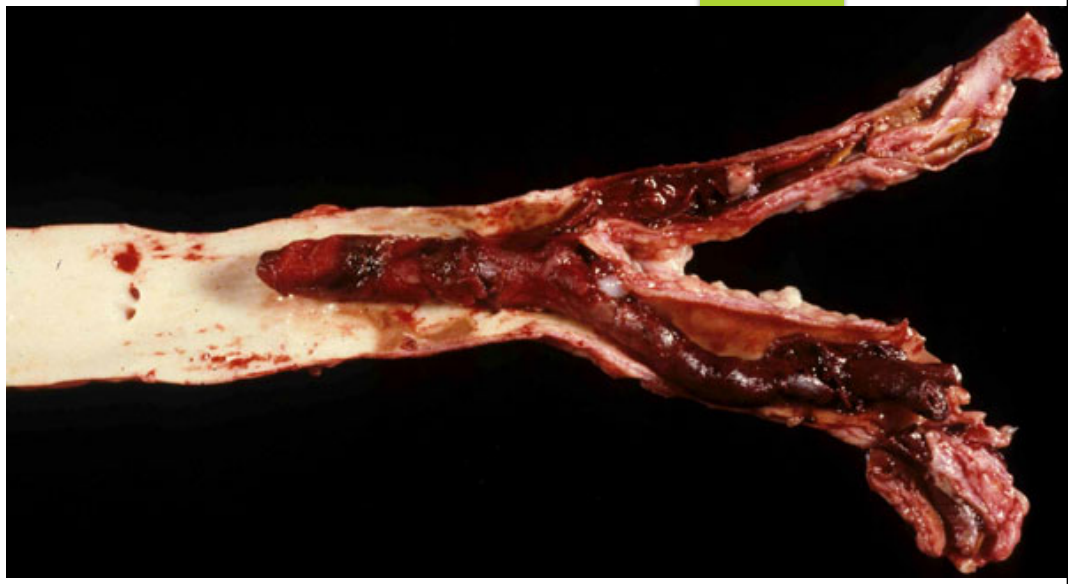
Foundation block Practical

THROMBO-EMBOLIC Disorders

Background information

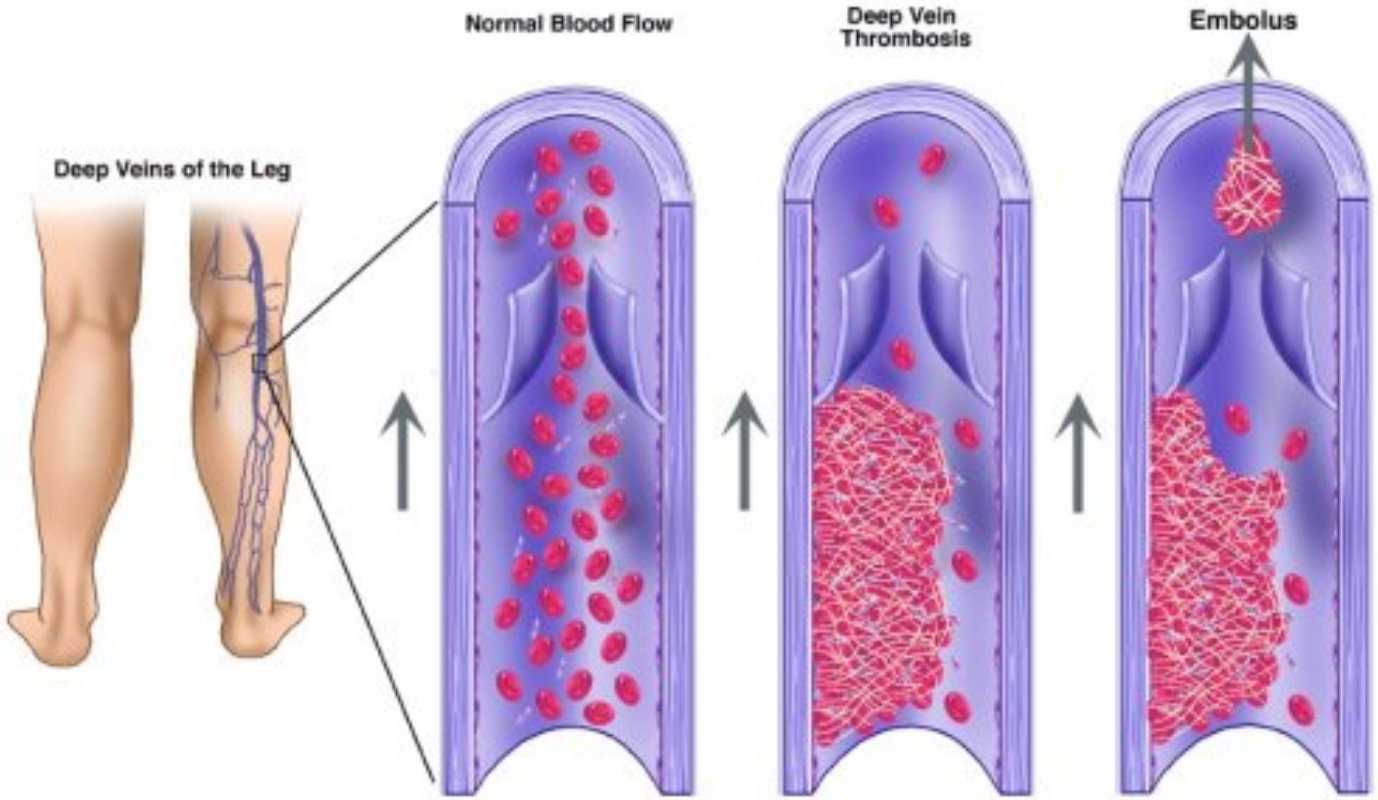
Background information: Thrombosis

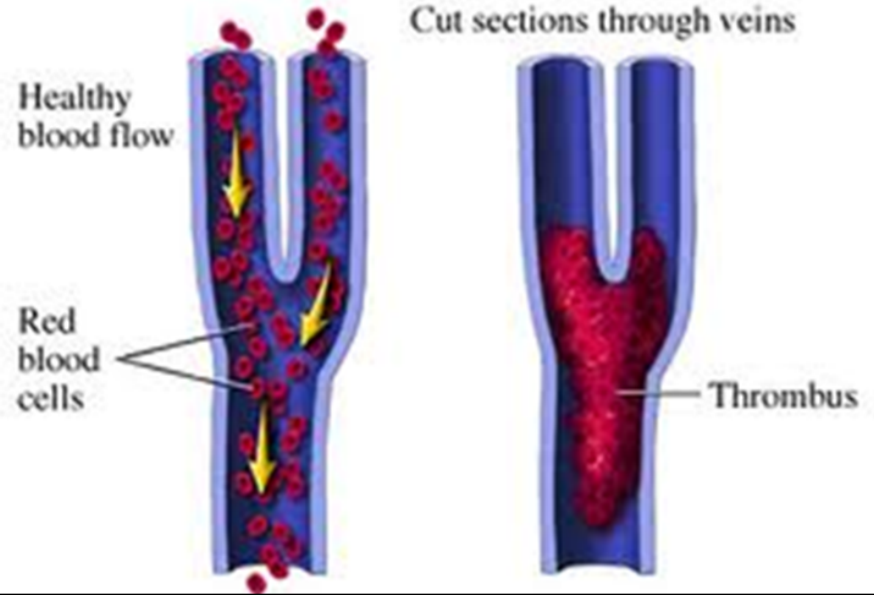
- Thrombosis is a process by which a thrombus is formed.
- A thrombus is a solid mass made up of blood constituents which develops in artery, vein or capillary.
- It is intravascular coagulation of blood and it can cause significant interruption to blood flow.
- Thrombi may develop anywhere in the cardiovascular system, the cardiac chambers, valve surface, arteries, veins, or capillaries. They vary in size and shape, depending on the site of origin.
- Thrombi in the vein are called **venous thrombi.** Thrombi in the artery are called **arterial thrombi.** When arterial thrombi arise in heart chambers or in aorta they are termed **mural thrombi**.
- Thrombi can grow. The propagating/growing tail of the thrombi is weak and is prone to fragmentation, creating an **embolus**



Mural thrombus.

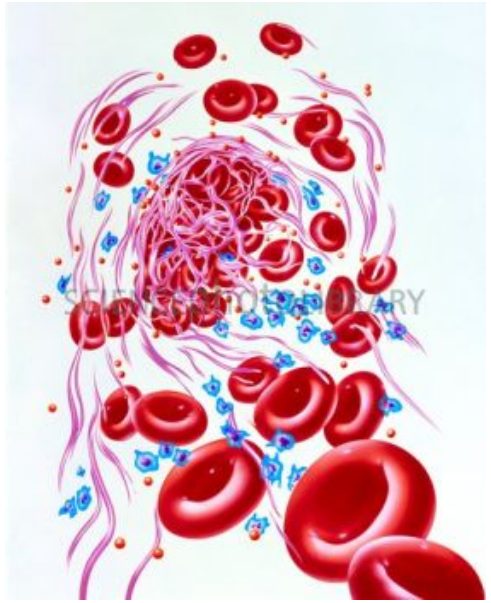
Background information:

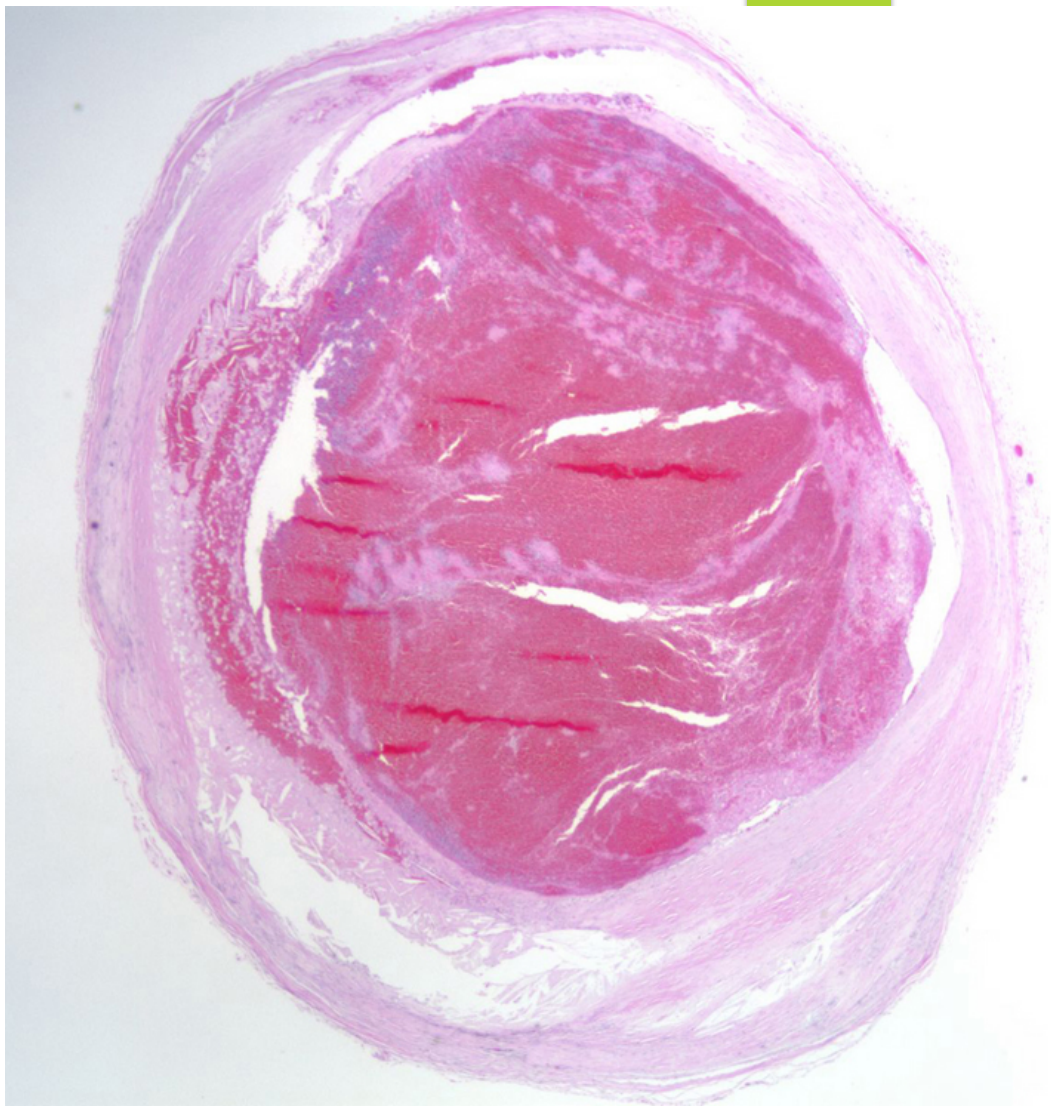




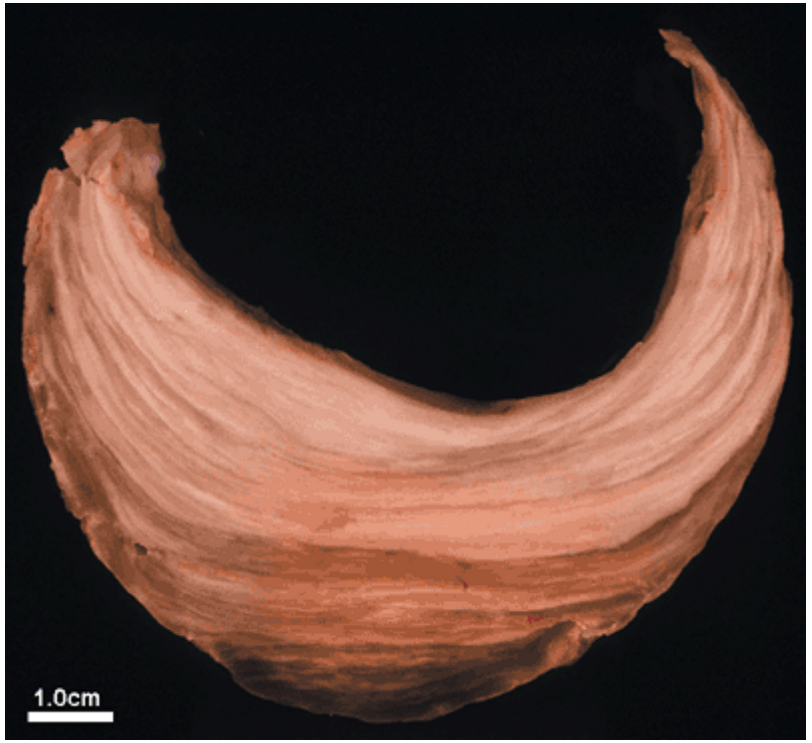
Background information: Morphology of thrombus

