Hemodynamic disorders
Pathology practical

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* *NOTES*

Hemostasis &Thrombosis*

- Normal hemostasis maintain blood in fluid, clot free state in normal vessel and induce localized hemostatic plug at site of vascular injury.

- Thrombosis is **inappropriate activation** of **normal hemostatic process** (eg formation of thrombus/blood clot) in un-injured vasculature or thrombotic occlusion of vessel after minor injury. *(opposite of hemostasis)*

** Thrombosis is the formation of blood clot.*

- **Normal fluid homeostasis** means ***maintaining blood as a liquid until such time as injury necessitates clot formation.***
- Clotting at inappropriate sites ***(thrombosis)*** or migration* of clots ***(embolism**)*** obstructs blood flow to tissues & leads to cell death ***(infarction).***

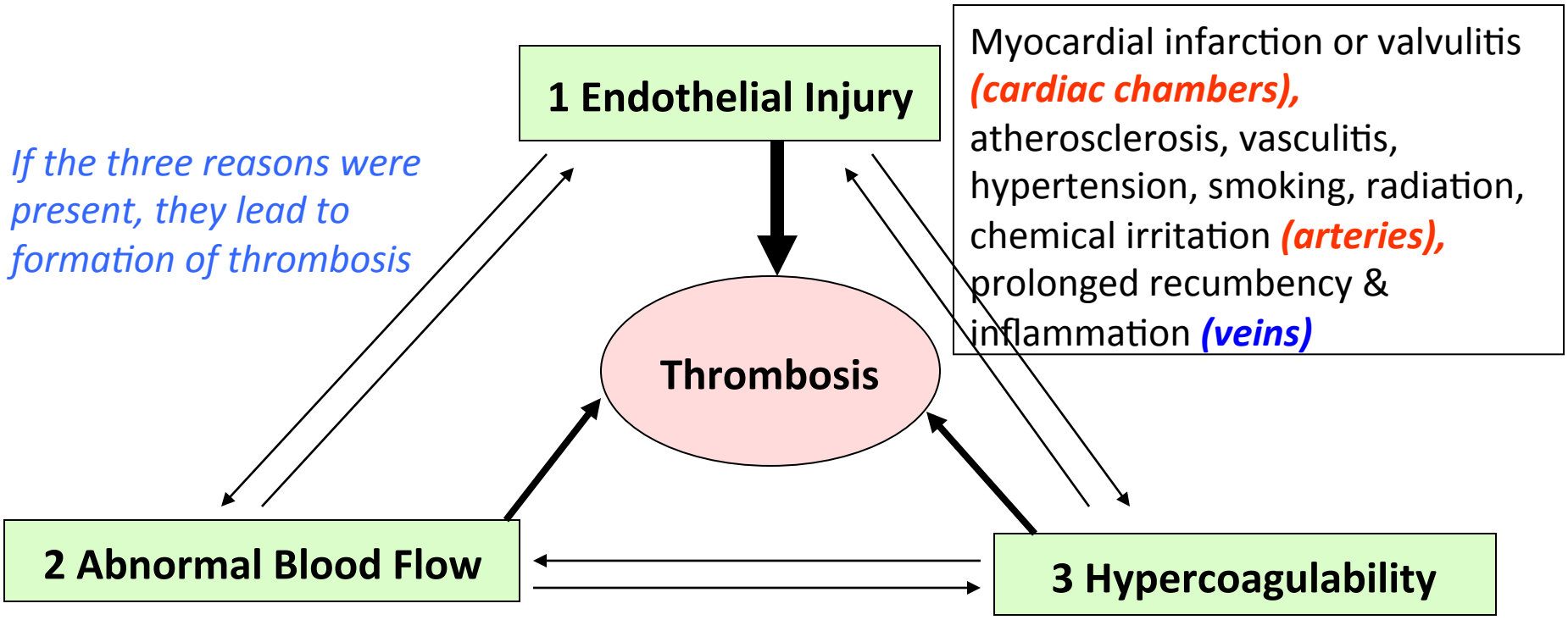
** Transfer of clots from one place to another.*
(like if the clot was in one organ (leg) and then it goes to another)

*** Embolism leads to infarction and cell death.*

Thrombosis (Important)

Pathogenesis (Causes= Predisposing Factors)

Three primary influences **predispos**e to thrombus formation called **Virchow's triad**



Turbulence *(arteries) &(cardiac chambers)*

Stasis *(veins)*

Atherosclerosis, aneurysm, dilated atria, atrial fibrillation, venous stasis, varicose veins

Primary causes (Genetic)
Secondary Causes (Acquired): High Risk Causes & Low Risk Causes

Fate of the Thrombus

If the patient survives the immediate effects of a thrombotic vascular obstruction, thrombi undergo some combination of the following **four events**

1.Propagation*: the thrombus may accumulate more platelets and fibrin eventually **obstructing** other critical vessel.

2.Dissolution: Thrombi may be **removed** by the fibrinolytic activity.

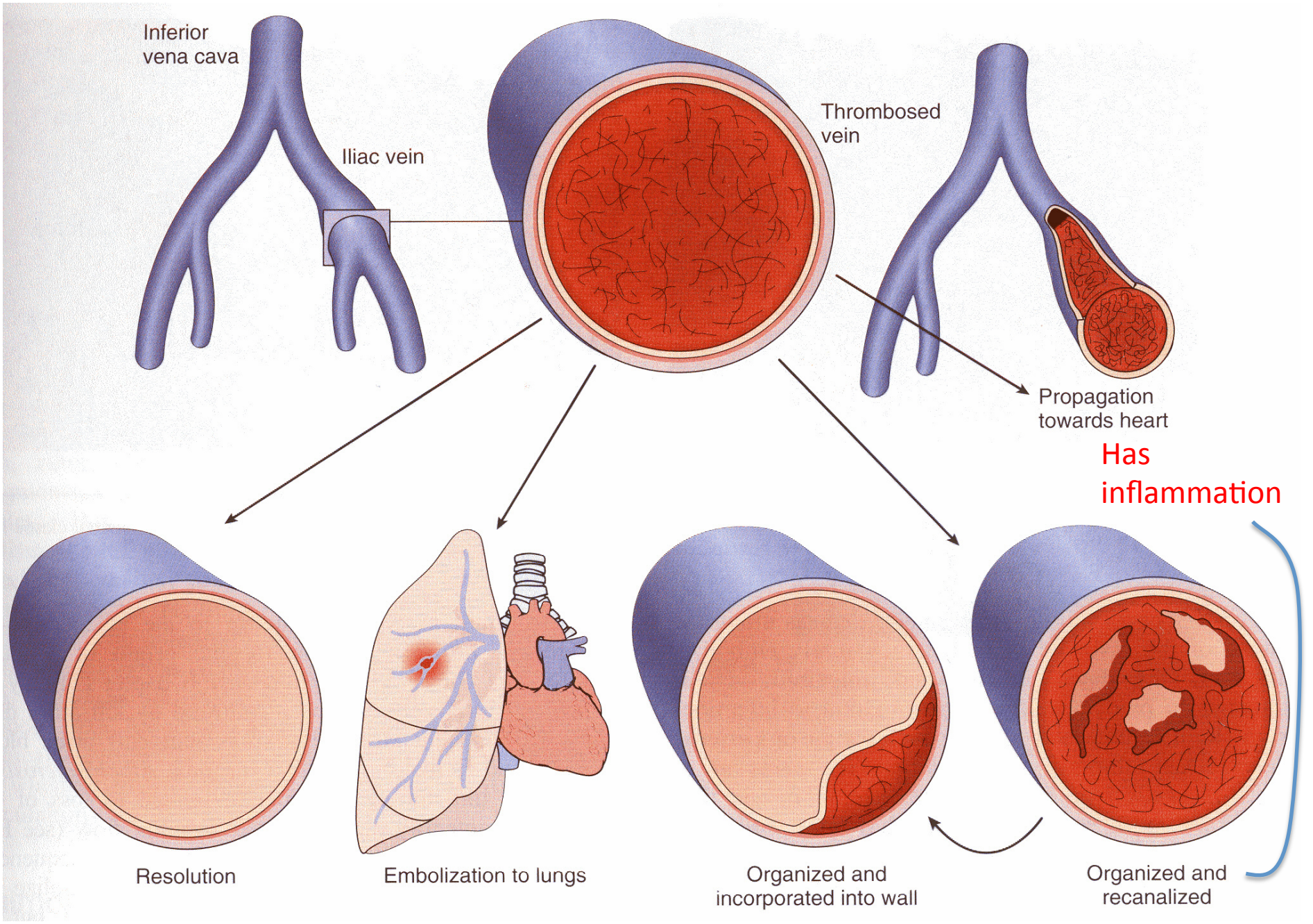
3.Embolization:** Thrombi may dislodge as **thrombotic emboli.**

4.Organization and Recanalization*:**
Thrombi may induce inflammation and fibrosis (organization) and may eventually **recanalize.**

** Increasing of the size.*

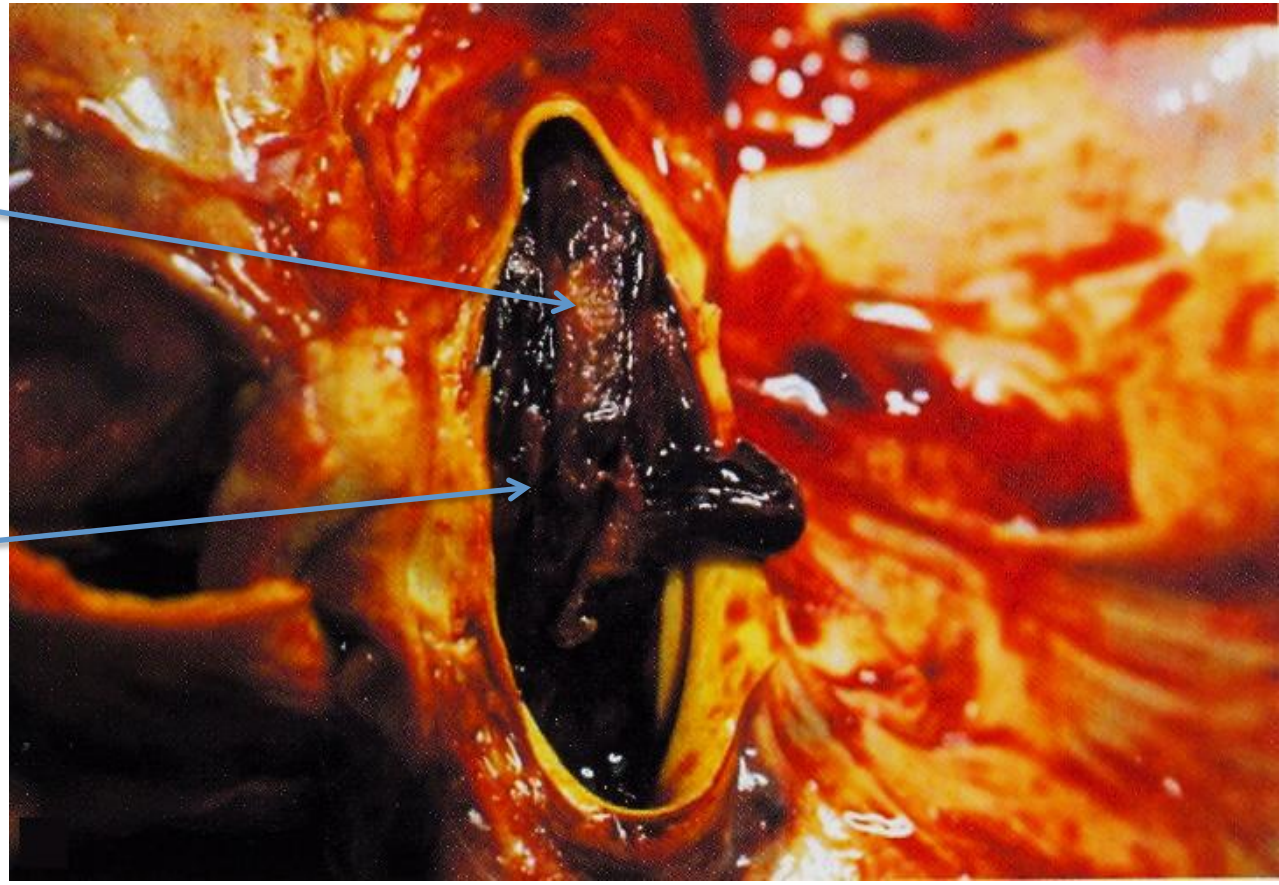
*** Moving from one place to another.*

**** Comes with inflammation.*


Inferior
vena cava
Iliac vein
Thrombosed
vein
Propagation
towards heart
Has
inflammation
Resolution
Embolization to lungs
Organized and
incorporated into wall
Organized and
recanalized

