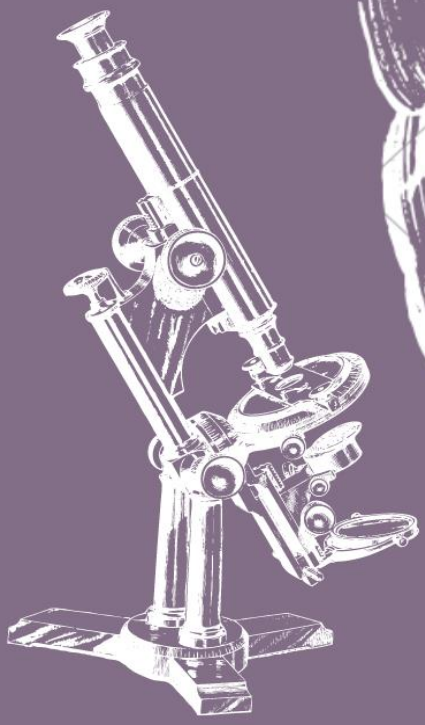
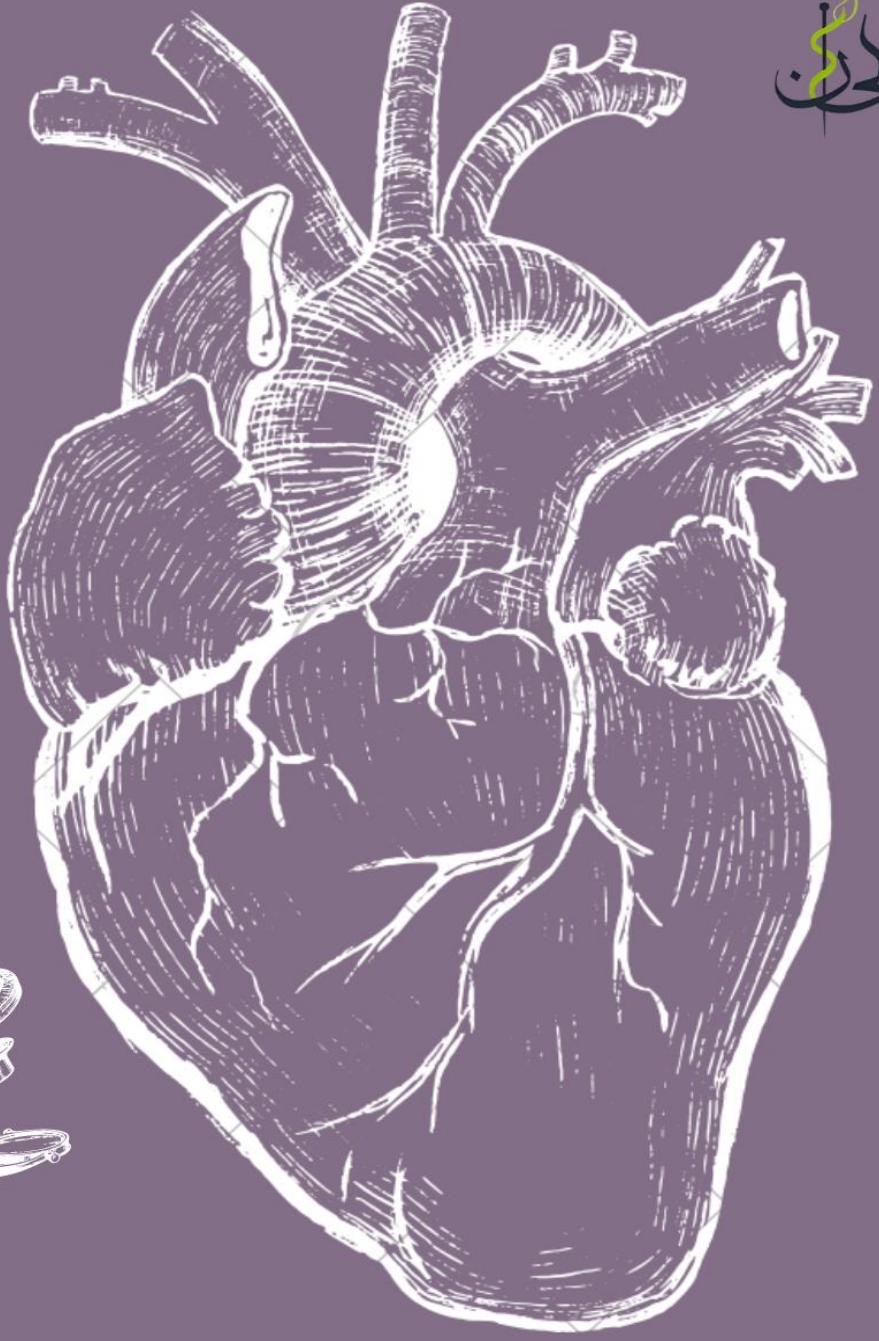
PATHOLOGY





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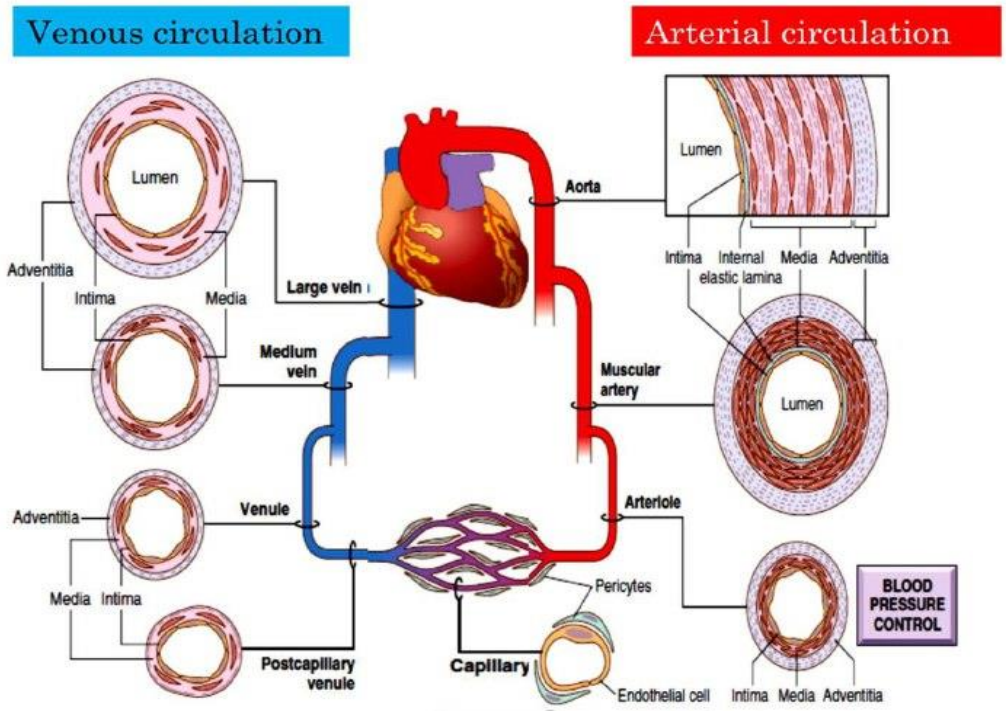
Sheet no. : 1