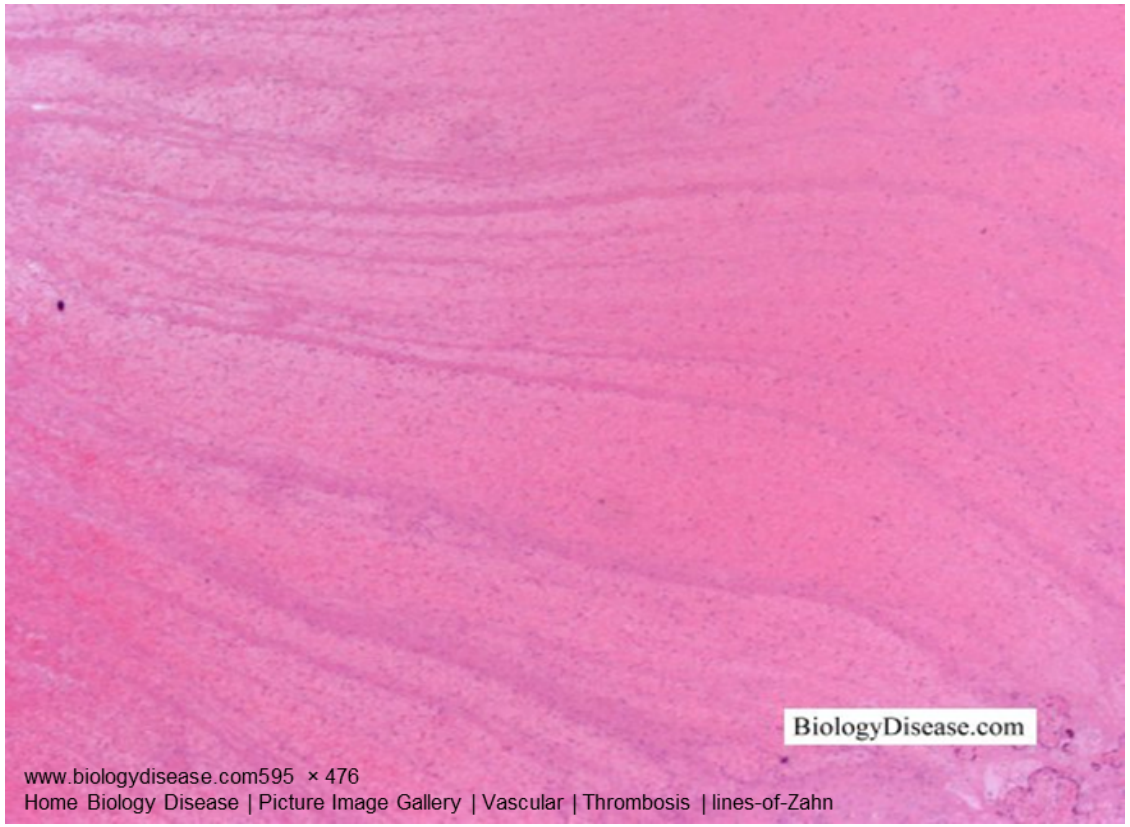
- A thrombus is made up of fibrin, platelets & red blood cells and some inflammatory cells.
- When formed in the heart or aorta or other large thrombi, thrombi may have laminations produced by alternating of pale and dark layers, called **lines of Zahn**; the pale layers contain platelets mixed with fibrin. The darker layers contain red blood cells.





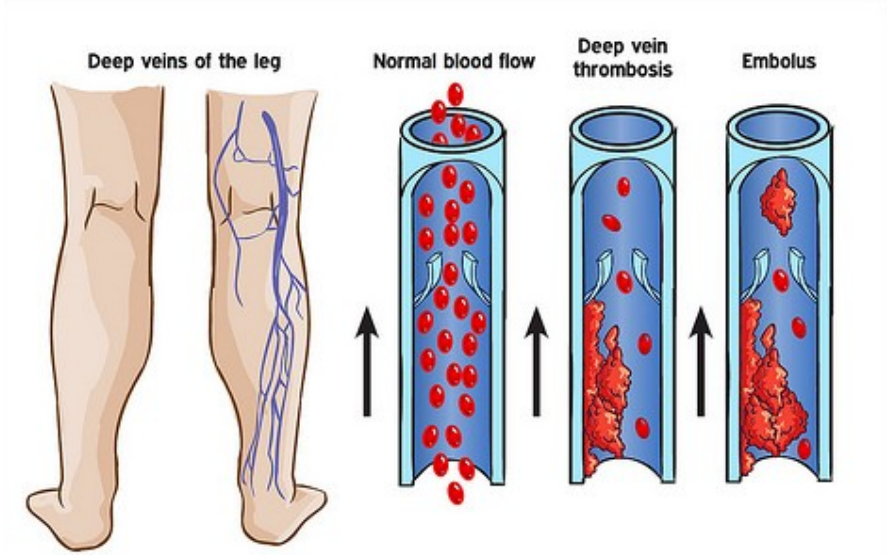
Background information: Lines of Zahn

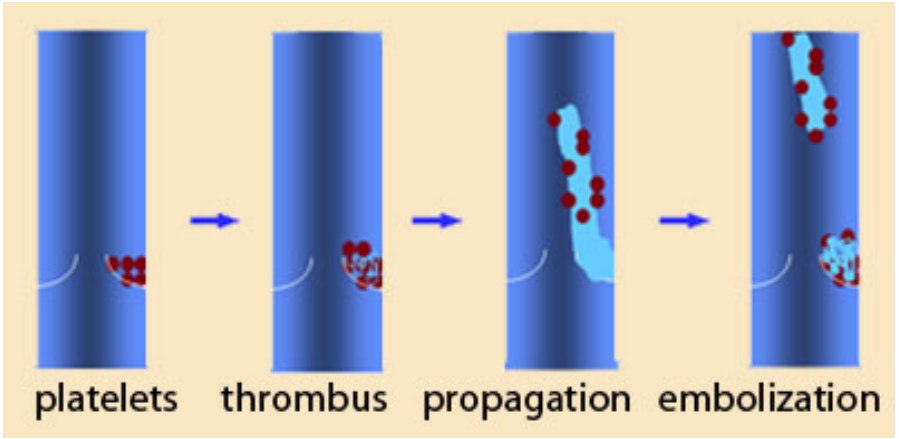




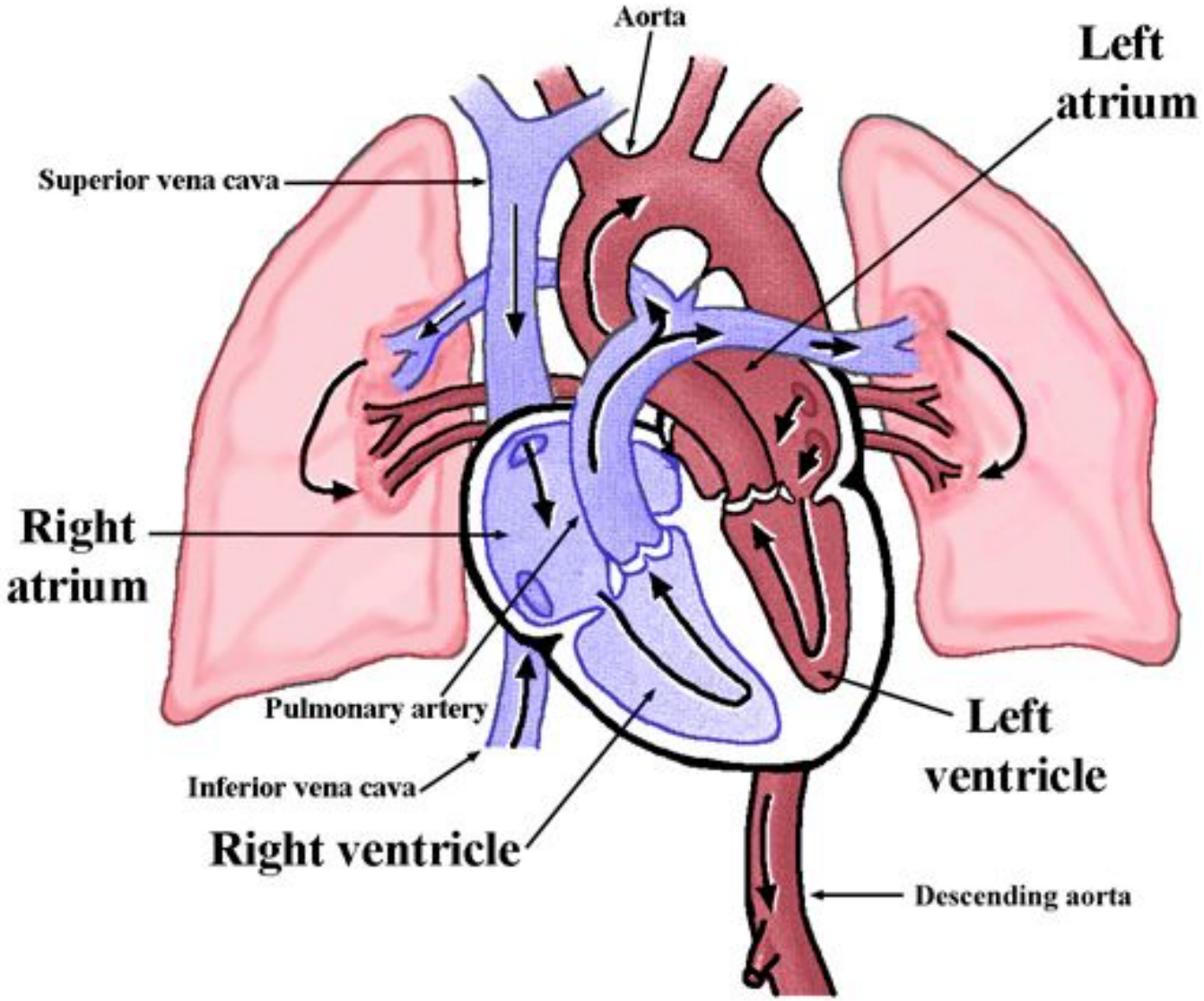
Background information: EMBOLISM

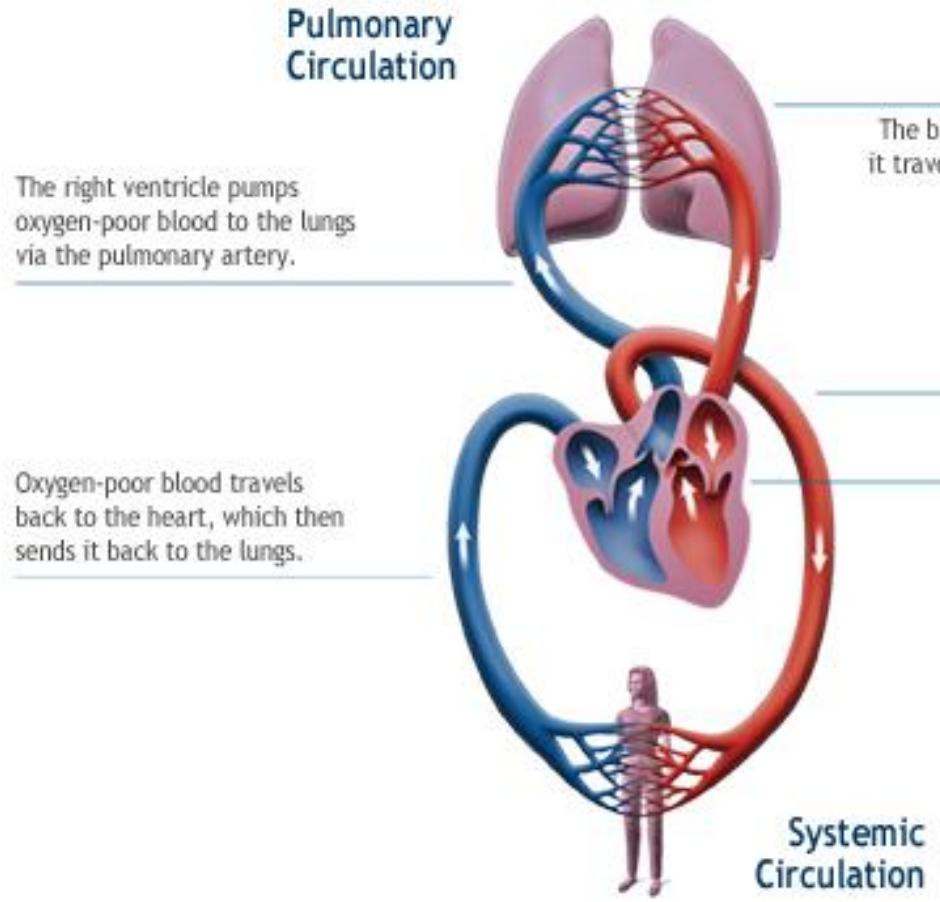
- *An embolus is a detached intravascular solid, liquid, or gaseous mass that is carried by the blood to a site distant from its point of origin.*
- Almost all emboli represent some part of a dislodged thrombus, hence the commonly used term *thromboembolism.*
- The emboli ultimately lodged in vessels too small to permit further passage, resulting in partial or complete vascular occlusion leading to ischemic necrosis of distal tissue (*infarction)*. Depending on the site of origin, emboli may lodge in the pulmonary or systemic circulations resulting in a **pulmonary embolus** or **systemic embolus**.