1- Organizing thrombus

Organizing thrombus in a case of pulmonary embolism

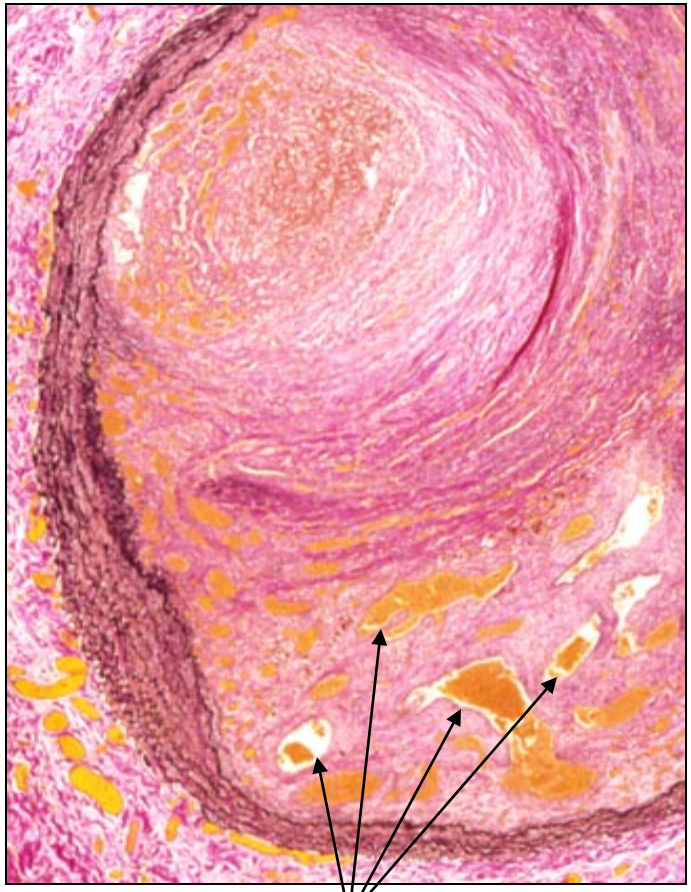


Whitish area means there is inflammation and trying to embolize

Predisposing factors for thrombus formation are: Prolonged bed rest, hypercoagulability syndrome and multiple bone fractures The patient can experience sudden onset of shortness of breath. Death may occur within minutes. Persons who are immobilized for weeks are at greatest risk.

Organising thrombus:

- reparative process
- ingrowth of fibroblasts and capillaries (similar to granulation tissue)
- lumen remains obstructed may eventually **recanalize**



Organization &Recanalization (multiple capillary channels)

Organizing thrombus:
Cross section of blood vessel shows:

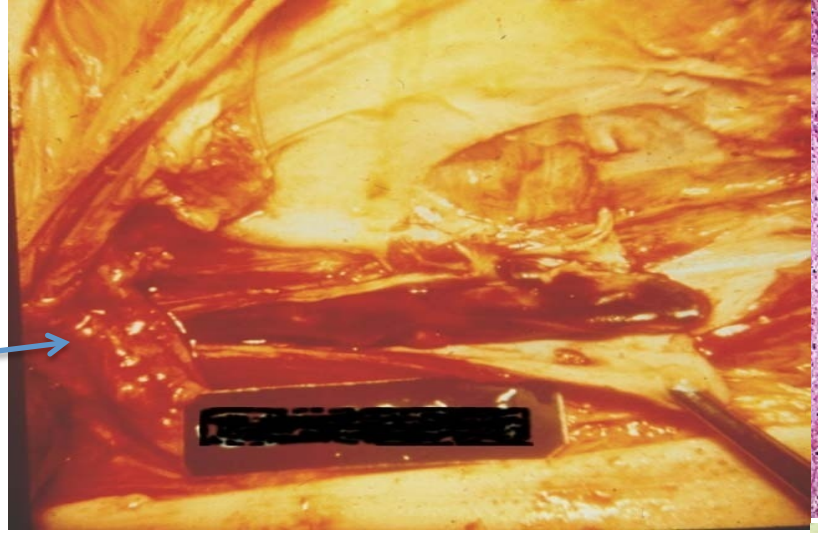
- The lumen is occluded by thrombus which consists of alternate layers of platelets with fibrin thread and clotted blood (line of Zahn*).

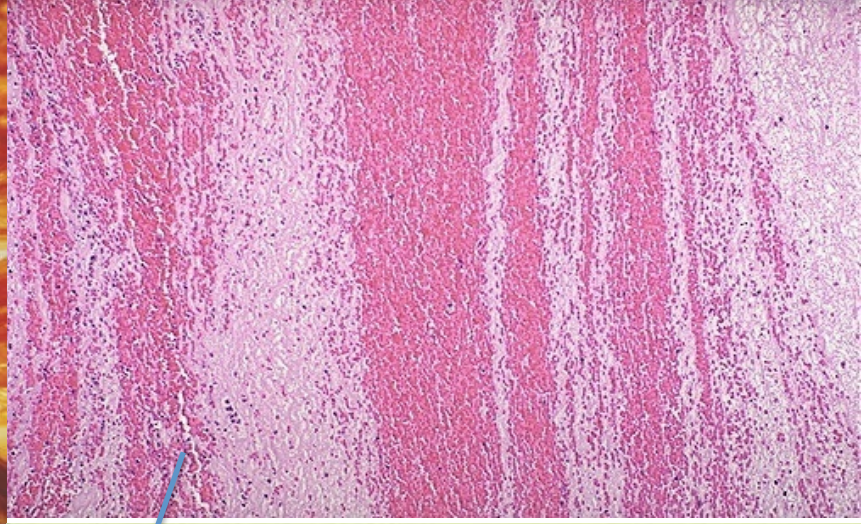
- Organization can be seen at the periphery of thrombus which includes formation of small capillaries & fibroblasts with chronic inflammatory cells.

- Recanalization is seen at one side.

** Seen in alternate layers of platelets with fibrin*

Before Death





After Death

Line of Zahan

Lines of Zahn, gross and microscopic, top, is evidence to prove a clot is PRE-mortem.
Clots appearing like current jelly or chicken fat are said to be POST-mortem.
In general, post mortem clots are rather AMORPHOUS and have no binding texture when you squeeze them

Embolism

Definition:

Embolism is the blockage of a blood vessel by solid, liquid or gas at a site distant from its origin.

And there is fat embolism

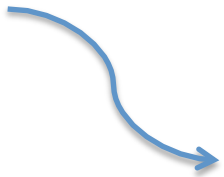
>90% of emboli are thrombo-emboli

Liquid example : amniotic fluid

Gas example : nitrogen

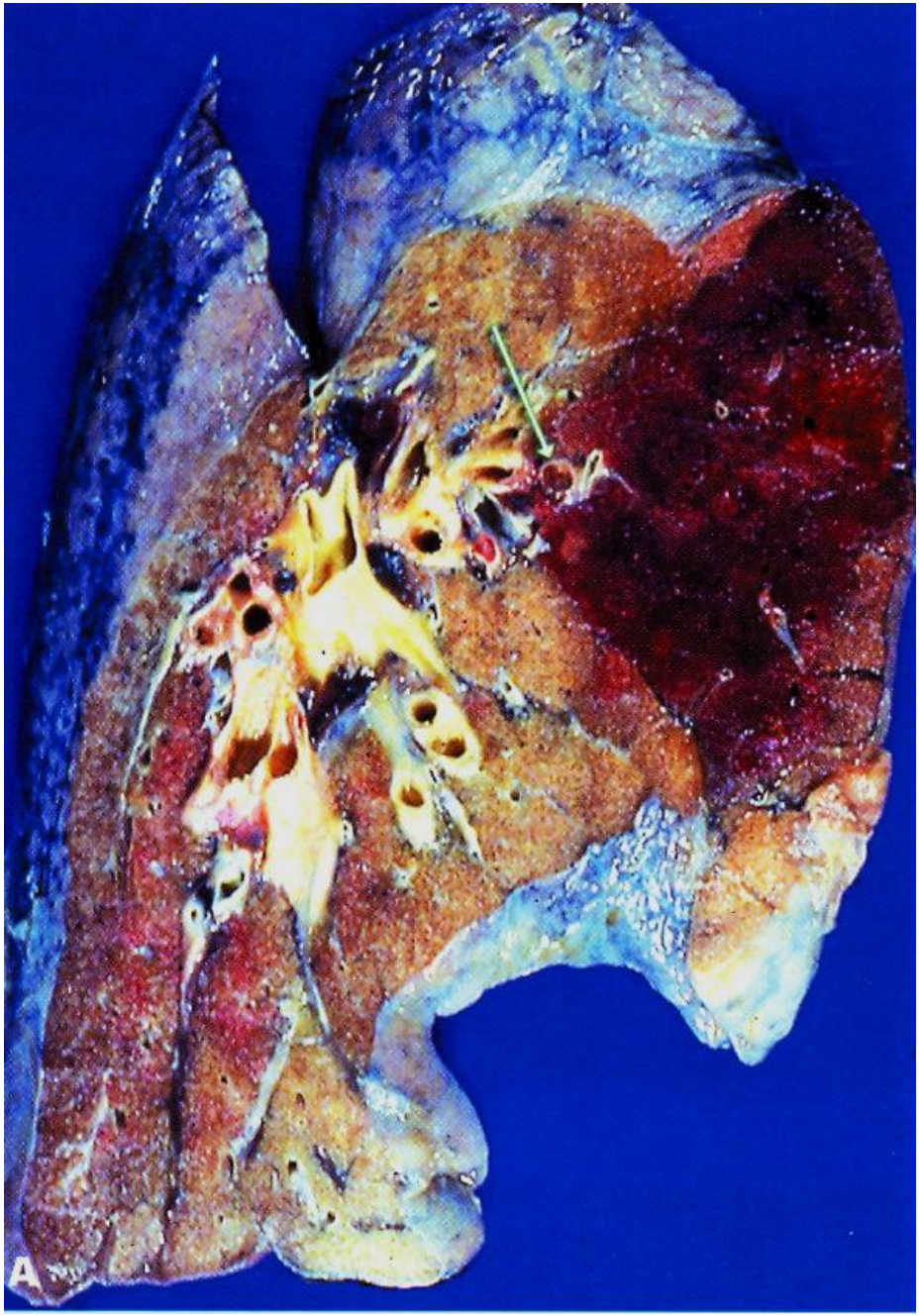
Embolism

- Other types
  - air
  - amniotic fluid
  - nitrogen
  - medical equipment
  - tumour cells



