Aneurysms & Dissections

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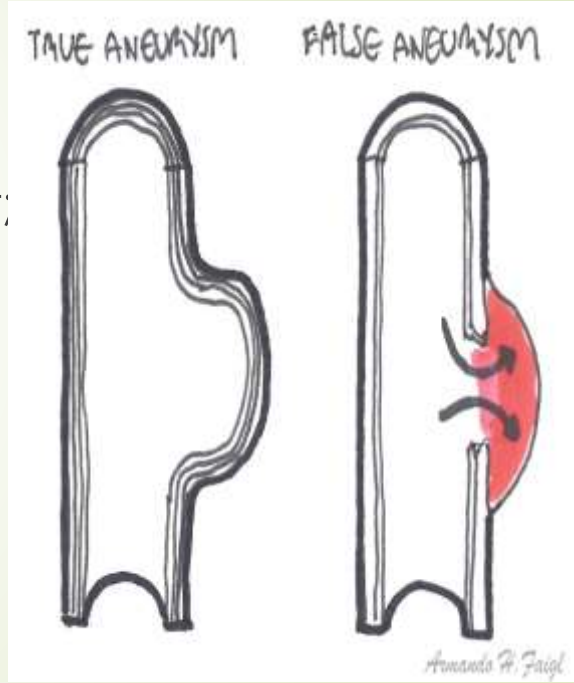
Aneurysms

- **A congenital or acquired dilations of blood vessels or the heart,** could be:

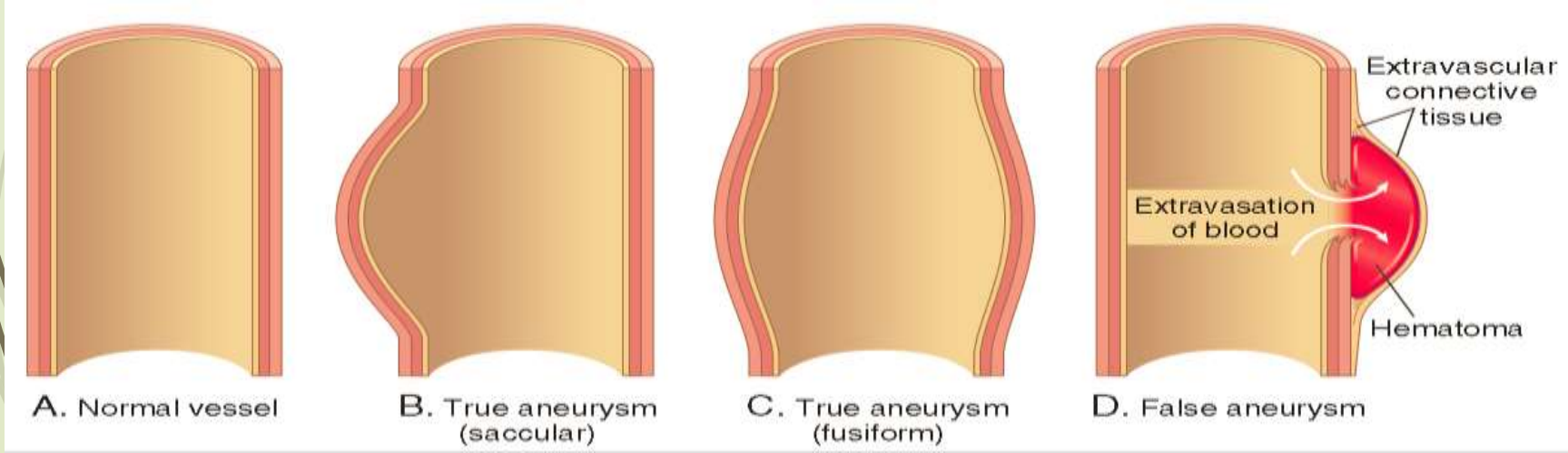
1. "**True**" : all three layers of the artery (intima, media, & adventitia) or the wall of the heart; e.g. atherosclerotic, congenital vascular aneurysms, ventricular aneurysms after MI

1. "**false**" : a wall defect leads to the formation of an extravascular hematoma that communicates with the intravascular space ("pulsating hematoma")



Aneurysms – Types by shape

- **Saccular aneurysms**: discrete outpouchings ranging (5-20 cm) in diameter, often with a contained thrombus.
- **Fusiform aneurysms:** circumferential dilations up to 20 cm in diameter, most commonly involve aortic arch, abdominal aorta, or iliac arteries.