Background information: EMBOLISM





Background information: PULMONARY THROMBOEMBOLISM

- Here the embolus get lodged in the pulmonary blood vessels.
- Depending on size of embolus, it may get stuck and block the main pulmonary artery or block the bifurcation of the pulmonary trunk *(saddle embolus)* or pass out into the smaller, branching arterioles of the pulmonary circulation.
- Most pulmonary emboli (60% to 80%) are clinically silent because they are small. Sudden death or cardiovascular problems occurs when 60% or more of the pulmonary circulation is obstructed with emboli.
- Embolic obstruction of small end-arteriolar pulmonary branches may result in infarction.

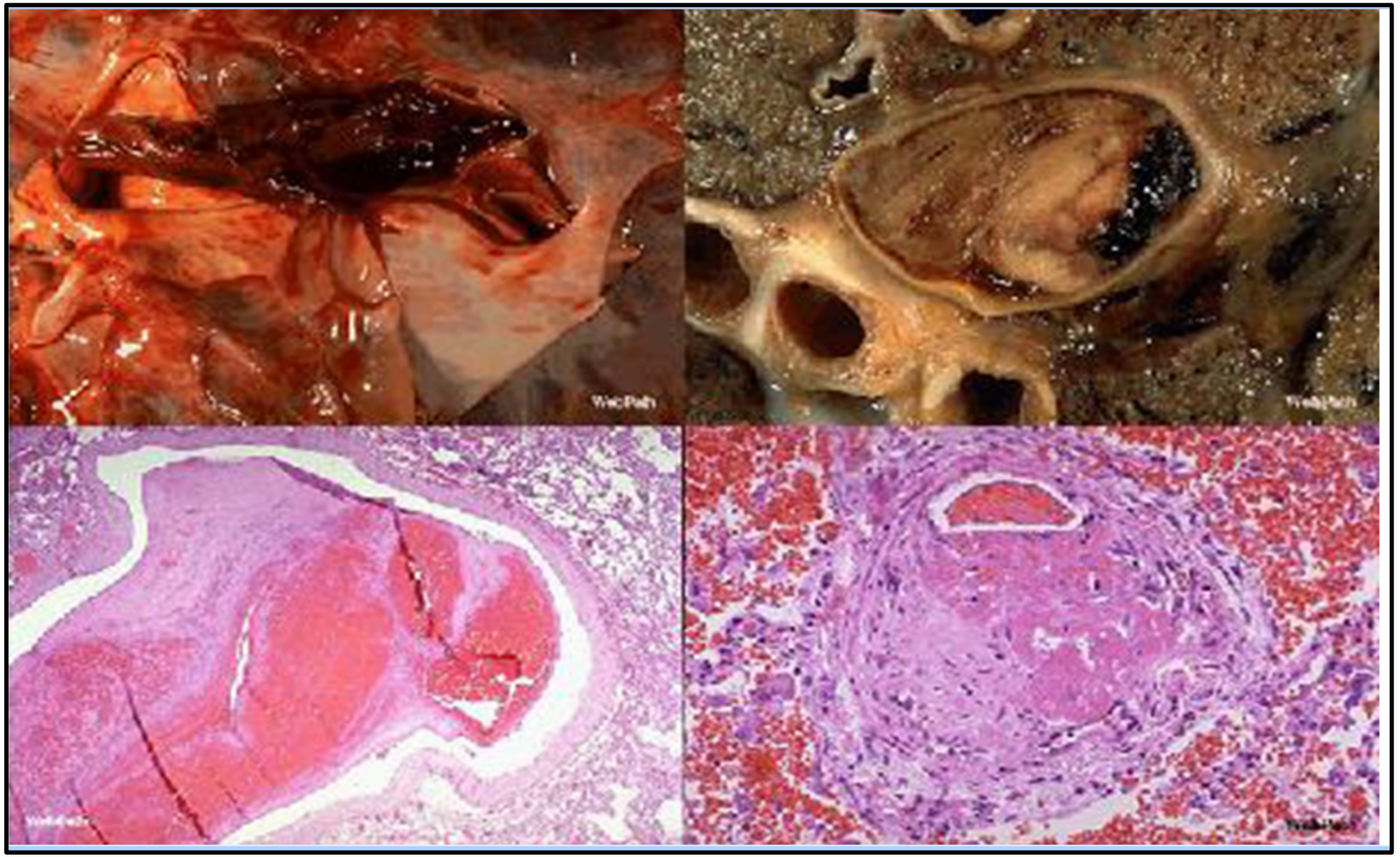
THROMBO-EMBOLIC Disorders Practical

Contents of this practical:

1. Organizing thrombus.
2. Pulmonary embolus with infarction.
3. Myocardial infarction.
4. Infarction of small intestine.

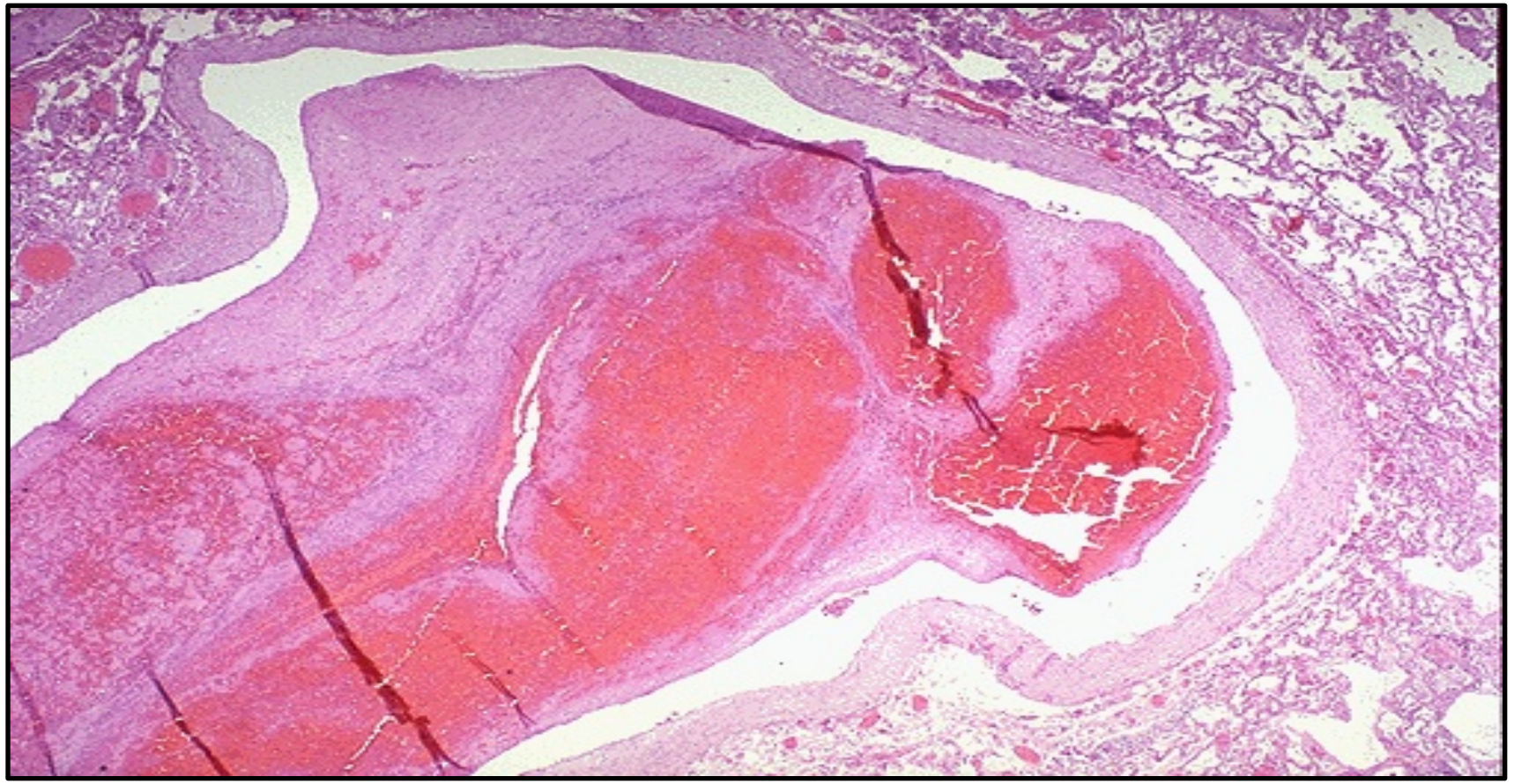
1- Organizing Thrombus

Organizing Thrombus



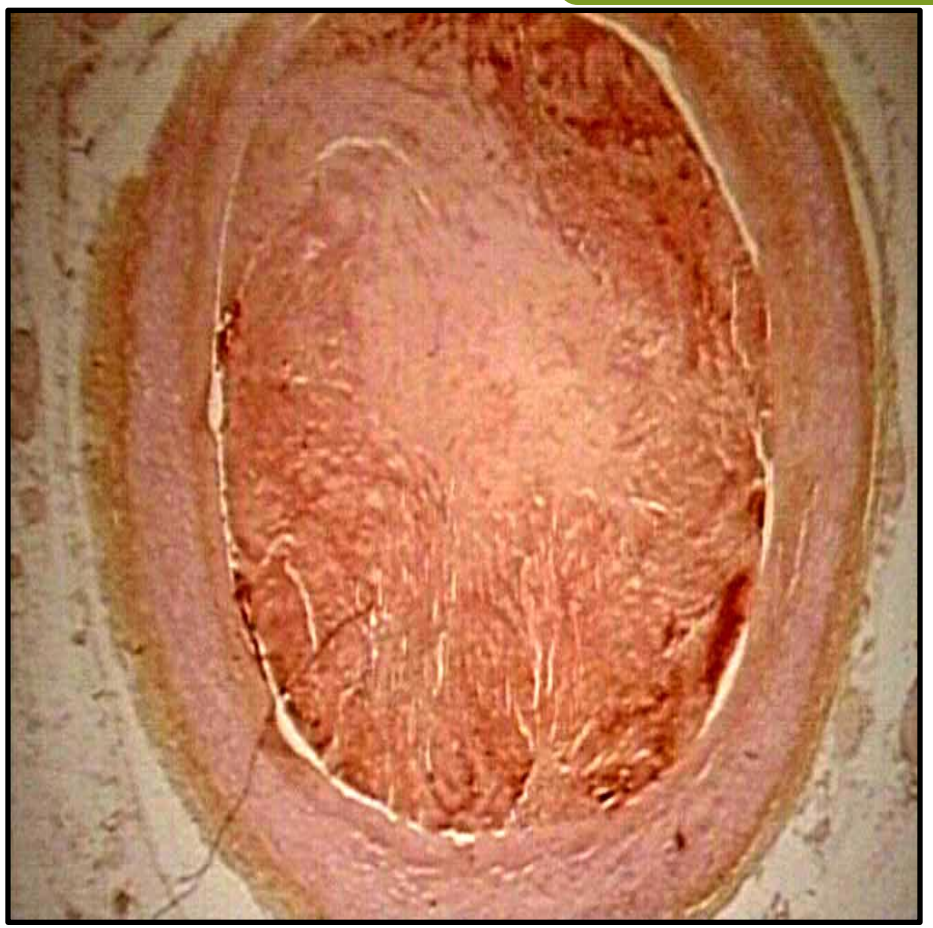
Organizing thrombus in a case of pulmonary embolism

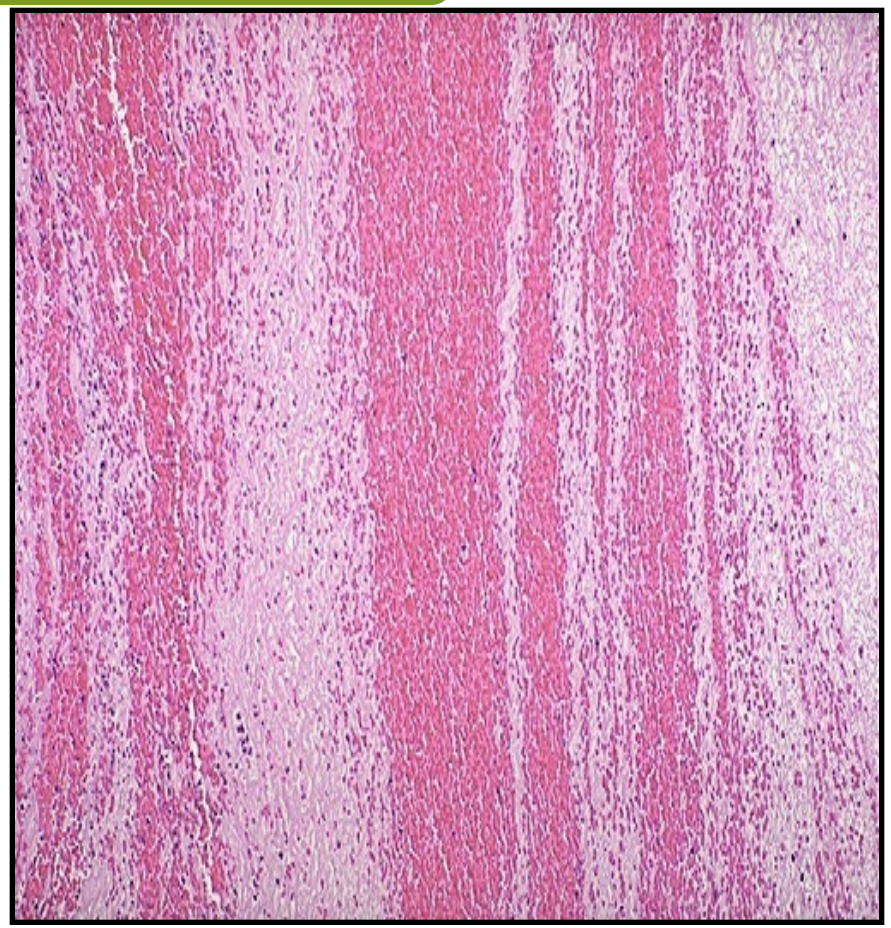
Organizing Thrombus with Lines of Zahn



This is the microscopic appearance of a pulmonary thromboembolus in a large pulmonary artery.