Metastasis

2- Pulmonary embolus with infarction



Region of lung infarction

** Because of the obstruction*

Infarction*

❖***It is an area of ischemic necrosis caused by occlusion of either the arterial supply or the venous drainage in a particular tissue.***

** Death of the tissue caused by the poor supplying of the blood*

Infarction

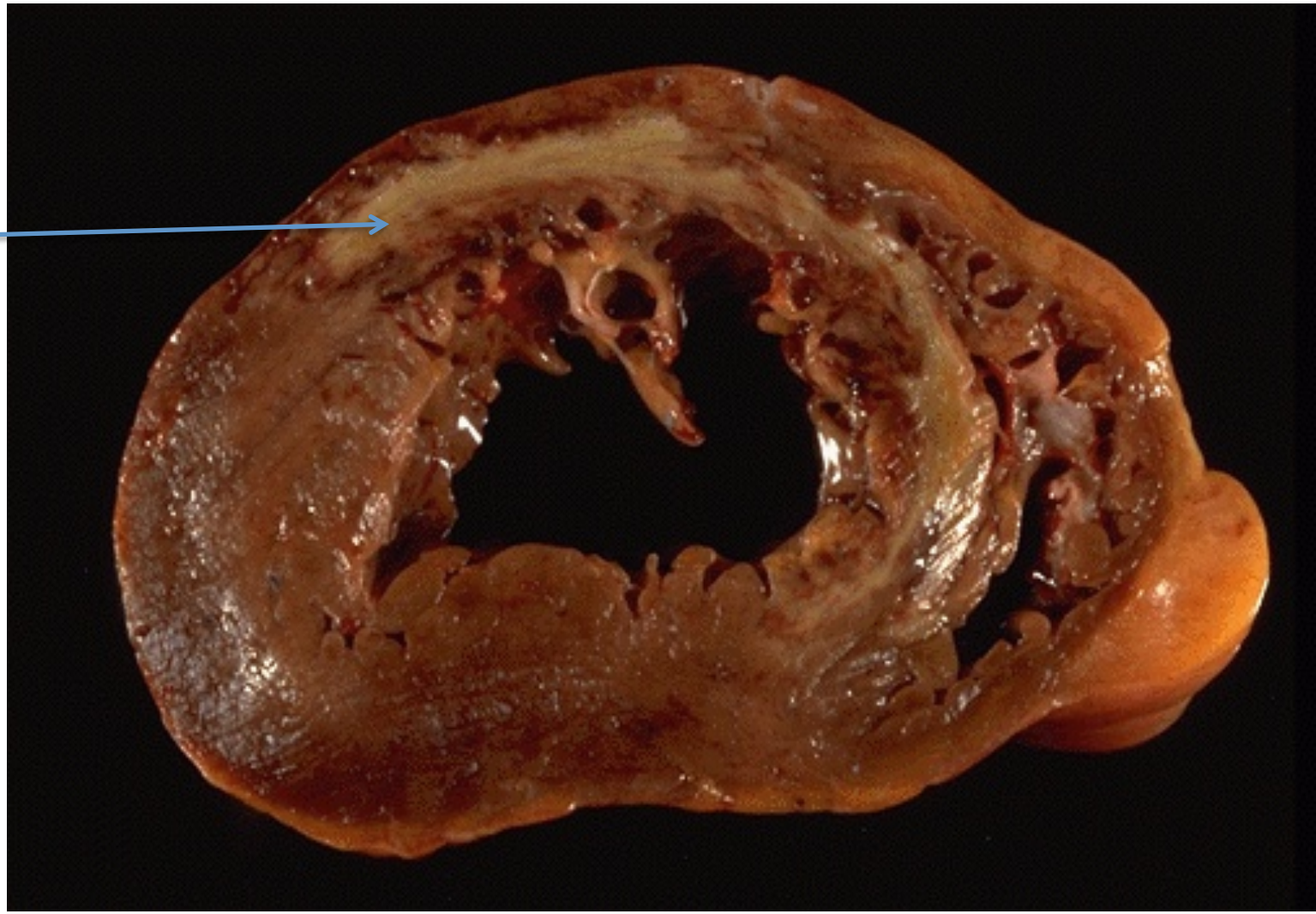
Causes:

1. Arterial occlusion due to **thrombotic or embolic** events (99%)
2. **Other arterial** causes:
   - Vasospasm
   - Swelling of atheroma
   - Extrinsic compression on vessel (by tumor …)
   - Twisting of vessels
   - Traumatic rupture
3. **Venous thrombosis**:

   in organs with single venous outflow e.g. testis, ovary

3- Myocardial infarction

Cross-section of the heart

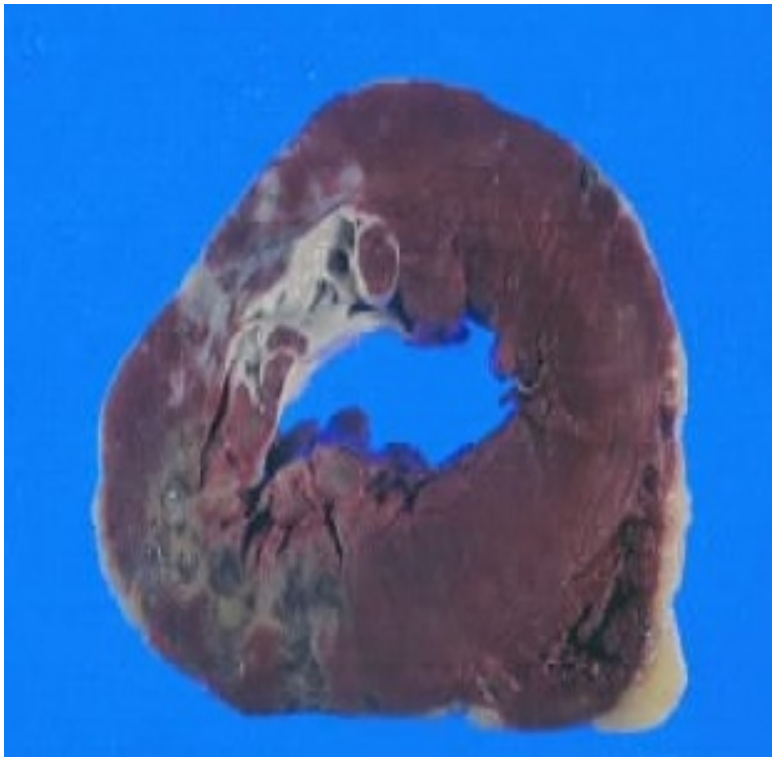


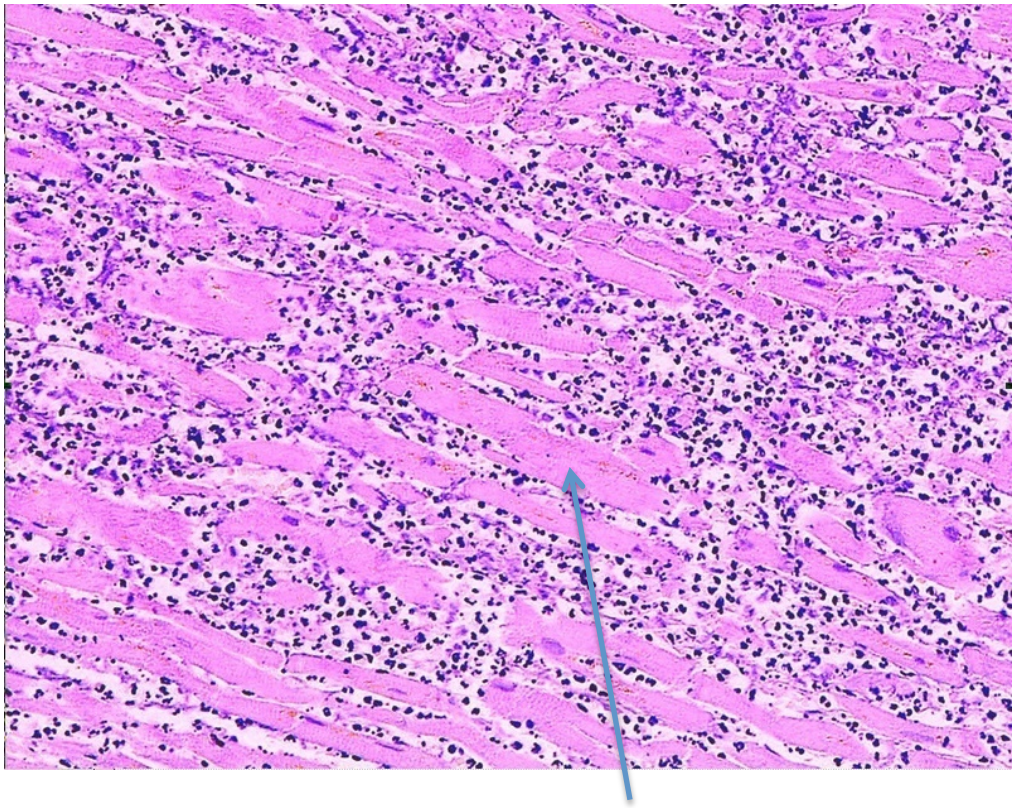
Whitish area is abnormal (dead cells)

But the dark region is normal

Infarction of many internal organs leads to a "pale" infarct from loss of the blood supply, resulting in coagulative necrosis. Shown here is a myocardial infarction from occlusion of a major coronary artery, here the left anterior descending artery.

Myocardial infarction (recent stage)

