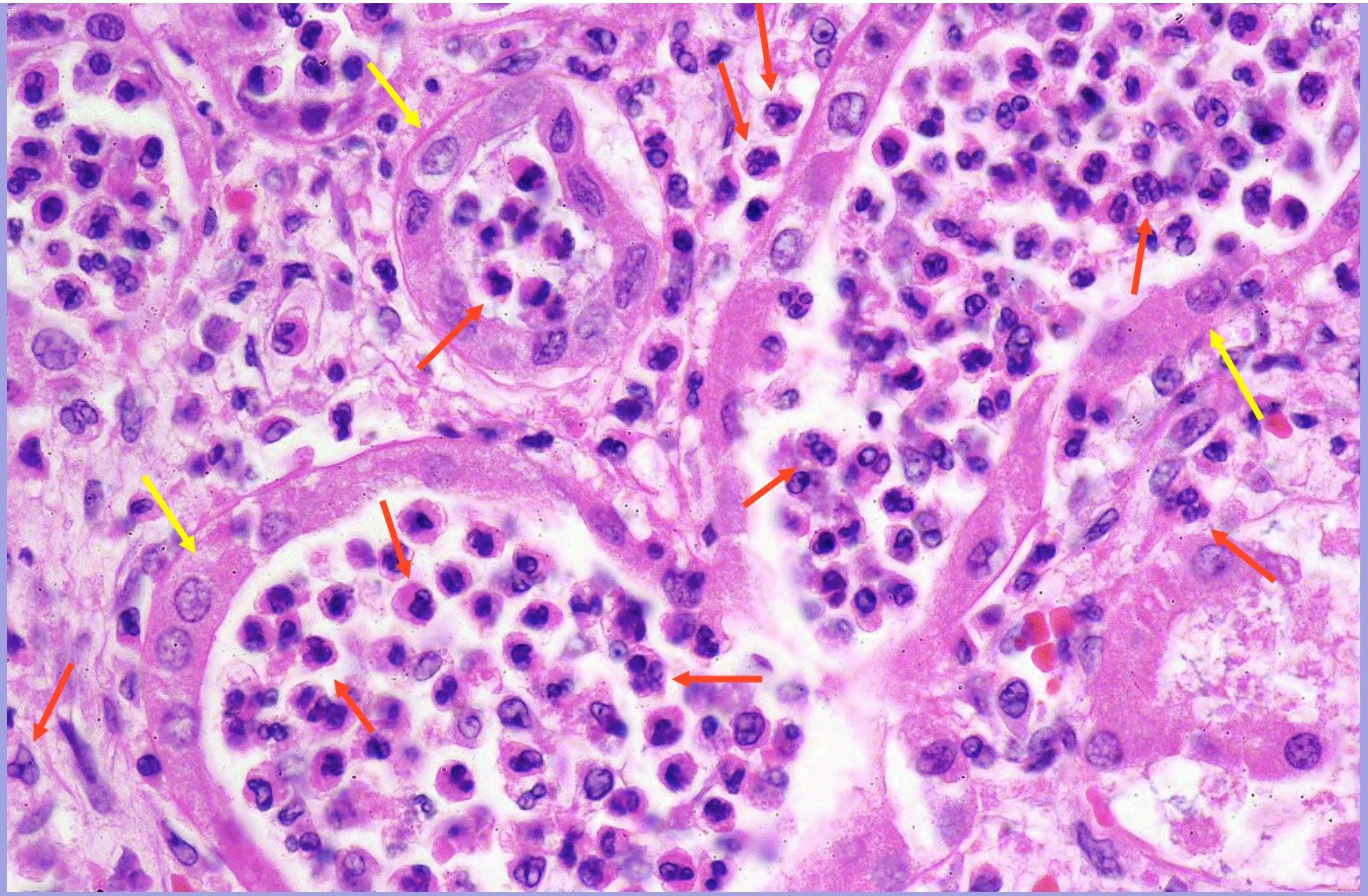
Acute appendicitis, high power. Neutrophils can be seen infiltrating between bundles of smooth muscle cells (black arrows) of the muscularis propria/externa. Note also extravasated red blood cells (red). Inflammation in appendicitis starts in the mucosa (early acute appendicitis). Abdominal pain is typically vague. Inflammation then spreads outwards and becomes transmural with the development of visceral and then parietal serosal inflammation, and the typical development of more severe localised pain. Necrosis eventually ensues as a result of tissue damaging lysosomal enzymes leaking from the numerous dying neutrophils and possibly secondary bacterial infection leading to the release of bacterial toxins. The mucosa ulcerates and the entire thickness of the appendix wall may undergo necrosis (acute gangrenous appendicitis) leading to perforation.