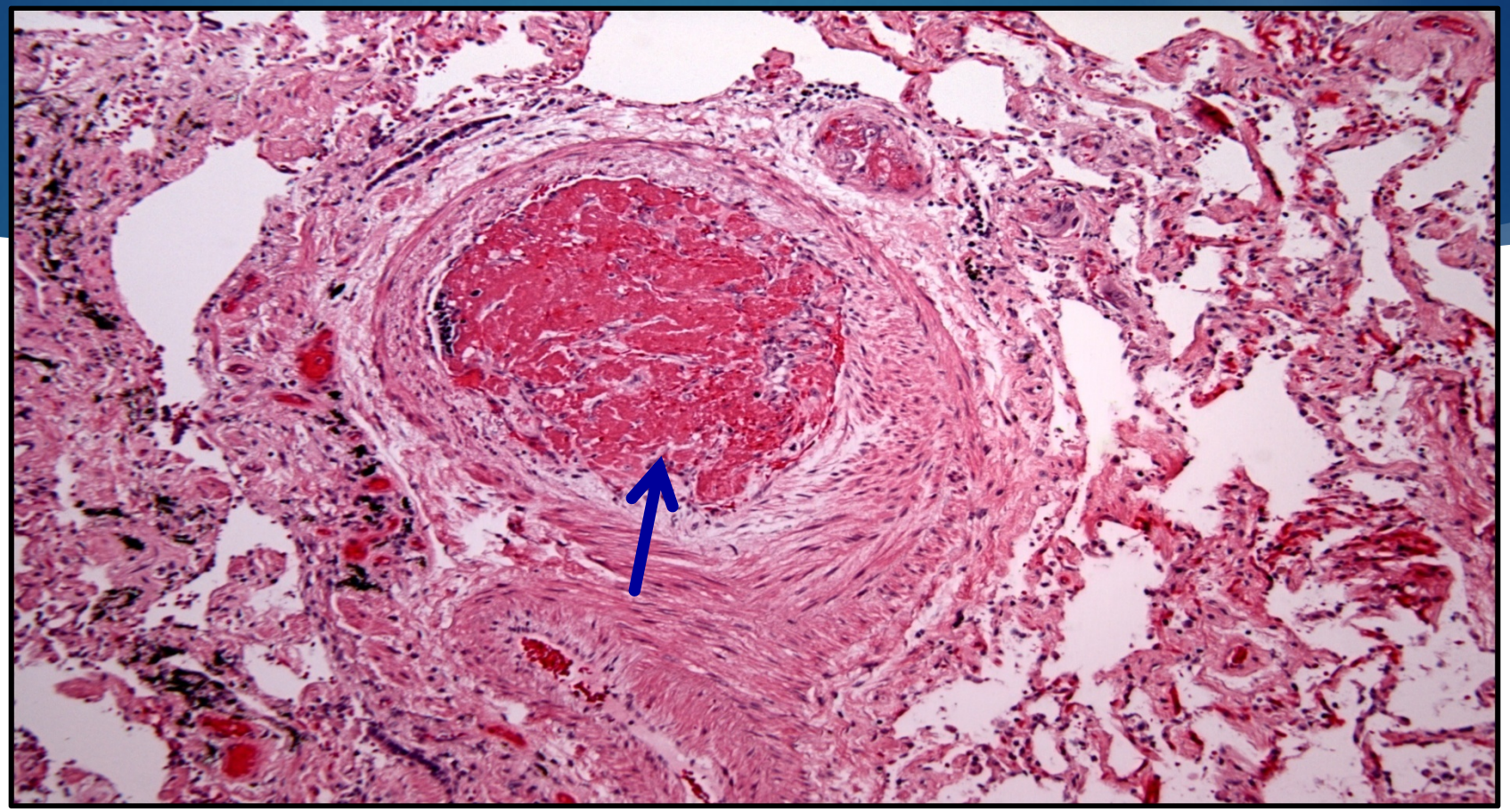
Lines of Zahn





There are interdigitating areas of pale pink and red that form the "lines of Zahn" characteristic for a thrombus. These lines represent layers of red cells, platelets and fibrin which are laid down in the vessel as the thrombus forms. Lines of Zahn, gross and microscopic, is evidence to prove a clot is pre-mortem and not post-mortem. These lines represent layers of red cells, platelets, and fibrin

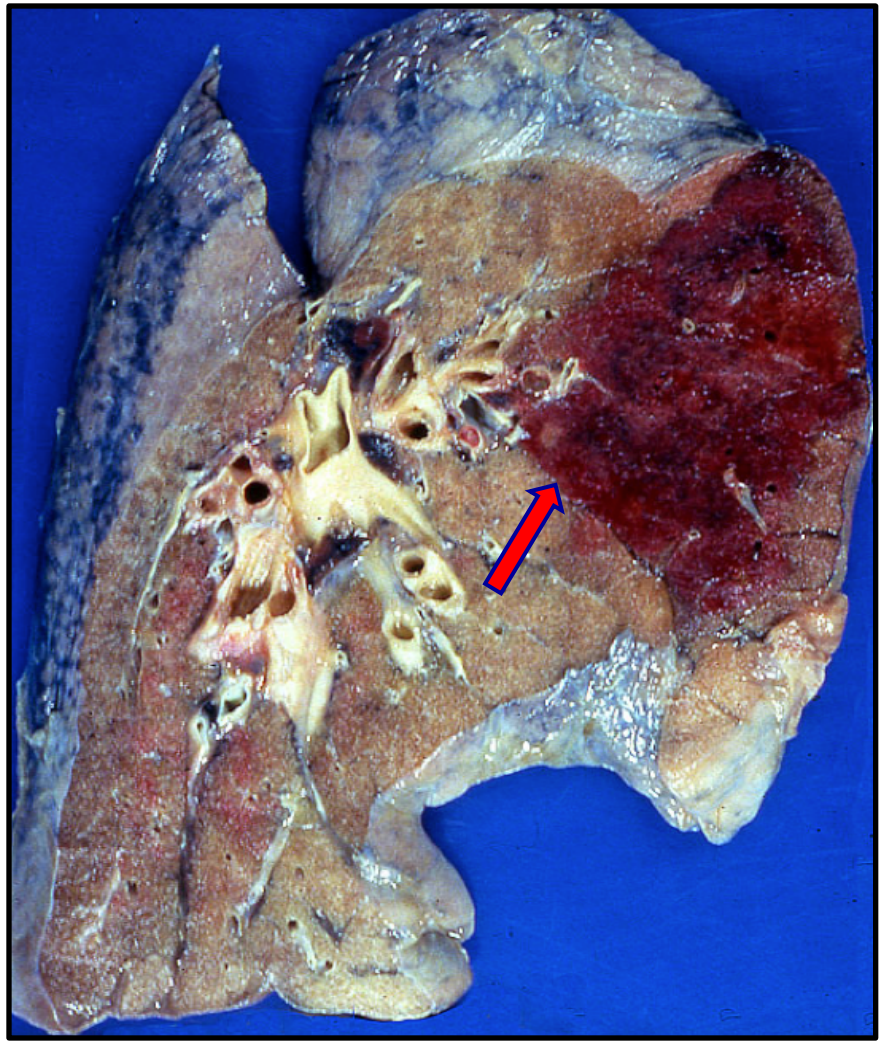
Thromboembolus in Pulmonary Artery

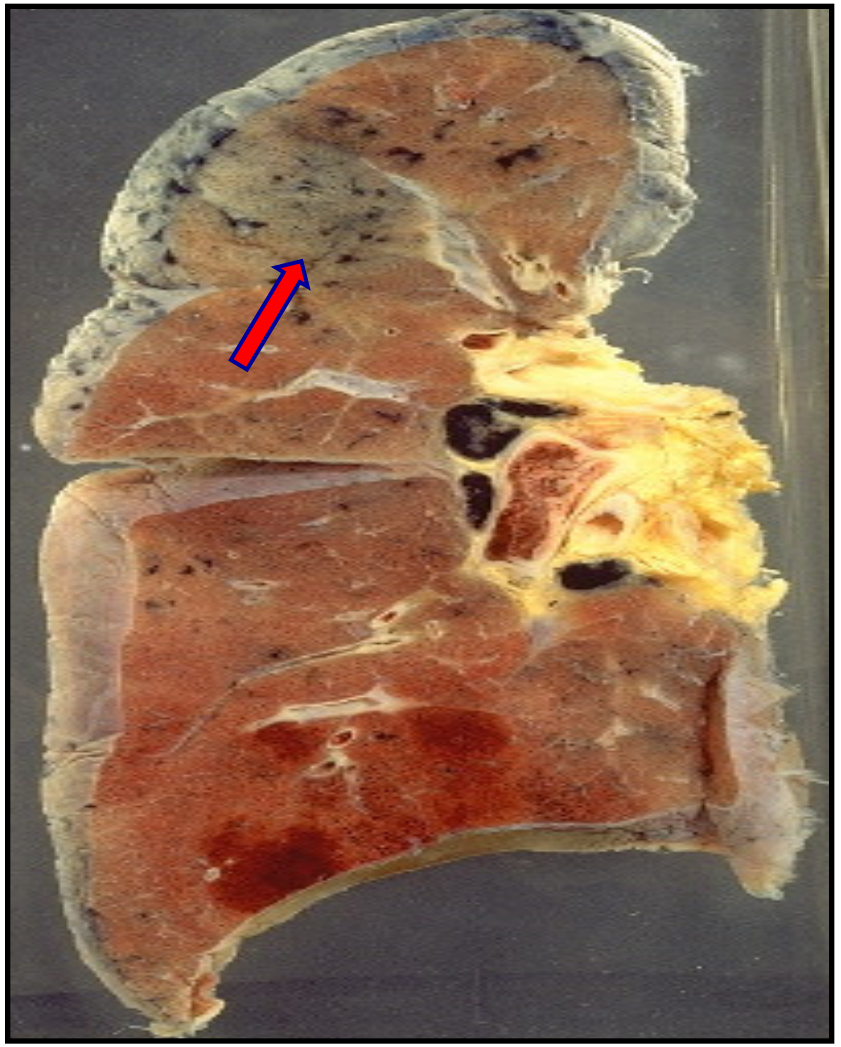


Pulmonary thromboembolus in a small pulmonary artery. The interdigitating areas of pale pink and red within the organizing embolus form the "lines of Zahn" (arrow) characteristic of a thrombus. These lines represent layers of red cells, platelets, and fibrin that are laid down in the vessel as the thrombus forms

2- Pulmonary Embolus with Infarction

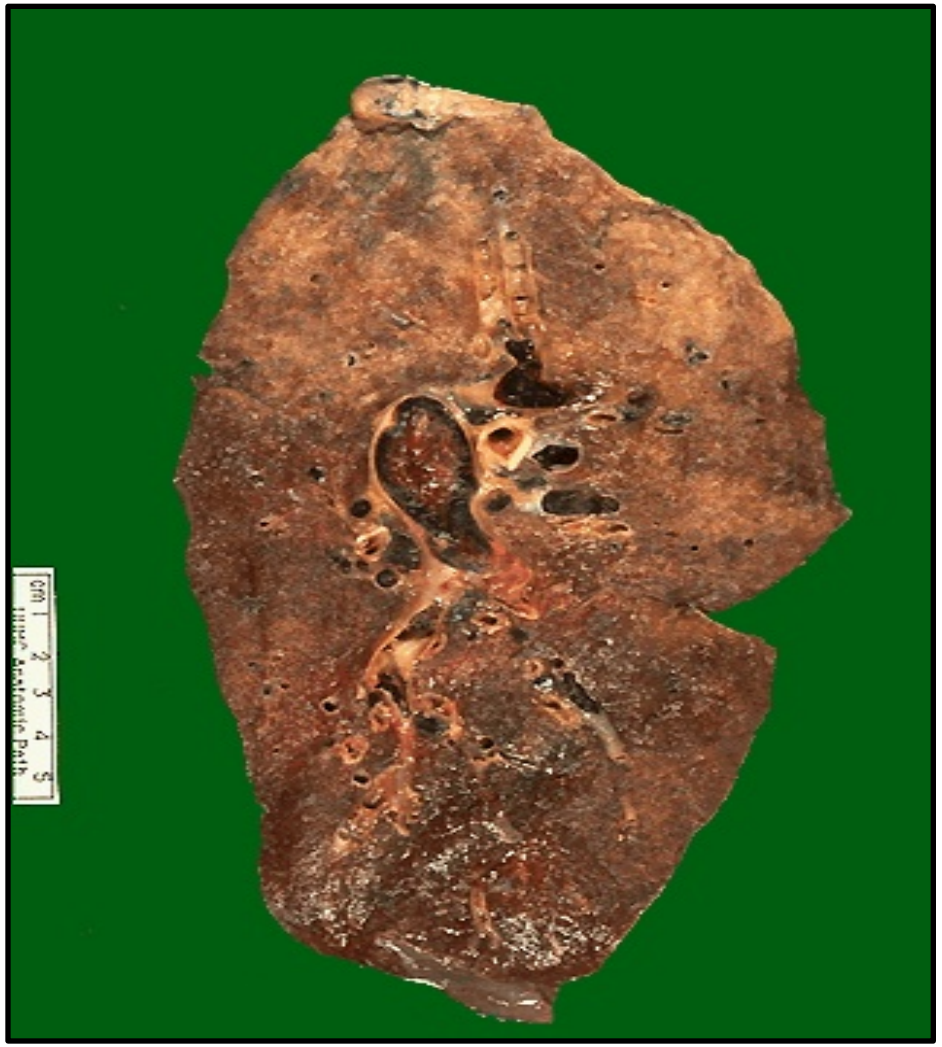
Pulmonary Embolus with Infarction





This specimen shows an area of dead lung tissue ("infarction") due to blockage of one of the major arteries to the lung by an embolus ("blood clot") commonly originating from the deep veins of the leg.

