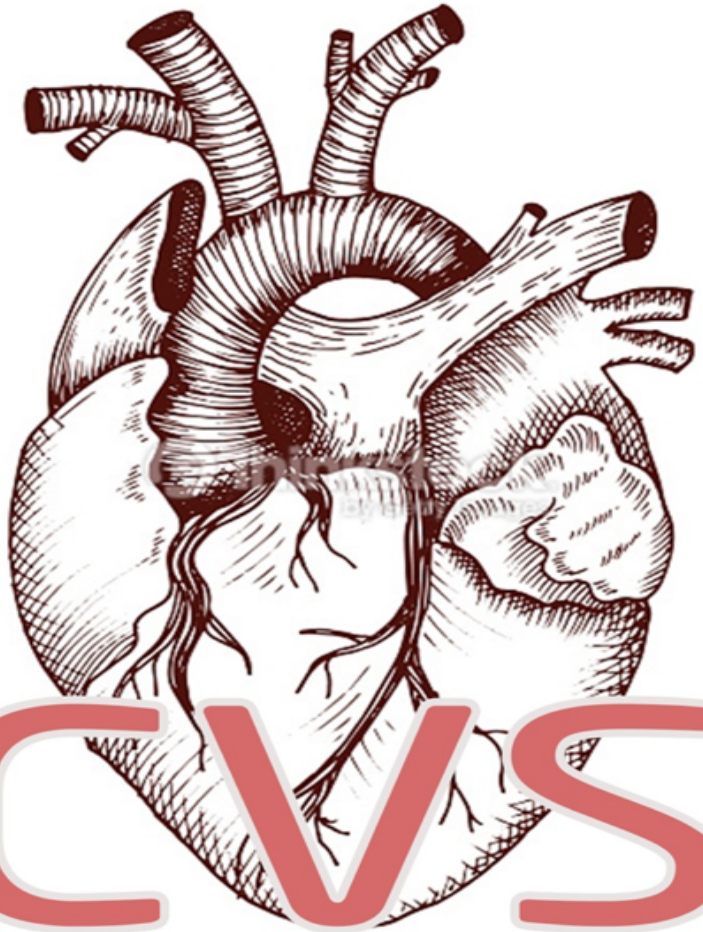


CVS

Pathology

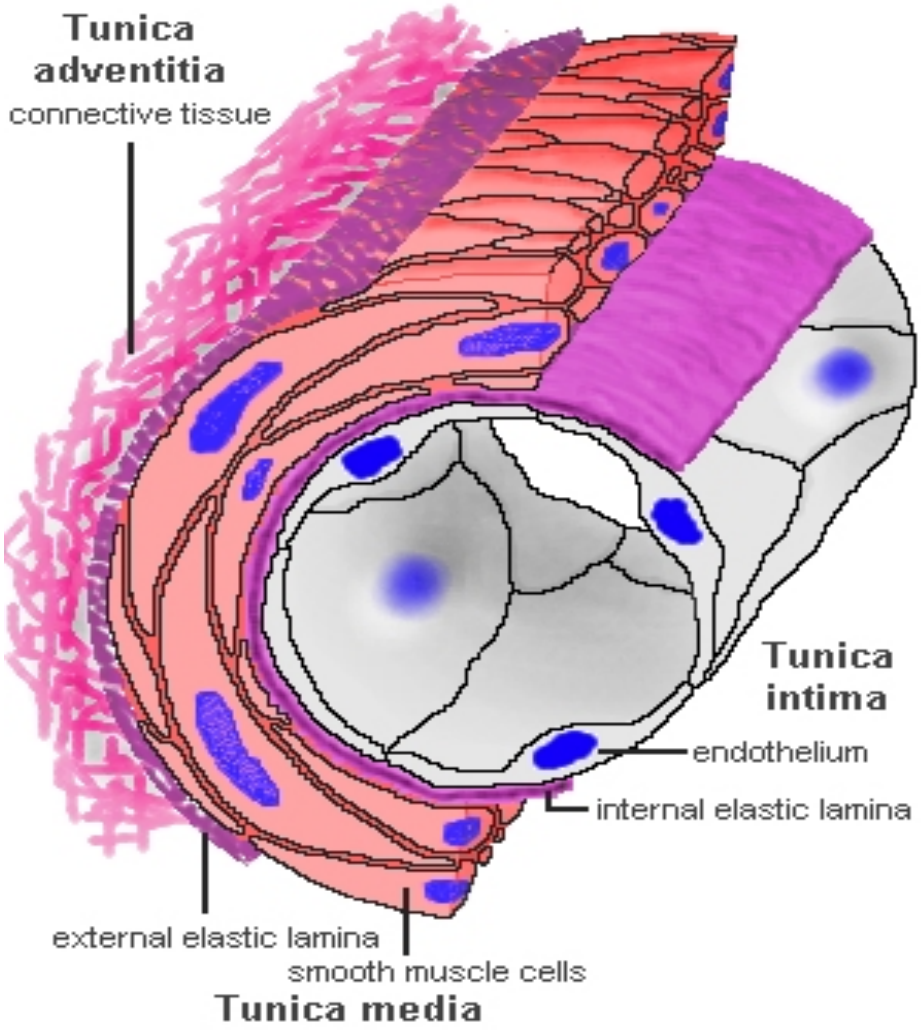
Slide

Sheet

Thrombosis

Dr. Nisreen Abu Shahin
Assistant Professor of Pathology
Pathology Department
University of Jordan

