ANEURYSMS AND DISSECTIONS

The doctor read everything in the slides

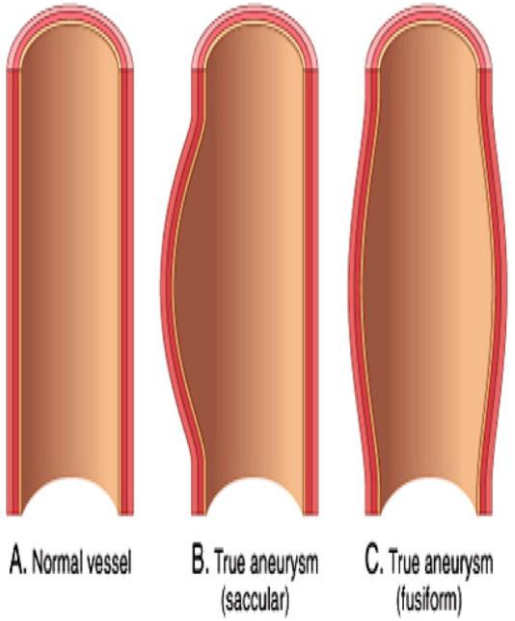
Aneurysm

- An *aneurysm* is a *localized abnormal dilation of* ***an artery*** *or the* ***heart***
- Types:

1-"true" aneurysm

→it involves **all three layers of the arterial wall** (intima, media, and adventitia) or the attenuated wall of the heart ,and is a result of weakness and the loss of the elastic recoil ability of the arterial wall.

→ e.g. **Atherosclerotic**, **syphilitic**, and **congenital** aneurysms, and **ventricular aneurysms** that follow transmural myocardial infarctions.



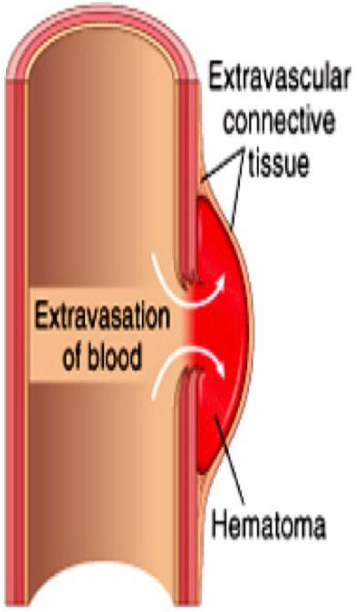
2- "false" aneurysm

→(also called ***pseudo-aneurysm***)

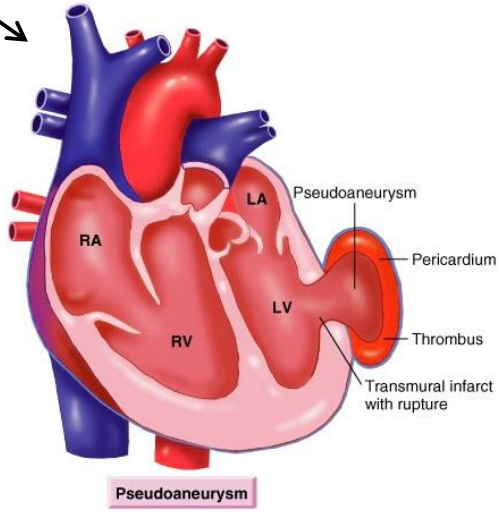
→is a **breach or a rupture involving all the three layers** (no weakness and dilation) in the vascular wall leading to an extravascular hematoma (contained by the **extravascular connective tissue**) that freely **communicates** with the intravascular space ("**pulsating hematoma**").

→E.g. ventricular ruptures after **MI** that are contained by a pericardial adhesion (**pericardium**)

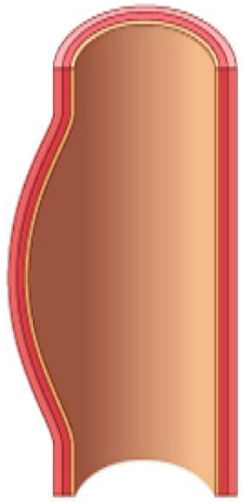
→E.g. a leak **at the junction** of a vascular graft with a natural artery *(at the junction/anastomotic site of a transplanted vessel e.g. Transportation of great saphenous vein to bypass the blockage of a coronary artery)* .



