THROMBOSIS- PATHOLOGICAL ASPECTS

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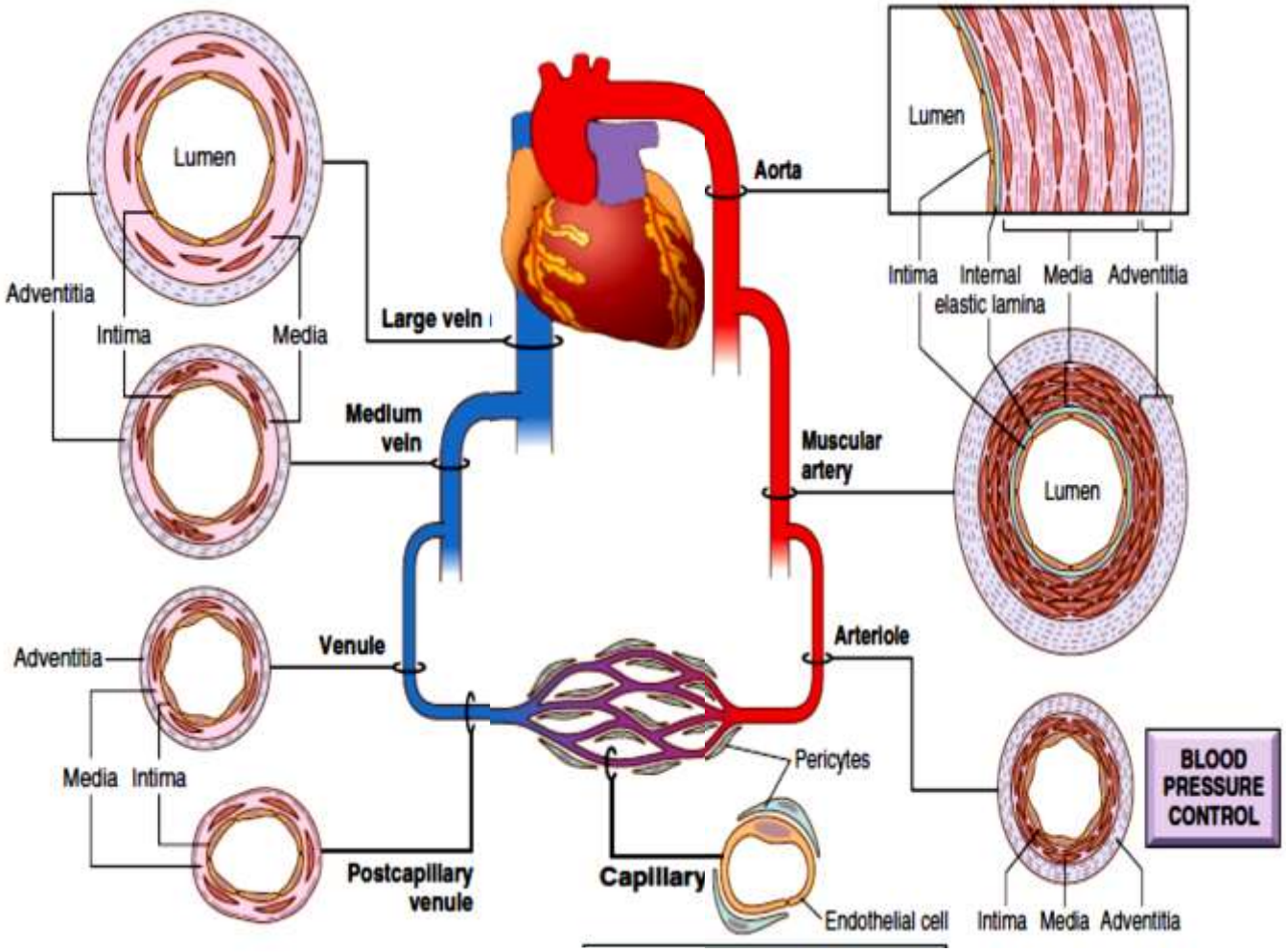
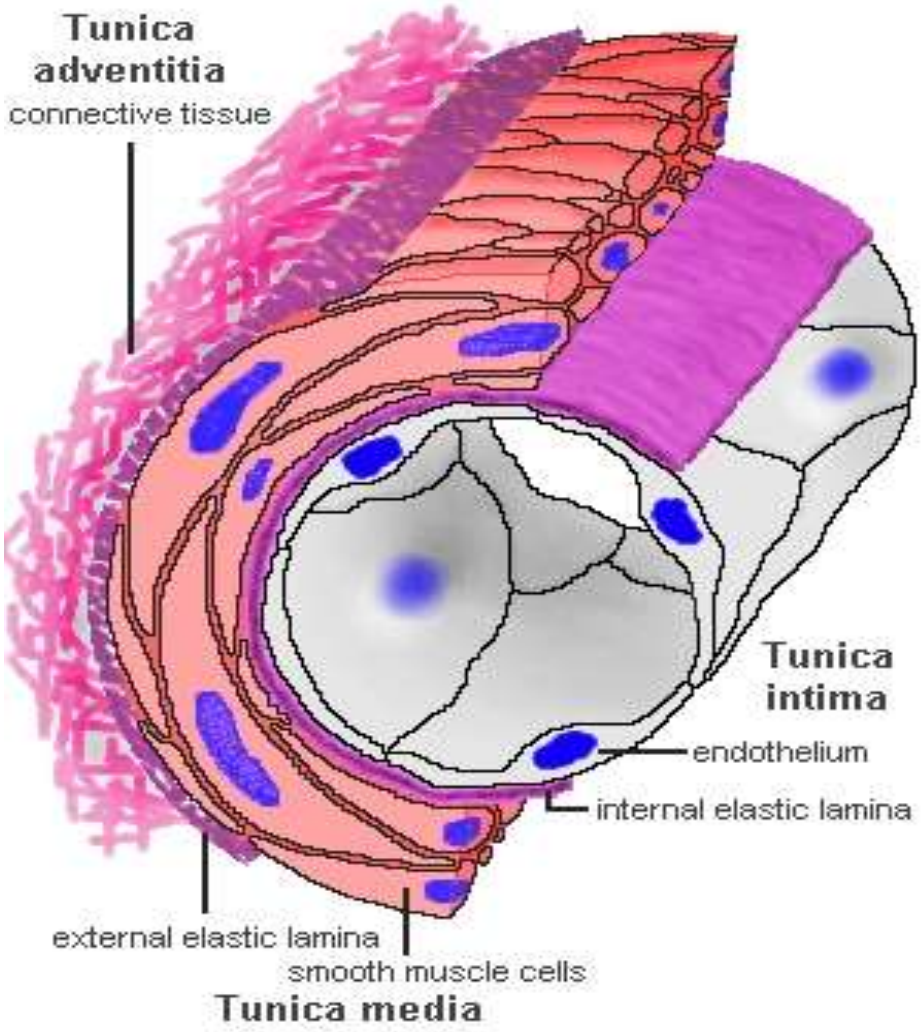
CARDIOVASCULAR SYSTEM