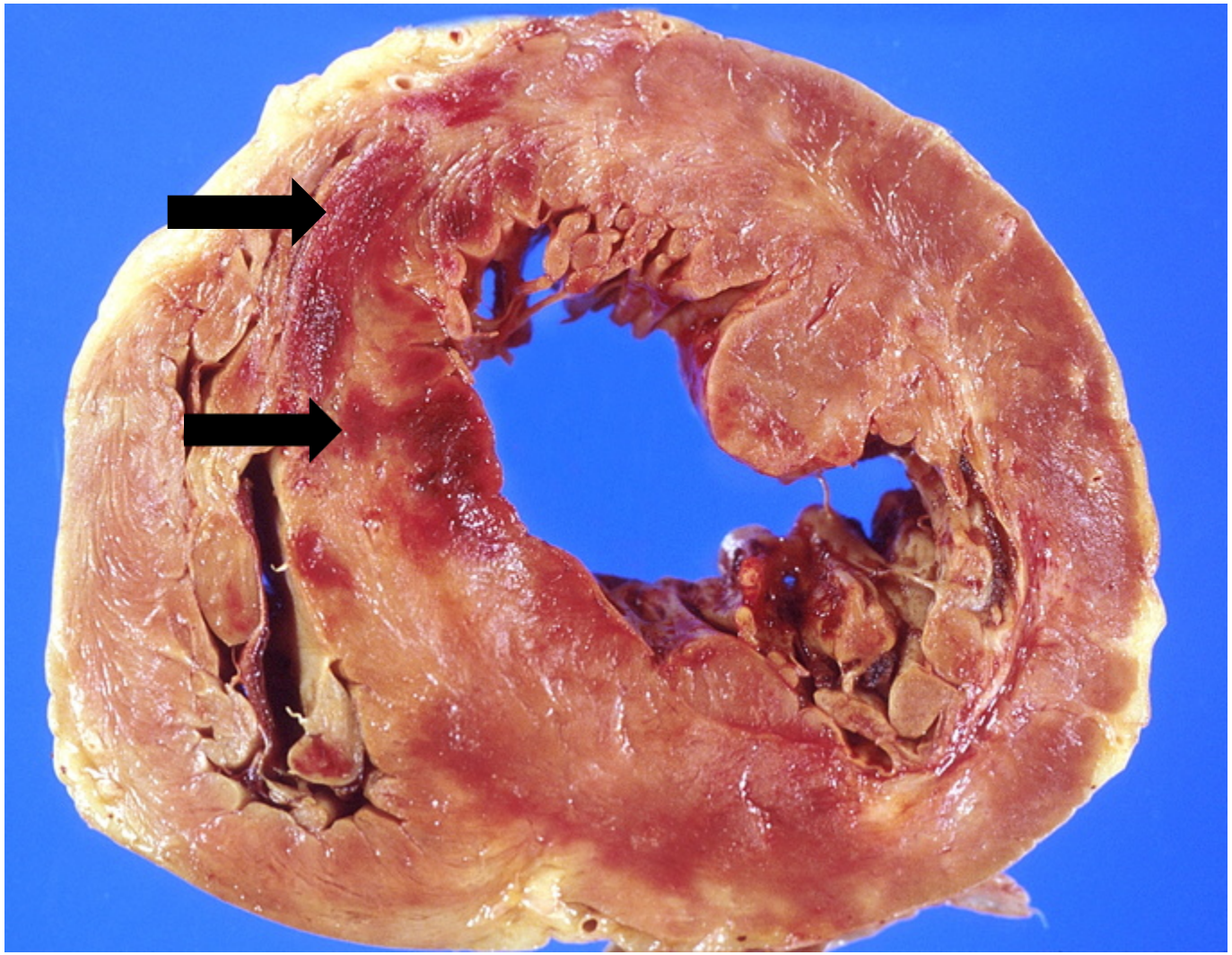
Pulmonary Embolus with Infarction

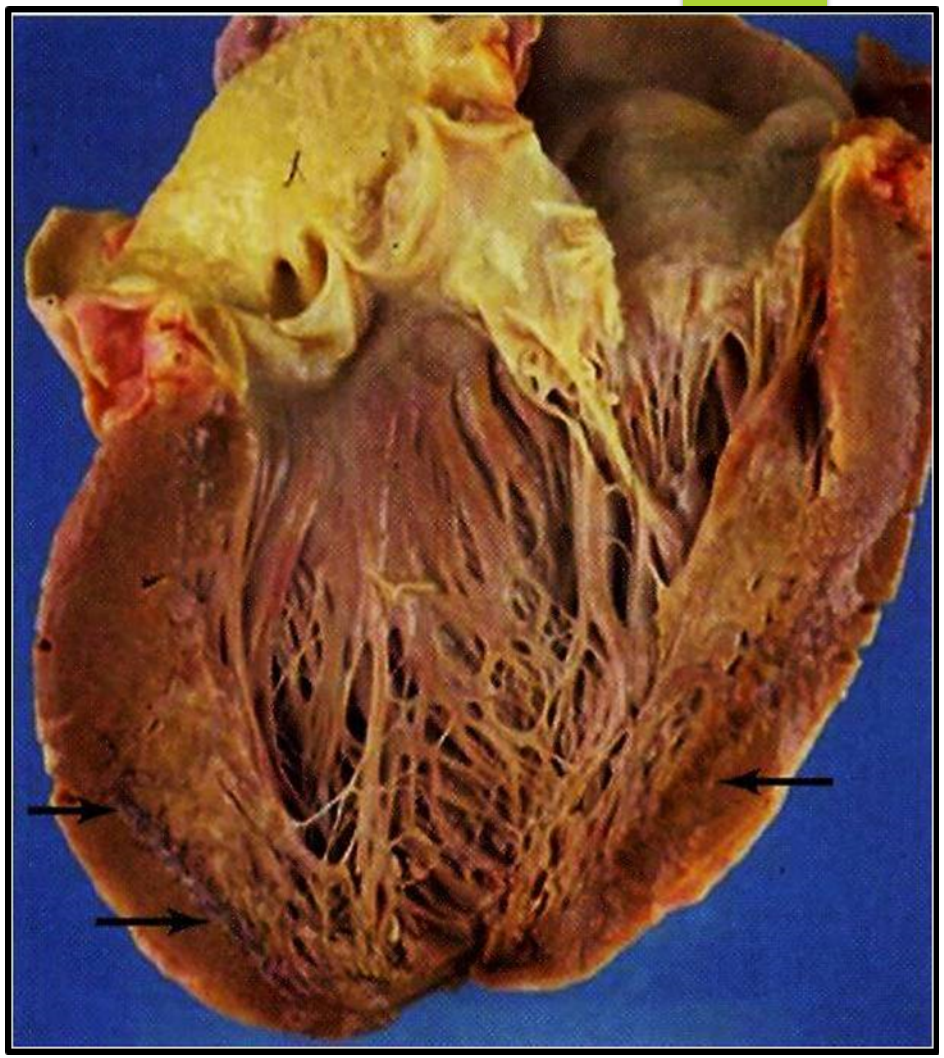


A large pulmonary thromboembolus is seen in the pulmonary artery of the left lung. Such thromboemboli typically originate in the leg veins or pelvic veins of persons who are immobilized

3- Myocardial Infarction

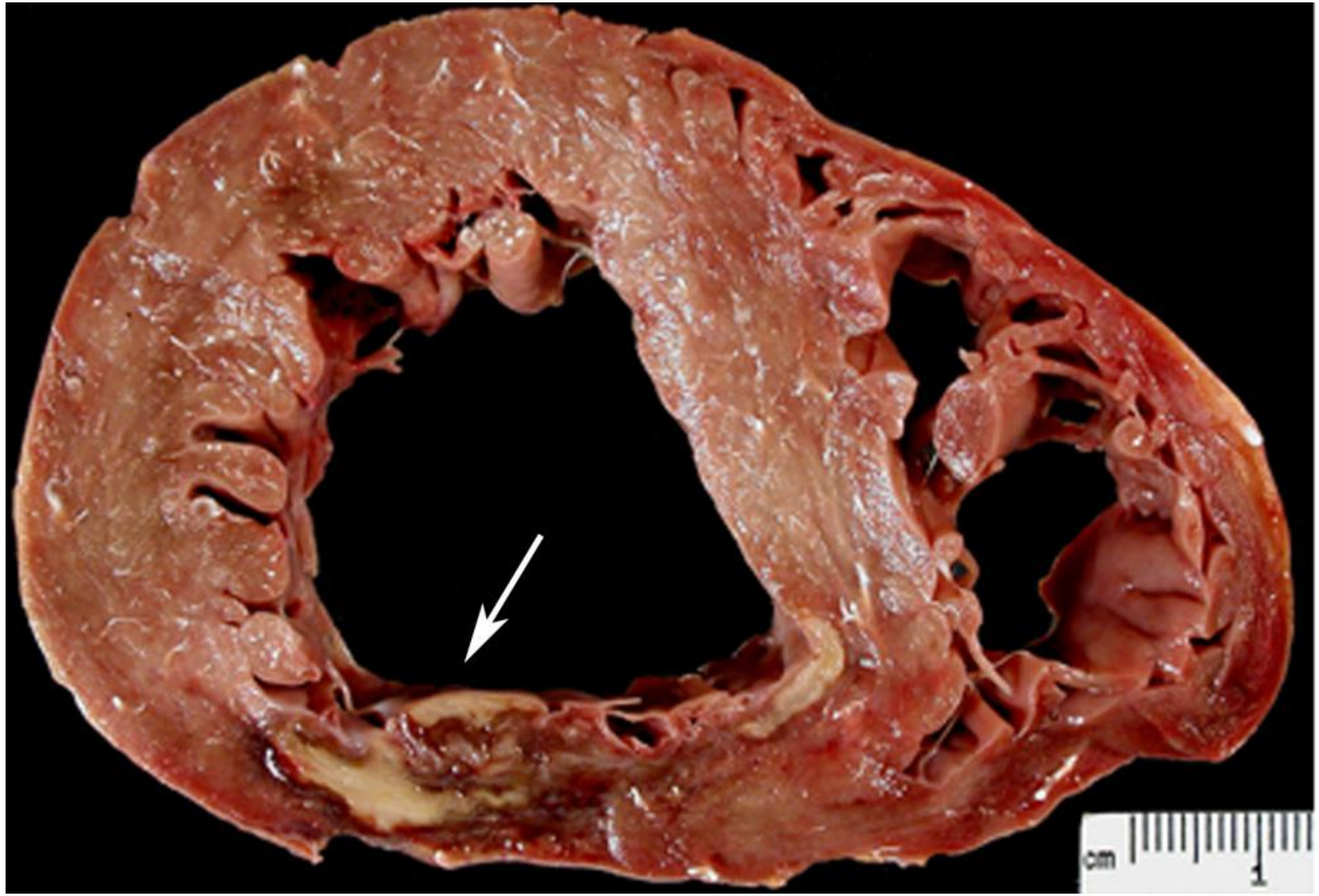
Myocardial Infarction





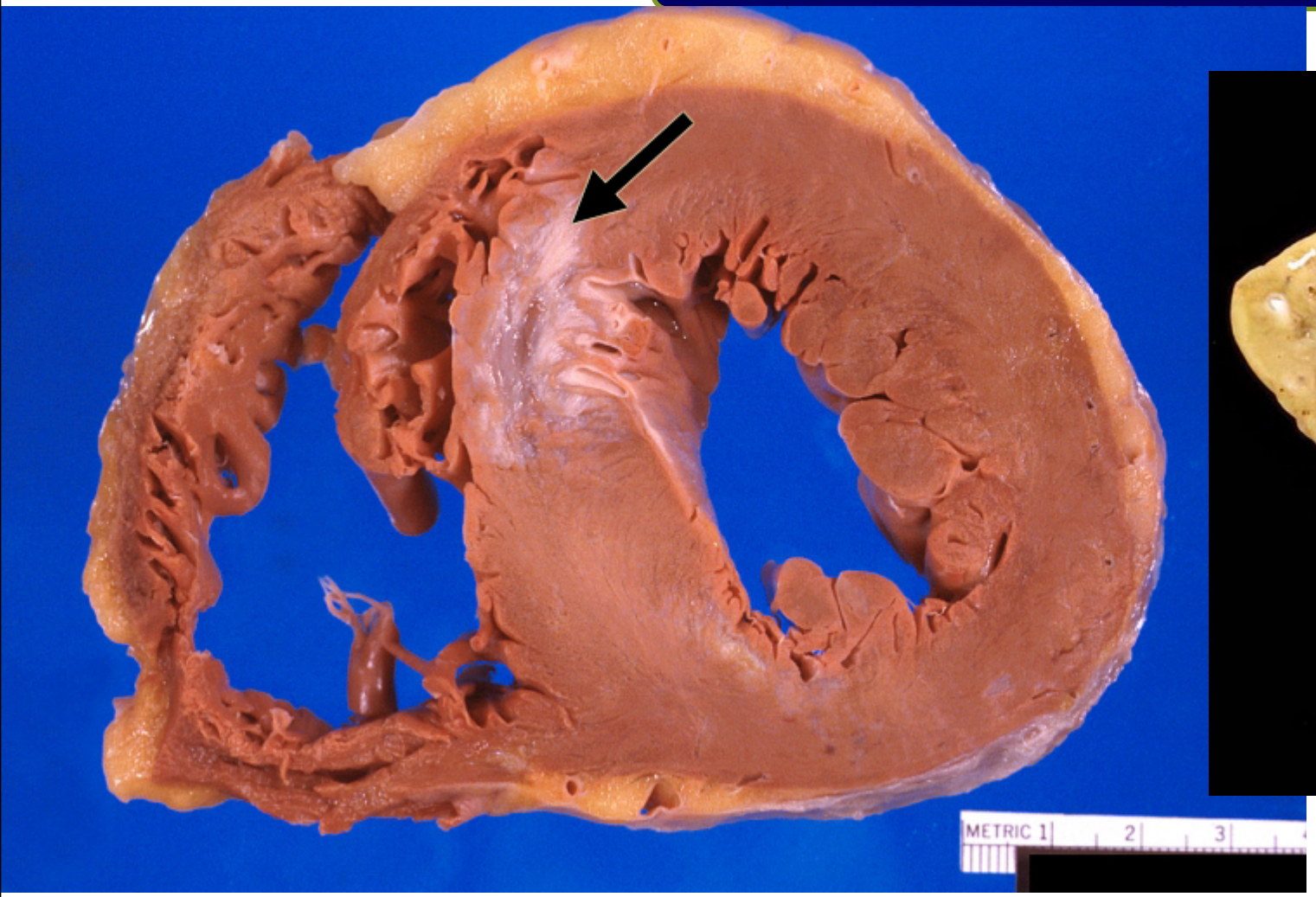
Acute MI: area of fresh myocardial infarction (arrows) in the left ventricle. Initially the area of fresh infarct appears red. The area of infarct becomes well defined by 2 to 3 days with a central area of yellow discoloration surrounded by a thin rim of hemorrhage. There is also some left ventricular hypertrophy.