Aneurysms – Pathogenesis

Alterations in SMCs or ECM → compromise **structural integrity** of the arterial media

1. *Inadequate or abnormal connective tissue synthesis:*

- *Marfan syndrome*: defective synthesis of scaffolding protein *fibrillin* → ↑TGF-β activity→ weak of elastic tissue → dilation in the aorta.

- *Ehlers- Danlos syndrome IV:* Defective type III collagen synthesis → weak vessels → aneurysm formation.

Aneurysms – Pathogenesis

2. *The balance of collagen degradation and synthesis is altered by inflammation and associated proteases..*

Increased matrix metalloprotease expression by macrophages in atherosclerotic plaque → aneurysm by degrading arterial ECM.

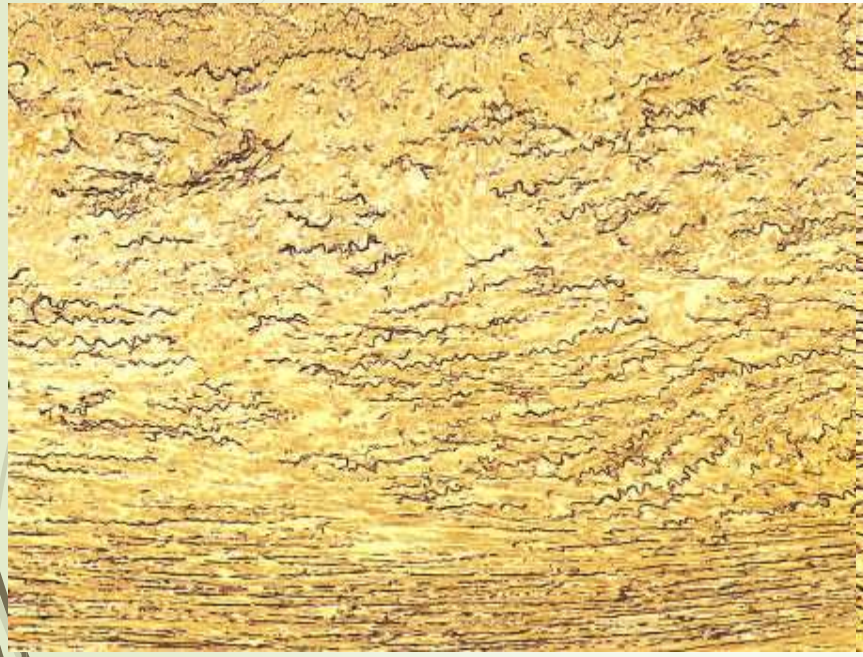
3. *The vascular wall is weakened through loss of smooth muscle cells or the synthesis of noncollagenous or nonelastic extracellular matrix.*