Normal blood vessel histology

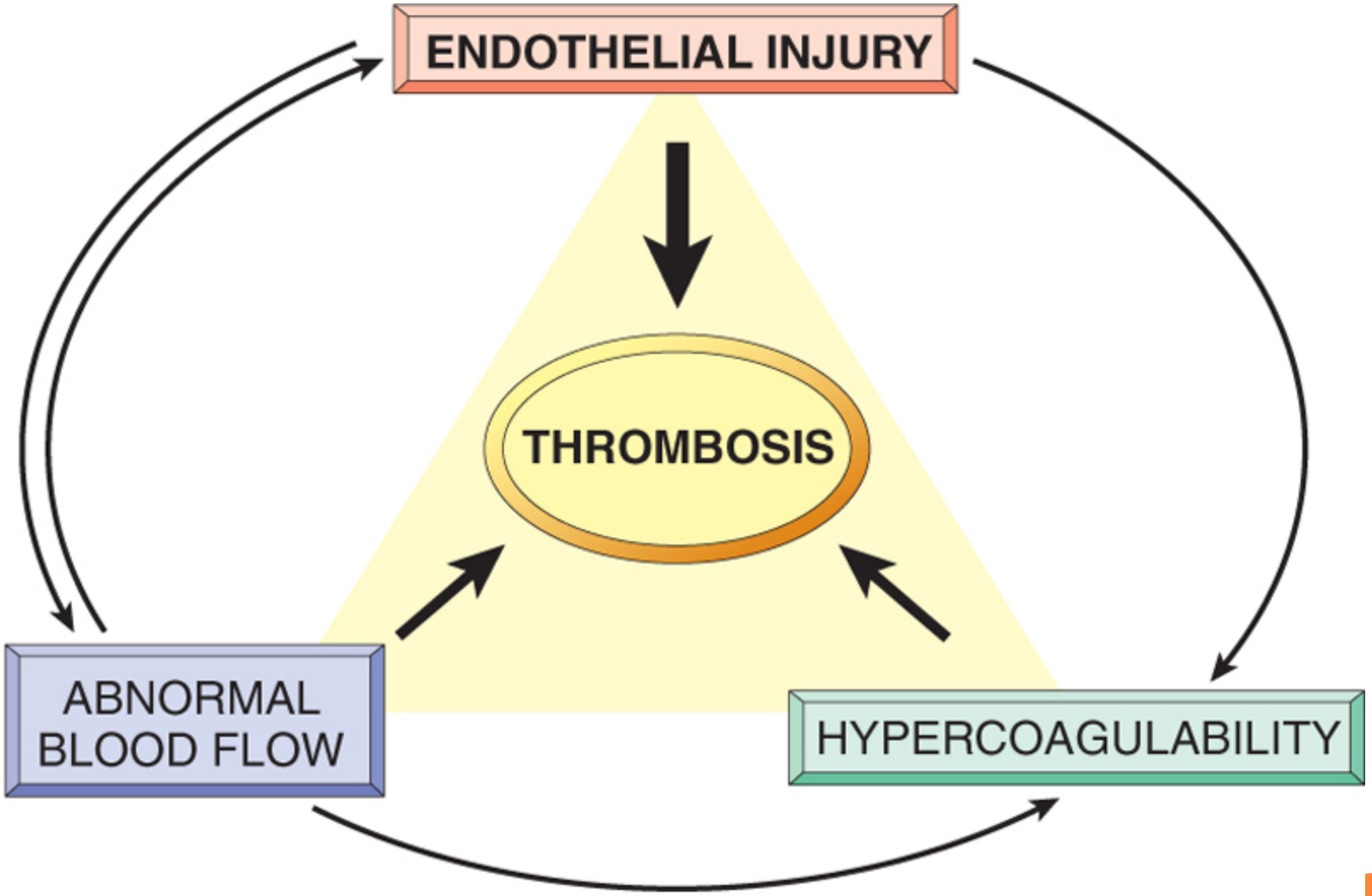


THROMBOSIS

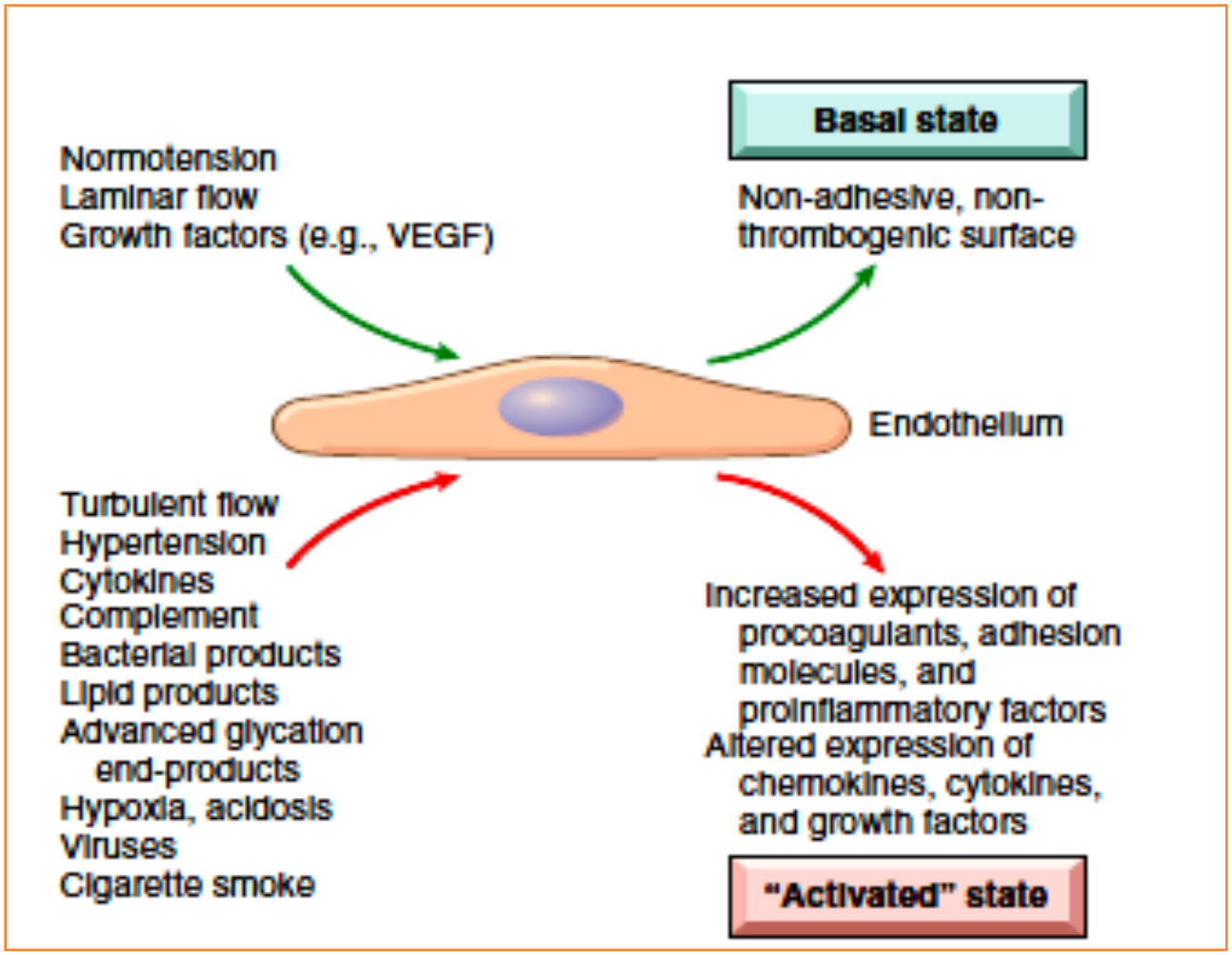
- **Pathogenesis (called *Virchow's triad*):**