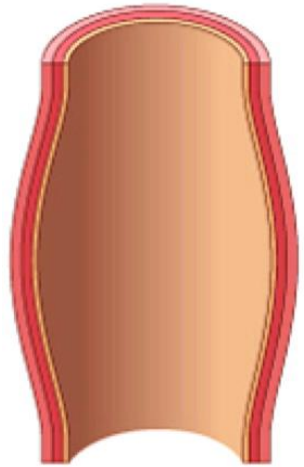
D. False aneurysm



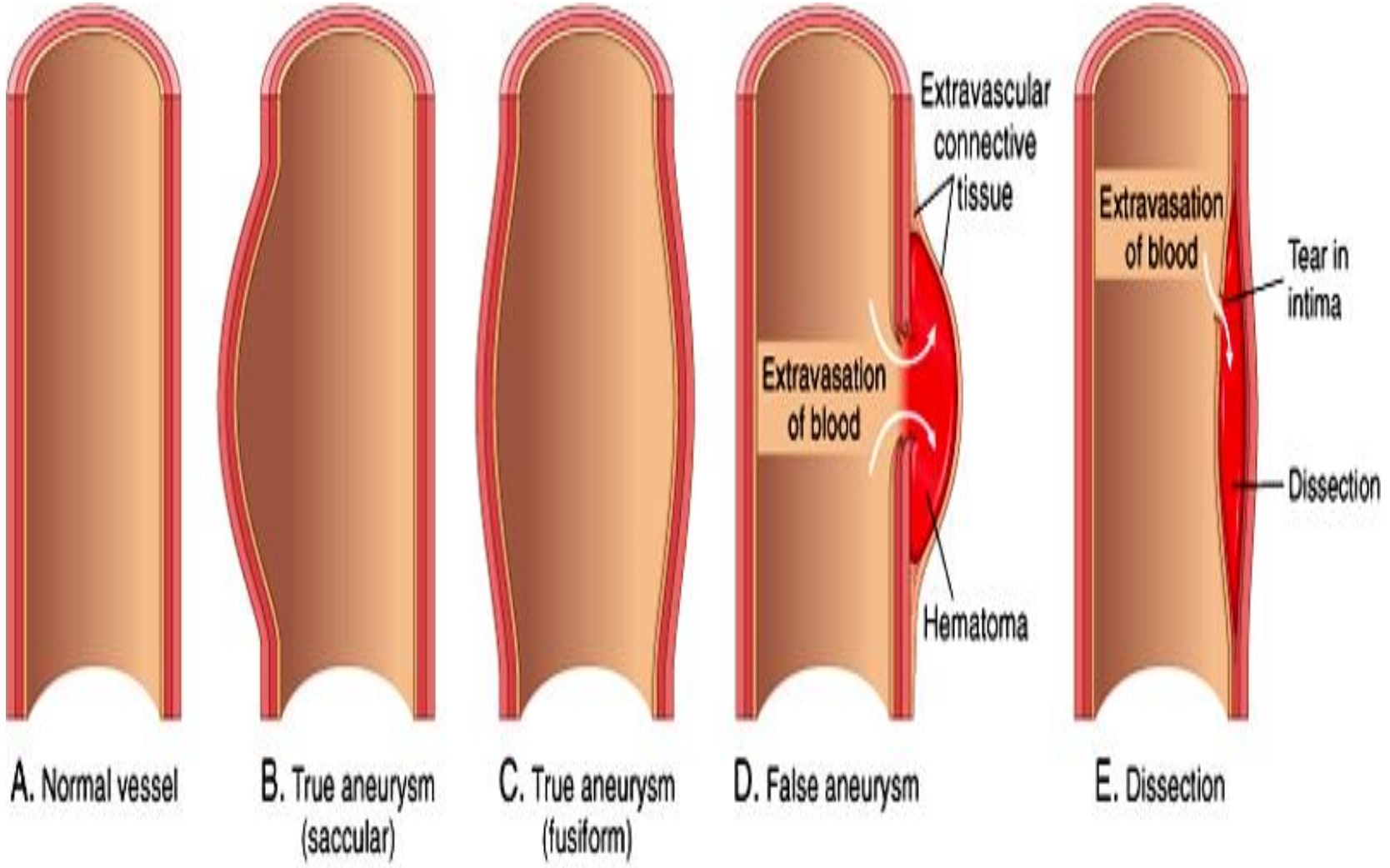
- aneurysms are classified by macroscopic shape and size (aspects of shape and size are ***not specific for any disease or clinical manifestations***) :
- ***Saccular* aneurysms**
- spherical outpouchings (involving only **one side** of the vessel wall, and often contain thrombi).
- ***Fusiform* aneurysms**
- diffuse, **circumferential** dilation of a long vascular segment;
- they vary in diameter and length and can involve extensive portions of the aortic arch, abdominal aorta, or even the iliacs.