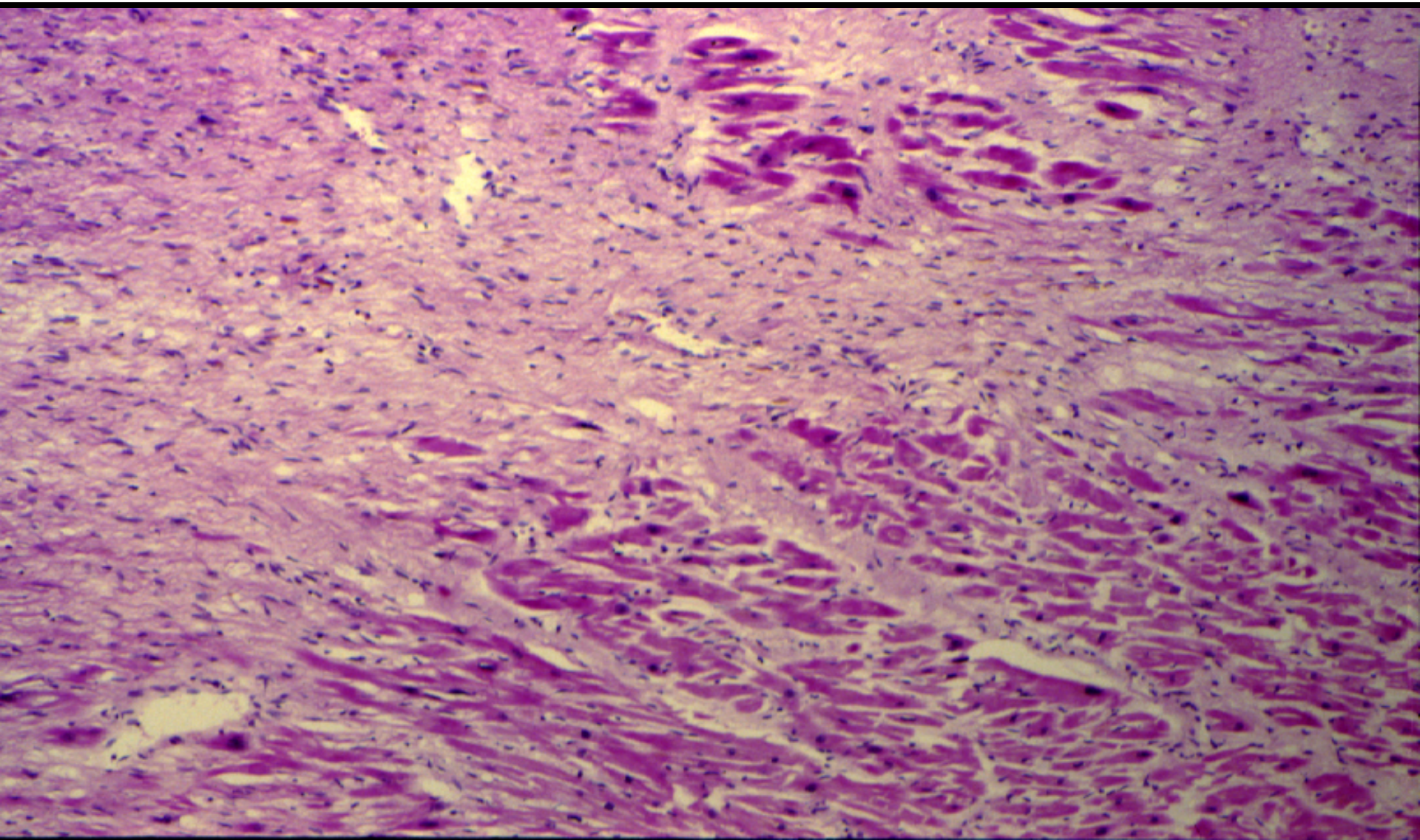




Coagulative necrosis

Few inflammatory cells mean there's acute myocardial infarction

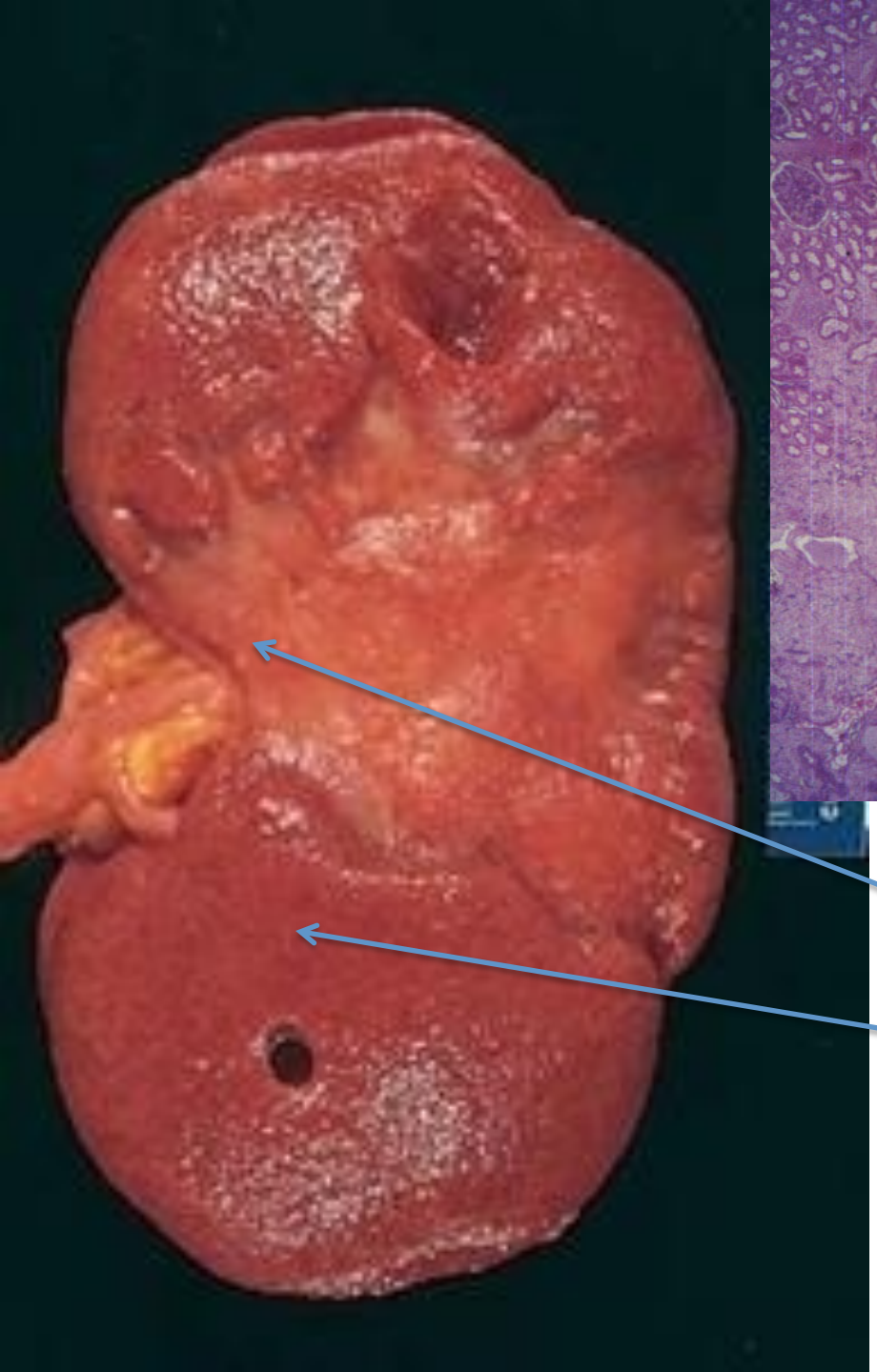
MYOCARDIAL INFARCTION (LATE STAGE)

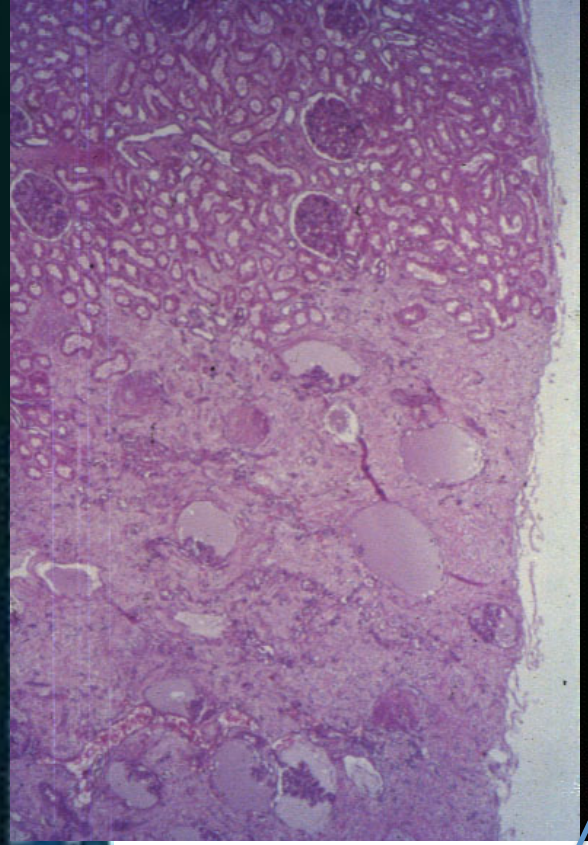


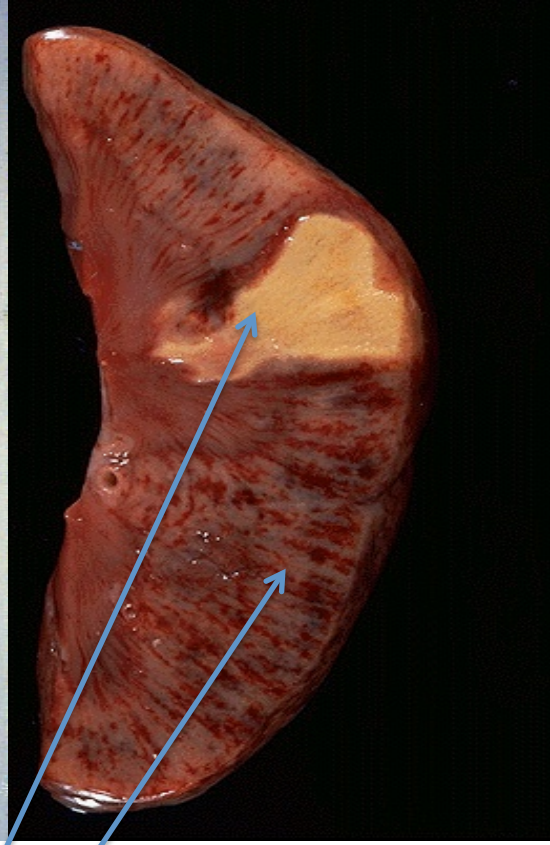
Myocardial infarction:
Section of myocardial shows:

- Patchy coagulative necrosis of myocardial fibres. The dead muscle fibres are structure less and hyaline.
- The necrotic muscle fibres are pale with loss of nuclei and striations. Infiltration of neutrophils in recent stage is seen .
- Later granulation tissue formation and fibrosis.