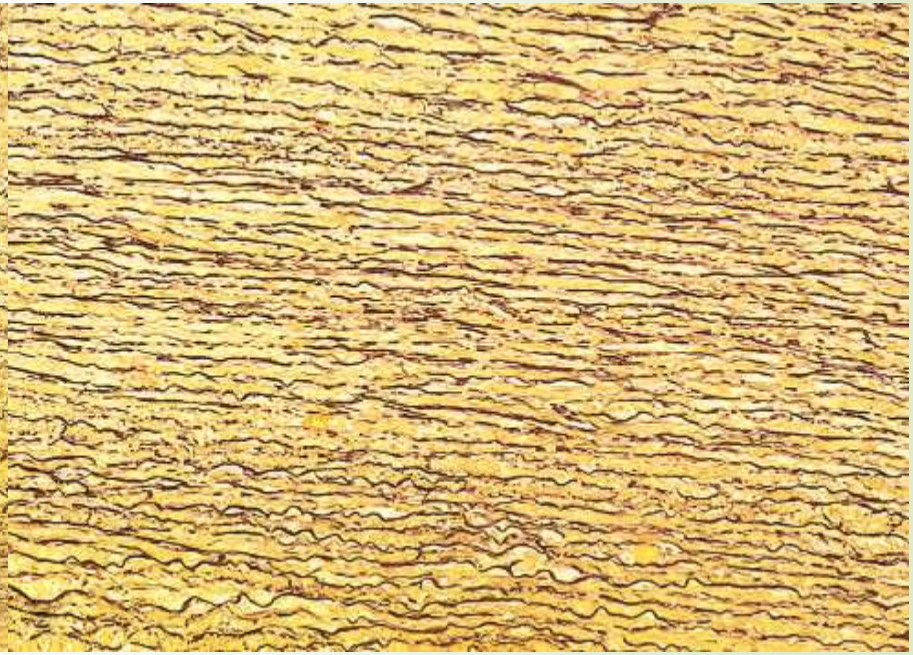
Aneurysms – Pathogenesis

- **Medial ischemia may lead to "degenerative changes" of the aorta**; Ischemia → smooth muscle cell loss → scarring and loss of elastic fibers → inadequate extracellular matrix synthesis → production of increasing amounts of amorphous ground substance (glycosaminoglycan).

- Histologically, these changes recognized as ***cystic medial degeneration***

Aneurysms – *cystic medial degeneration*





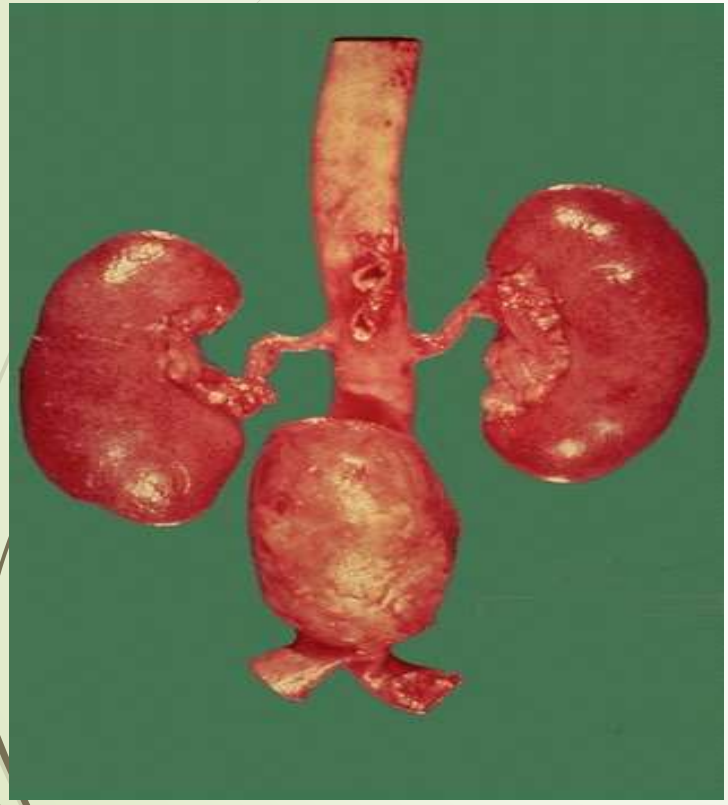
Abdominal Aortic Aneurysm (AAA)