Venous circulation

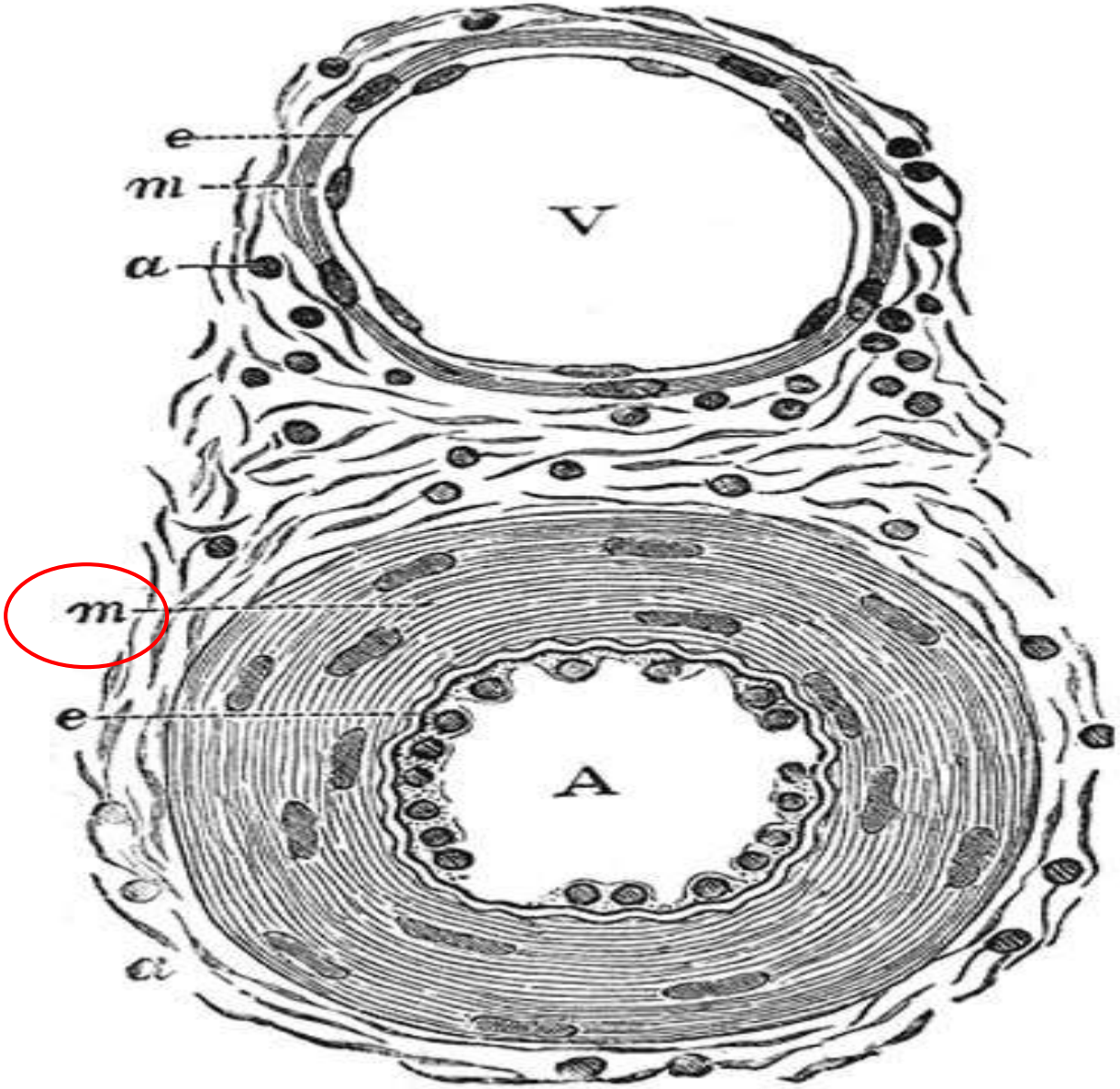
Arterial circulation



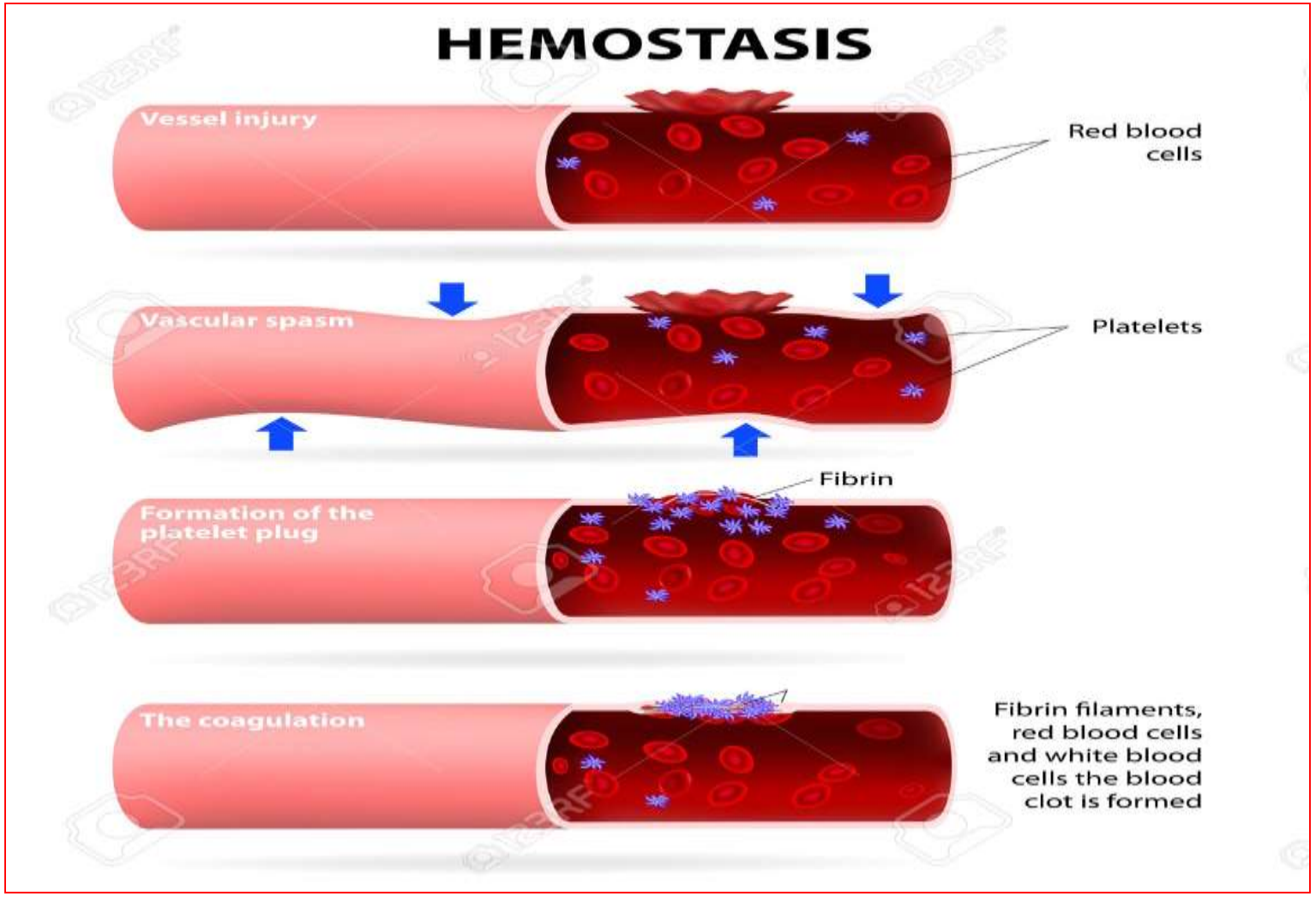
Normal blood vessel histology



ARTERY (A) VS VEIN (V)



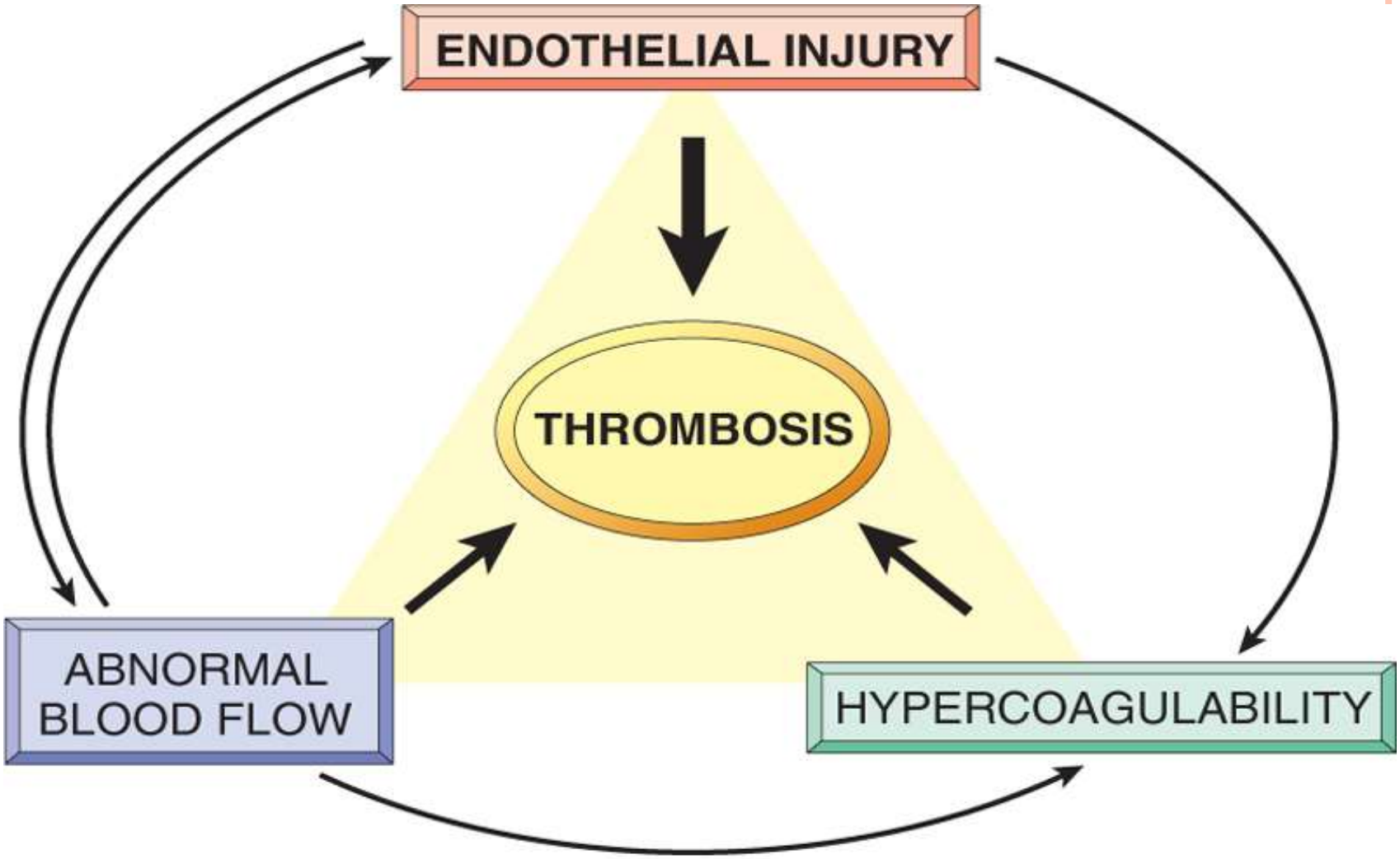
Physiology of thrombosis



THROMBOSIS- PATHOLOGICAL ASPECTS

- Blood coagulation is a very 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**):