بسم الله الذي لا يضر مع اسمه شيء في الارض ولا في السماء وهو السميع العليم

Now take a deep breath and let the game begin

Thrombosis

Topic 1: Cardiovascular system



According to differencies in the function of the blood vessles we devide them into venous circulation and arterial circulation

Venous circulation begins inside the tissues goinig straight to the heart (specificly the right side of the heart) with 2 major veins which are SVC and IVC,then goes to the lung for gas exchanging ending up with oxygenated blood (carrying oxygen) making its way to the left side(left atrium then ventricle),finaly it will be pumped out(through the aorta) to each part of the body.

We end up by reaching the tissues with small artrerioles and venules making a network called capillary.

Venous circulation	Arterial circulation
From the tissues	To the tissues
Deoxygenated	Oxygenated
Into the RT side of the heart	into the LF side of the heart
Venuoles in capillary	arterioles in capillary

Understand the histology & you will understand the pathology!

The blood vessles generally speaking have 3 components(inner to outer):

1-tunica intima

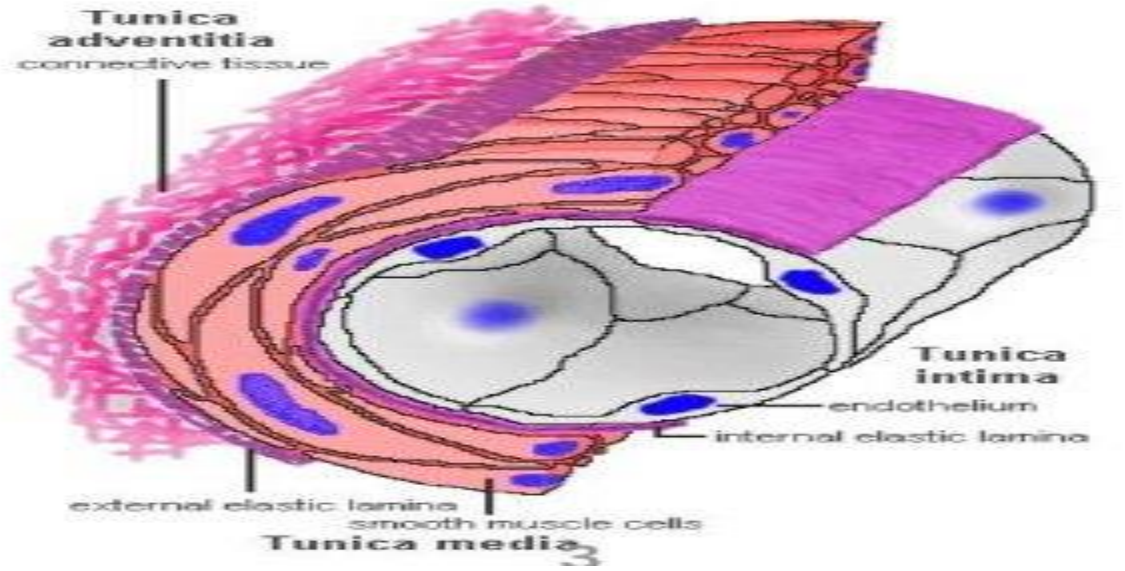
Inner most layer,composed of endothelial cells

2-tunica media

Basically composed of smooth muscle cells,ECM protiens mainly elastin

3-tunica adventitia

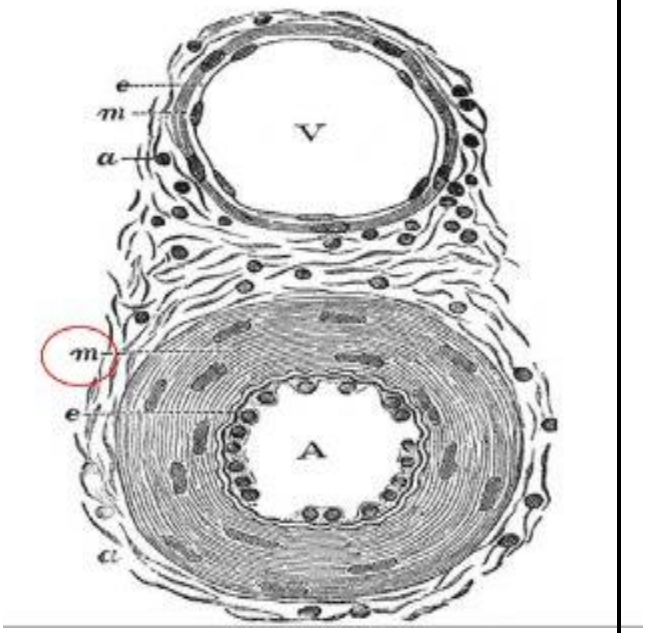
Connective tissue giving support to the wall,and it gives the BV nerve and blood supply so it contains vasa vasorum (the nerve supply)