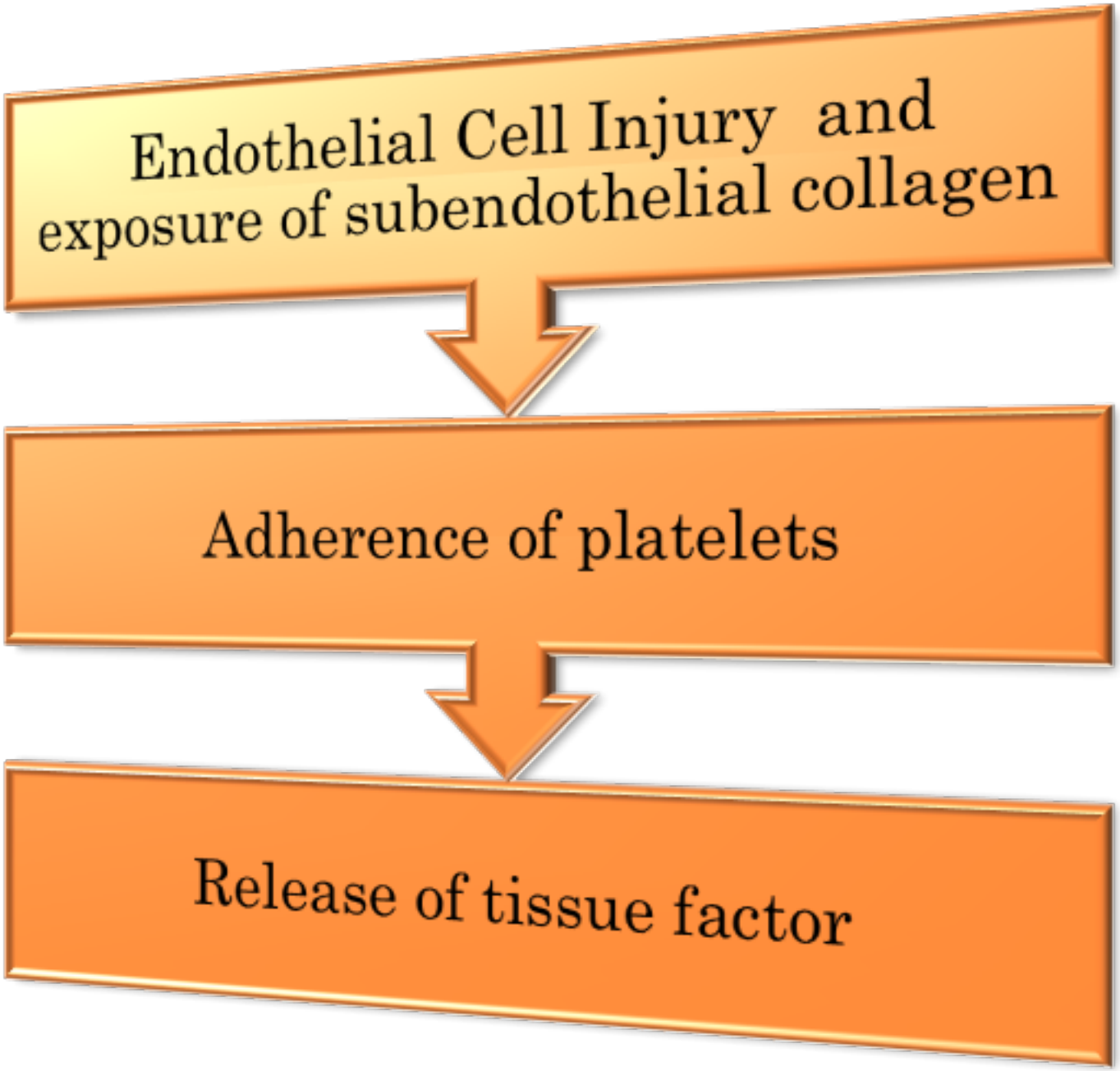
1. ***Endothelial* Injury (Heart, Arteries)***
2. ***Stasis (abnormal blood flow)***
3. ***Blood Hypercoagulability***

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



Contribution of Endothelial Cells to Coagulation




Endothelial Cell Injury and exposure of subendothelial collagen
Adherence of platelets
Release of tissue factor