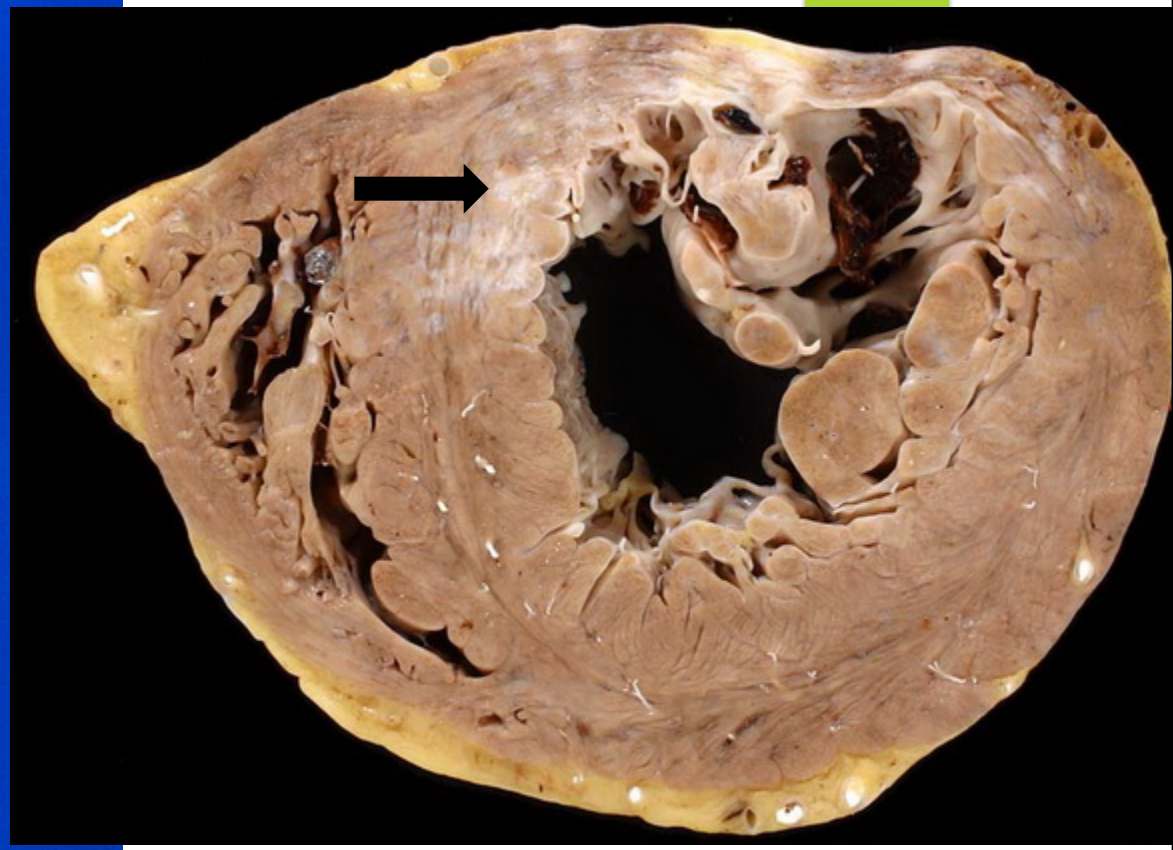
Myocardial Infarction



Acute myocardial infarct. At 3 days, there is a zone of yellow necrosis surrounded by darker hyperemic borders. The arrow points to an infarct in the wall of the left ventricle.

Myocardial Infarction



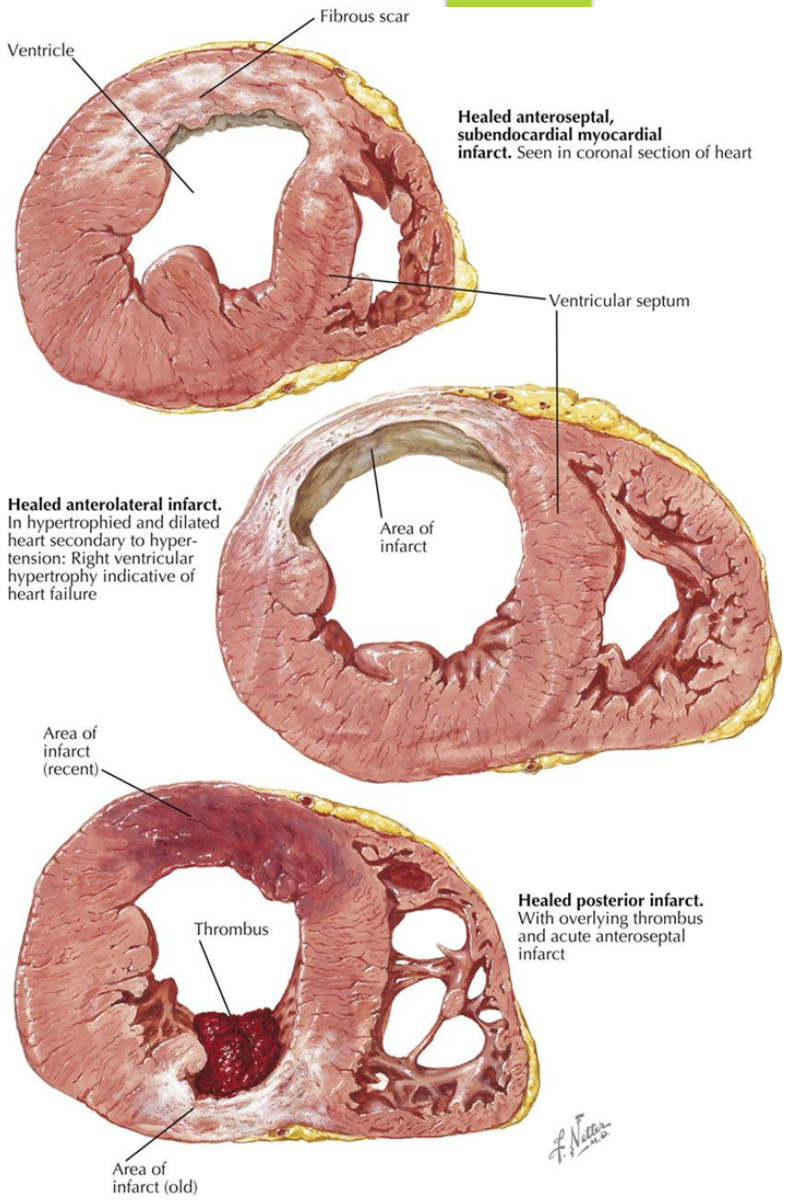


Healed myocardial infarct: cross section of the left and right ventricles shows a pale and irregular area of fibrosis (arrow) in the left ventricular wall. There is also increased thickness of the left ventricular wall (left ventricular hypertrophy).

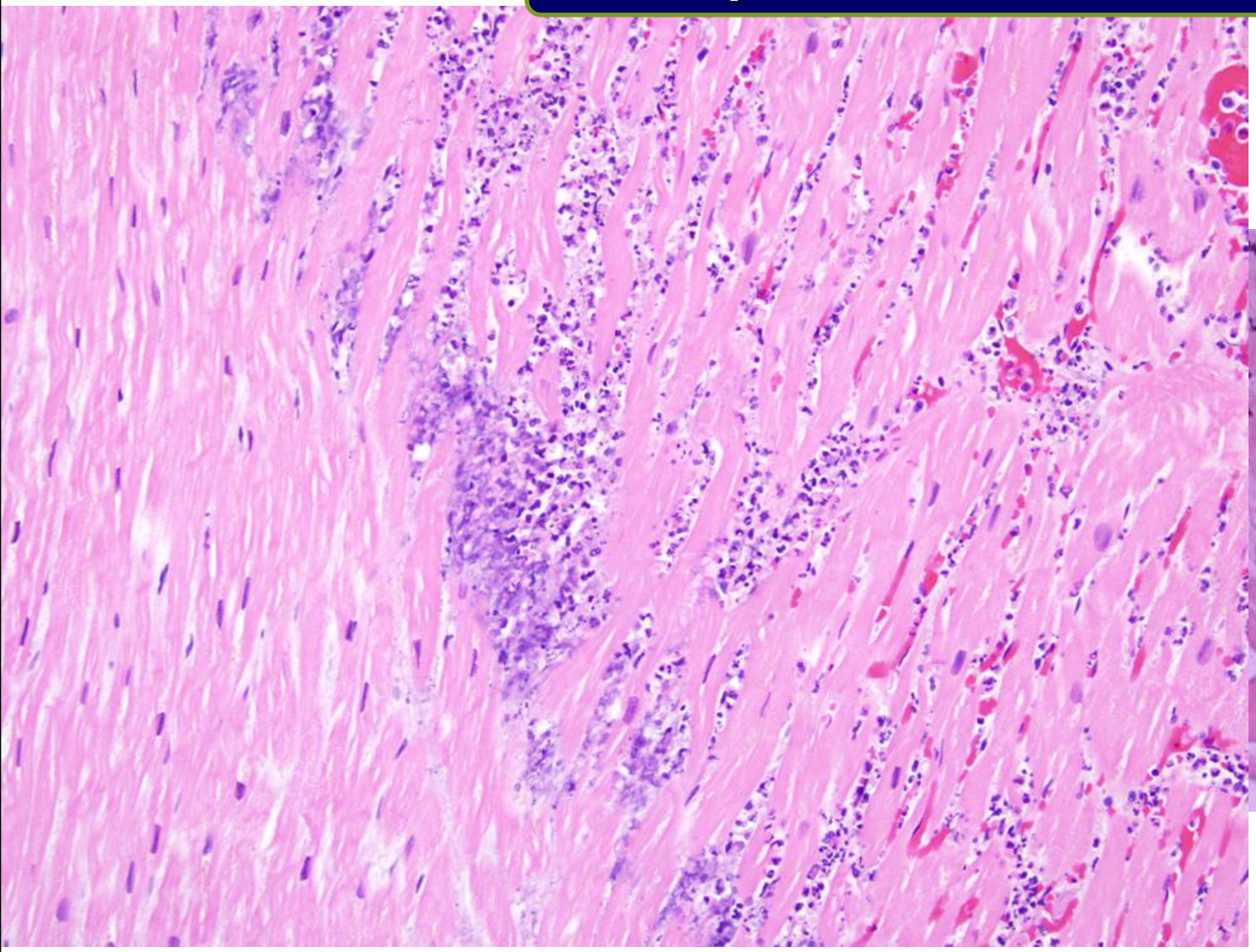
Background information

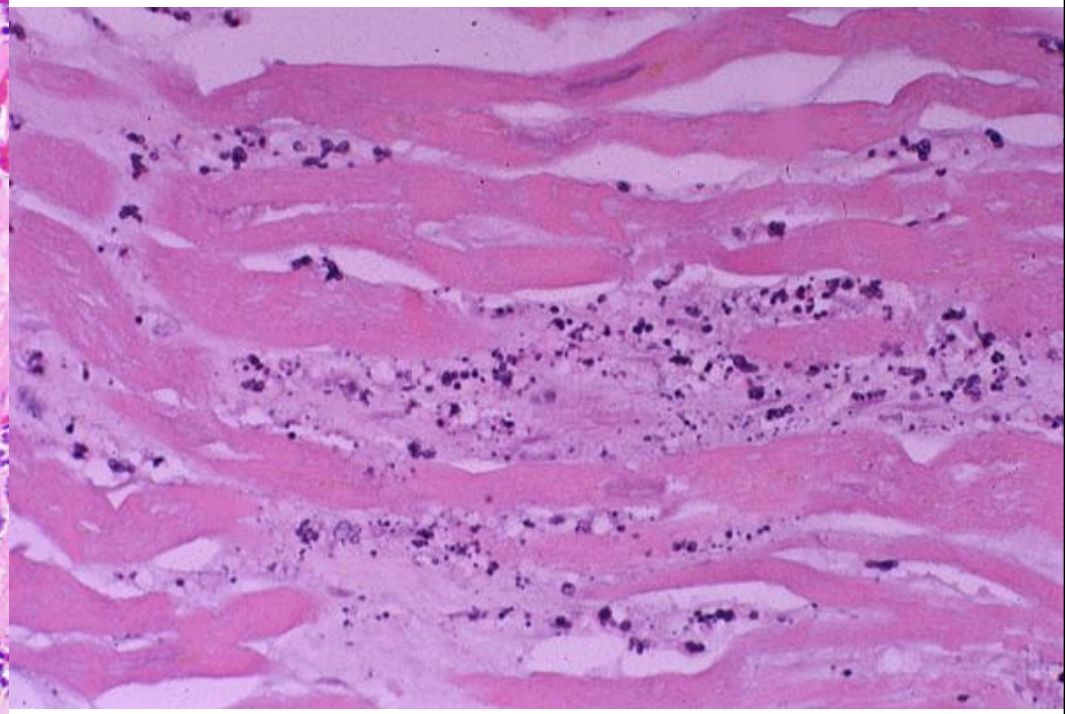
Changes in myocardial Infarction

Time	Gross changes	Microscopic changes
0-4 hours	None	None
4-12 hours	Mild Mottling (hemorrhagic look)	Coagulation necrosis
12-24 hours	Dark Mottling	More coagulation necrosis; neutrophils come in
1-7 days	Yellow infarct center with surrounding red borders	Neutrophils die, macrophages come to eat dead cells
1-2 weeks	Yellow infarct center with red gray borders	Granulation tissue
2-8 weeks	Scar	Collagen and fibrosis