Topic 2: cross-sectional for the BV

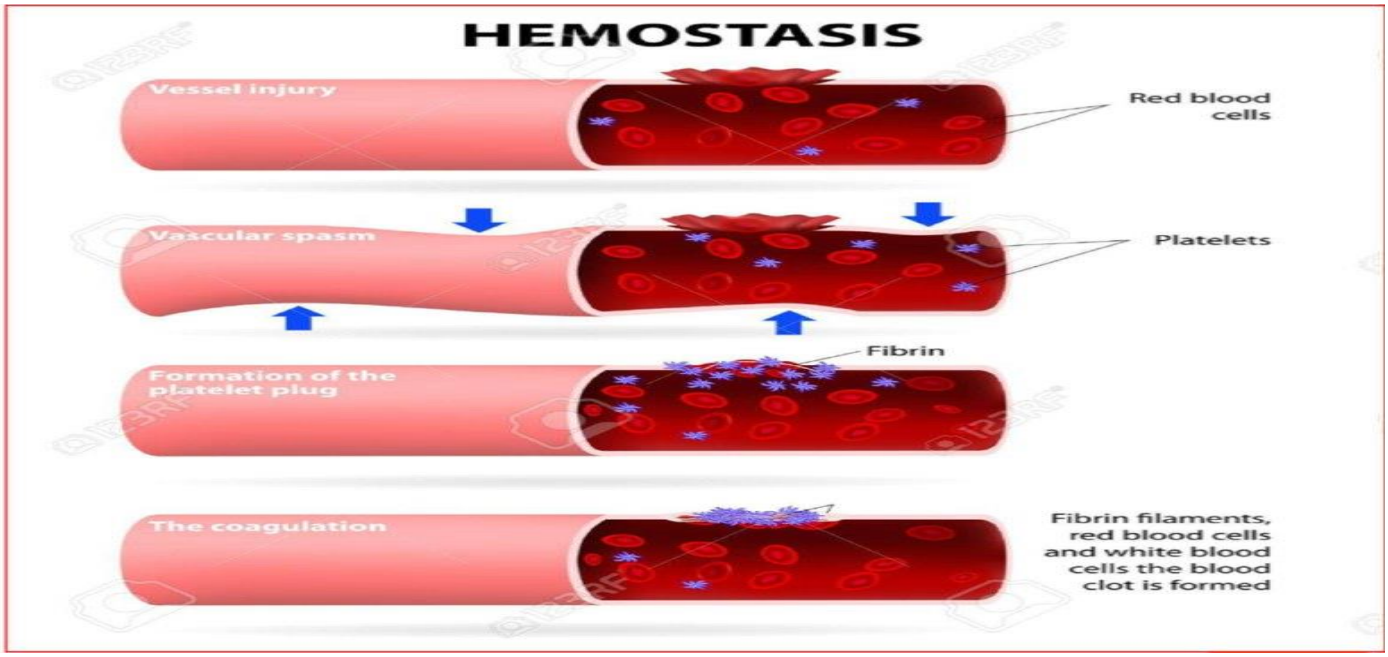
Main difference is in the media layer which is thicker in the artery

Purpose?it's for the higher capacity for contraction ,take the Aorta as an example ,while in the vein there's no need to , makes sense!



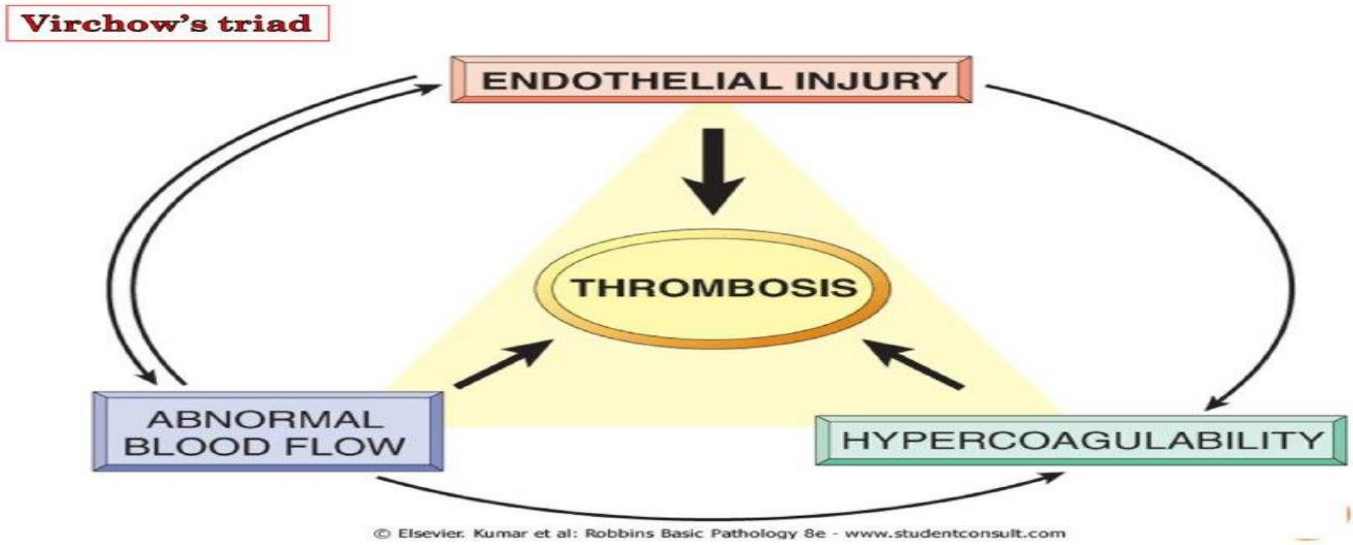
Topic 3:Thrombosis

Whenever we have an abnormal or unnecessary thrombosis we call it pathological thrombosis,but remeber that thrmbosis is a physiological process to maintain the hemostasis in case of blood loss and it starts with blood coagulation.



THROMBOSIS- PATHOLOGICAL ASPECTS

- Blood coagulation is an important physiological event to protect our hemostasis, and life
- However, at certain points, this process can be pathological that may endorse injury and cause harm to our body
- This happens whenever unnecessary blood clotting is activated
- The “pathological” thrombosis is caused by the presence of at least one of 3 factors (together called Virchow’s triad)



Pathogenesis (called Virchow's triad):

1. Endothelial* Injury (Heart, Arteries)

2. Stasis (abnormal blood flow)

3. Blood Hypercoagulability

* Endothelial cells are a special type of cells that cover the inside surface of blood vessels and heart.