Virchow's triad



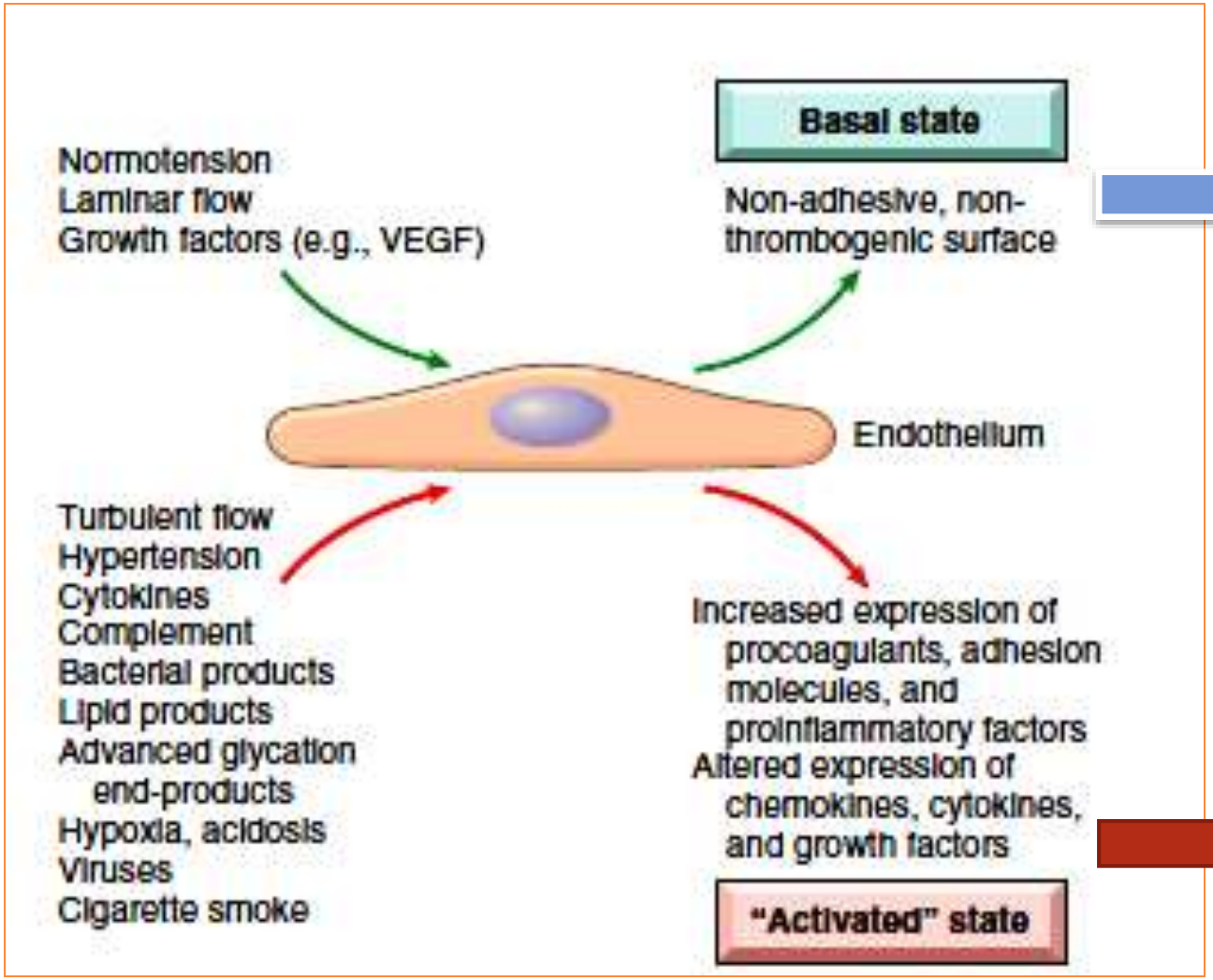
Thrombosis- pathological aspects

- **Pathogenesis (called *Virchow's triad*):**

1. ***Endothelial* Injury (Heart, Arteries)***
2. ***Stasis (abnormal blood flow)***
3. ***Blood Hypercoagulability***

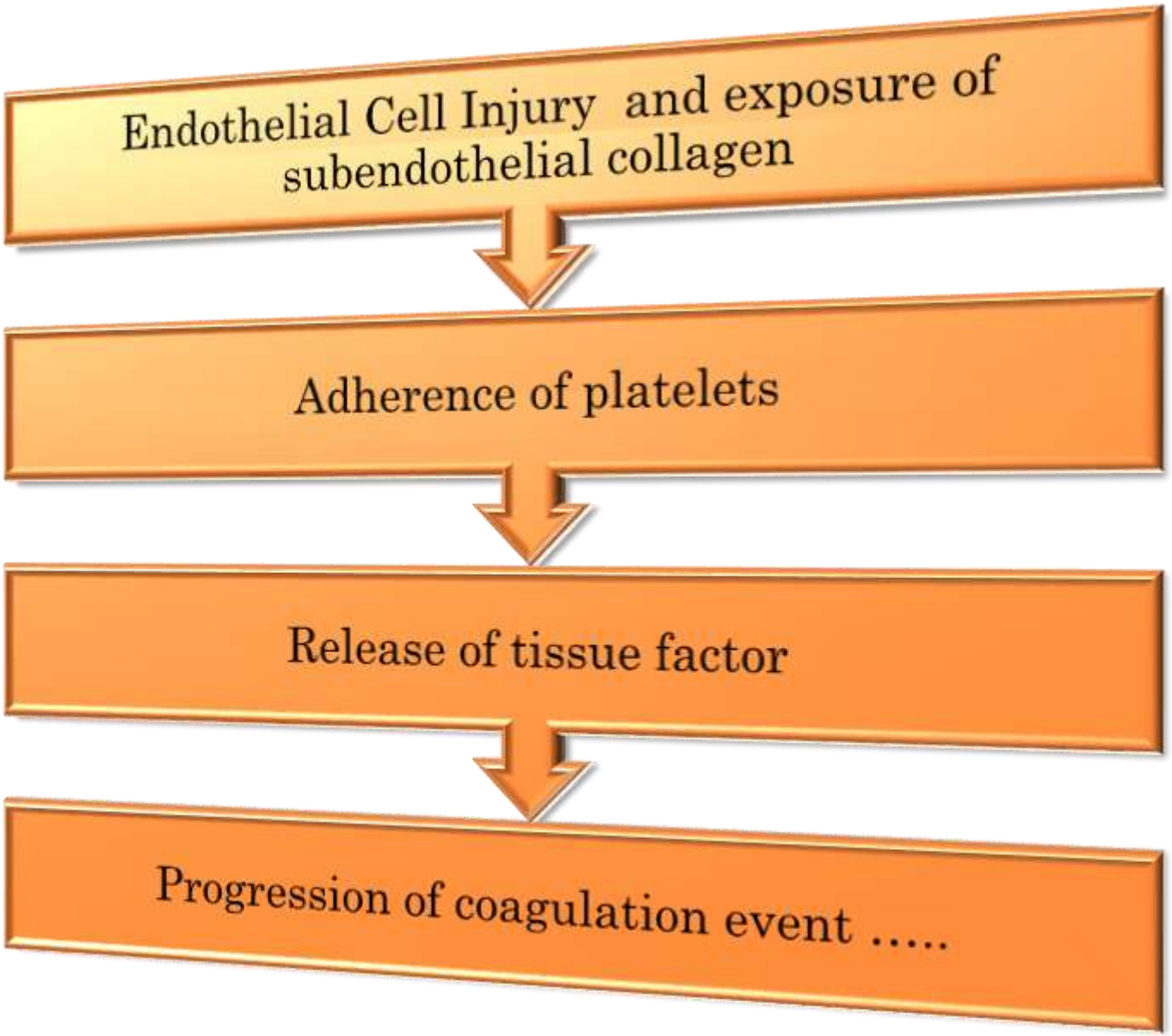
*** Endothelial cells are special type of cells that cover the inside surface of blood vessels and heart.**