4- Infarcted kidney



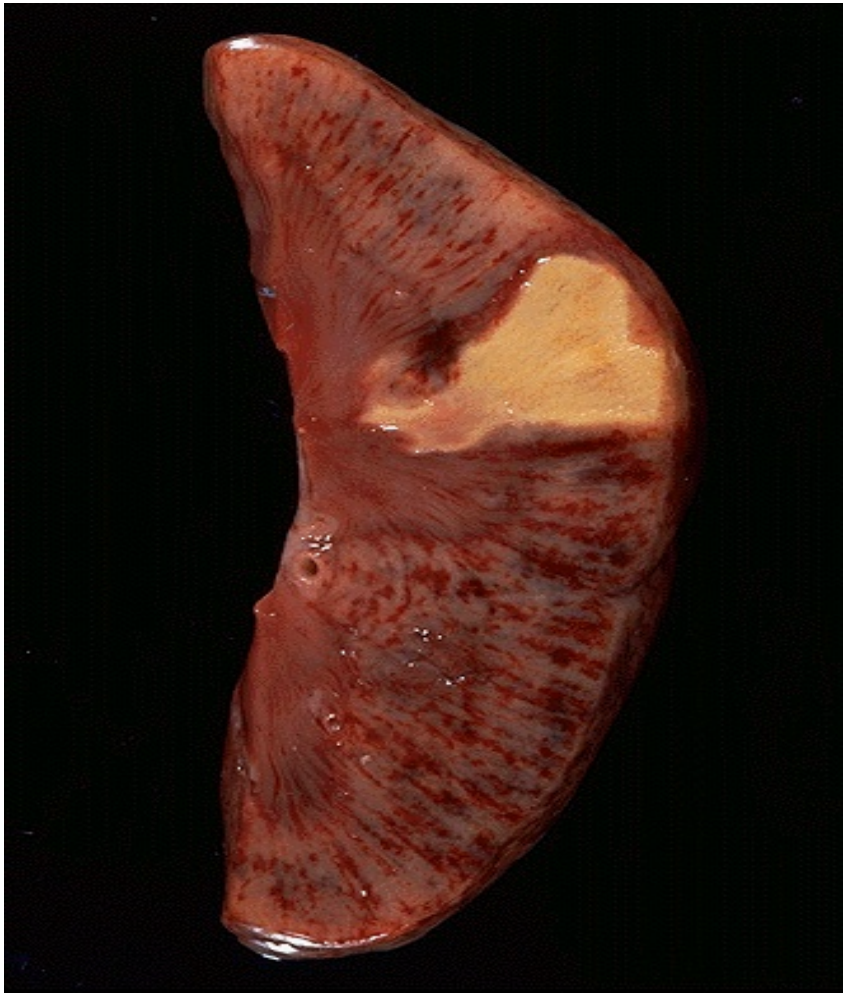




Whitish is abnormal

Brownish is normal

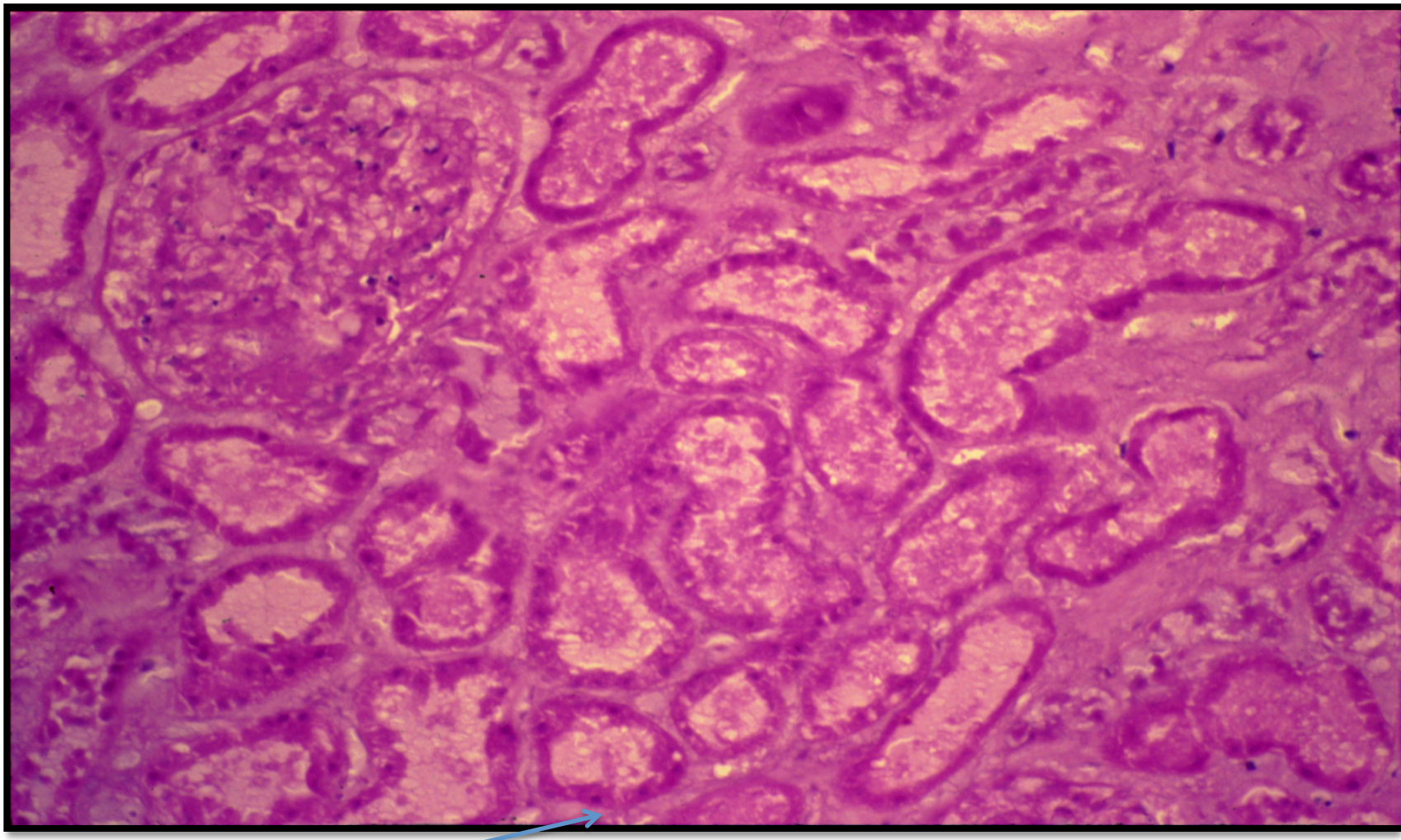
WEDGE SHAPED SCARRED INFARCT following the distribution of a end artery branch of the renal artery. FIBROSIS implies that it is old (months to years)



Organ: Kidney Dx:Coagulative necrosis

the kidney shows a pale triangular cortical infarct .

INFARCTED KIDNEY



There is no neucleus

If we can’t see nucleus that’s mean it’s normal

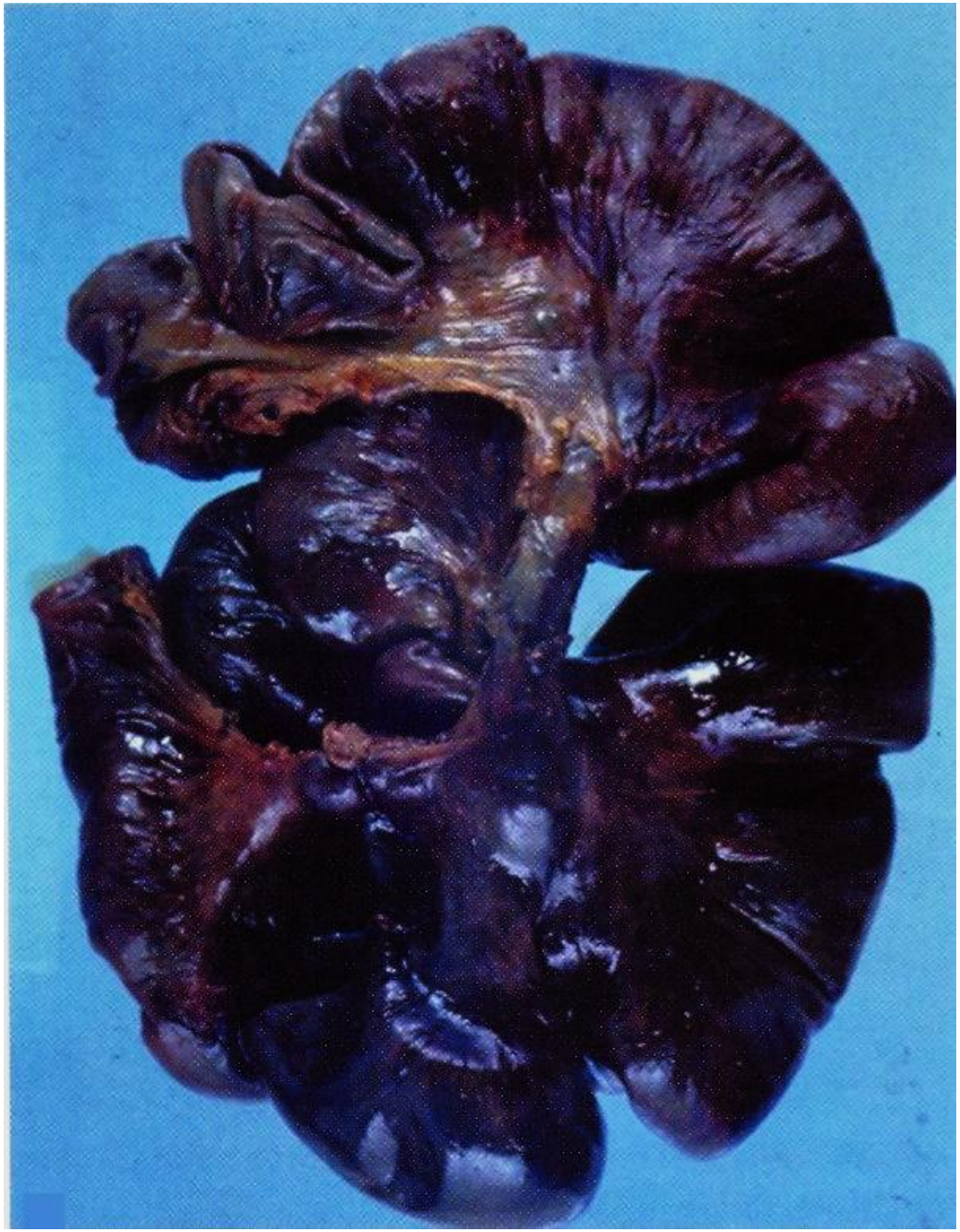
Infarcted kidney:

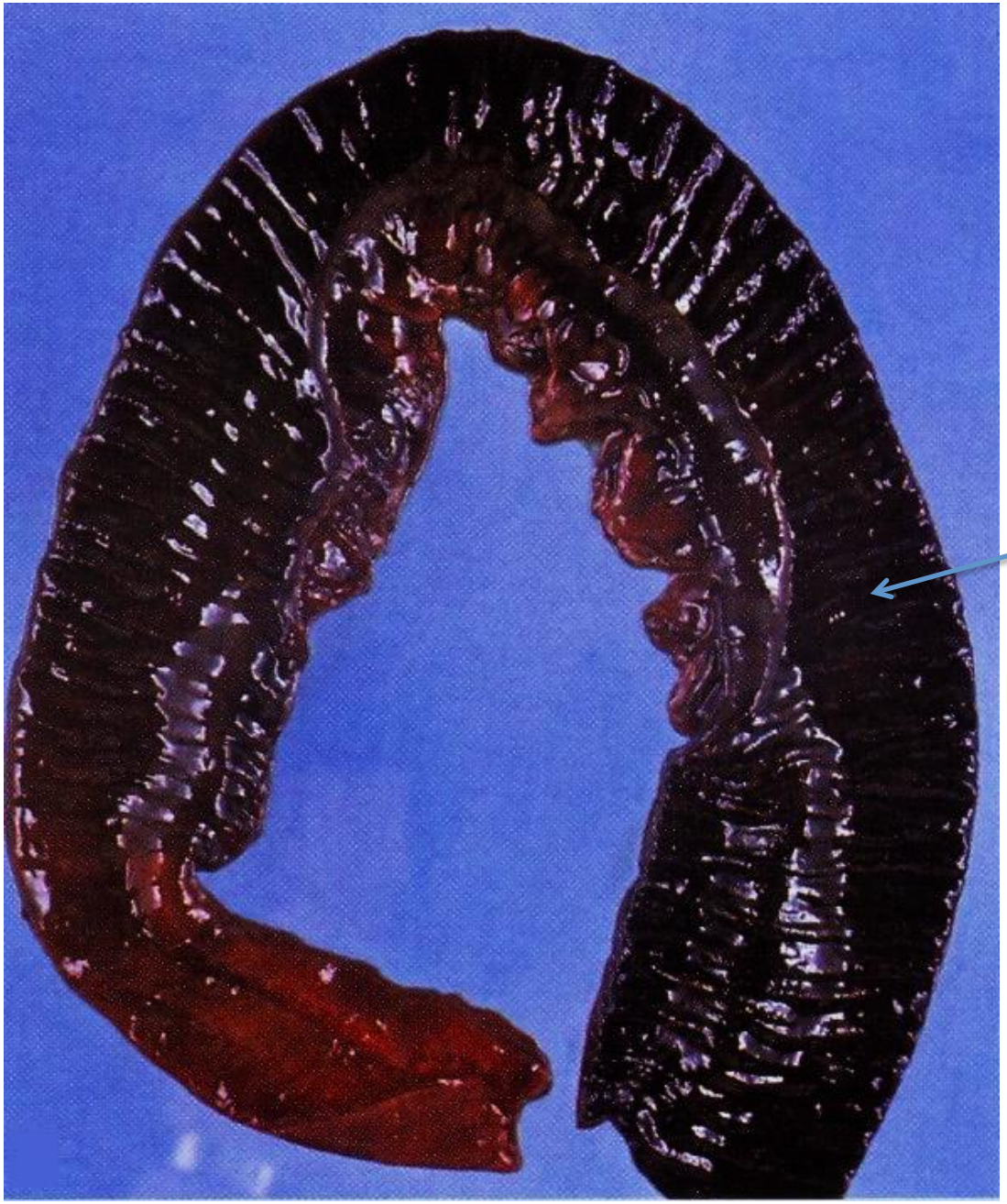
Section of kidney shows:

- A cortical infarct showing coagulative necrosis of glomeruli, tubules and interstitial tissue with loss of cell nuclei.