-*One factor = pathological thrombois*

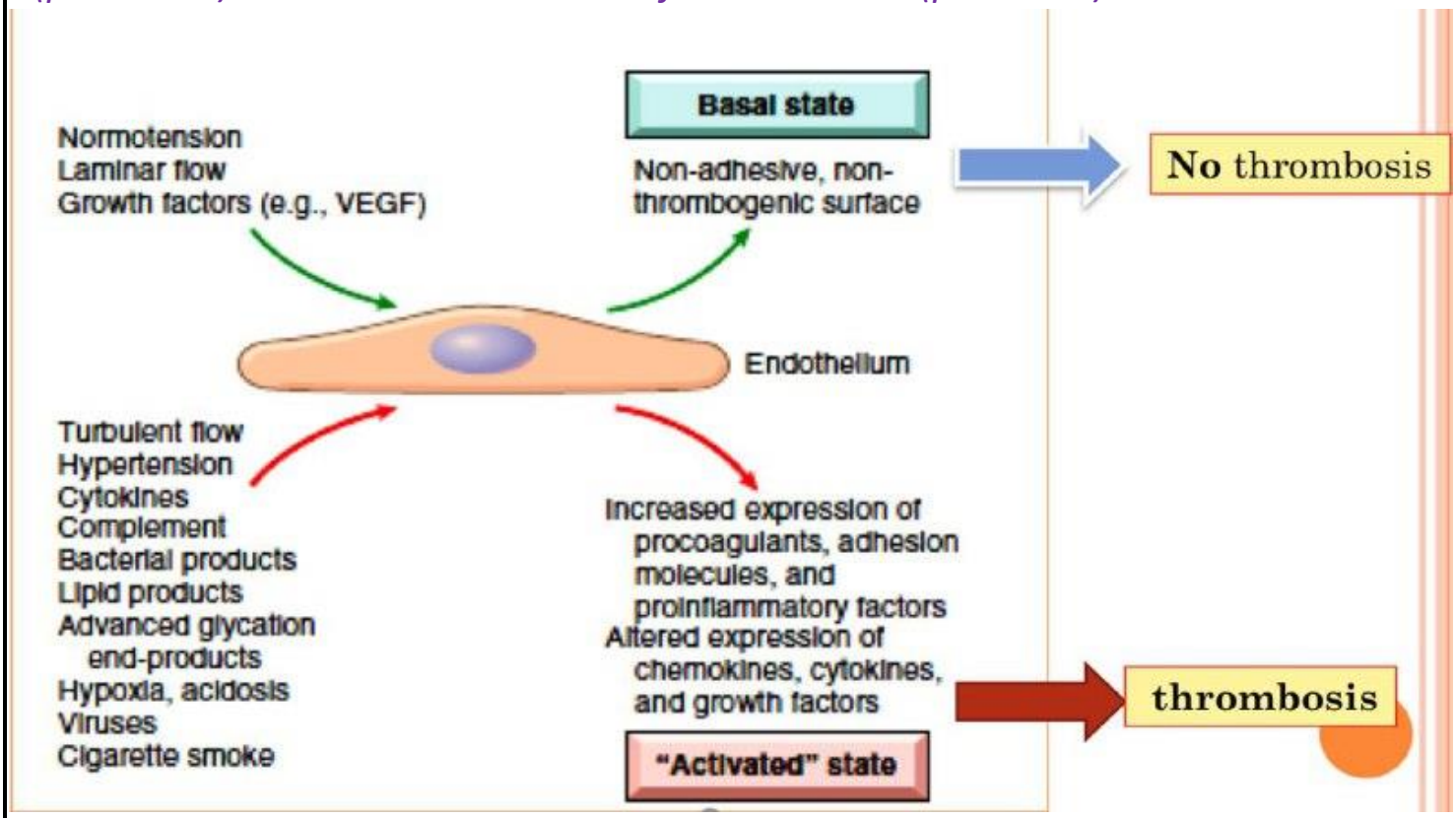
-More than one at the same site= pathological thrombois

-One leads to the other factor = pathological thrombois

Starting with endothelial cells ,they are a special type of cells which line the inside of the blood vessels &the heart

Main state of the endothelial cells is called basal state or resting ,during this state the cells have non-adhesive /non-thrombogenic surface which is the normal environment for the endothelial cells ,<u>But any variation could lead to pathological thrombosis</u>

In case of activation which is an endothelial cell injury,it will transform from (protective)cell inot an activated cell for thrombosis (promoter)



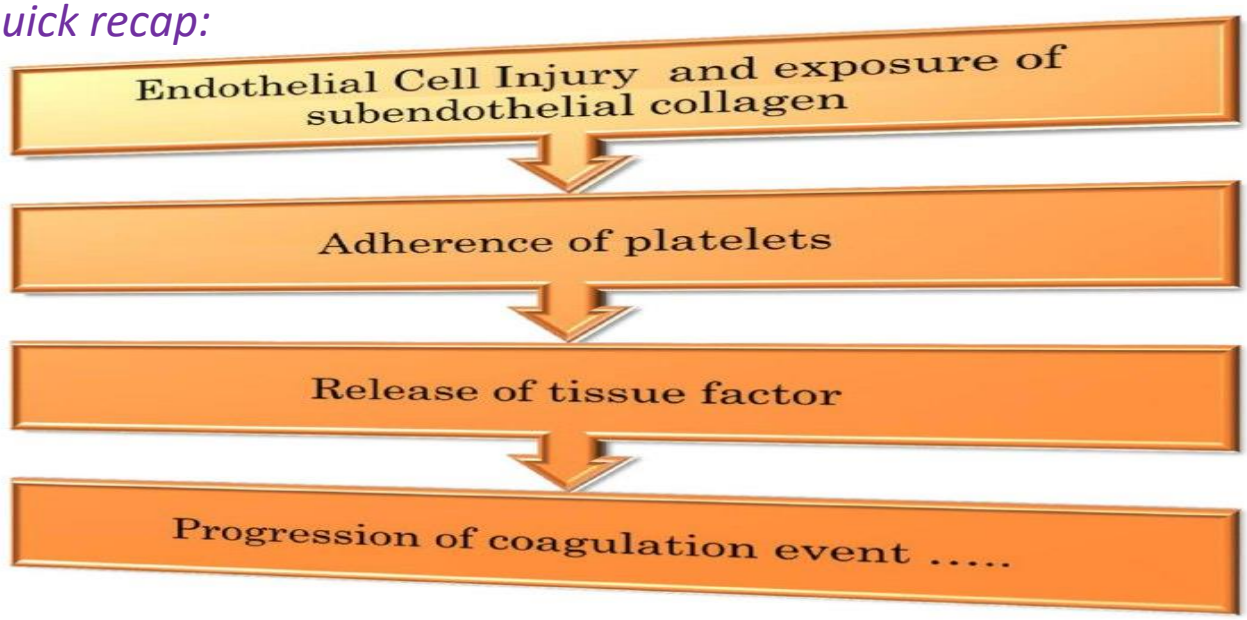
- Causes of Endothelial injury (not to memorize them!)