Contribution of Endothelial Cells to Coagulation

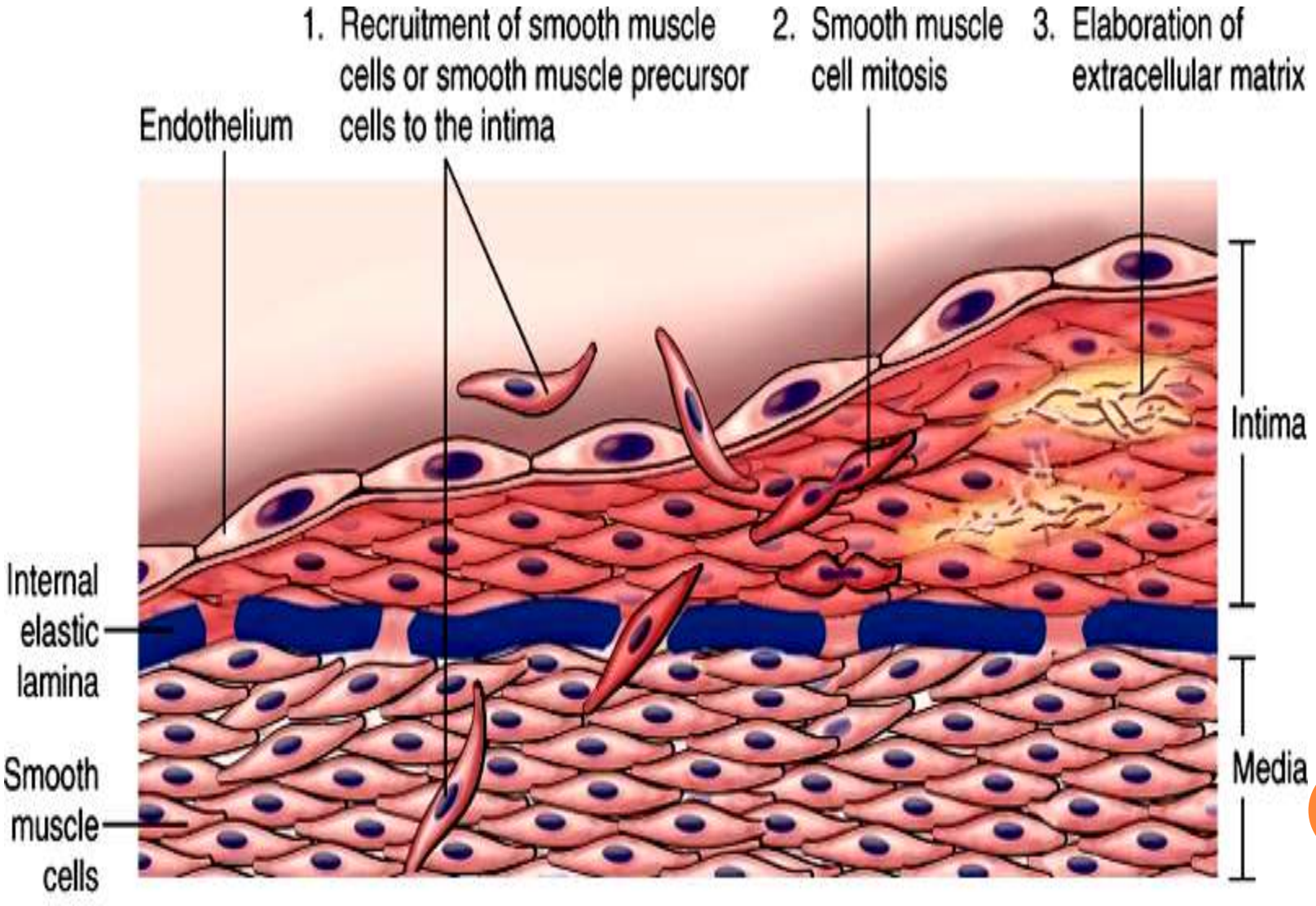


No thrombosis

thrombosis


Endothelial Cell Injury and exposure of subendothelial collagen
Adherence of platelets
Release of tissue factor
Progression of coagulation event .....

Response of Vascular Wall Cells to Injury



Response of Vascular Wall Cells to Injury

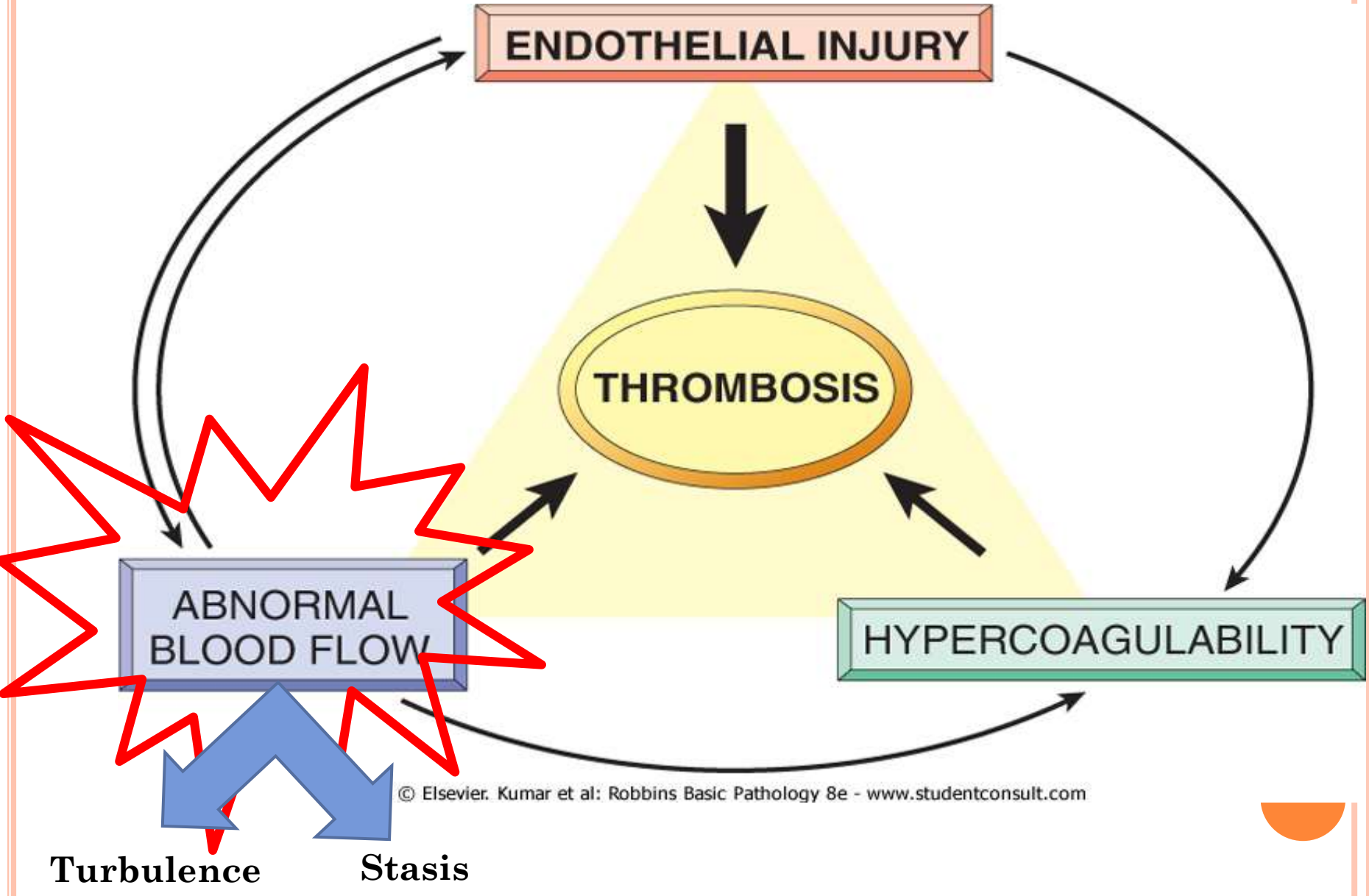
- **Injury results in a healing response**

- **Pathologic effect of vascular healing:**

Excessive thickening of the intima →→
luminal stenosis & blockage of vascular flow