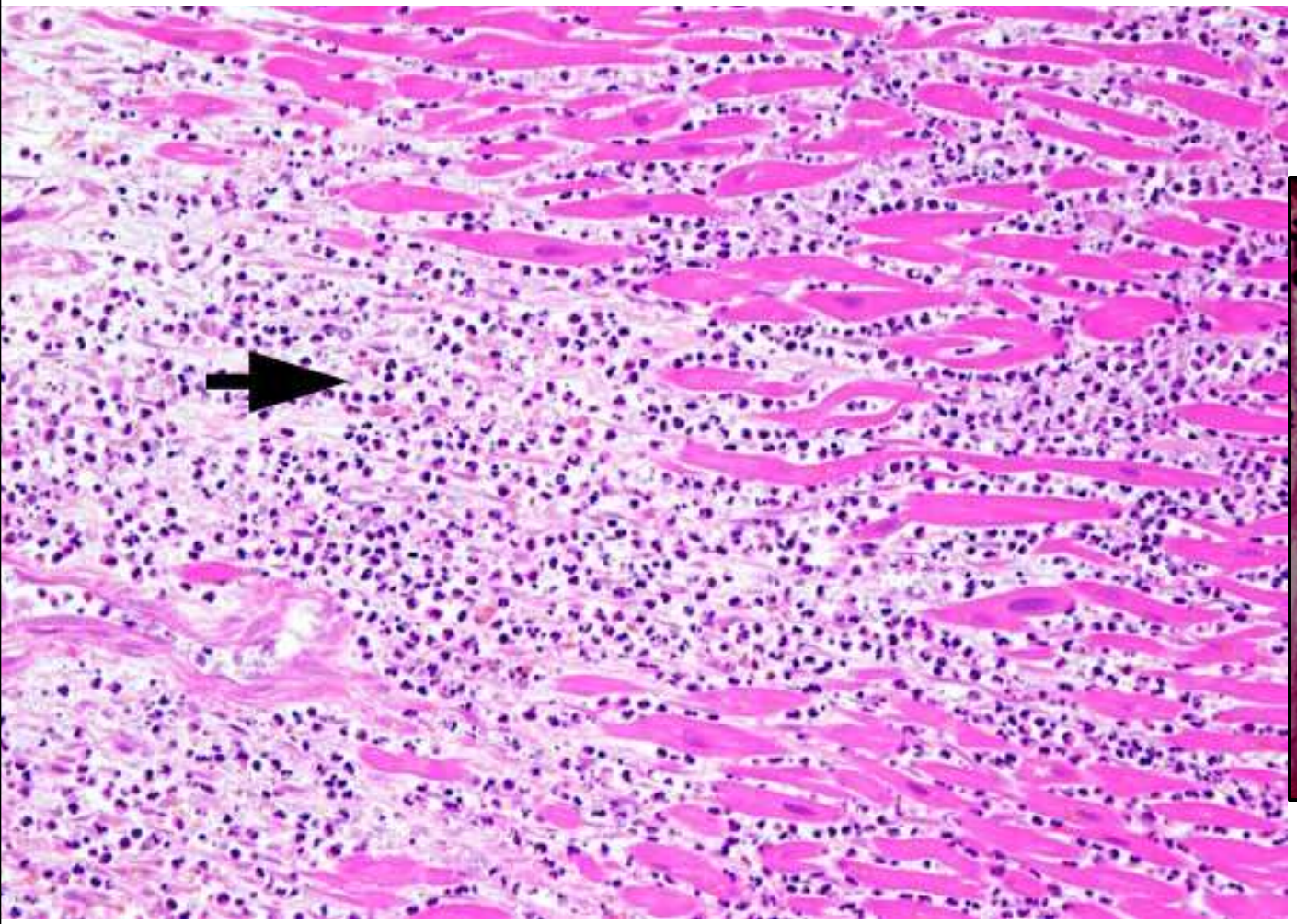
Myocardial Infarction

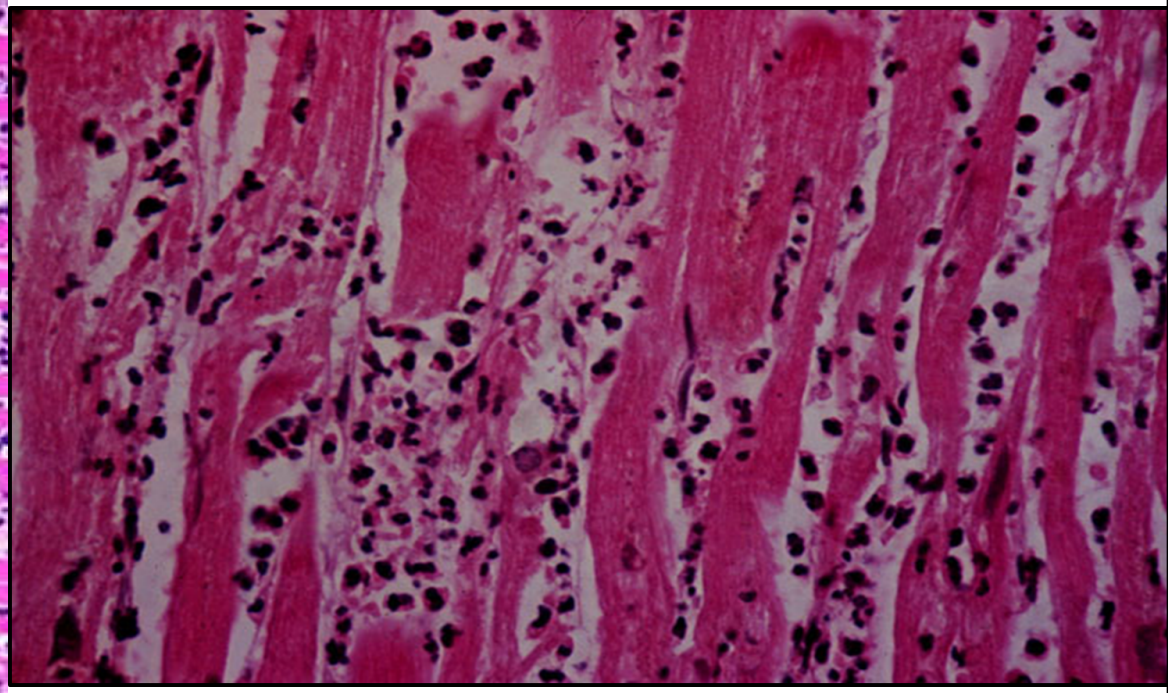




Acute myocardial infarct (after 24 hours) there is a neutrophilic infiltrate at the border of the infarct. Viable myocardium is at the left, and neutrophils are seen infiltrating the necrotic muscle. Note: the nuclei are not clearly visible in most of the necrotic cells.

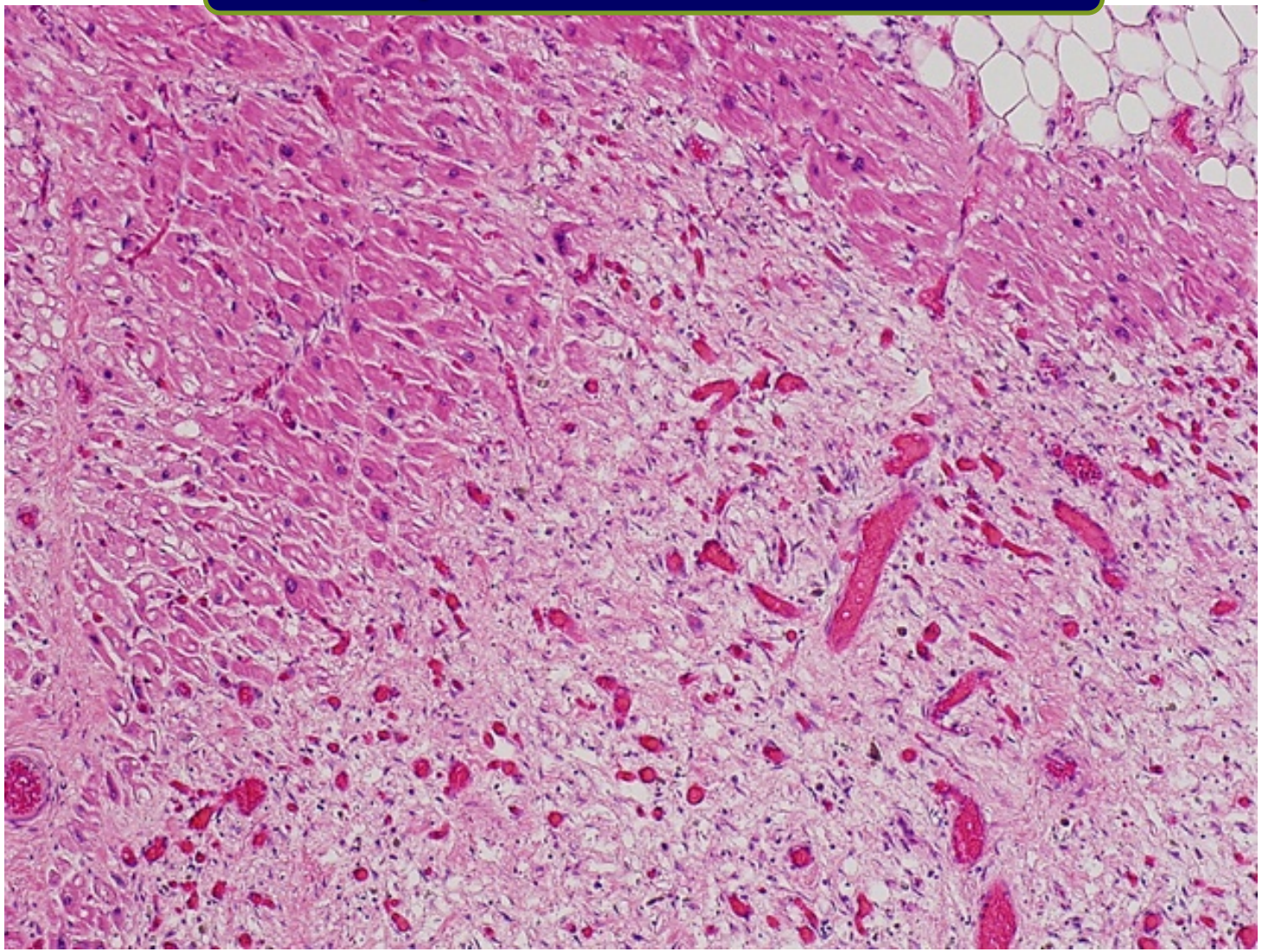
Myocardial Infarction





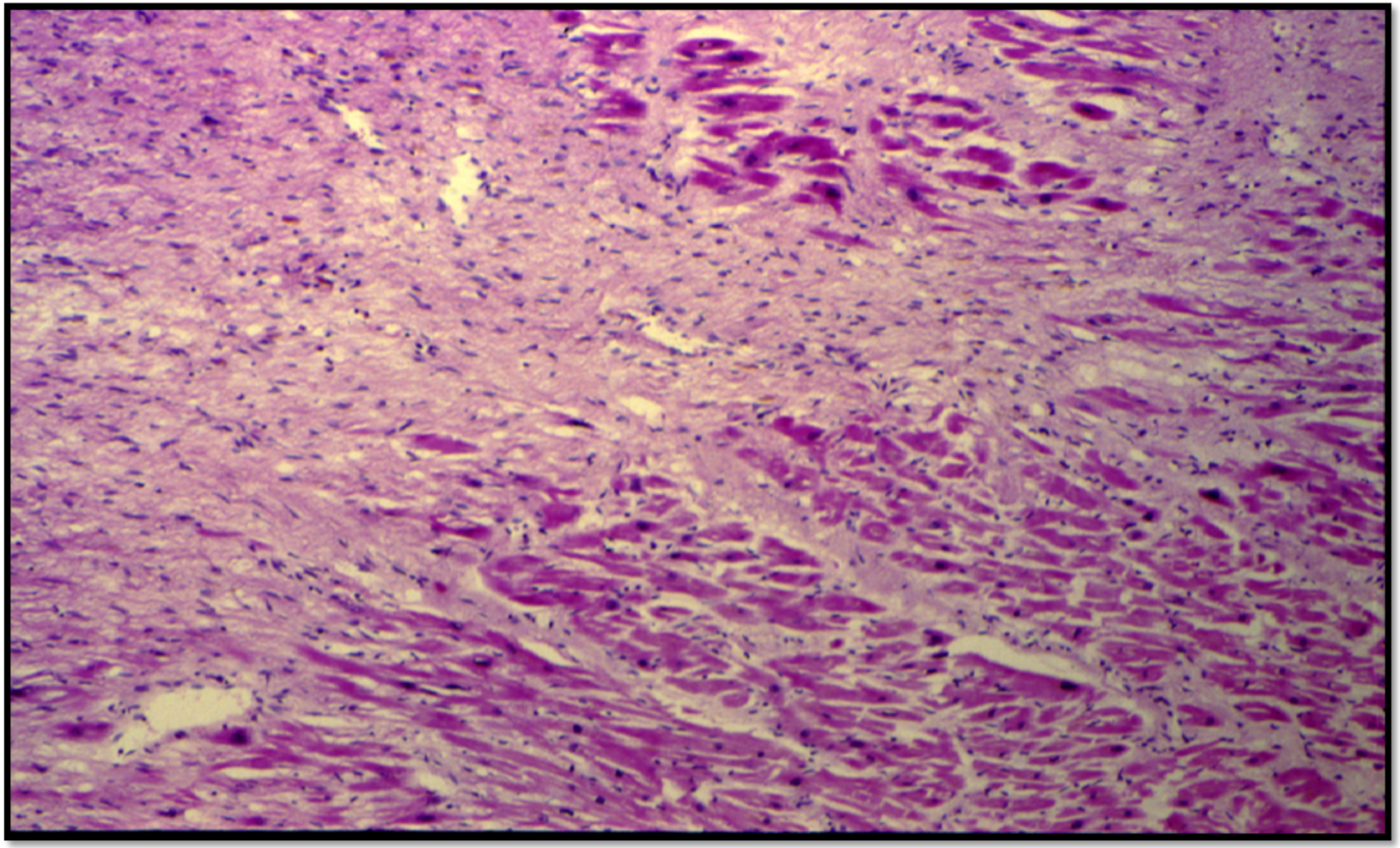
ACUTE MYOCARDIAL INFARCTION: a 3-day old acute infarct showing necrosis of myocardial cells (cardiomyocytes) infiltrated by a heavy neutrophilic infiltrate (arrow). The neutrophils release enzymes that help dissolve dead cell bodies which will be phagocytized by macrophages. With time the neutrophils begin to die and replaced by an influx of macrophages.