1. Valvulitis 2. MI 3. Atherosclerosis

4. Traumatic or inflammatory conditions 5. Hypertension 6. Endotoxin

7. Hypercholesterolemia 8. Radiation 9. Smoking 10. ……..etc.

as a quick recap:

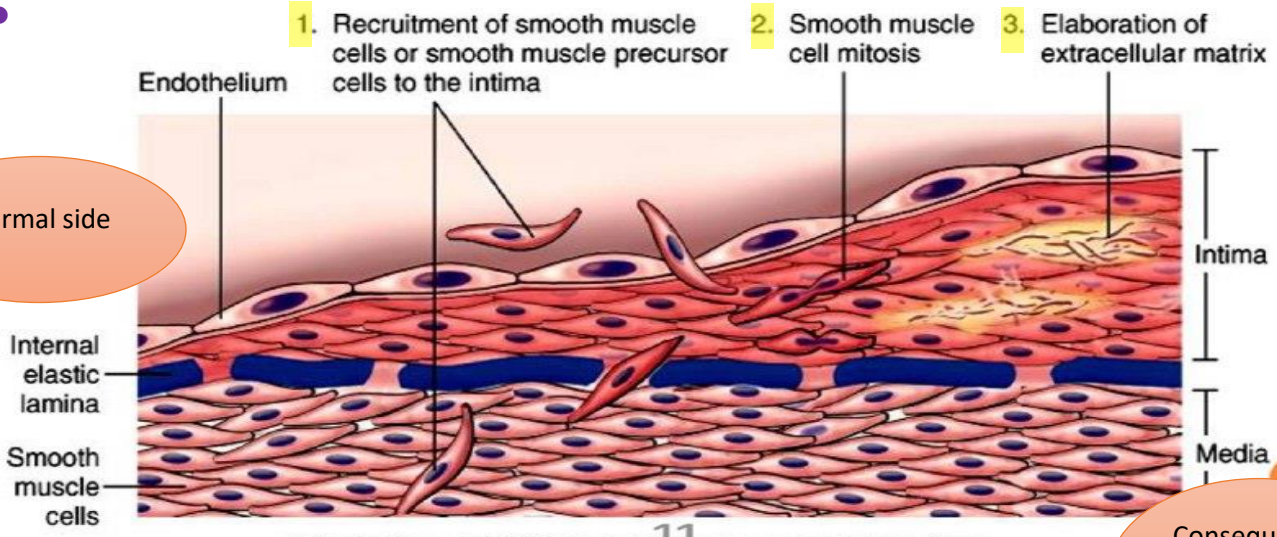


Topic 4: changes inside the layers of the vessel

- *Lumen: clot formation /thrombosis site*

The difference is in the thickness that would be on the expanse of the lumen(diameter).

- *Now read the steps in the figure they're important*
- *Regarding the third note possibly it will end up with blockage of the blood flow after narrowing the lumen which leads to ischemia to the downstream tissue(the tissues that are distal to a particular artery & taking blood supply from it).*
-



Normal side

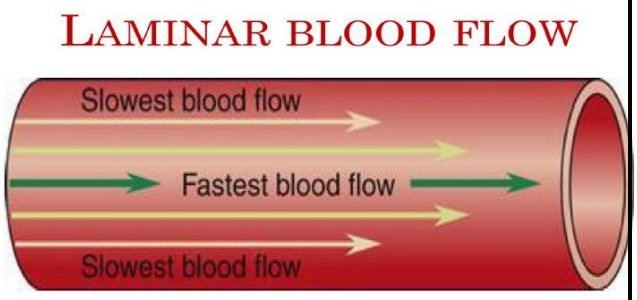
Consequences of the cell's injury

Never let monkeys eat bananas! I'm kidding

But please Never forget these processes (Cell injury-Inflammation-healing and repair).

Topic 5: second factor of the triad is Blood flow

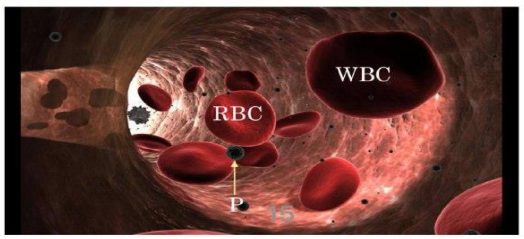
In order to recognize the abnormal flow you should first know the normal



it's called laminar which means layered

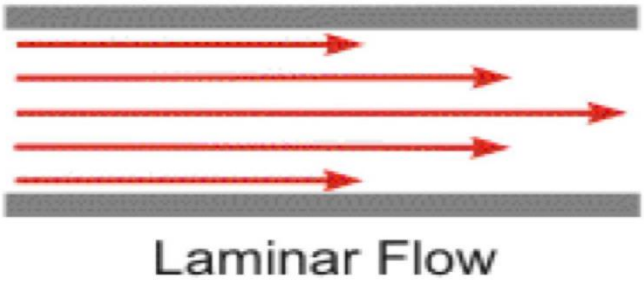
- *Fastest blood flow is almost at the center*
- *Going toward the wall its getting slower*

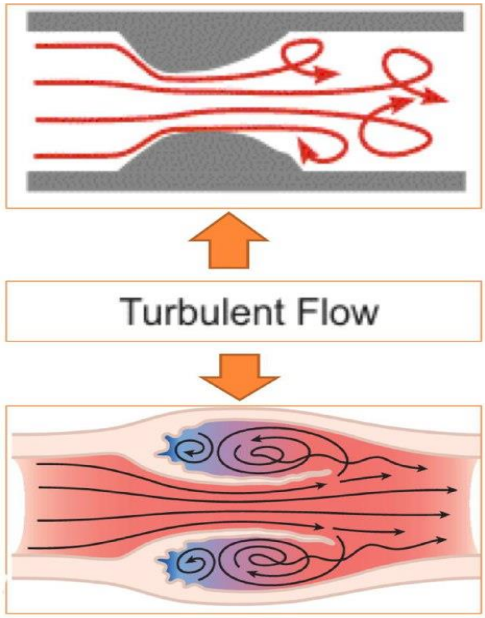
When normal blood flow is maintained you're Still on the safe side ,but once it gets closer to the walls ,the chance of forming a thrombus is high so ,logically the platelets should be at the center. Abnormal blood flow could be:



1. *Stenosis*
2. *Reduced blood flow*
3. *Abnormal dilation*
4. *And a lot more will be disused later inshallah*