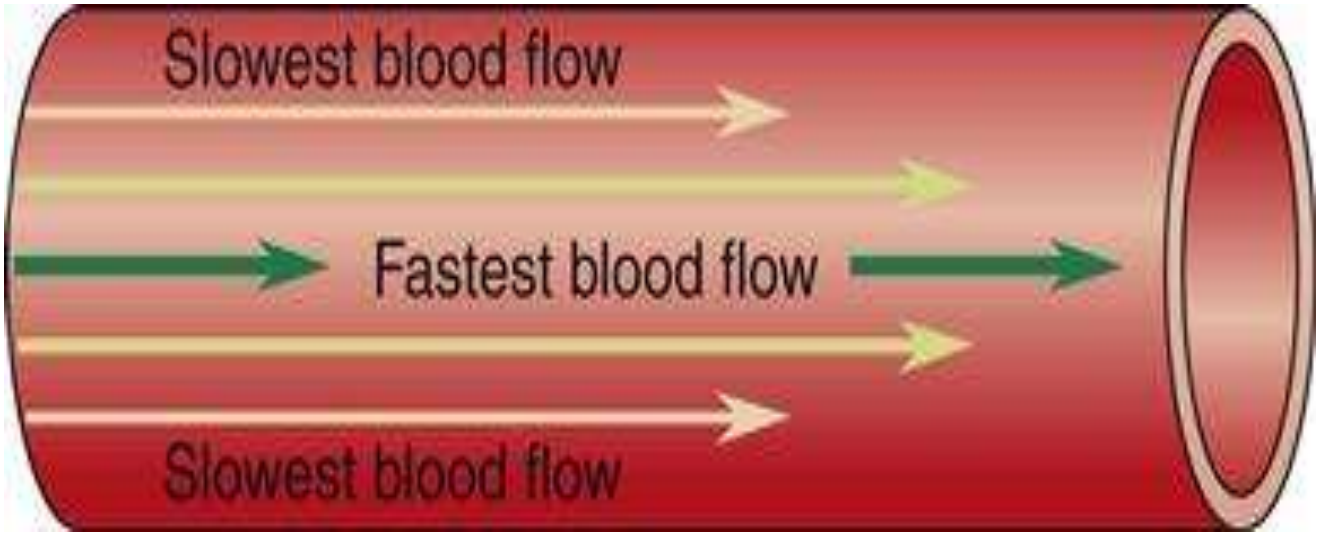
Causes of Endothelial injury

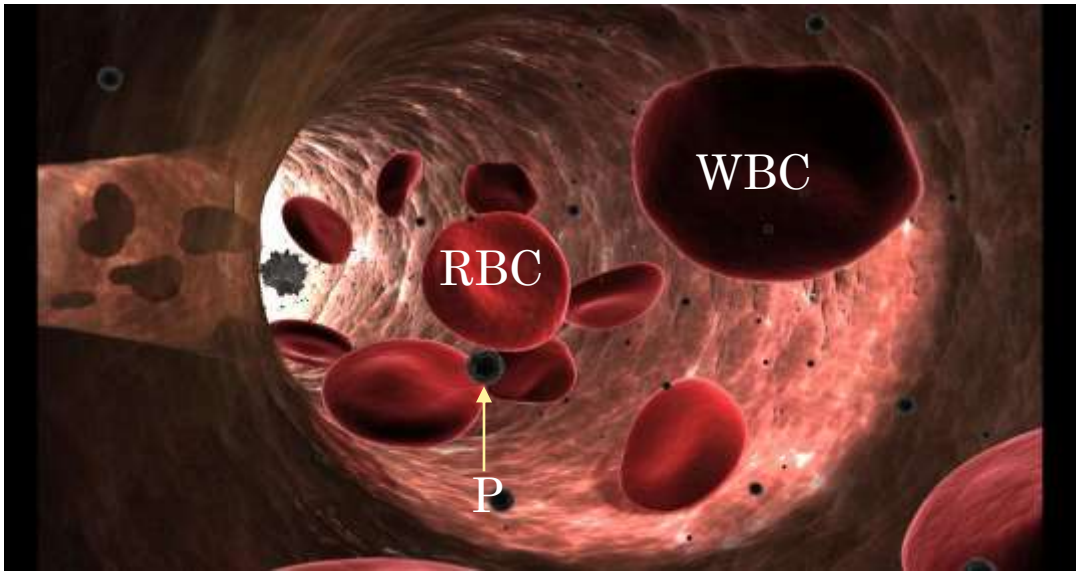
1. ***Valvulitis***
2. ***MI***
3. ***Atherosclerosis***
4. ***Traumatic or inflammatory conditions***
5. ***Hypertension***
6. ***Endotoxins***
7. ***Hypercholesterolemia***
8. ***Radiation***
9. ***Smoking***
10. ***……etc.***



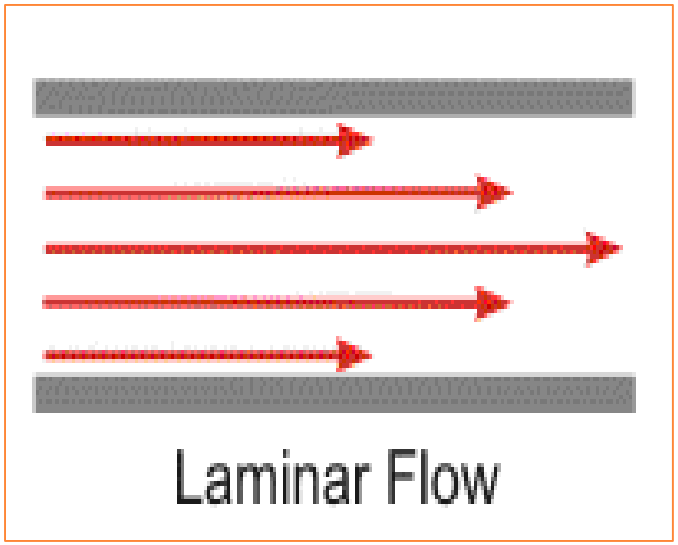
Turbulence **Stasis**

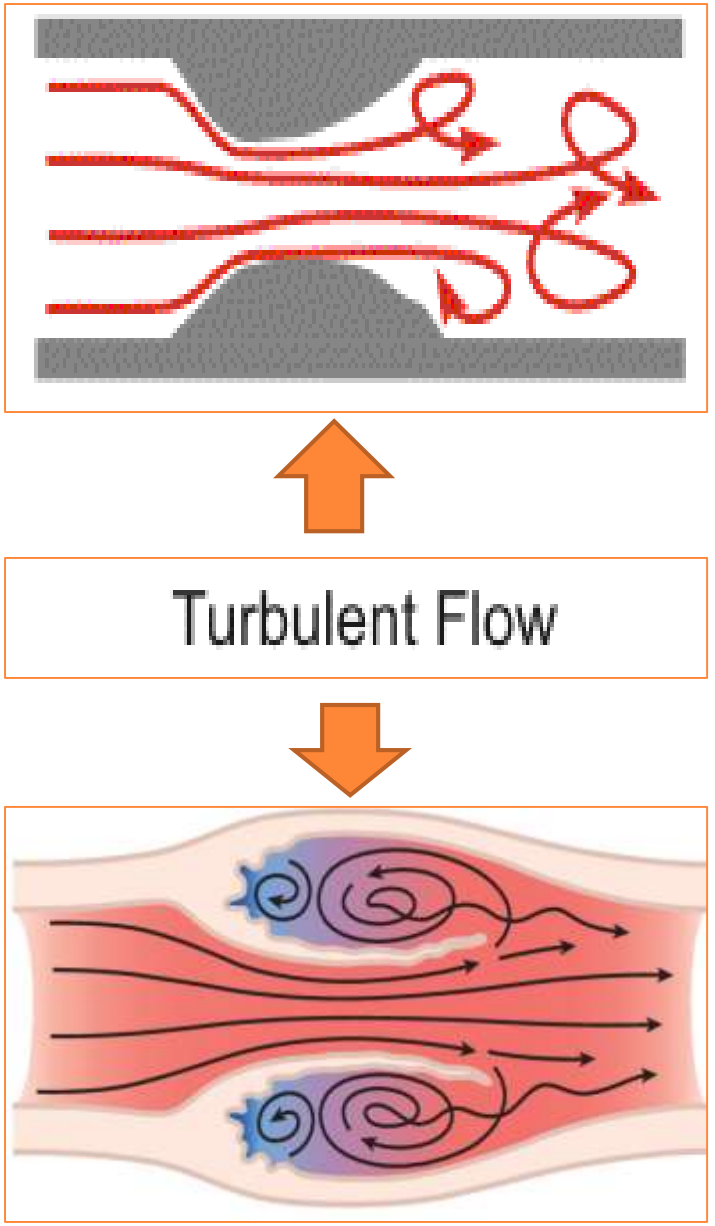
Laminar blood flow (normal)