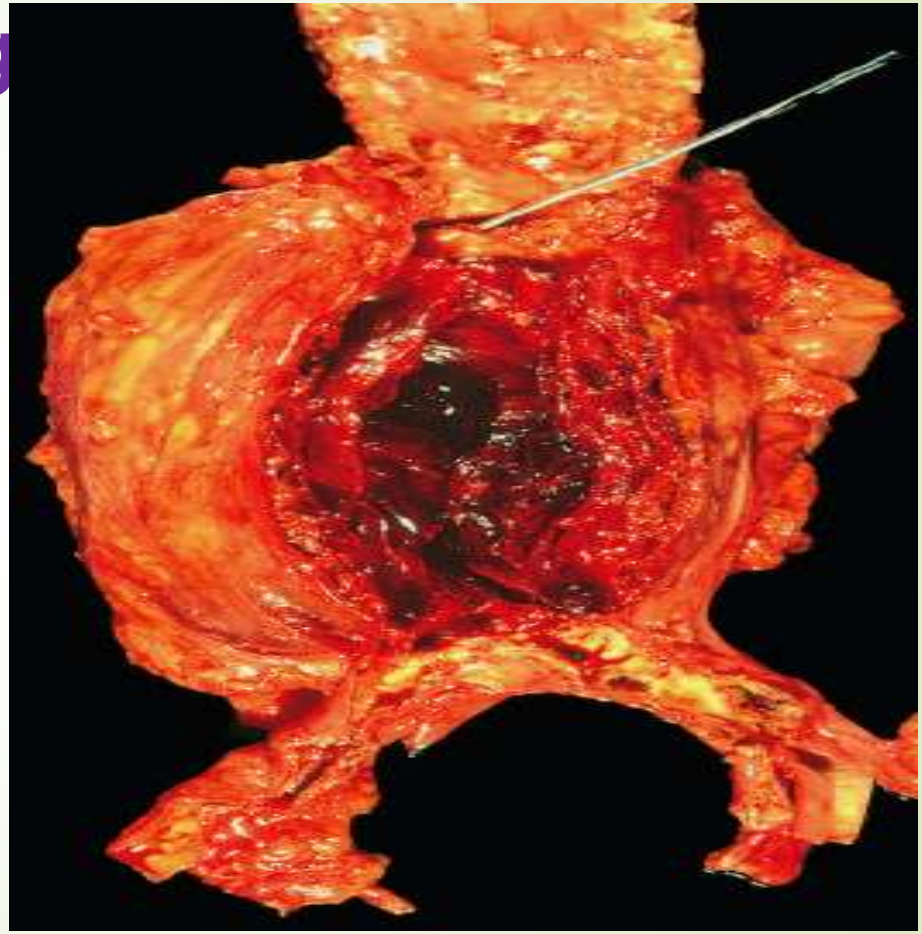
- Aneurysms occurring as a consequence of atherosclerosis form most commonly in abdominal aorta & common iliac arteries.
- More frequently in men & in smokers & rarely before 50.
- Atherosclerotic plaques → compromise the diffusion of nutrients & wastes between vascular lumen & arterial wall → deleterious effects on SMCs.
- Also atherosclerotic lesions → Inflammatory infiltrates → release proteolytic enzymes → ECM degradation
- Combination of these, the media undergoes degeneration & necrosis → arterial wall thinning → dilation

AAA- Morphology

- AAAs typically occur between the renal arteries & the aortic bifurcation; can be saccular or fusiform & up to 15 cm in diameter and 25 cm in length.
- In the vast majority extensive atherosclerosis is present, with thinning & focal destruction of the underlying media.
- The aneurysm sac usually contains bland, **laminated**, poorly organized mural thrombus …can fill much of the dilated segment.
- Not infrequently, AAAs are accompanied by smaller iliac artery aneurysms.

AAA- Morpholog





AAA- Clinical Manifestations

- Most AAA are asymptomatic & discovered incidentally as ***An abdominal mass*** (often palpably pulsating) that simulates a tumor.
- ***Obstruction*** of a vessel branching off the aorta.
- ***Embolism***.
- ***Impingement on adjacent structures.***
- ***Rupture*** into the peritoneal cavity or retroperitoneal tissues → massive, often fatal hemorrhage.