B. True aneurysm (saccular)



C. True aneurysm (fusiform)



Aortic aneurysms

- *One of the most commonly affected arteries.*
- *The two most important causes are:*

1- **<u>atherosclerosis :</u> the most common cause**

→ Although it resides in the intima causes thinning and weakening of the media by: The intimal plaques **compress** the underlying media and also **compromise nutrient and waste diffusion** from the vascular lumen into the arterial wall. The media consequently undergoes **degeneration and necrosis**, thus allowing the dilation of the vessel

<u>2- cystic medial degeneration of the arterial media.</u>

- Other causes include trauma, congenital defects (e.g., *berry* aneurysms), infections (*mycotic* aneurysms), systemic diseases, such as vasculitis.

Extra :p : from robbins fibrosis (replacing distensible elastic tissue), inadequate ECM synthesis, and accumulation of increasing amounts of amorphous proteoglycans in the media. Histologically, these changes are collectively called **cystic medial degeneration**, although no true cysts are formed. Such changes are nonspecific; they can occur whenever ECM synthesis is defective, including in genetic disorders such as Marfan syndrome and metabolic syndrome such as scurvy.
So its not a cause and just a histological finding?
** and the 2nd most common cause is hypertension
But of course stick with what the doctor says :p

Abdominal Aortic Aneurysm

- ***Atherosclerotic aneurysms occur most frequently in the abdominal aorta* (*abdominal aortic aneurysm,* often abbreviated AAA)**
- the common iliac arteries, the arch, and descending parts of the thoracic aorta can also be involved
- **Pathogenesis**
- AAA occurs more frequently in men and rarely develops before age 50.
- **Atherosclerosis** is a major cause of AAA ---> affects elders
- other contributors include: hereditary defects in structural components of the aorta, that will result in cystic medial degeneration (e.g., defective fibrillin production in Marfan disease affects elastic tissue synthesis)
- Can also be a result of altered balance of collagen degradation and synthesis mediated by local inflammatory infiltrates and the destructive proteolytic enzymes

Treatment:
It depends on the size of the aneurysm:
-if it was a small one (less than 5cm), we can use conservative management (to avoid the risk of rapture), then an elective(اختيارية) surgery is offered
-if it was a large aneurysm, surgery must be done faster, because the risk of rapture is very high.

Morphology

- Usually positioned below the renal arteries and above the bifurcation of the aorta
- AAA can be **saccular or fusiform**
- as large as 15 cm in diameter *(normally 2cm)*, and as long as 25 cm.
- Microscopically: atherosclerosis with destruction and thinning of the underlying aortic media
- the aneurysm frequently contains a laminated mural thrombus

The clinical consequences of AAA include:

- **Rupture** →massive potentially fatal hemorrhage , might result in **hypovolumic shock**(large amounts of the blood goes to the abdominal cavity)

  - risk of rupture is directly related to the size of the aneurysm (of 5 cm or more the risk of rapture is high) *we can know this by using radiographical imaging like MRI and CT scan.*

  - operative mortality for unruptured aneurysms 5%, emergency surgery after <u>rupture the mortality rate is more than 50%</u>

- **Obstruction** ***commonly caused by a thrombus secondary to stasis*** of downstream vessel → tissue **ischemic** injury((e.g. iliac (leg), renal (kidney), mesenteric (gastrointestinal [GI] tract), or vertebral (spinal cord) arteries))
- **Embolism** →from atheroma or mural thrombus most commonly will go to the lower limb (caused by stasis)
- **Impingement** and **compression** on an adjacent structure (e.g.ureter or vertebrae)
- Presentation as an **abdominal mass** (often palpably pulsating) that simulates a tumor

Clinical scenario :
An elder (probably, but might be a young person if he has a congenital problem like marfan disease) will come to the ER due central abdominal pain, he might come unconscious because of hypovulomic shock, low blood pressure.rearly , neurological manifestation might occur due to the compression of the adjacent nerves or vertebras by abdominal mass.
After physical examination, a pulsating mass might be palpable which represents the dilated aorta .