So the direction of the blood would be changed Making turbulence blood flow



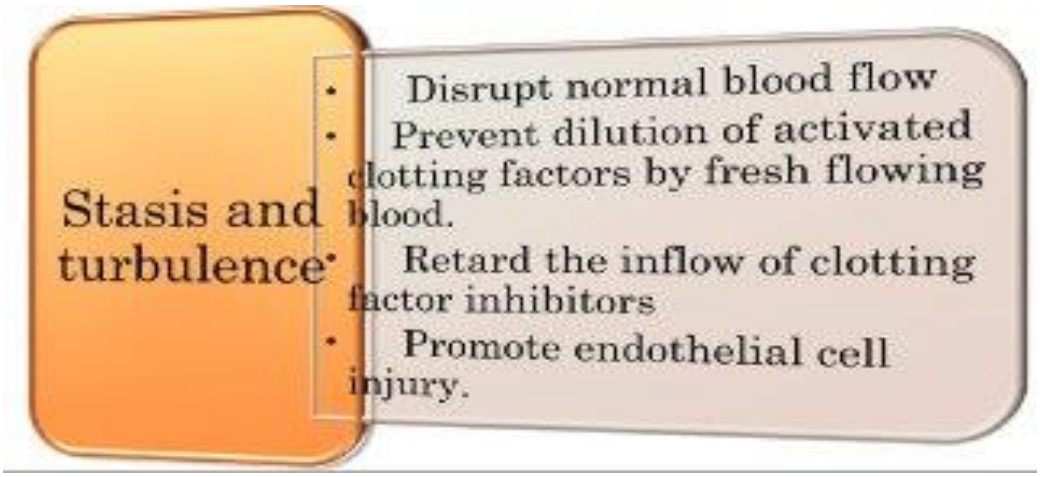


Topic 6: stasis

-Stasis is a major factor in venous thrombi

-Normal blood flow is laminar (platelets flow centrally in the vessel lumen, separated from the endothelium by a slower moving clear zone of plasma)

-Stasis and turbulence cause the following:



Causes of Stasis: (do not memorize)

1. Atherosclerosis
2. Aneurysms
3. Myocardial Infarction (Non-cotractile fibers)
4. Mitral valve stenosis (atrial dilation)
5. Hyper viscosity syndrome (PCV and Sickle Cell anemia)

Topic 7: third factor of the triad is Hypercoagulability

It means higher tendency to have clots formed

A. Genetic (primary):*less common*

- most common >> mutations in factor V gene and prothrombin gene

B. Acquired (secondary):*more common*

- multifactorial & more complicated

- causes include Immobilization, MI(*myocardial infarction*), AF(*atrial fibrillation*), surgery, fractures, burns, Cancer, Prosthetic cardiac valves(صمامات القلب الصناعيه)

MORPHOLOGY OF THROMBI :

Can develop anywhere in the CVS (e.g., in cardiac chambers, valves, arteries, veins, or capillaries).

- Arterial or cardiac thrombi: begin at sites of endothelial injury or turbulence; and are usually superimposed on an atherosclerotic plaque.
- Venous thrombi : occur at sites of stasis. Most commonly the veins of the lower extremities (90%) .
- Thrombi are focally attached to the underlying vascular surface.
- The propagating portion of a thrombus (free)is poorly attached :fragmentation and embolus formation

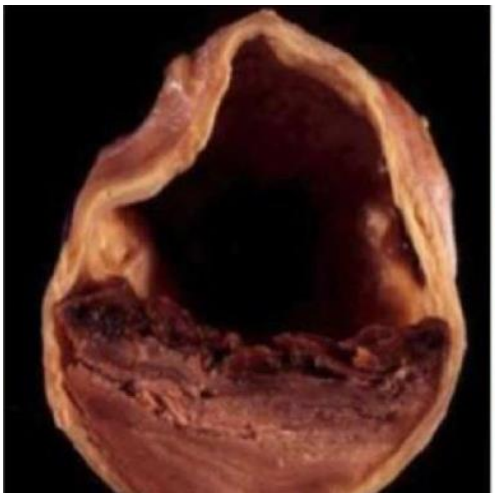
TERMS TO REMEMBER ….

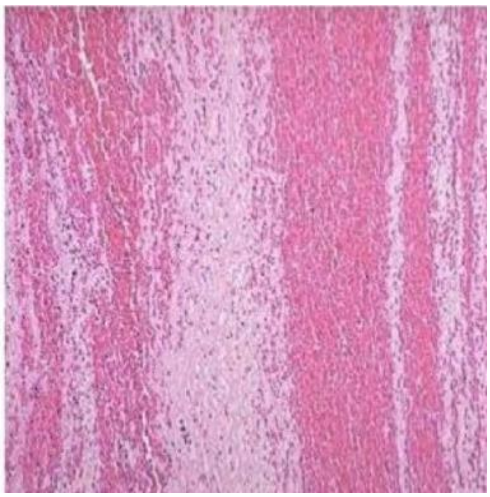
LINES OF ZAHN

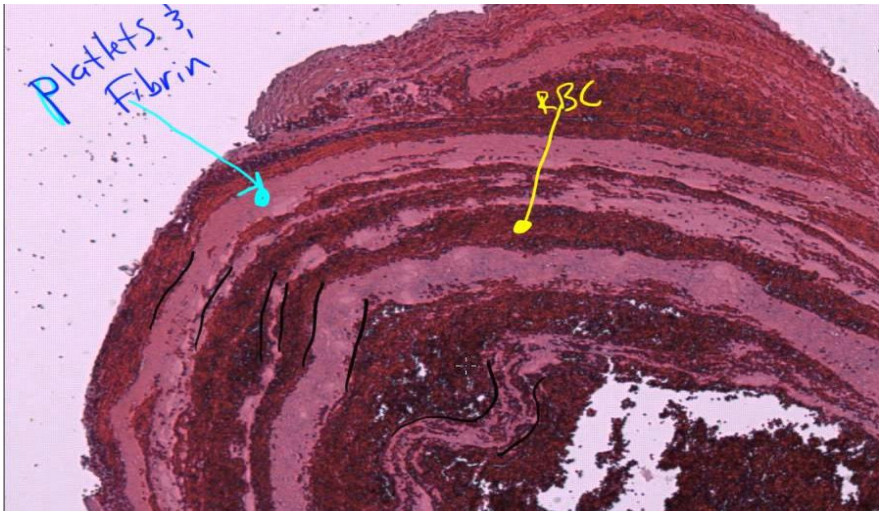
-gross and microscopically apparent laminations
-represent pale platelet and fibrin layers alternating with darker erythrocyte-rich layers
-Significance?
distinguish antemortem (*before death*) thrombosis from postmortem (*after death*) clots , *so determination the cause of death and whether the blood clots are formed during life or just some gravity effect that leads to blood clot formation after death*