AAA- Clinical Consequences

- The risk for rupture is related to the size of AAAs. (4 cm in diameter or less almost never burst, 4-5 cm do so at a rate of 1% per year, 11% per year for AAAs 5-6 cm, & 25% per year for aneurysms >6 cm in diameter.

- Aneurysms 5 cm in diameter or larger are managed surgically.

- Timely intervention is critical; (mortality in elective procedures ~ 5%, whereas in emergency surgery after rupture ~ 50%)

Thoracic Aortic Aneurysm

Most commonly associated with **hypertension**, bicuspid aortic valves, & Marfan syndrome & Manifest with the following signs & symptoms:

- *Respiratory or feeding difficulties*
- *Persistent cough* from irritation of the recurrent laryngeal nerves.
- *Pain* caused by erosion of bone.
- *Cardiac disease* due to valvular insufficiency, narrowing of the coronary ostia, or aortic valvular incompetence.
- *Aortic dissection or rupture.*

Aortic Dissection

- Aortic dissection occurs when blood separates the laminar planes of the media to form a blood-filled channel within the aortic wall.
- Catastrophic if the dissection ruptures through the adventitia & hemorrhages into adjacent spaces.

- two groups of patients:

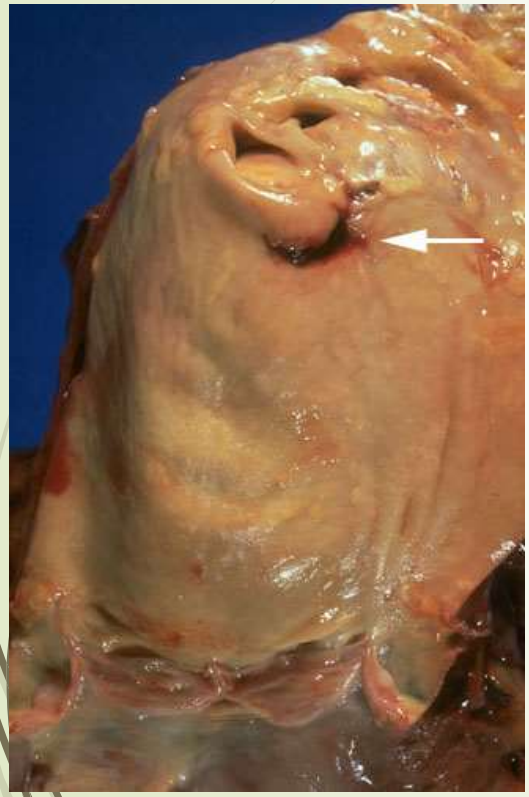
(1) Men **40-60 years** with antecedent hypertension (>90% of cases).

(2) **Younger** adults with systemic or localized abnormalities of connective tissue affecting the aorta (e.g., Marfan syndrome).