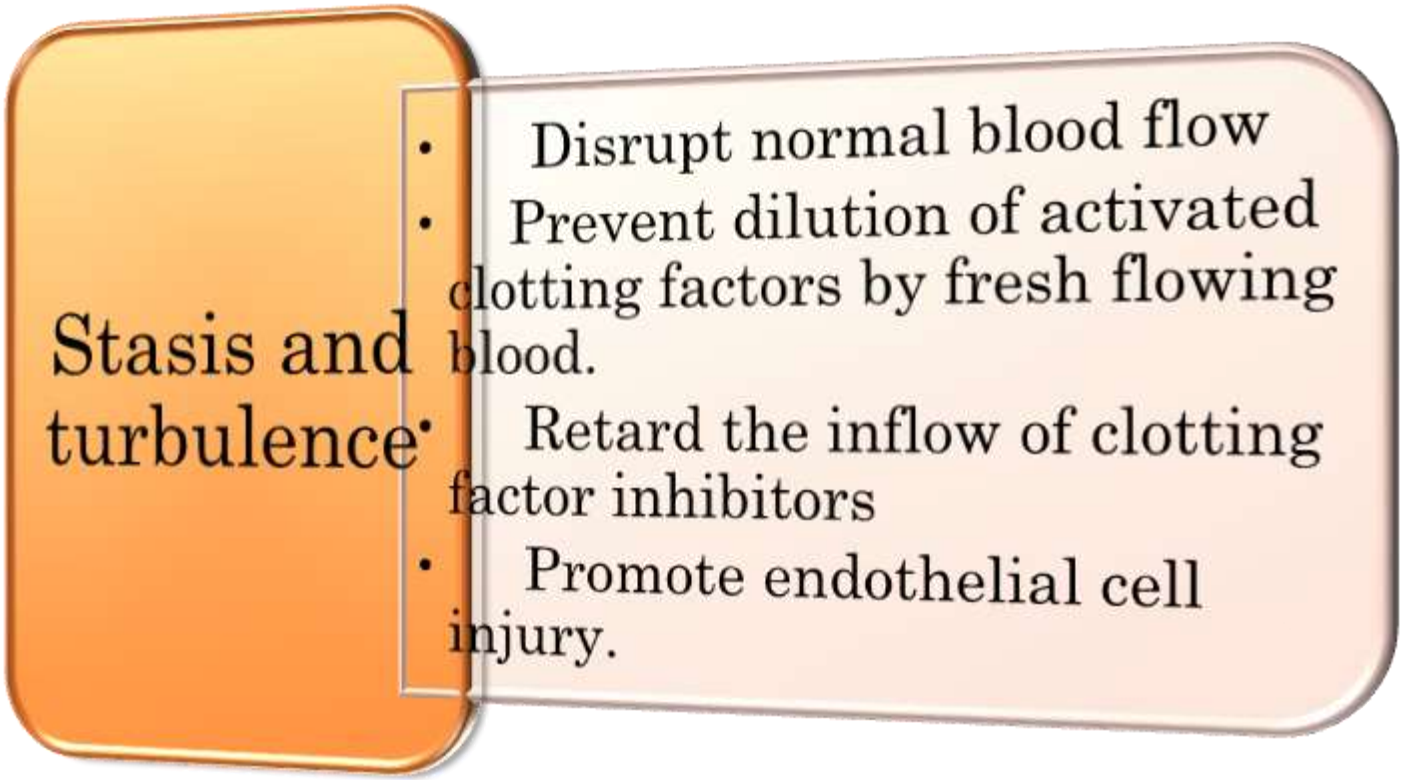
Laminar vs turbulent blood flow





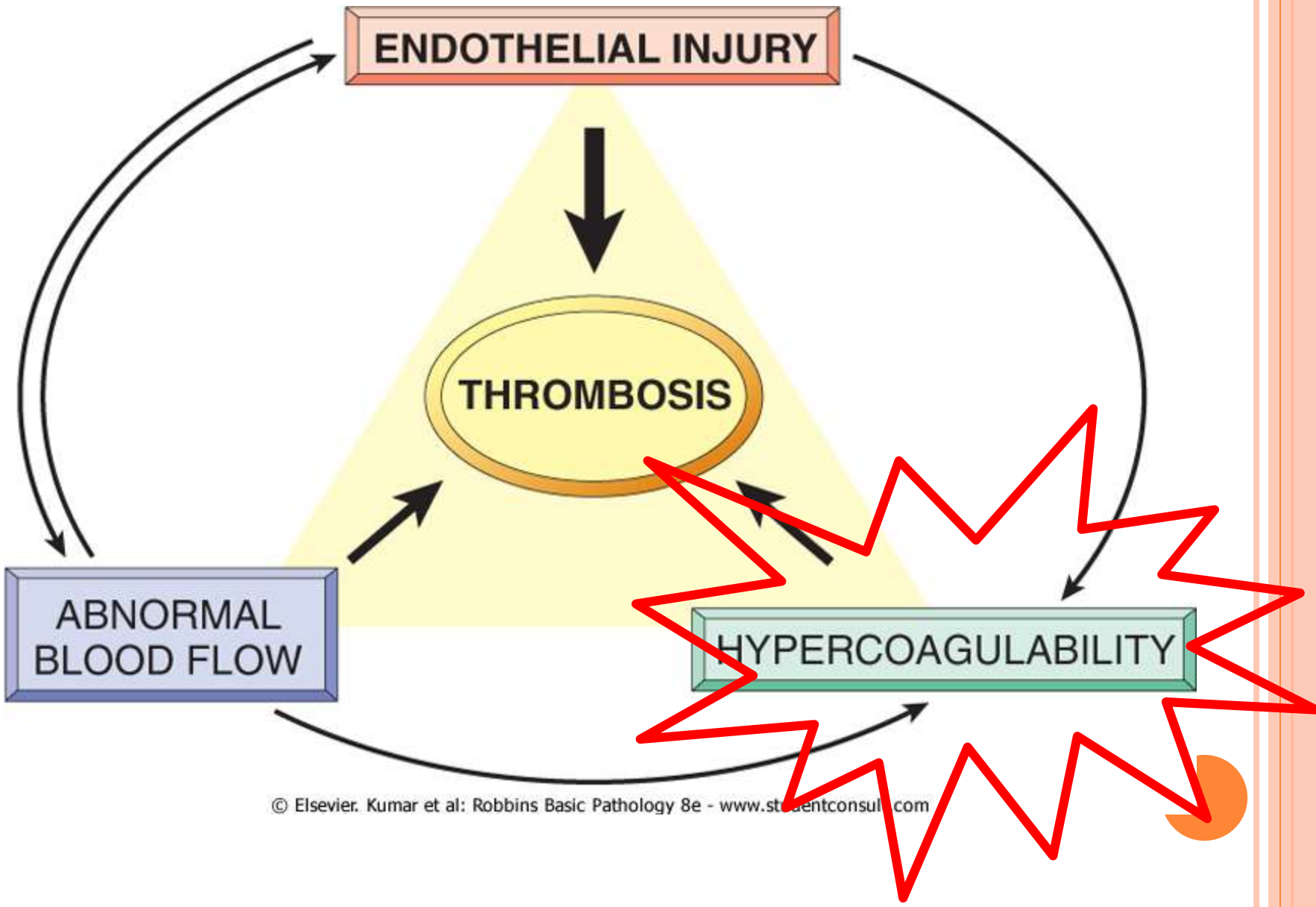
Stasis

- *Stasis is a major factor in **venous** thrombi*
- Normal blood flow is ***laminar (p***latelets flow centrally in the vessel lumen, separated from the endothelium by a slower moving clear zone of plasma)
- Stasis and turbulence cause the followings:



Causes of Stasis

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)***
6. **……..**


ENDOTHELIAL INJURY
THROMBOSIS
ABNORMAL BLOOD FLOW
HYPERCOAGULABILITY
© Elsevier. Kumar et al: Robbins Basic Pathology 8e - www.studentconsult.com

Hypercoagulability

A. Genetic (primary):

- Inherited mutations in clotting factors or anti-clotting factors
- mutations in factor V gene and prothrombin gene are the most common causes of primary

B. Acquired (secondary):

- Much more frequent than primary causes
- multifactorial & more complicated
- causes include: Immobilization, MI, AF, surgery, fractures, burns, Cancer, Prosthetic cardiac valves …etc

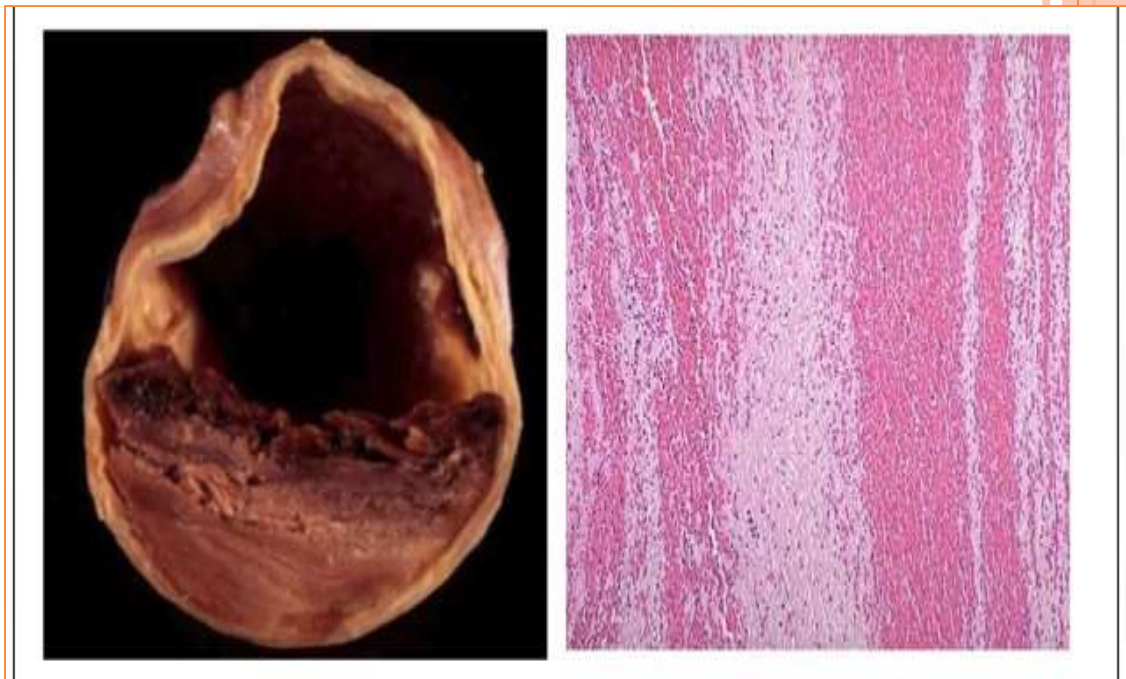
MORPHOLOGY OF THROMBI

- **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 is poorly attached →fragmentation and embolus formation**