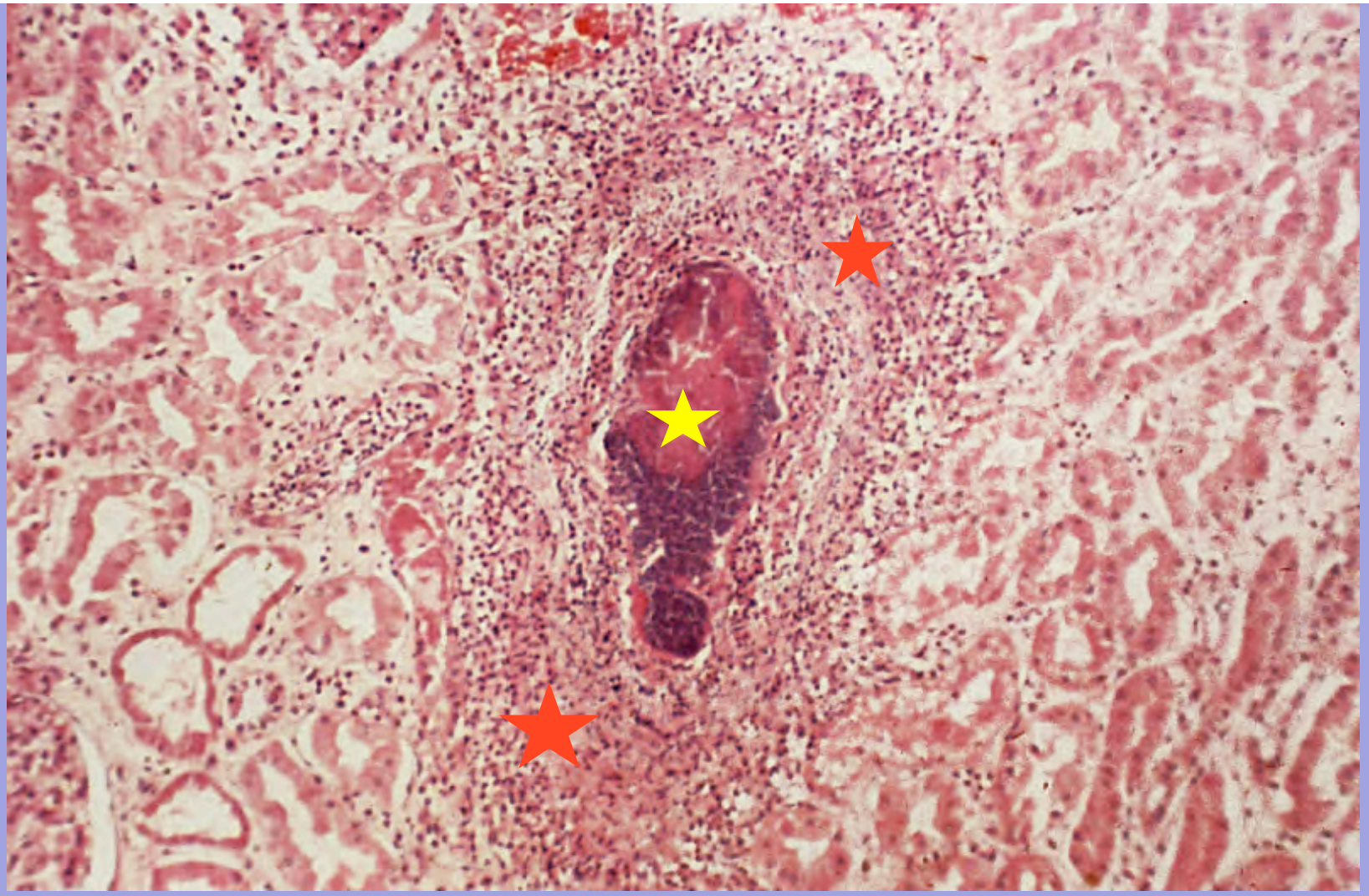
Bronchopneumonia differs from lobar pneumonia by its pattern. The pattern of pneumonia varies depending on virulence of the causative organism and susceptibility of the individual. In bronchopneumonia infection and inflammation spread from the bronchi and bronchioles to involve the lung parenchyma in a patchy fashion. Consolidation is thus initially patchy as seen above on a low power view of the lung.



In bronchopneumonia there is acute bronchial inflammation, seen here on medium (left image) and high power views (right image), and pulmonary inflammation. Note neutrophils in the bronchial lumen and dilated blood vessels.



Numerous neutrophils (e.g. red arrows) in tubules and interstitium in acute pyelonephritis (medium-high power). Note the cuboidal renal tubular epithelium (yellow arrows). Note that the neutrophil filled tubules look different to glomeruli. Acute pyelonehphritis results from bacterial infection, generally having ascended from the bladder. It thus primarily affects the tubules and interstitial tissue rather than the glomeruli.



In cases of acute inflammation where neutrophils are abundant, generally associated with certain types (pyogenic) of bacterial infection, necrosis may develop. Bacterial toxins cause necrosis and tissue damaging lysosomal enzymes and free radicals leak from the numerous neutrophils as they die leading to liquefaction. When confined within a solid organ, an abscess may form. The image demonstrates a microabscess in the kidney. Colonies of bacteria are present centrally (yellow star). These are surrounded by necrotic debris and neutrophils (red stars).