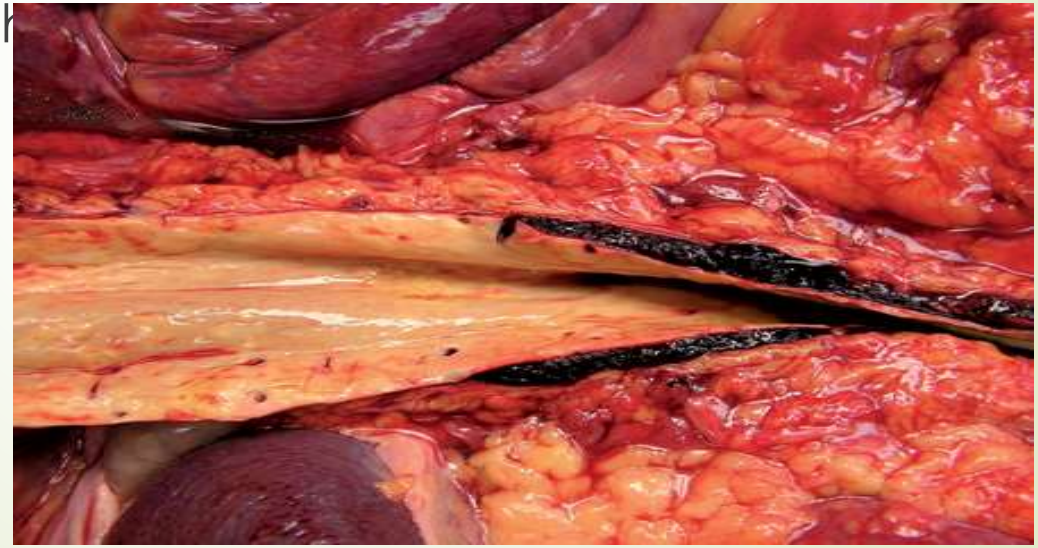
Aortic Dissection - Pathogenesis

- **Hypertension is the major risk factor for aortic dissection.**
- Narrowing of the vasa vasorum → diminished flow through vasa vasorum → degenerative changes in ECM & variable loss of medial SMCs.
- Abrupt , transient increase in blood pressure, as may occur with cocaine abuse, is also known to cause aortic dissection.
- The trigger for the intimal tear is not known in most cases. Nevertheless, once the tear has occurred, blood under systemic pressure dissects through the media along laminar planes.

Aortic Dissection - Morphology



mostly the intimal tear marking origin point is found in the ascending aorta within 10 cm of the valve. Dissection plane can extend retrograde toward the h