Response of Vascular Wall Cells to Injury

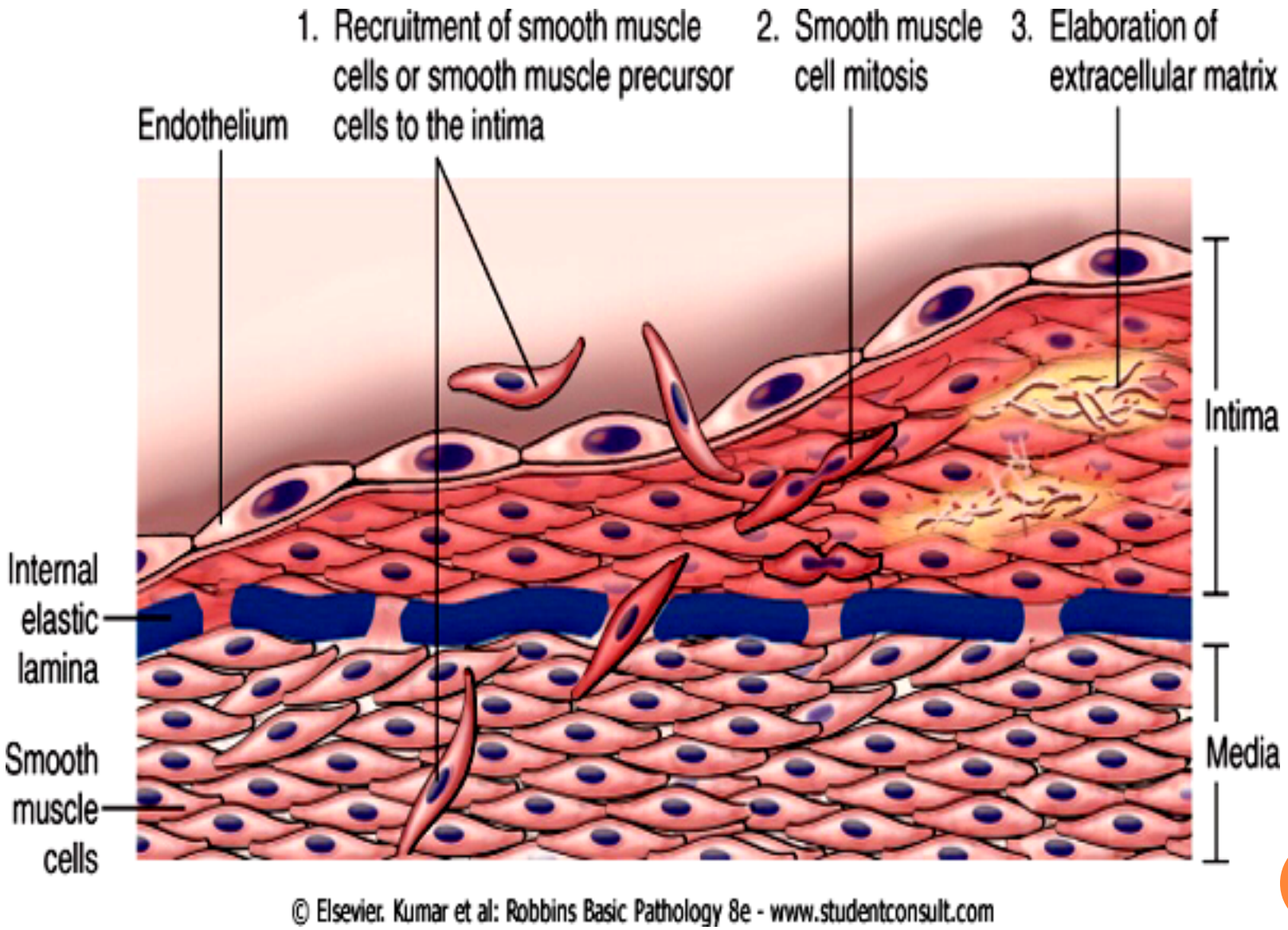
- **Injury results in a healing response:**

1- Intimal expansion (proliferating SMCs; new ECM)

2- recruitment and activation of SMCs

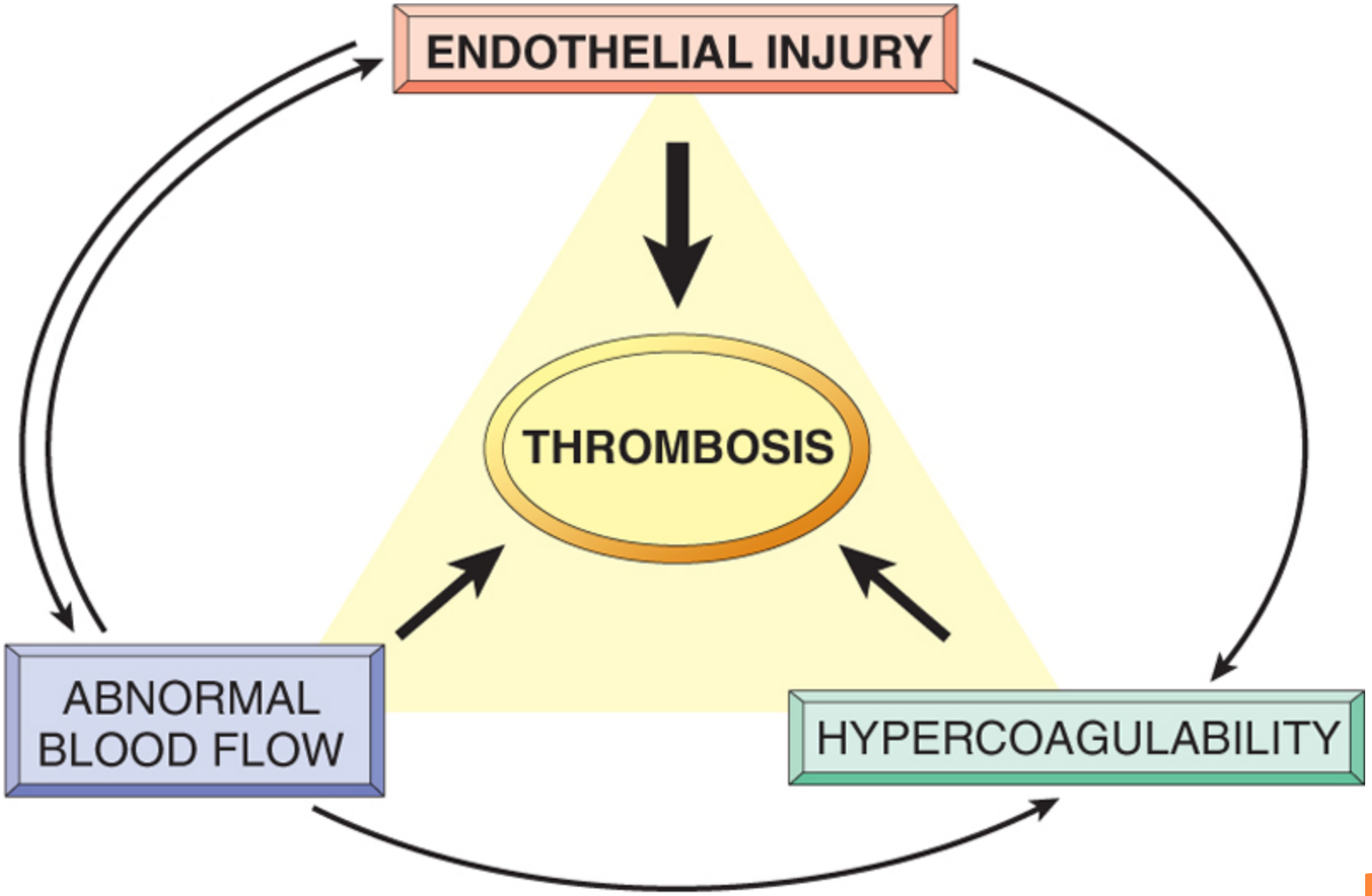
- **Pathologic effect of vascular healing→**