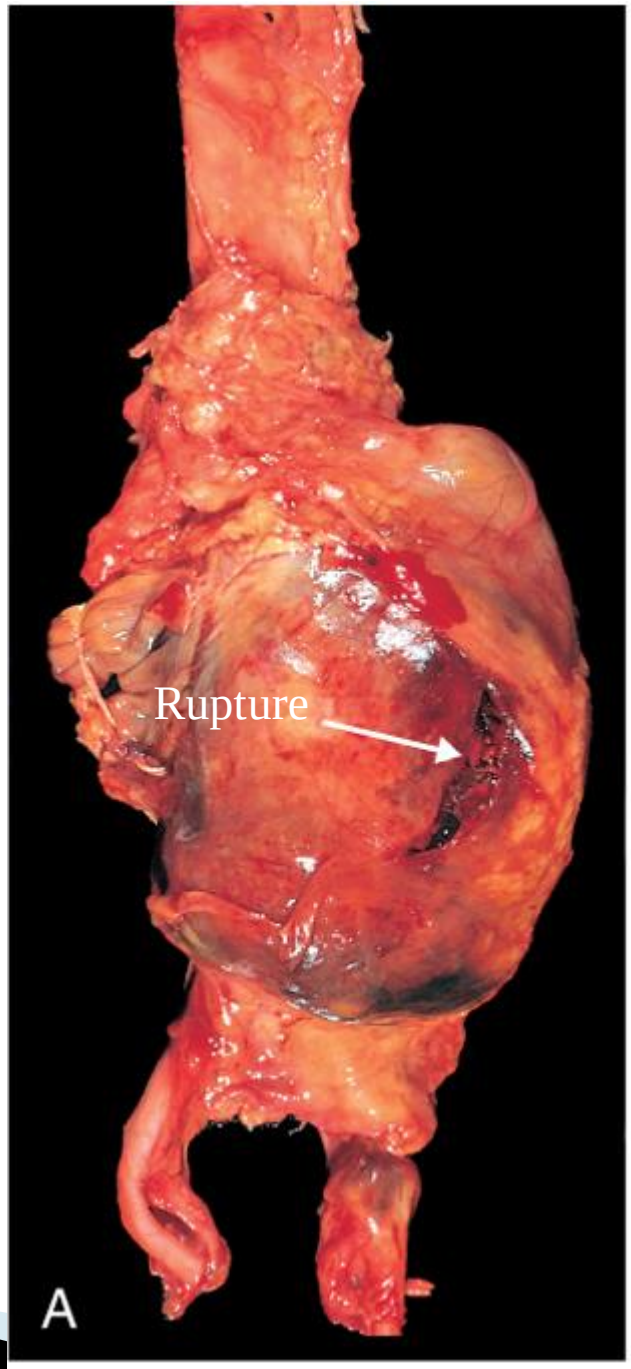
Abdominal aortic aneurysm

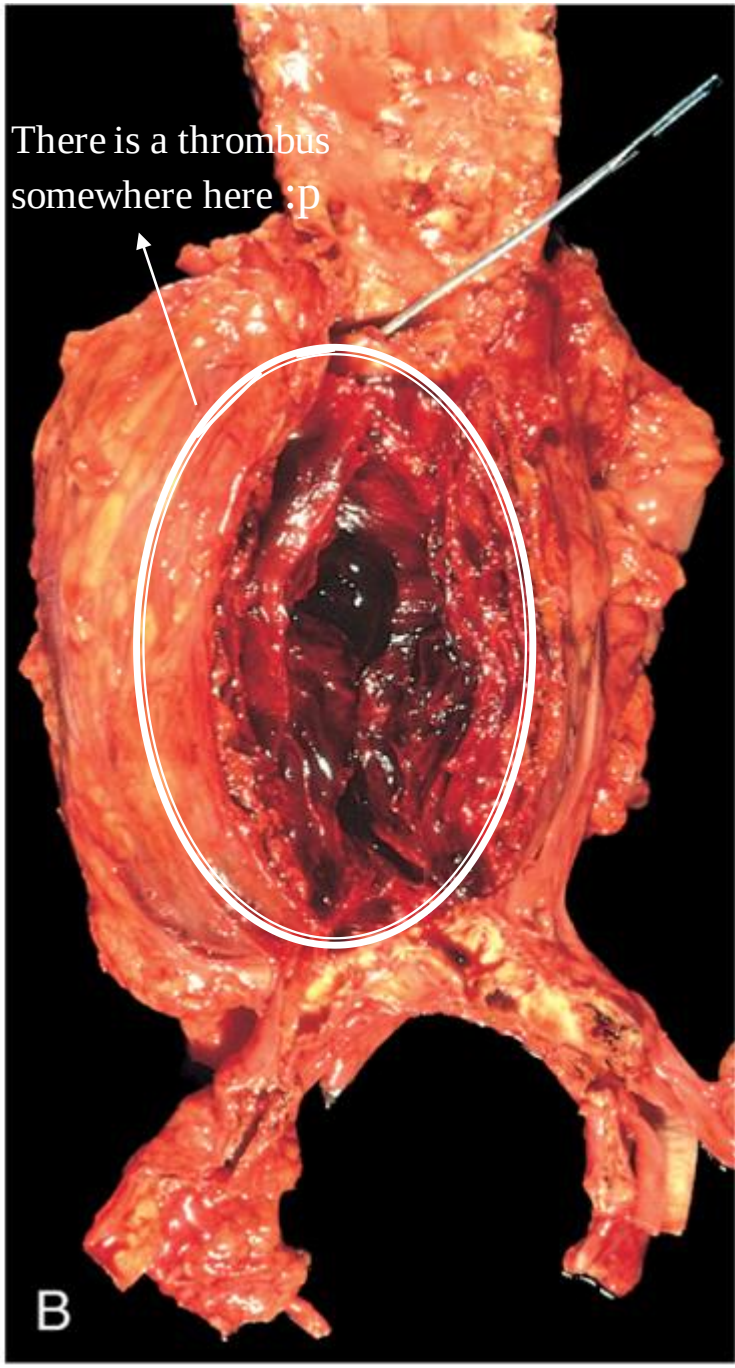
In microscopic examination:

-We can know the underlying cause (in atherosclerosis for example we'll see a fibrous cap and a lipid core ..etc , a complication like thrombus forming might be present)

-the media will be very thin.

Probably fusiform in shape , but it doesn't matter to know the shape





Mycotic aneurysms

Remember:
A thrombus might lead to a superimposed infection (like a mycotic infection)

- Infection of a major artery ***(The organism is in the wall)*** that weakens its wall is called a *mycotic aneurysm*
- possible complications: thrombosis and rupture.
- can originate from:

(1) embolization of a septic/infected thrombus, usually as a complication of infective endocarditis

(2) extension of an adjacent suppurative process;

(3) circulating organisms directly infecting the arterial wall

- (the doctor didn't read this paragraph)**Mycotic AAAs** are atherosclerotic lesions infected by lodging of circulating microorganisms in the wall, e.g. bacteremia from a primary *Salmonella* gastroenteritis. suppuration further destroys the media, potentiating rapid dilation and rupture

Syphilitic Aneurysm

- *Caused by* The spirochetes *T. pallidum*
- A vanishingly rare complication in the U.S. and West thanks to early recognition and treatment of syphilis
- Tertiary stage of syphilis can cause *obliterative endarteritis*(vasculitis) of the involve small vessels in any part of the body, including the **vasa vasorum of the aorta** (that supply the media)
- this results in **ischemic <u>medial</u> injury**, leading to aneurysmal dilation of the aorta (aortic arch in specific) and aortic annulus(the venriclulo-aortic junction site), and eventually valvular insufficiency.