-postmortem blood clots are non-laminated clots (no lines of Zahn)cross sectional of the BV:

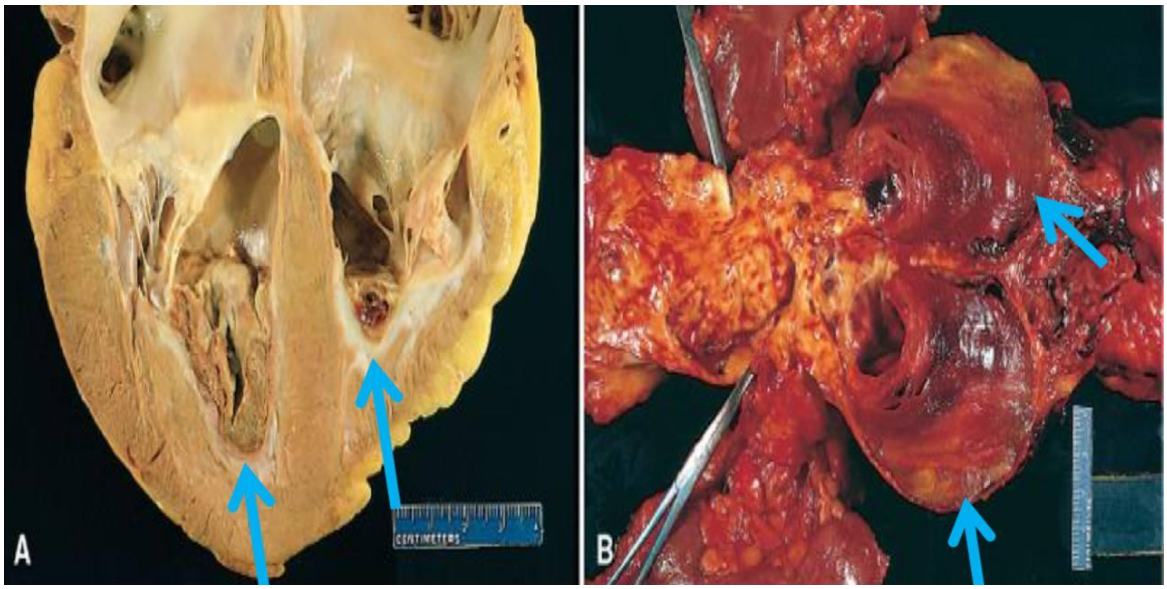
Someone asked since the patient is dead how come blood clots will be formed inside his BV!
He is dead-no blood flow movement-turbulence-clots formation .