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


1. Recruitment of smooth muscle cells or smooth muscle precursor cells to the intima
2. Smooth muscle cell mitosis
3. Elaboration of extracellular matrix
Endothelium
Internal elastic lamina
Smooth muscle cells
Intima
Media
© Elsevier. Kumar et al: Robbins Basic Pathology 8e - www.studentconsult.com

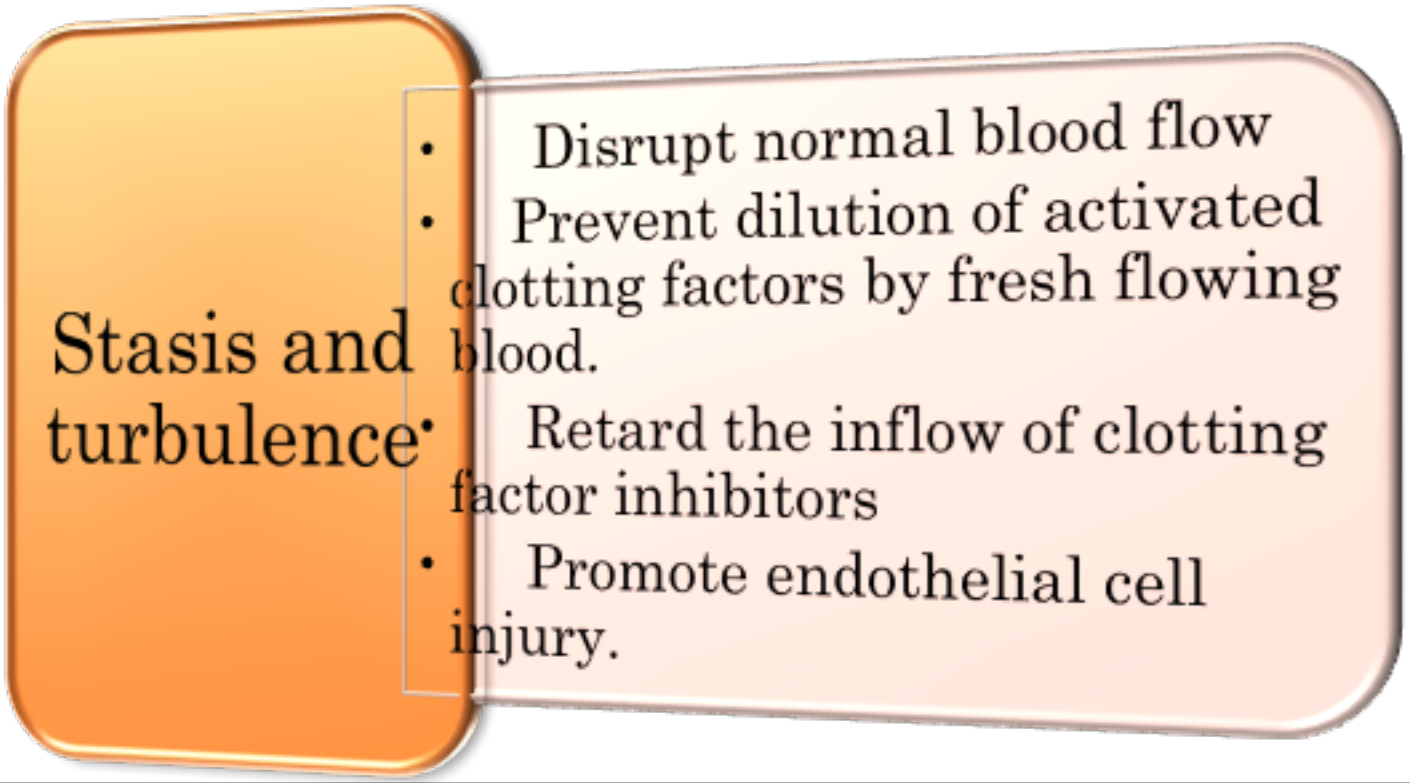
Causes of Endothelial injury

1. *Valvulitis*
2. *MI*
3. *Atherosclerosis*
4. *Traumatic or inflammatory conditions*
5. *Increased Blood Pressure*
6. *Endotoxins*
7. *Hypercholesterolemia*
8. *Radiation*
9. *Smoking*