According to the doc. In the tertiary stage there is <u>no active organism</u> , and an inflammatory process causes obliterative endarteritis

Morphology of Syphilitic Aneurysm

- **Obliterative endarteritis:**

Became Extremely rare because of the antibiotics

- luminal narrowing and obliteration, scarring of the vessel wall, and a dense surrounding rim of lymphocytes and plasma cells that may extend into the media
- With destruction of the media, the aorta loses its elastic recoil and may become dilated, producing an aneurysm.
- valvular insufficiency and massive volume overload lead to hypertrophy of the left ventricle.
- The greatly enlarged heart when seen in X-ray is called **"cor bovinum" (cow's heart).**

Arterial *dissection*

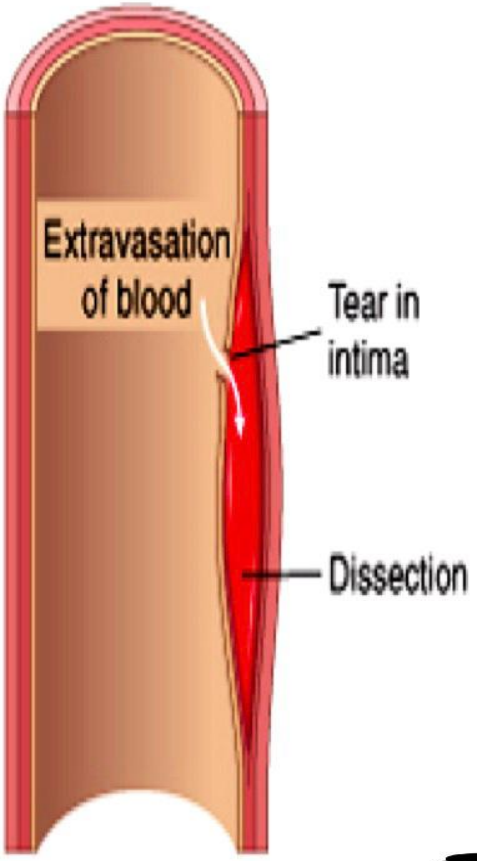
- arises when blood enters the wall of the artery *through a tear in intima*, as a hematoma dissecting **between its layers(inside the media).**
- Dissections are often but not always aneurysmal.
- Both true and false aneurysms as well as dissections can rupture, often with catastrophic consequences

Aortic dissection

- is a catastrophic event whereby blood dissects apart the media to form a blood-filled channel within the aortic wall
- Complications are :

  - massive hemorrhage --> inside the wall(the doc said) :/ :/ :/

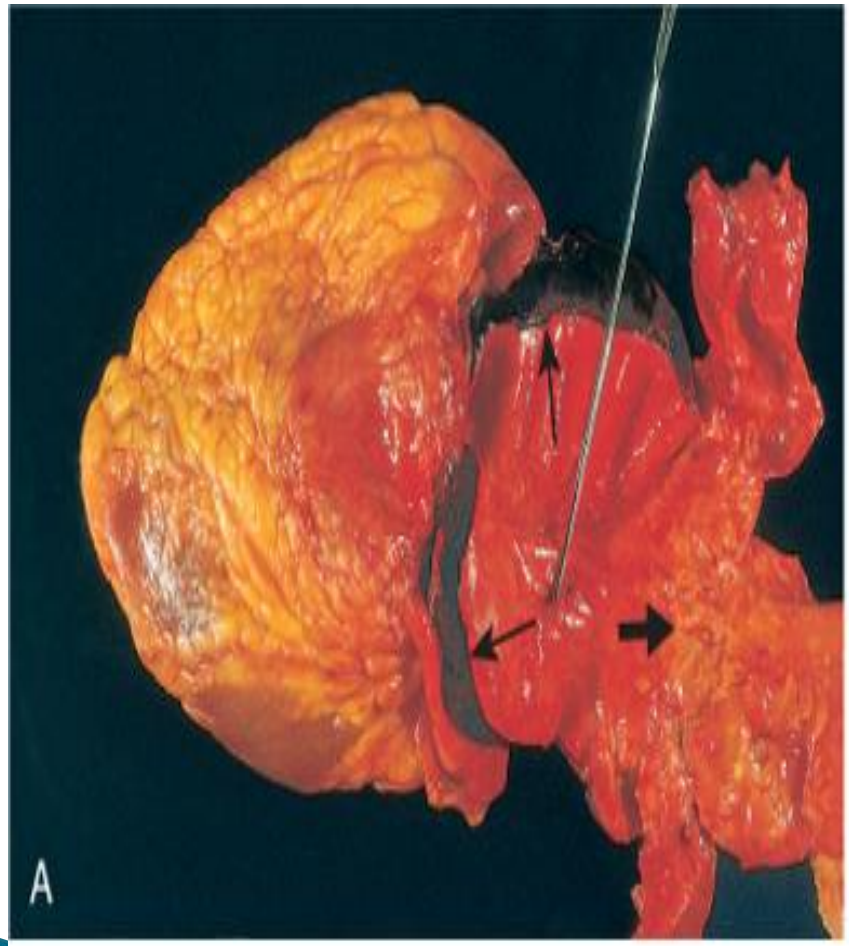
  - cardiac tamponade *if the dissection happens inside the wall of the heart* (hemorrhage into the pericardial sac).