- The haemorrhagic zone at the periphery of the infarct shows dilated and congested blood vessels and cellular infiltrate by neutrophils, red blood cells and lymphocytes.

5- Infarction of the small intestine





Dead intestine

Hyperemia* & Congestion**

- **Increased blood in an area (inside blood vessels)**

compared to normal.

** Increasing in the size of the vessel leads to increasing in the blood flow. (Need energy inflow)*

*** A blocked vein of an organ leads to increasing in the blood flow.*
(Doesn't need energy)

Compare between:

"Hyperemia & Congestion"

Hyperemia:

- is an "active process"
- resulting from augmented tissue inflow
- due to arteriolar dilation
- (e.g. acute inflammation)

Congestion

- is a "passive process"
- resulting from impaired outflows from a tissue
- Impaired venous return
- (e.g. cardiac failure or venous obstruction)

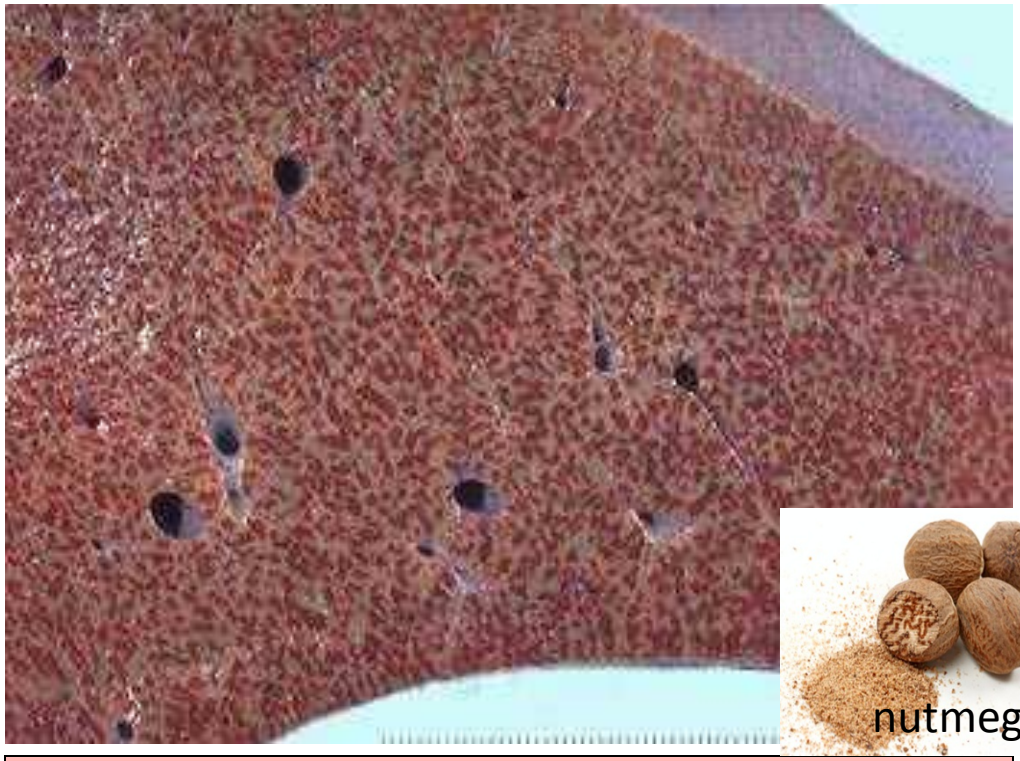
6- Chronic venous congestion of the liver

Happens because of heart failure (left side of heart leads to the right side failure leads to kidney).

Pathological Features of Chronic Venous Congestion in Liver

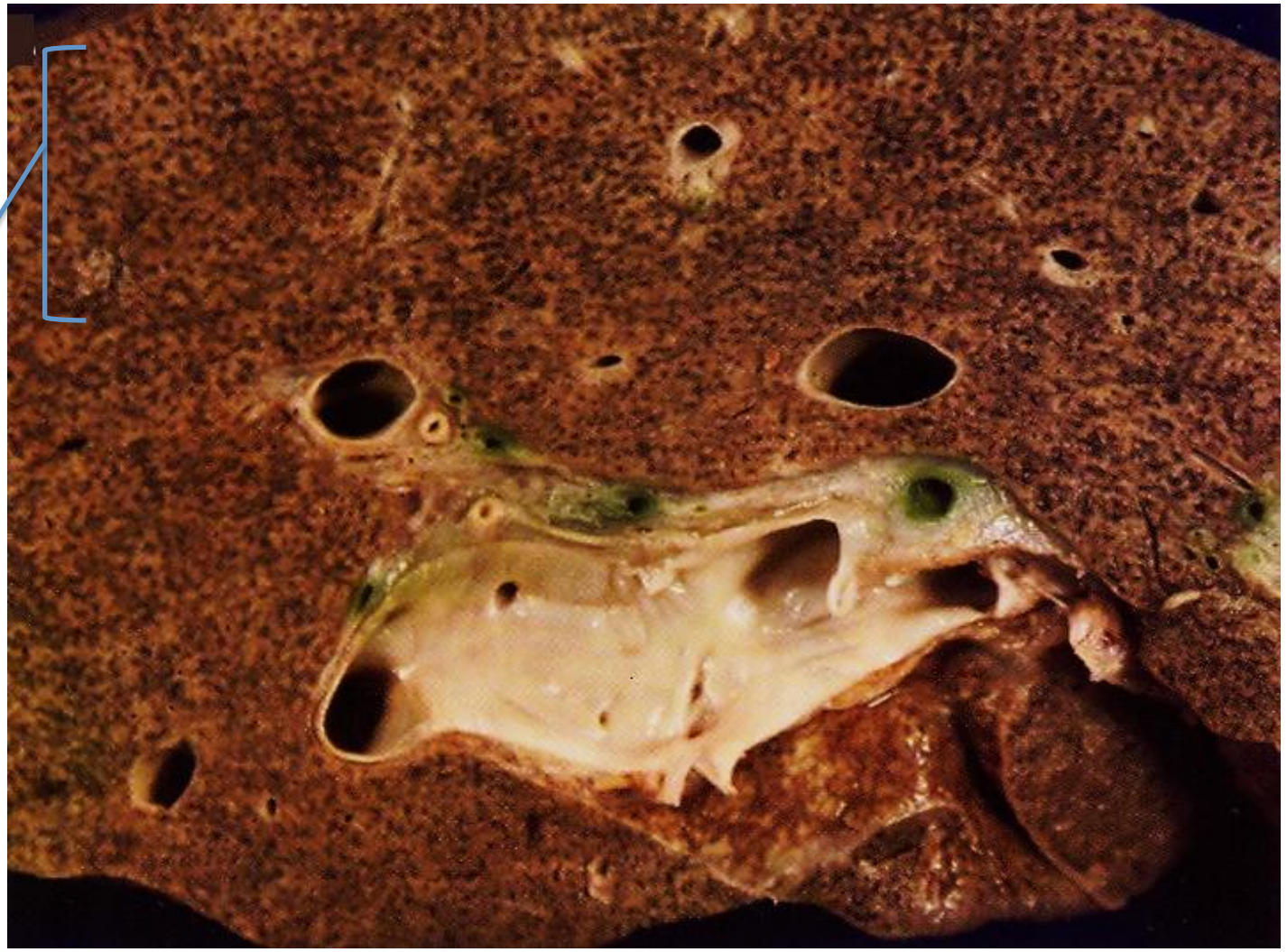
1.Liver: It is mainly due to <u>right sided heart failure.</u>

Gross Picture: The liver is **enlarged** , **firm** and the cut surface shows alternating **dark** areas of **congestion** with **pale** areas of **fatty change**, giving the liver the **nutmeg** appearance.

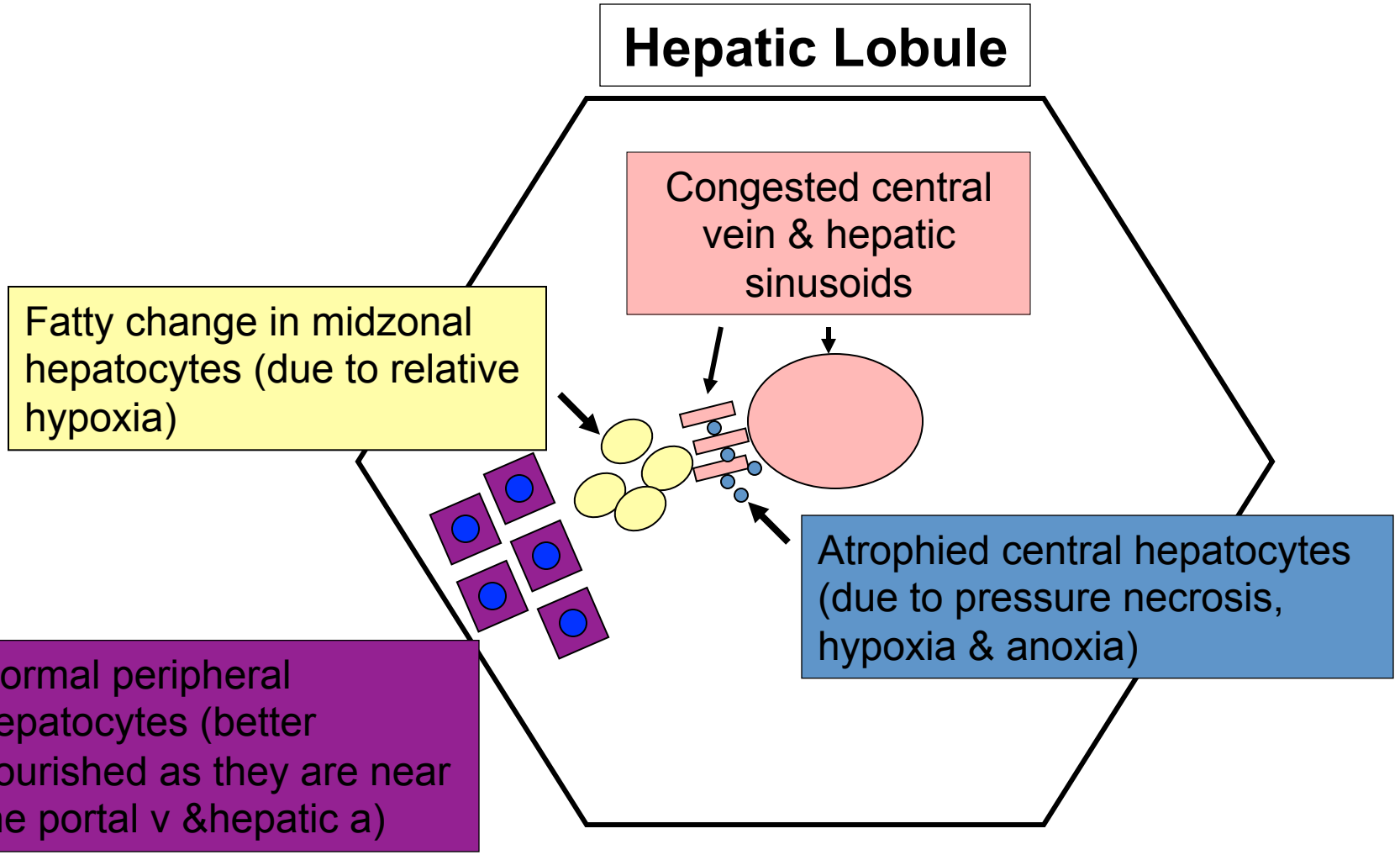


Nutmeg cut surface of the liver (dark areas of congestion alternating with pale areas of fatty change)