Myocardial Infarction



Recent MI with early healing changes (3 weeks post MI) → shows granulation tissue (growth of capillaries and fibroblasts) and the collagen is being laid down to form a scar. The non-infarcted myocardium is present on the left and upper part of the picture.

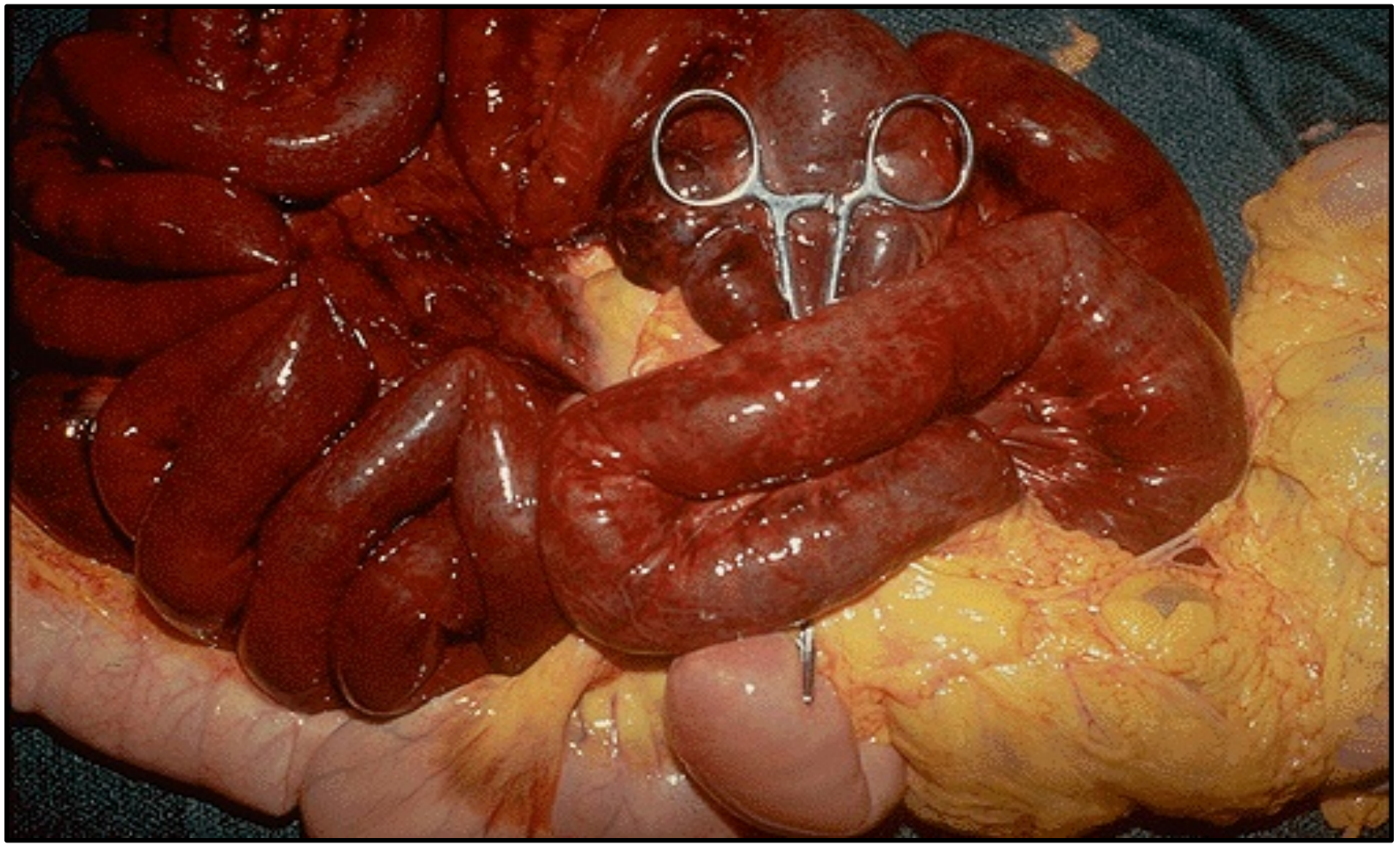
Myocardial Infarction



Healed myocardial infarct: in it there is replacement of the necrotic cells by a dense collagenous scar. The myocardium shows fibrosis with collagenization (scar) following healing of a myocardial infarction. Residual viable red myocardial fibers are present. This stage is reached about 2 months post MI.

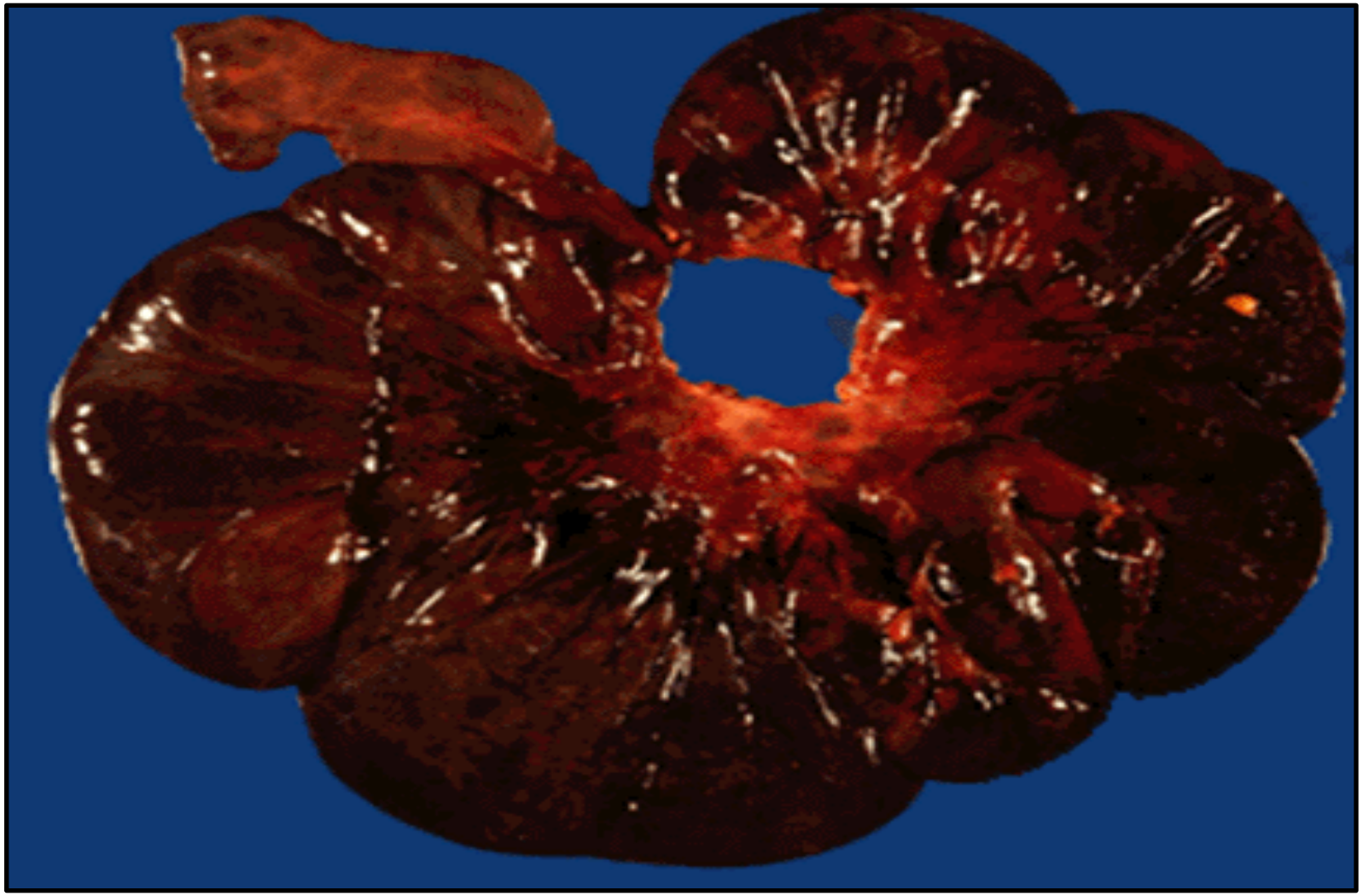
4- Infarction of the Small Intestine

Infarction of the Small Intestine



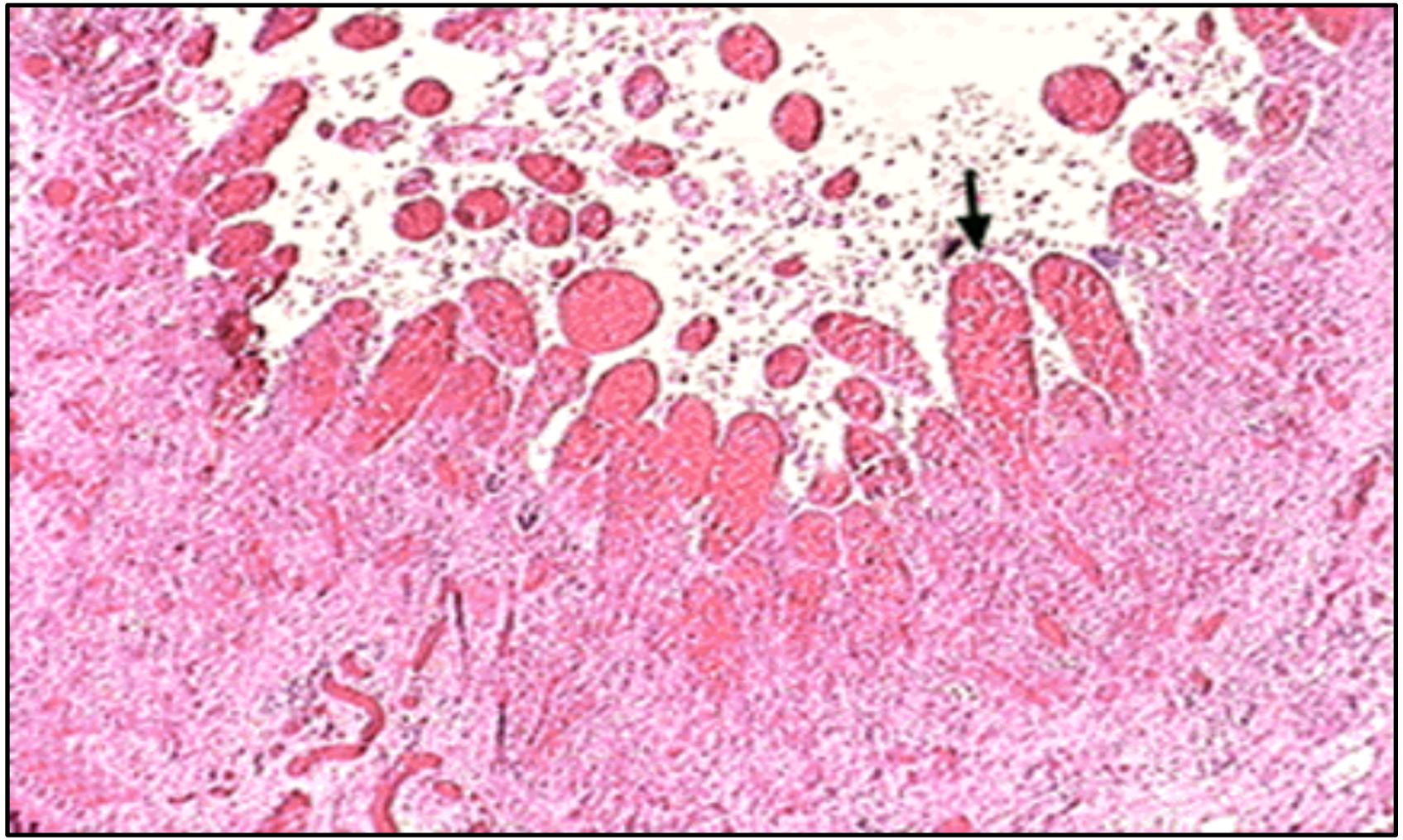
The dark red infarcted small intestine contrasts with the light pink viable bowel. (note: the forceps extend through an internal hernia in which a loop of bowel and mesentery has been caught. This is one complication of adhesions from previous surgery. The trapped bowel has lost its blood supply

Infarction of the Small Intestine



Diffuse violacious red appearance is characteristic of transmural hemorrhagic intestinal infarction

Infarction of the Small Intestine



Intestinal infarction typically begins in the villi, which are end vasculature without anastomoses. There is complete loss of the mucosal epithelium. Broad areas of hemorrhage with moderate inflammatory infiltrate is present