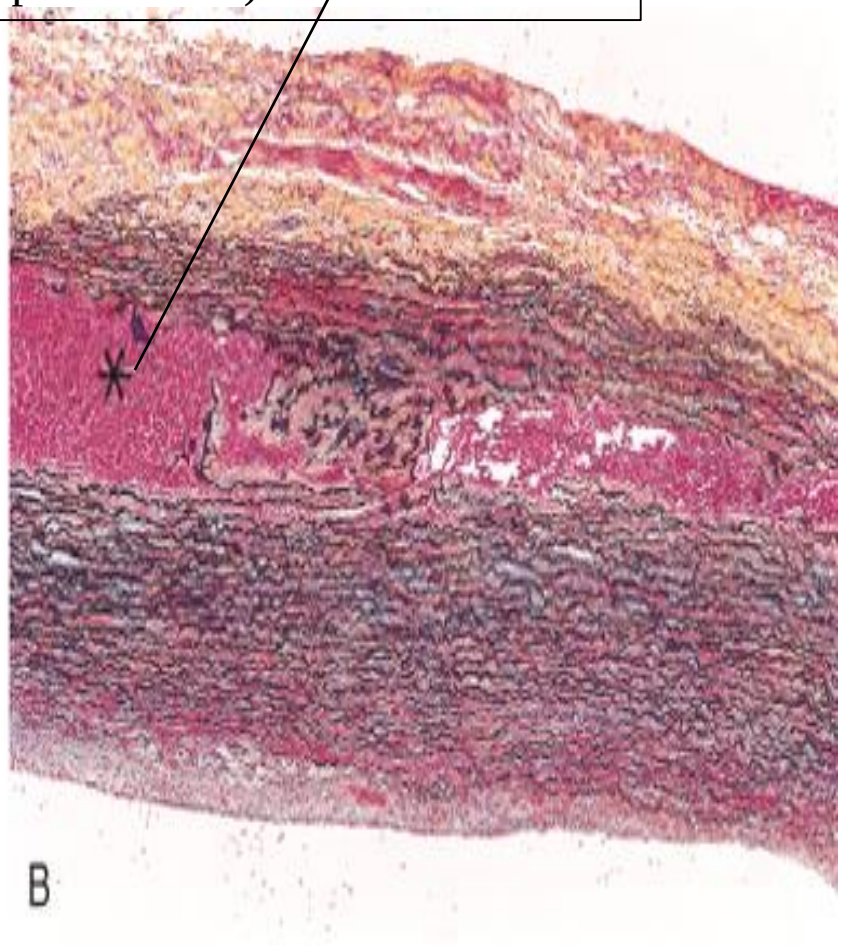


E. Dissection

Aortic dissection

Accumulation of blood in the media (we know the media by staining the elastic fibers that appear black in color using that specific stain)





Pathogenesis of Aortic dissection

- 1- Hypertension is ***the*** **major risk factor**
- pressure-related mechanical injury and/or ischemic injury.
- 2- inherited or acquired connective tissue disorders causing abnormal vascular ECM
- (e.g., Marfan syndrome, Ehlers-Danlos syndrome, vitamin C deficiency, copper metabolic defects)

Marfan syndrome

- The most common cause among the inherited or acquired connective tissue disorders assosiated with Aortic dissection
- it is an autosomal dominant disease of **fibrillin**, an ECM scaffolding protein **required for normal elastic tissue synthesis**.
- Patients have skeletal abnormalities (elongated axial bones) and ocular findings (lens subluxation) in addition to the cardiovascular manifestations (have a higher risk of developing aortic aneurysms and dissections).