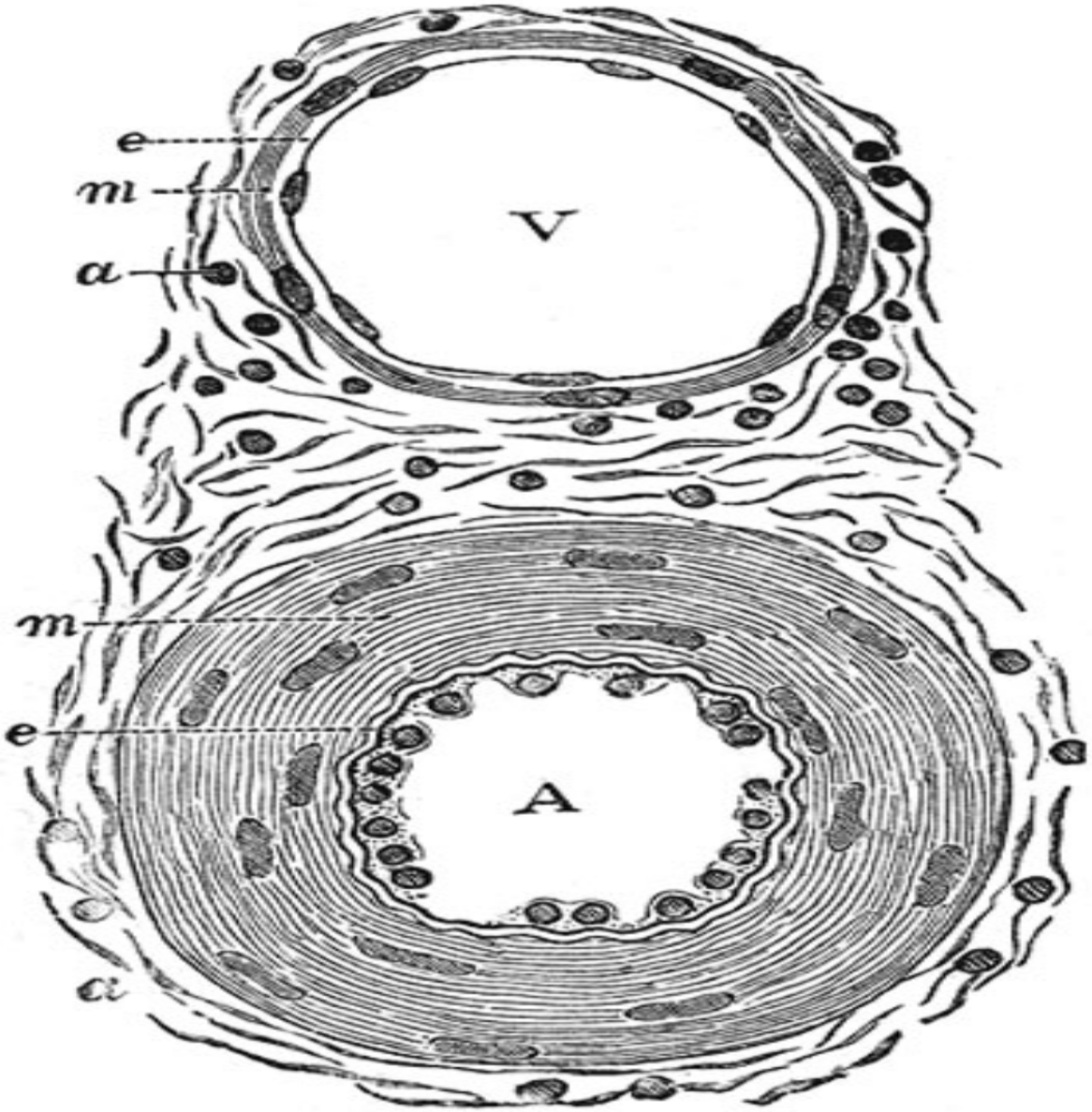


Stasis

- *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:



Artery (A) vs vien (V)



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)***

Hypercoagulability

A. Genetic (primary):

- mutations in the factor V gene and the prothrombin gene are the most common

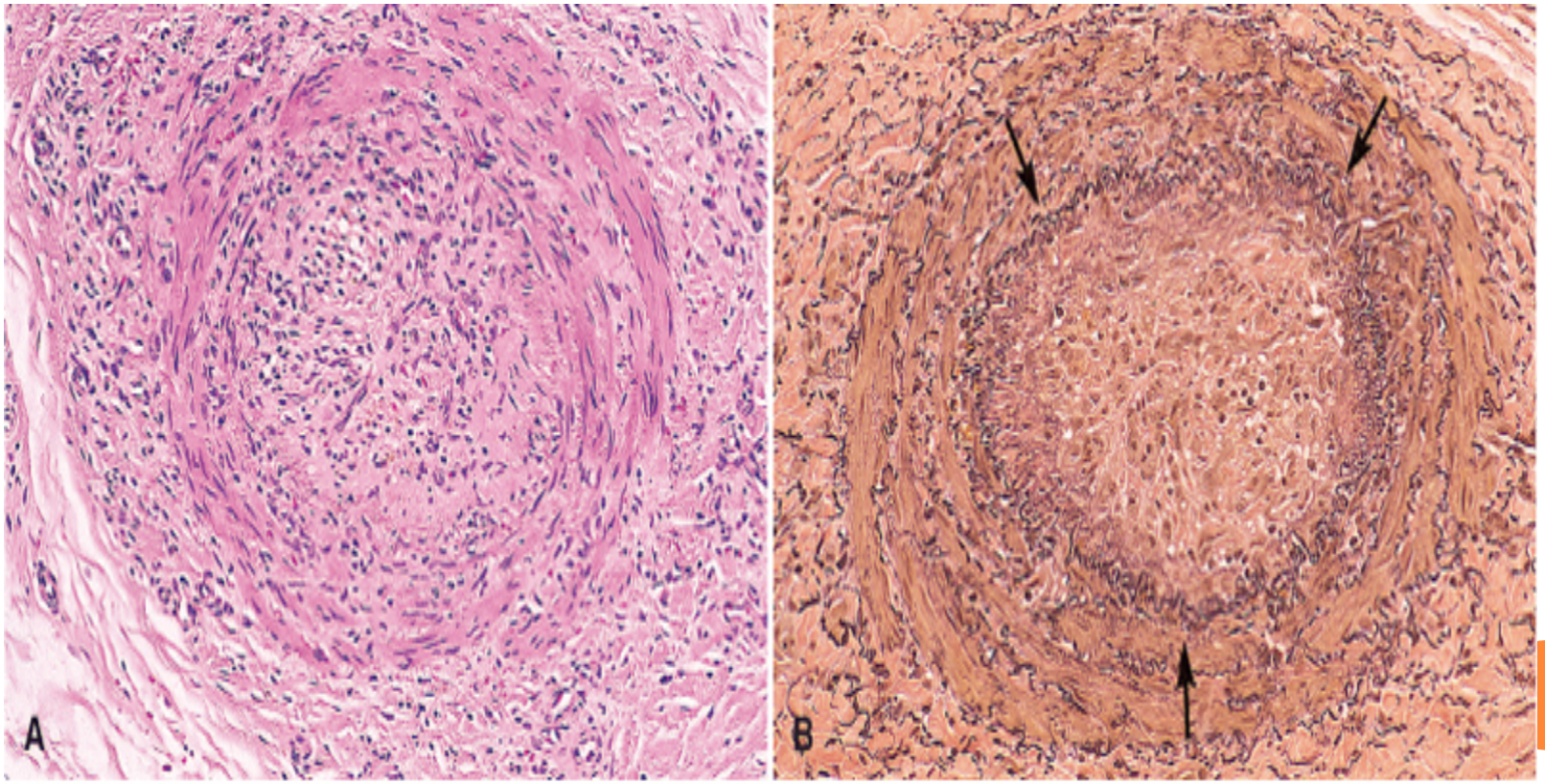
B. Acquired (secondary):