Aortic Dissection - Morphology

Dissection plan usually lies between the middle and outer thirds of the media.

Aortic Dissection – Clinical presentation

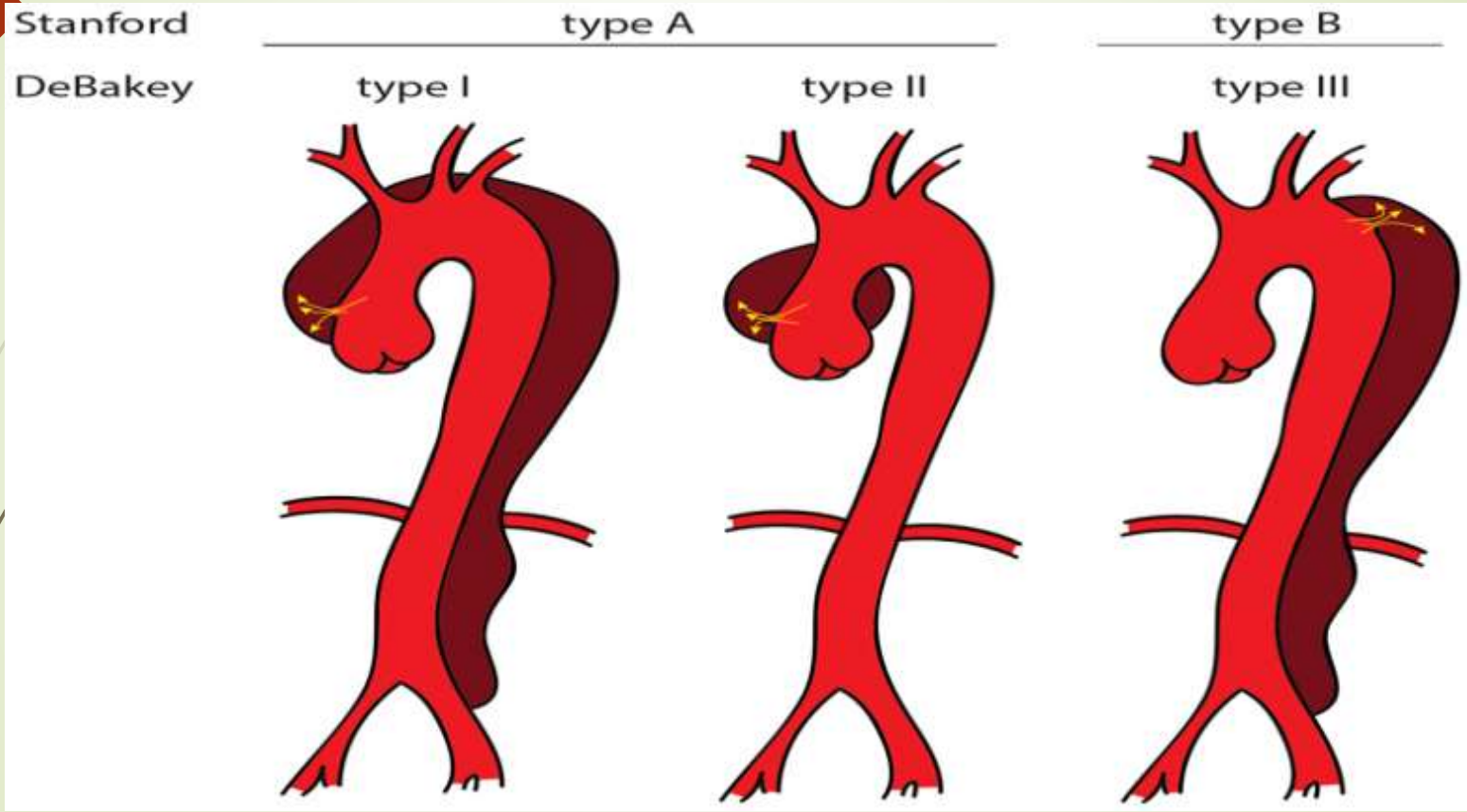
- The classic clinical symptom of aortic dissection is the **sudden onset of excruciating tearing or stabbing pain** in the anterior chest, radiating to the back between the scapulae, & moving downward as the dissection progresses.
- The most common cause of death is rupture of the dissection into the pericardial, pleural, or peritoneal cavity.
- Common clinical presentations stemming from cardiac involvement include tamponade, aortic insufficiency, and myocardial infarction.