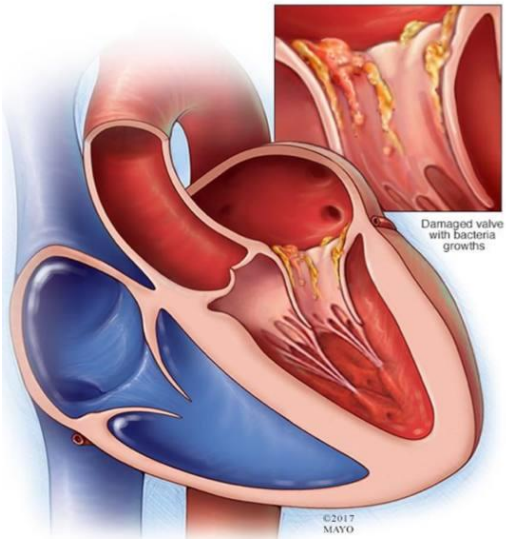


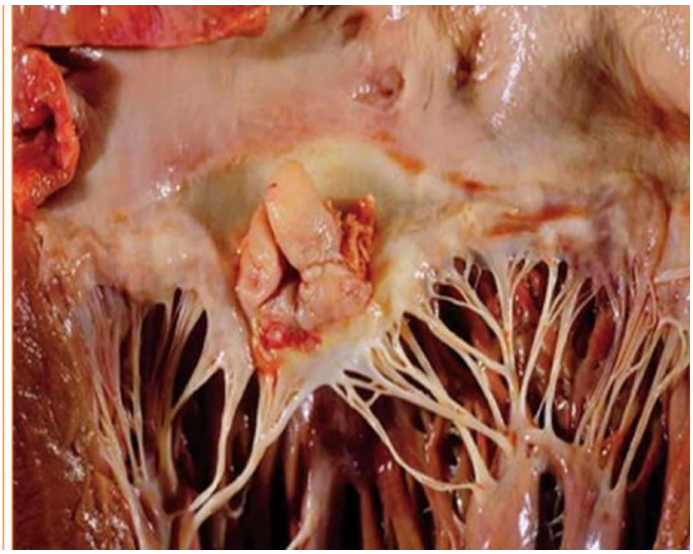


MURAL THROMBI= IN HEART CHAMBERS OR IN AORTIC LUMEN



CARDIAC VEGETATIONS





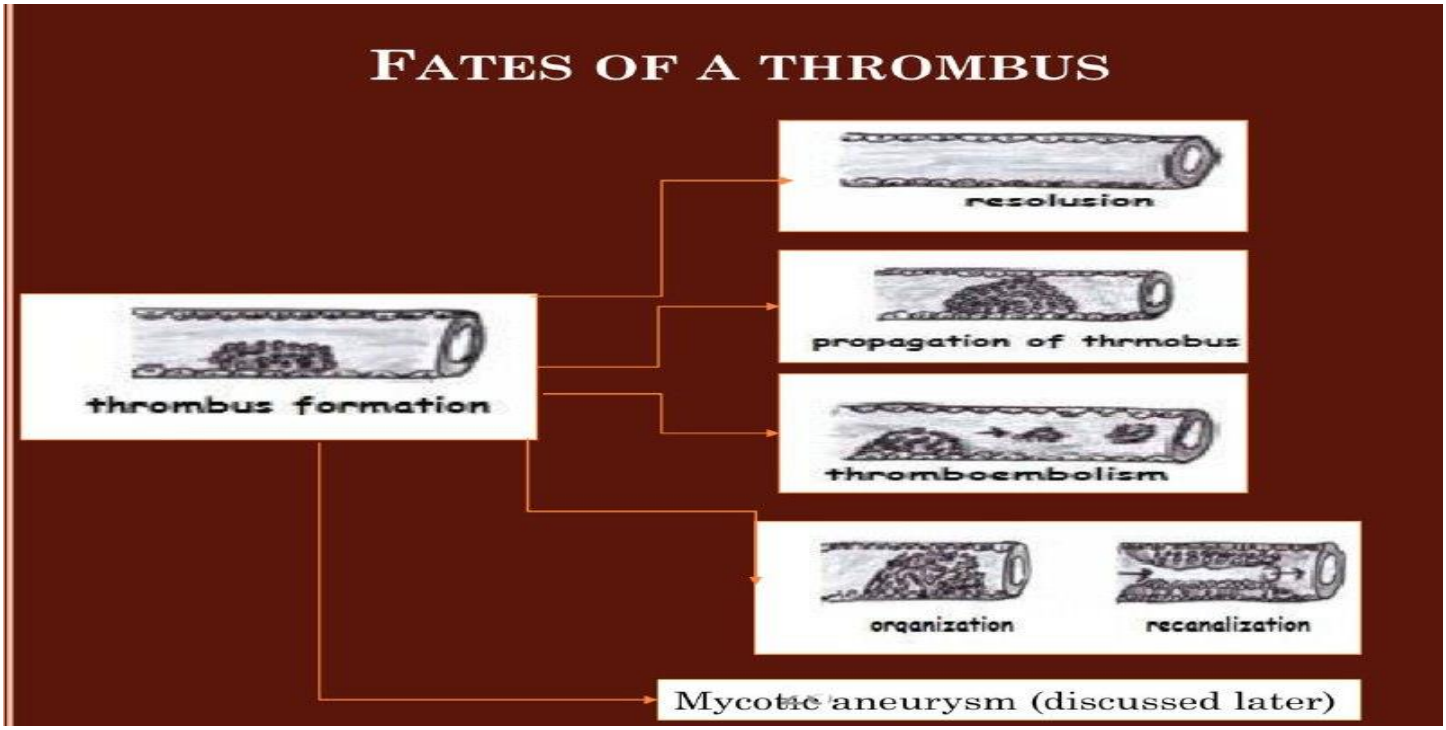
-Thrombi on heart valves

Types:

1- infectious (Bacterial or fungal blood-borne infections) e.g., infective endocarditis
2- non- infectious: e.g., non-bacterial thrombotic endocarditis

note: infectious means that the thrombus itself is infectious (thrombus+ microorganism inside of it)

Fates of a thrombus



First fate: Resolution which is the perfect fate, it means dissolving of the thrombus only if it was

- *small*
- *recent thrombi within hours*

Second fate: Propagation and getting larger, resulting in occlusion

Third fate: Thromboembolism-there will be a discharged, freely, small thrombi in the circulation

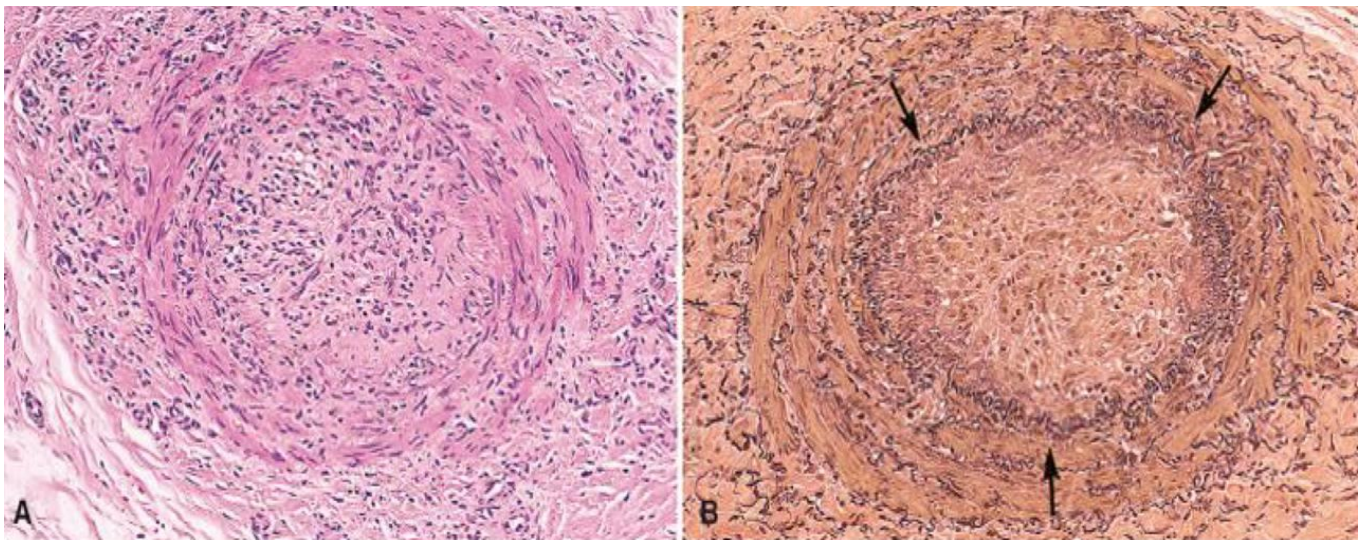
Fourth fate: Organization /Recanalization

Organization -which is same thrombus ,same site but with different components(more resilient , irreversible & permanent)

Recanalization -to make an opening inside this permanent thrombus ,but it's smaller than the BV diameter & impeding the rapidity of blood flow .

Fifth fat : Mycotic aneurysm ,it's rare & will be discussed later inshallah

ORGANIZED ARTERIAL THROMBUS



Lumen under microscope would be seen as whitish color if not containing any blood ,but in this figure I can not recognize any lumen

Both figures having organization but with different stains.

Fate of thrombi

1. ***Propagation*** → accumulate additional platelets and fibrin, eventually causing **vessel obstruction**
2. ***Embolization*** → Thrombi dislodge or fragment and are transported elsewhere in the vasculature
3. ***Dissolution*** → Thrombi are removed by fibrinolytic activity (only in **recent** *thrombi)*
4. ***Organization* and recanalization*** → Thrombi induce inflammation and fibrosis. These can *recanalize* (re-establishing some degree of flow), or they can be incorporated into a thickened vessel wall

**Organization refers to the ingrowth of endothelial cells, smooth cells and fibroblasts into the fibrin rich thrombus.*

5. ***Superimposed infection (Mycotic aneurysm)***

No motivational quote for today's sheet

if you want to give up You're free to do so.

Love,Kotkot!