- multifactorial and is therefore 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**

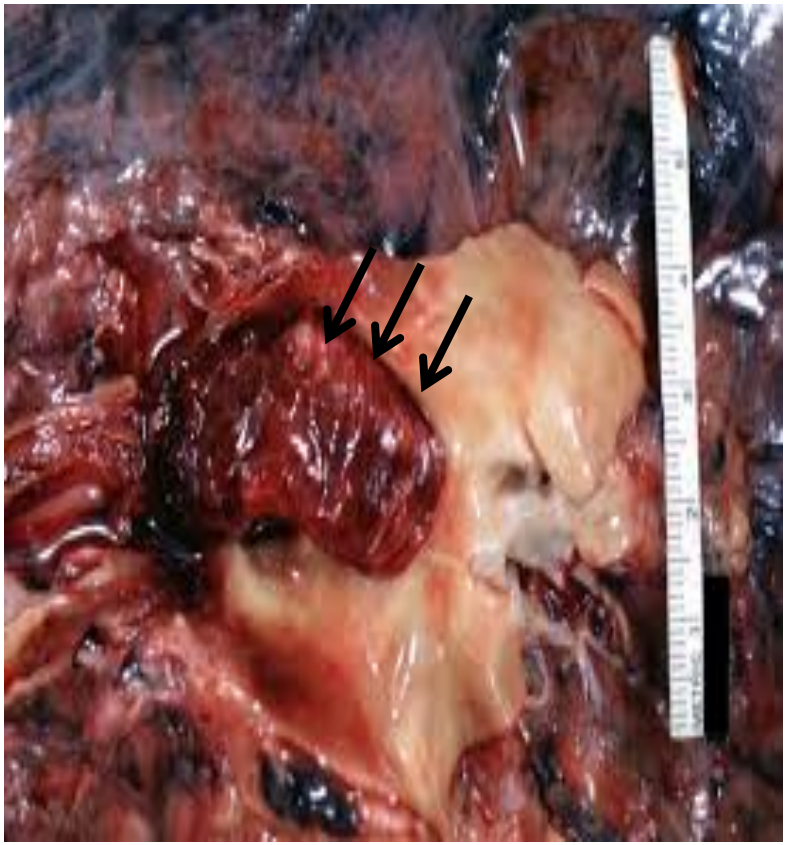
ARTERY WITH AN OLD THROMBUS. A, H&E-STAIN. B, STAIN FOR ELASTIC TISSUE.
THE ORIGINAL LUMEN IS DELINEATED BY THE INTERNAL ELASTIC LAMINA (*ARROWS*) AND IS TOTALLY FILLED WITH ORGANIZED THROMBUS, NOW PUNCTUATED BY A NUMBER OF RECANALIZED CHANNELS (WHITE SPACES).