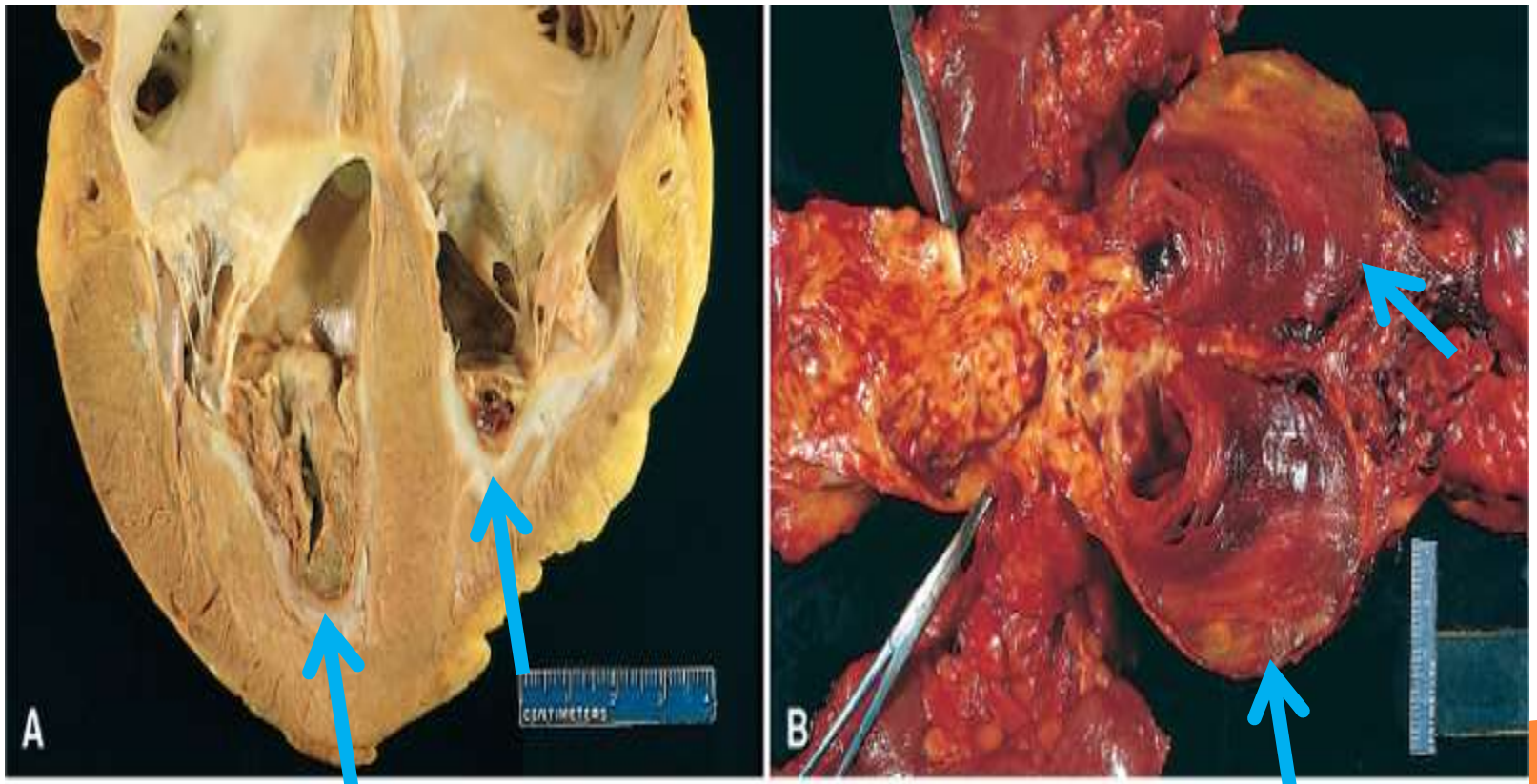
Terms to remember ….

LINES OF ZAHN

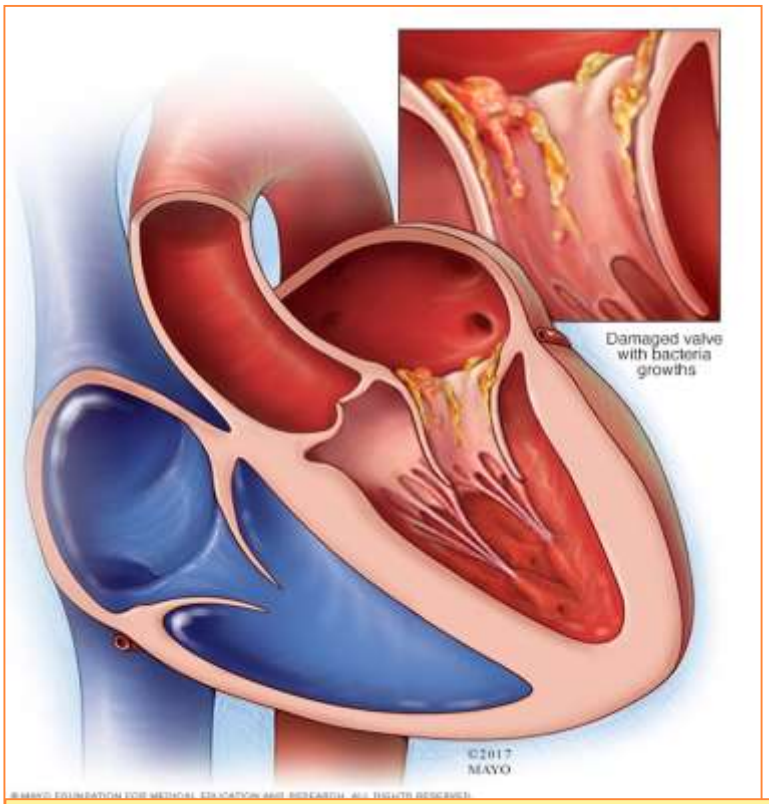
- gross and microscopically apparent laminations
- represent pale platelet and fibrin layers alternating with darker erythrocyte-rich layers
- Significance? distinguish **antemortem** thrombosis from postmortem clots
- postmortem blood clots are non-laminated clots *(no lines of Zahn)*

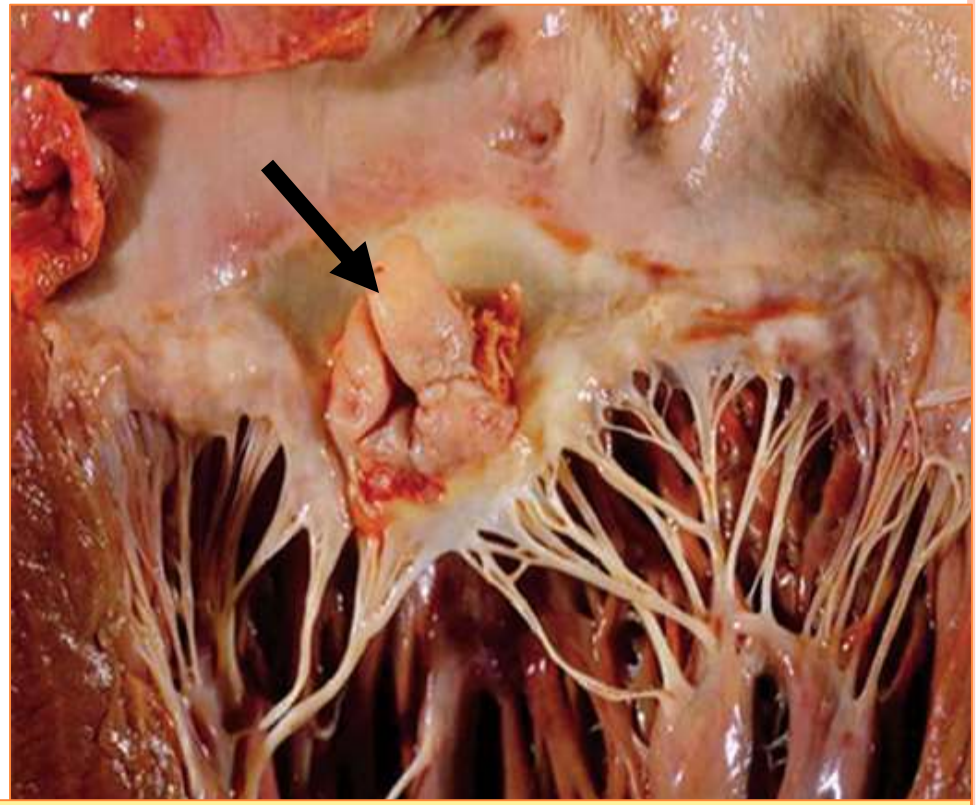


Mural thrombi= - in heart chambers or in aortic lumen



Cardiac Vegetations





= Thrombi on heart valves

Types:

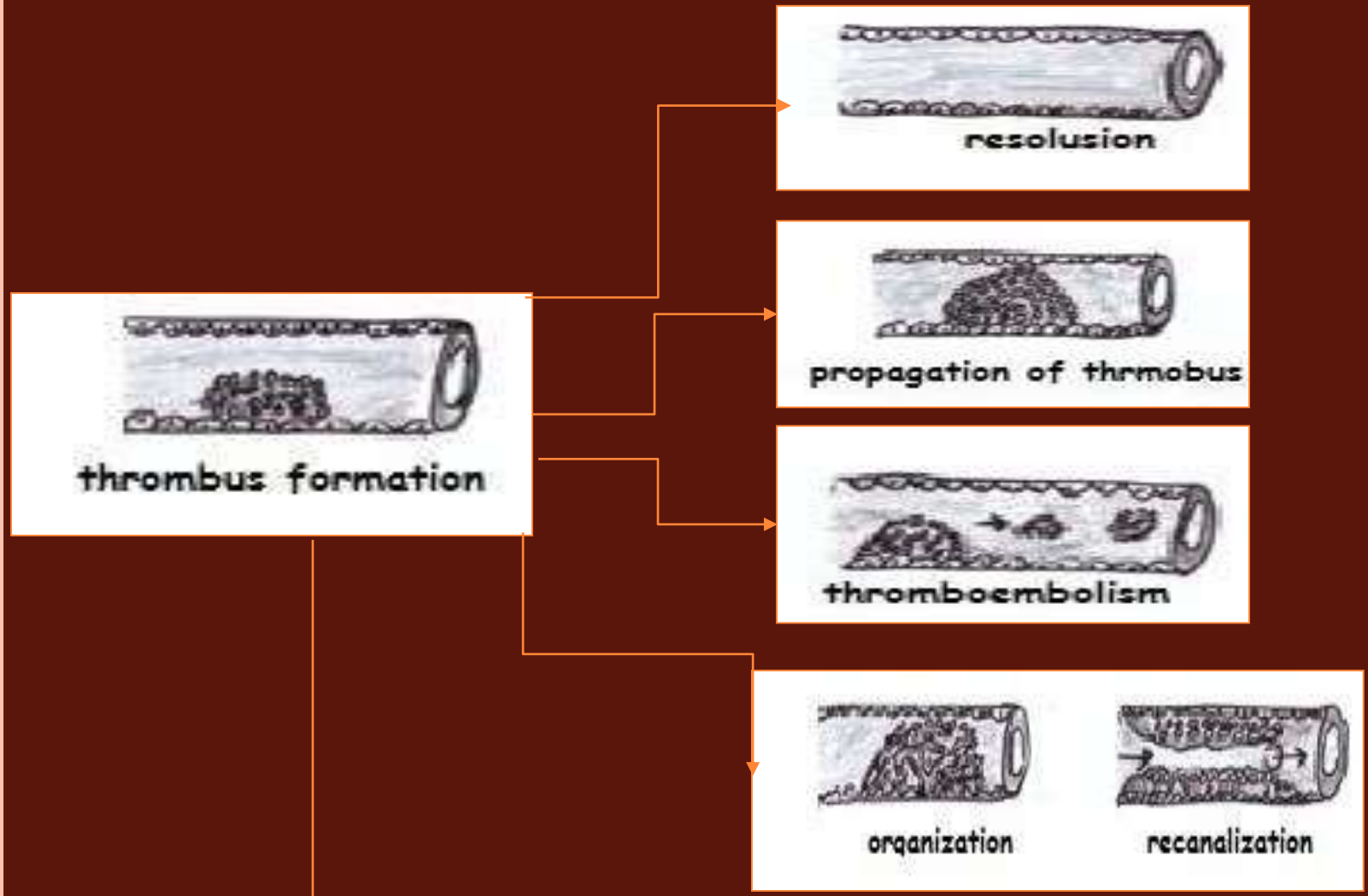
1- infectious (Bacterial or fungal blood-borne infections)

e.g. infective endocarditis

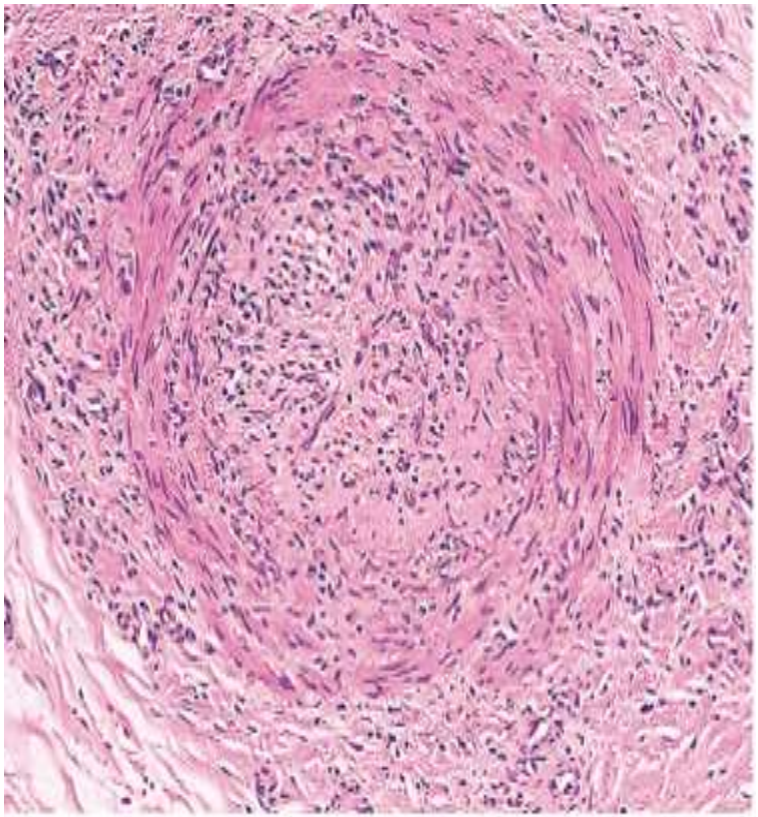
2-non- infectious:

e.g. rheumatic; non-bacterial thrombotic endocarditis

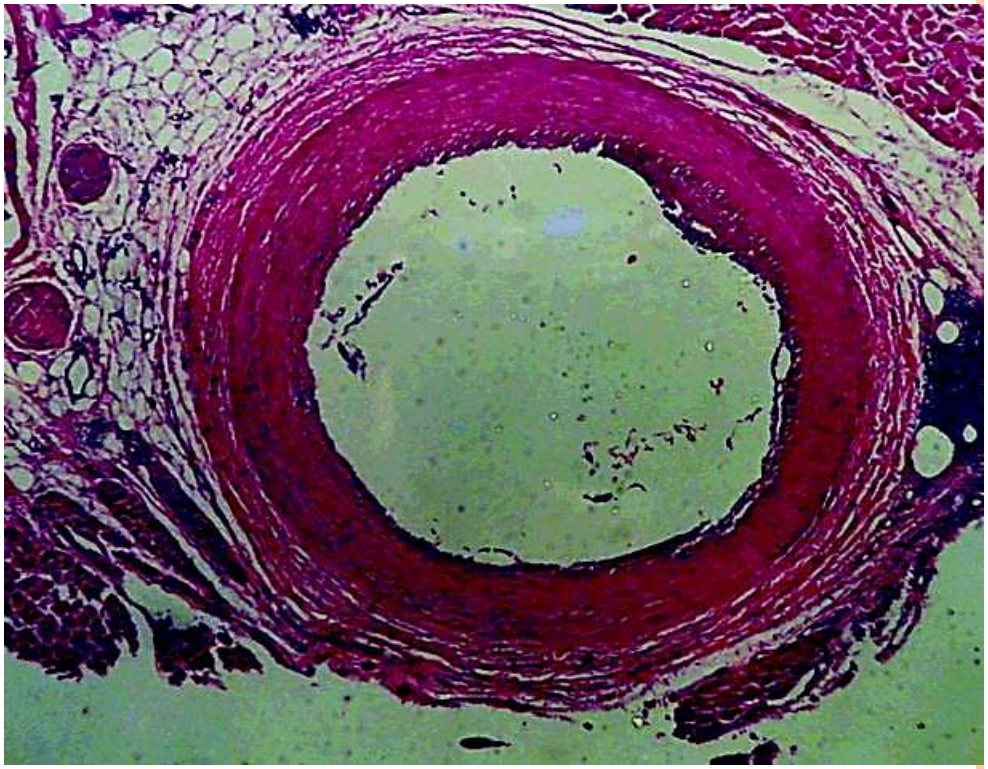
Fates of a thrombus



Organized arterial thrombus



A normal artery cross section for comparison



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)***