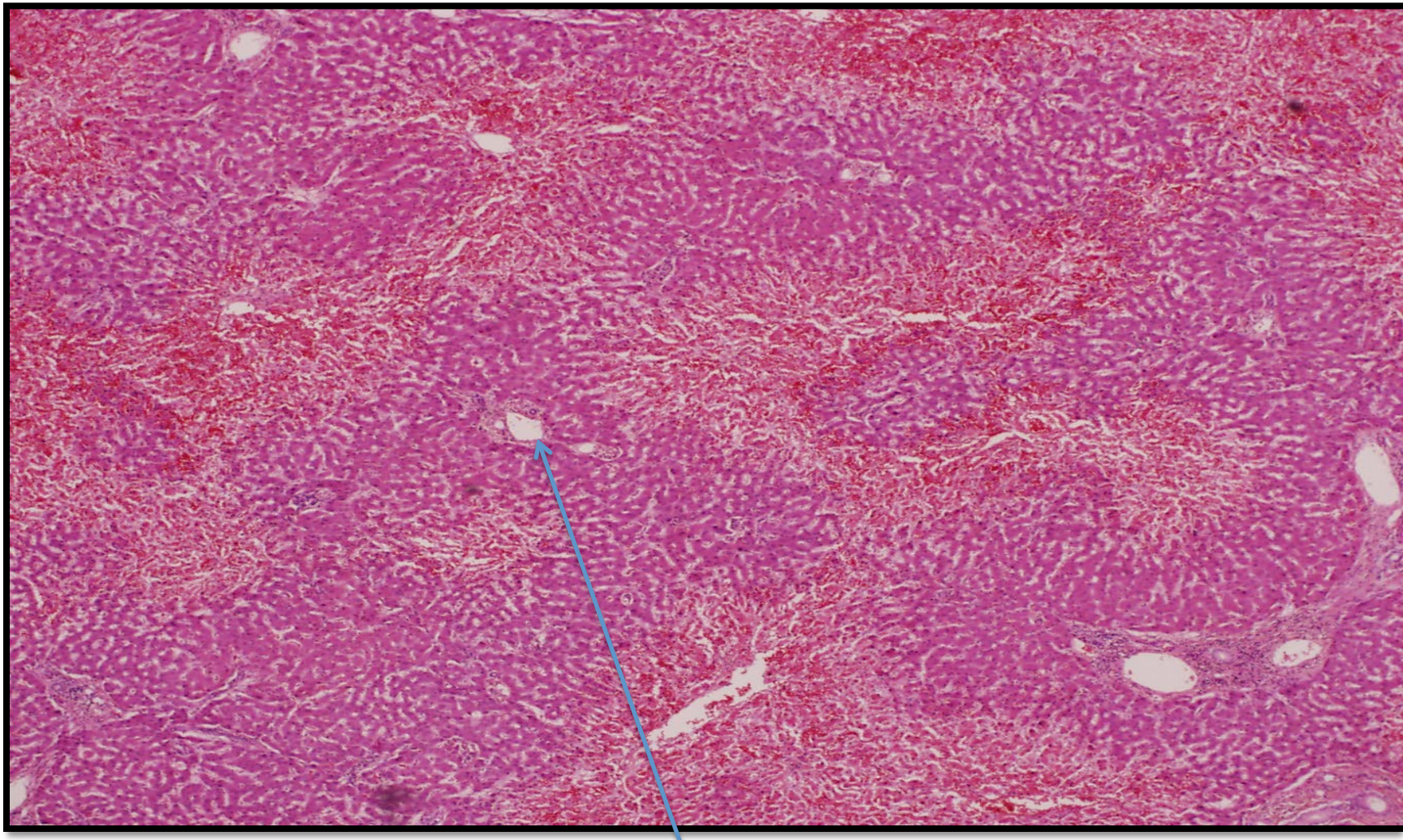
Light and dark area



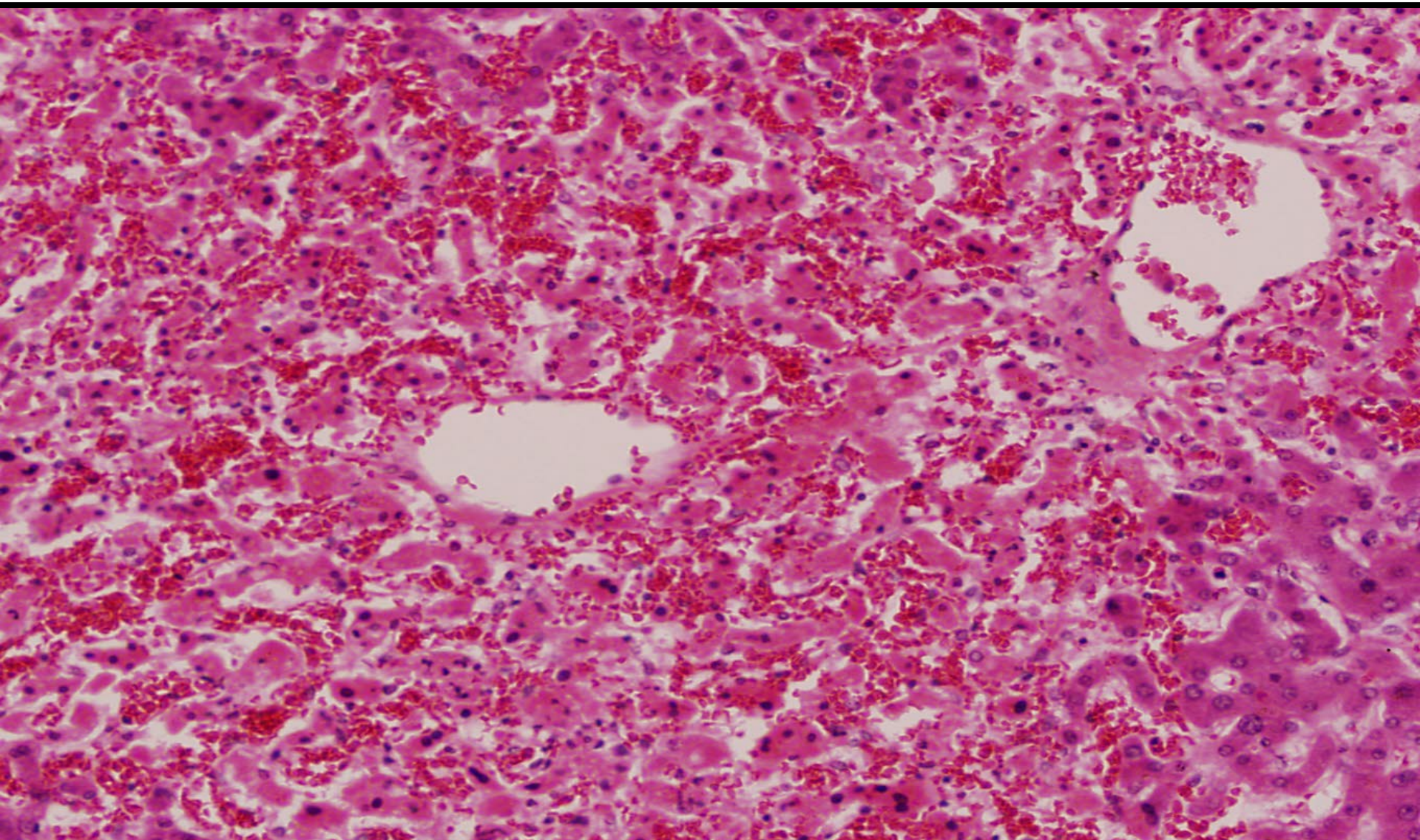
Pathological Features of Chronic Venous Congestion in Liver



PASSIVE CONGESTION OF THE LIVER (NUTMEG LIVER)



PASSIVE CONGESTION OF THE LIVER (NUTMEG LIVER)



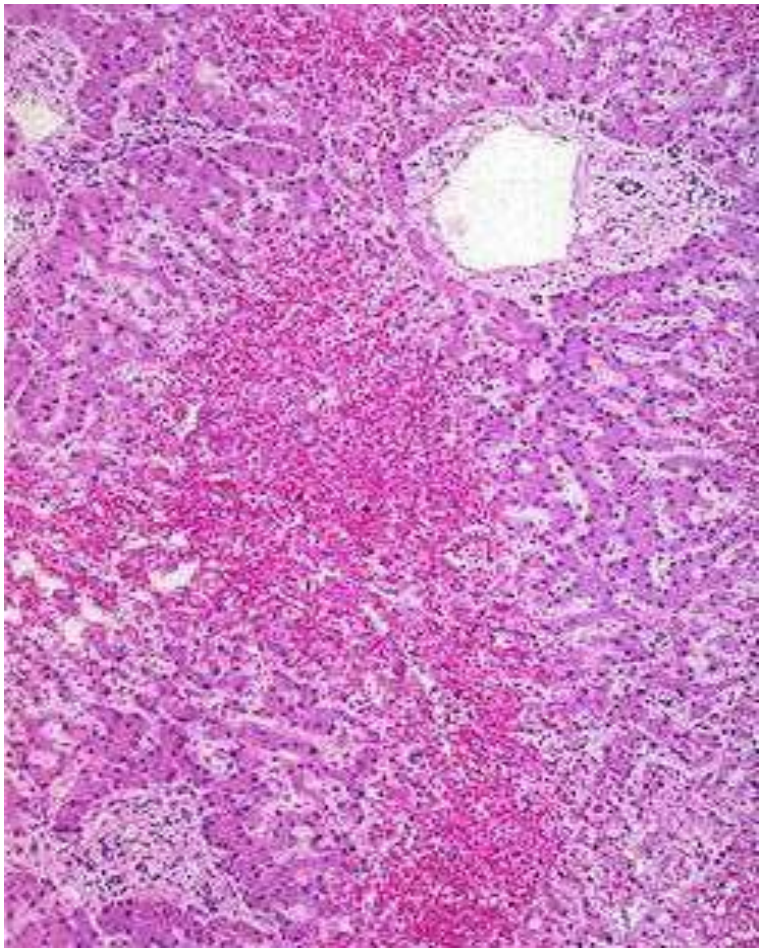
Pathological Features of Chronic Venous Congestion in Various Organs (Liver)

Microscopic Picture of Liver in Chronic Venous Congestion

1. The **central vein** in hepatic lobule is **congested** as well as the **hepatic sinusoids** in the central area.
2. The **central hepatocytes** will show **atrophy** and **necrosis**.
3. The **mid zonal hepatocytes** may show **fatty change** due to relative hypoxia.
4. The **peripheral hepatocytes** are **normal.**