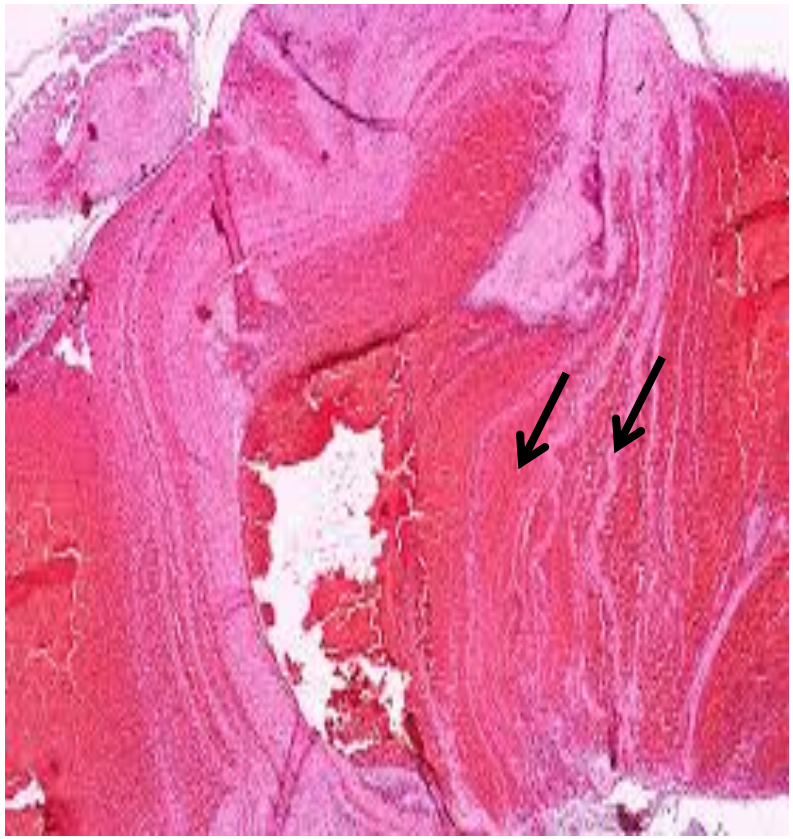


LINES OF ZAHN

- Thrombi can have grossly (and microscopically) apparent laminations called **lines of Zahn**; these represent pale platelet and fibrin layers alternating with darker erythrocyte-rich layers.
- Such lines are significant in that they represent thrombosis of **flowing** blood (can potentially distinguish antemortem thrombosis from postmortem clots)
- postmortem blood clots are bland non-laminated clots *(no lines of Zahn)*

LINES OF ZAHN





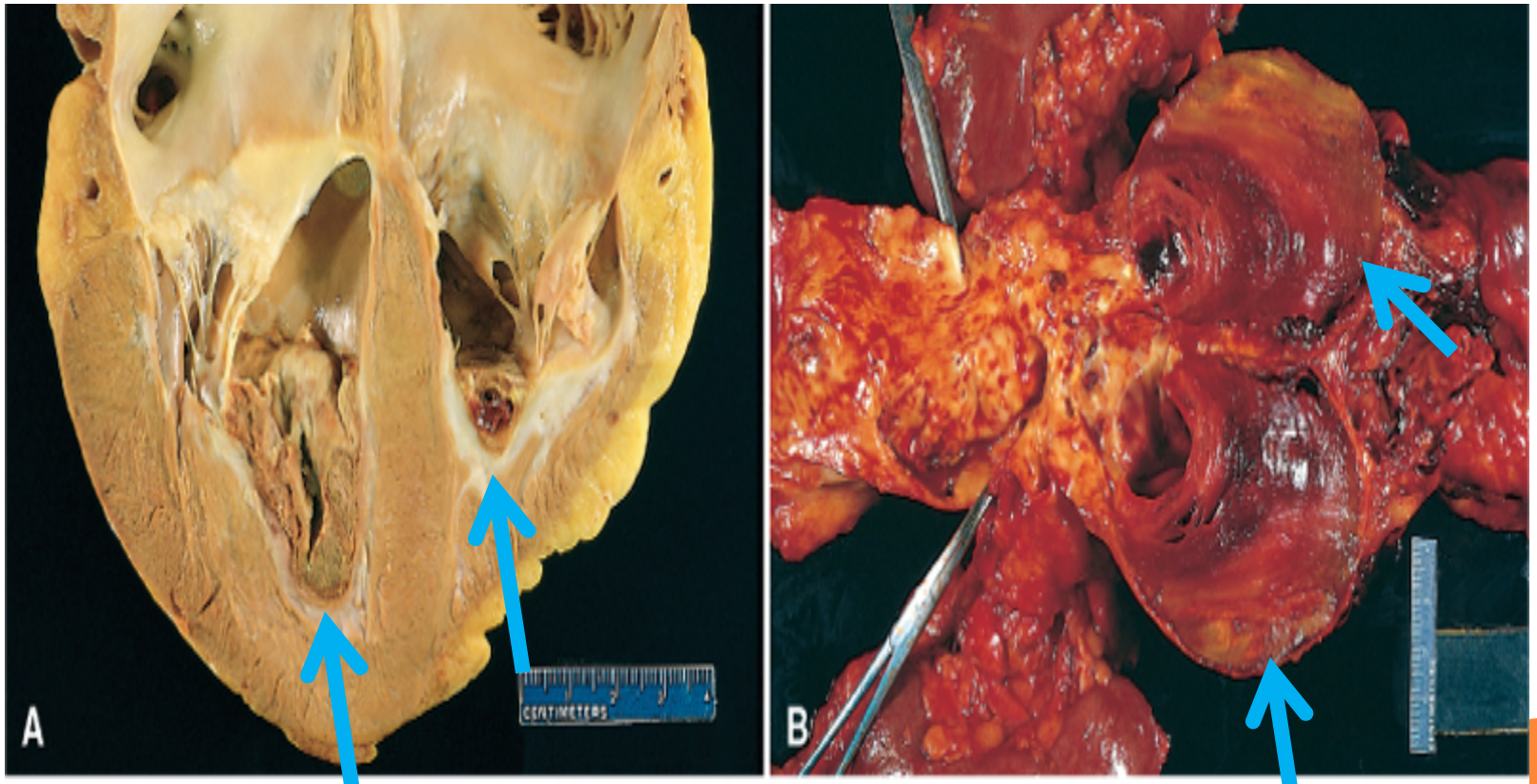
- **Mural thrombi**
- Thrombi occurring in heart chambers or in the aortic lumen.
- Causes include: Abnormal myocardial contraction (e.g. arrhythmias, dilated cardiomyopathy, or MI) or endomyocardial injury (caused by myocarditis, catheter trauma)
- **Vegetations**
- Thrombi on heart valves are called **vegetations:**

Types:

1- infectious (Bacterial or fungal blood-borne infections)→(e.g. infective endocarditis,).

2-Non-bacterial thrombotic endocarditis occur on sterile valves**.**

Mural thrombi



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)***

- **Venous thrombi**
- (veins of the legs) are most common

a. ***Superficial****: e.g. Saphenous veins.*

   - can cause local congestion, swelling, pain, and tenderness along the course of the involved vein, but they rarely embolize

a. ***Deep****: e.g. Popliteal, Femoral and iliac vein.*

- more serious because they may embolize
- can occur with stasis or in a variety of hypercoagulable states