Aortic Dissection – Clinical presentation

- Aortic dissections generally are classified into two types

Proximal lesions (type A dissections), involving the ascending aorta, with or without involvement of the descending aorta (DeBakey type I or II, respectively)… Needs rapid diagnosis & institution of intensive anti-hypertensive therapy coupled with surgery.. **Worse outcome** (most common)

Distal lesions (type B dissections), beginning beyond the subclavian artery (DeBakey type III) … Mostly can be managed conservatively.. Better outcome; 75% survival rate whether they are treated with surgery or with antihypertensive only.


Stanford
type A
type B
DeBakey
type I
type II
type III

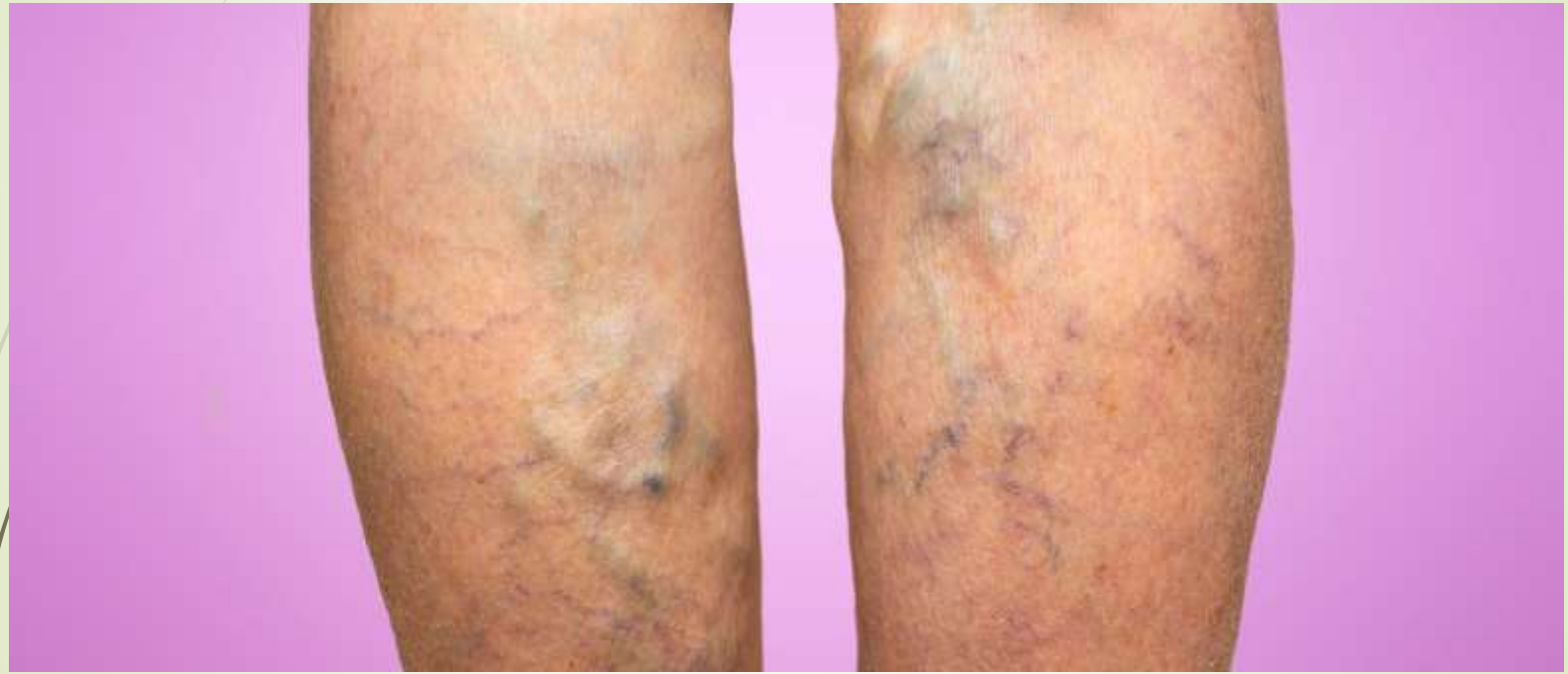
Veins and Lymphatics

90% of clinical venous disease caused by Varicose veins and phlebothrombosis/thrombophlebitis

Varicose veins

- **Abnormally dilated tortuous veins produced by chronically (1)increased intraluminal pressures & (2)weakened vessel wall support.**
- Venous valves incompetent → lower-extremity stasis, congestion, edema, pain, & thrombosis.
- **Locations:** typically, superficial veins of the upper & lower leg.
- **Risk factors:** Obesity, female sex, pregnancy, & familial tendency.
- **Clinical features:** persistent edema & secondary ischemic, skin changes, including stasis dermatitis and ulcerations.

Varicose veins– Clinical presentation



Thrombophlebitis & Phlebothrombosis

- **Two terms are largely interchangeable designations for venous *thrombosis* accompanied by *inflammation*.**
- 90% of cases are due to thrombosis of deep leg veins **(DVT)**
- **Risk factors for DVT: *Prolonged immobilization***, postoperative state, congestive heart failure, pregnancy, oral contraceptive use, malignancy, obesity, male sex, & age over 50 year.
- **Clinical manifestation:** few reliable signs or symptoms; distal edema, cyanosis, superficial vein dilation, heat, tenderness, redness, swelling, & pain. However, many DVTs are asymptomatic, and the absence of findings does not exclude their presence.

Thrombophlebitis & Phlebothrombosis

- **Pulmonary embolism is a common & serious clinical complication of DVT, resulting from fragmentation or detachment of the venous thrombus.**
- In many cases, the first manifestation of thrombophlebitis is a pulmonary embolus.
- Depending on the size and number of emboli, the outcome can range from resolution with no symptoms to death