Macrophages in the liver

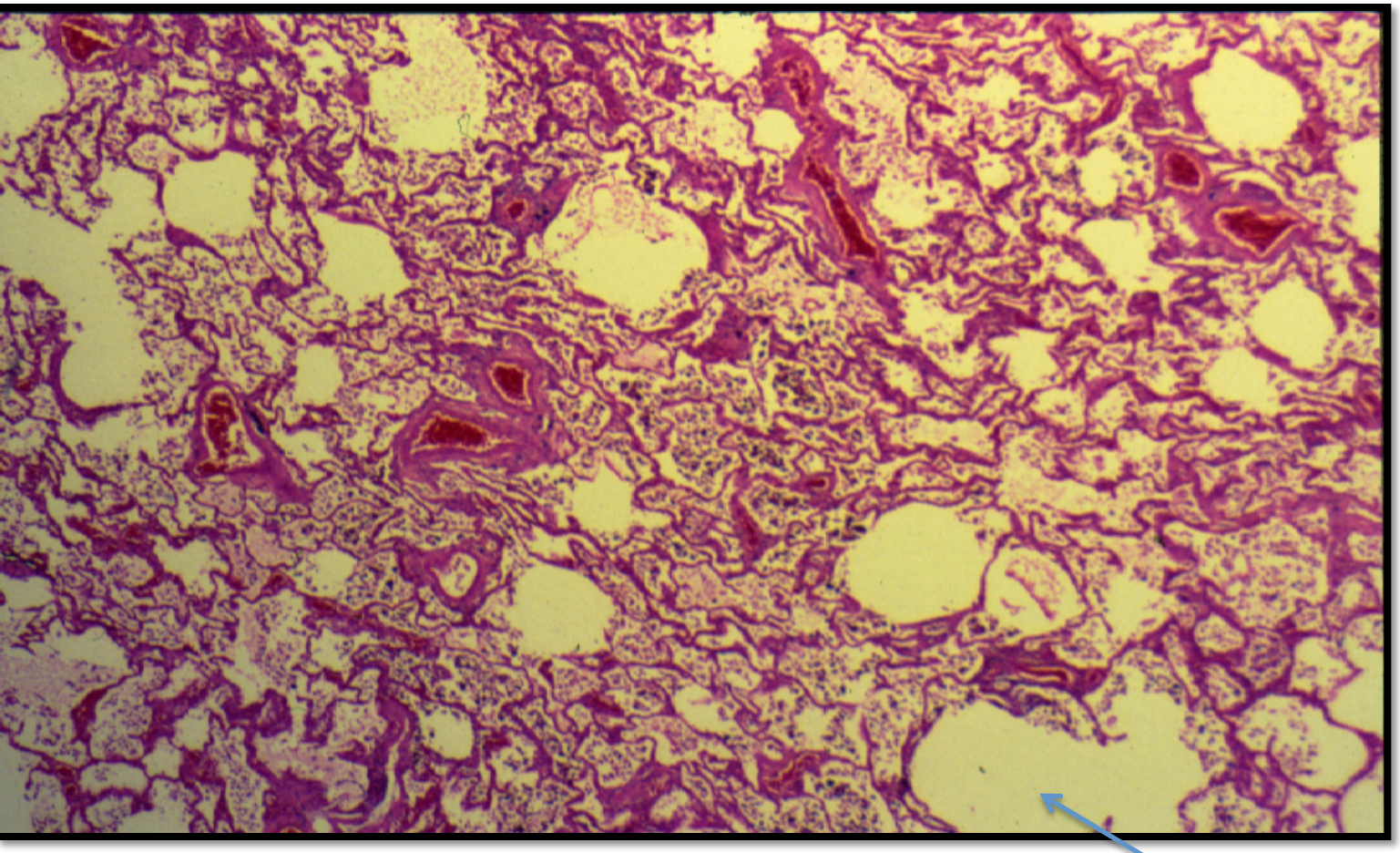
5. Kupffer cells contain few brown haemosiderin pigment granules



Early CVC of liver (congestion of central vein &central hepatic sinusoids)

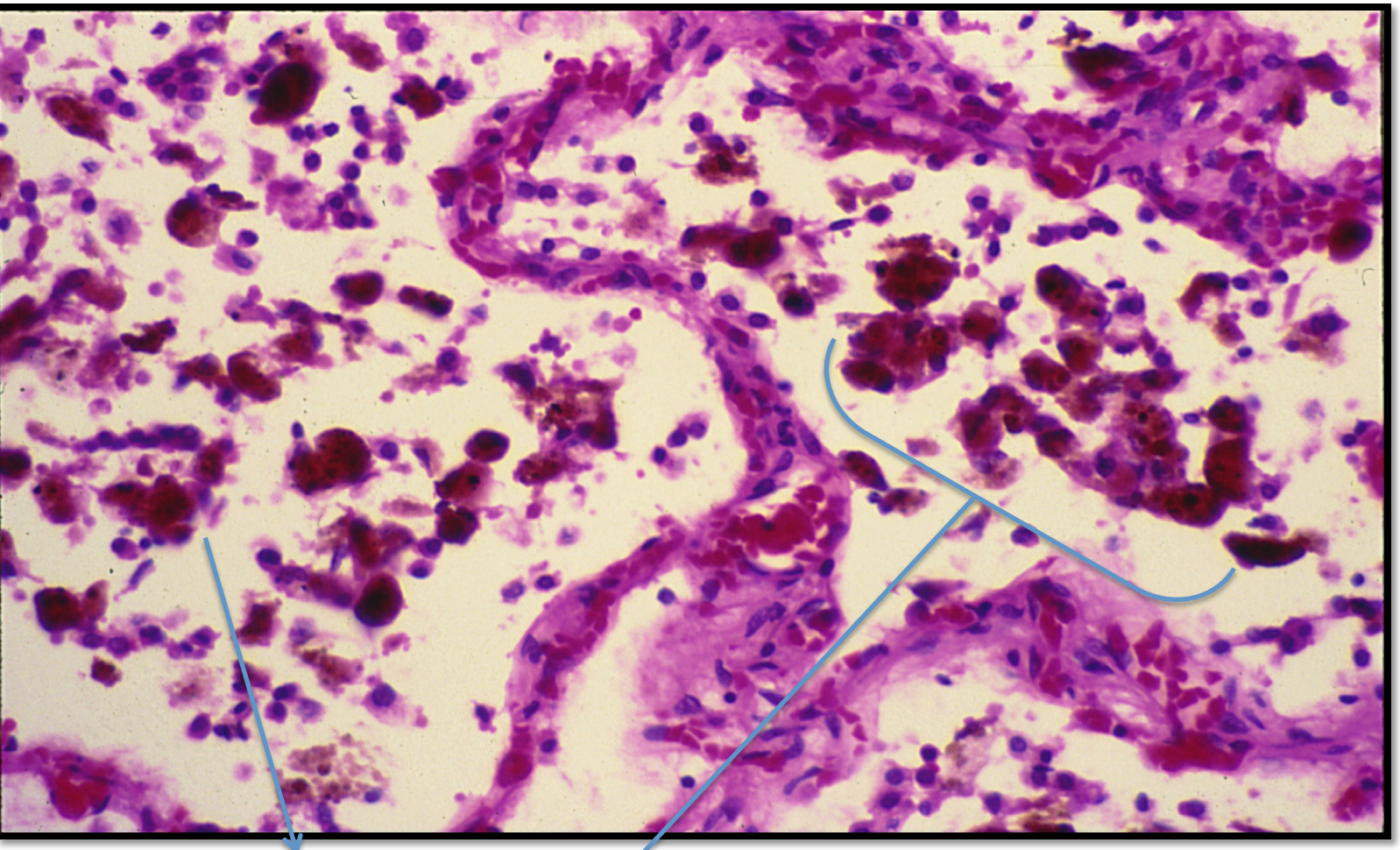
7- Chronic venous congestion of the lung

CHRONIC VENOUS CONGESTION (LUNG)



Lung air space

CHRONIC VENOUS CONGESTION (LUNG)



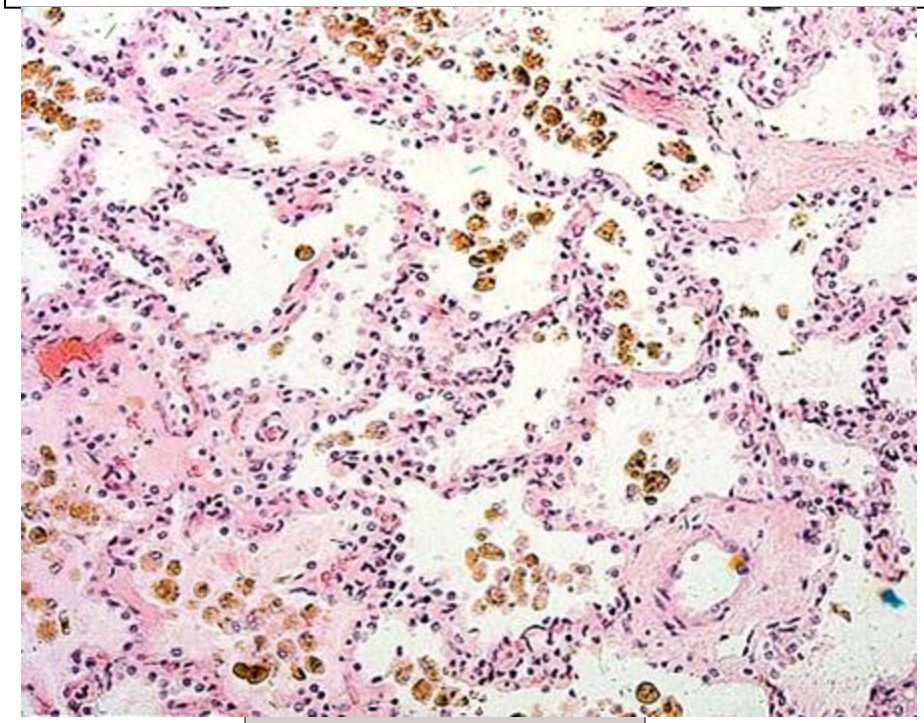
Macrophages known as “heart failure cells”

Pathological Features of Chronic Venous Congestion in Various Organs (Lung)

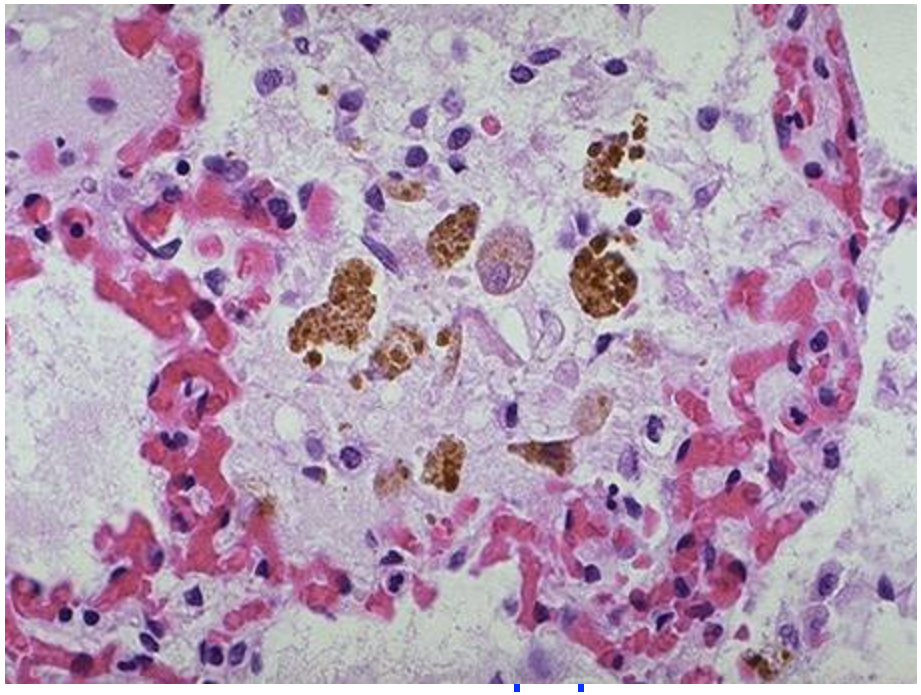
Gross Picture: The lungs are **enlarged, heavy, firm** and **deep red** in colour **(brown induration).** Frothy blood oozes from the cut surface on squeezing.

Microscopic Exam.: The **alveolar septa** are thickened by congested dilated capillaries and oedema fluid followed **later** by fibrosis.

- As a result of microhaemorrhages, the **alveoli** contain oedema fluid, RBCs either intact or haemolysed and haemosiderin laden macrophage **(heart failure cells).**



Thickened alveolar septa (fibrosis)



heart failure cells