Aortic dissections are generally classified (according to the initial location of dissection) into two types:

Because it affects the major branches of the arch of the aorta (will affect the blood supply of the brain)

- 1- The more common (and dangerous) *proximal* lesions (called *type A dissections*), involving either the **ascending aorta** only(type II DeBakey) or both **the ascending** and descending aorta (type I DeBakey)

- *2- Distal lesions* ***not involving the ascending part*** and usually beginning distal to the subclavian artery (called *type B dissections* or DeBakey type III

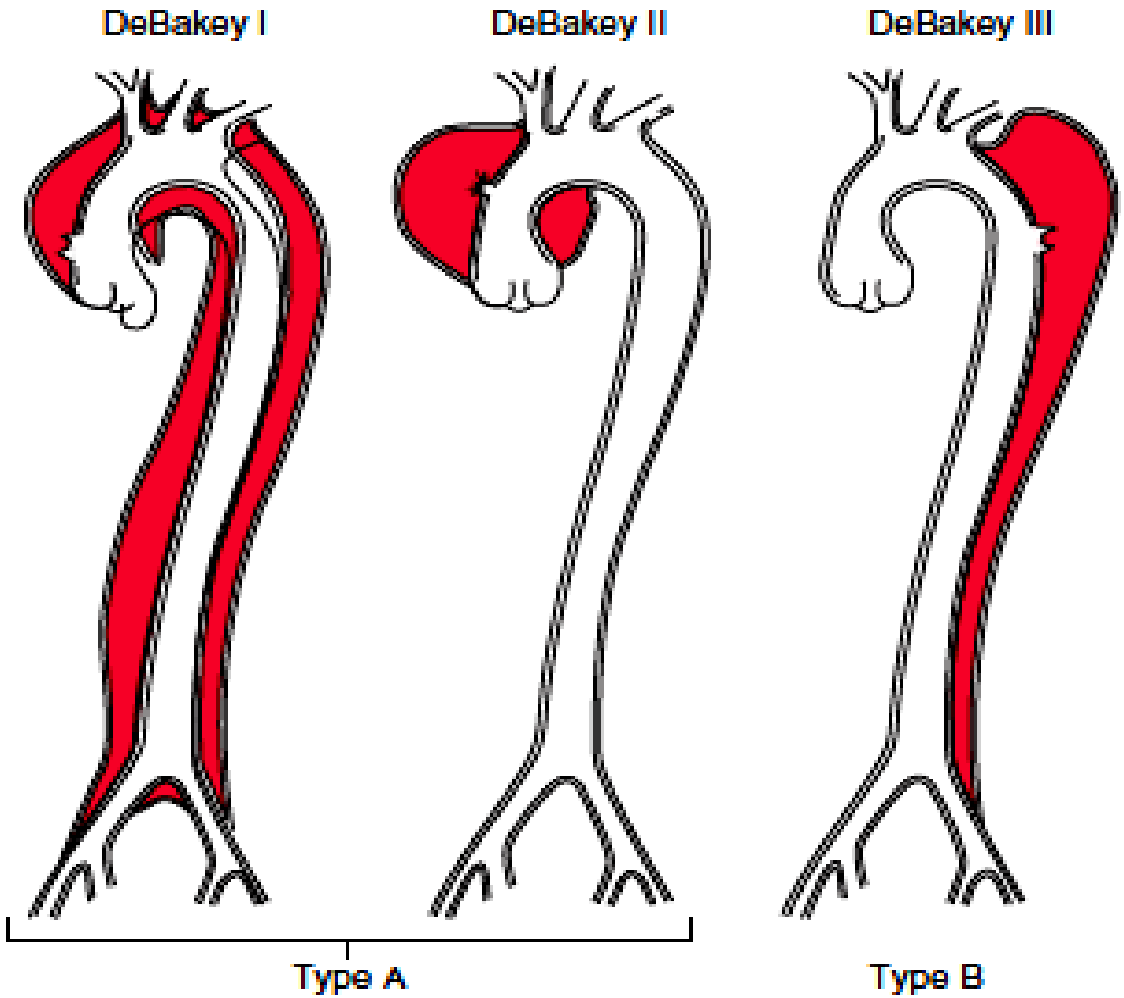


Figure 9–21 Classification of dissections. Type A (proximal) involves the ascending aorta, either as part of a more extensive dissection (DeBakey type I), or in isolation (DeBakey type II). Type B (distal, or DeBakey type III) dissections arise after the takeoff of the great vessels.

Clinical course

- Previously, aortic dissection was typically fatal, but the prognosis has markedly improved.
- Rapid diagnosis and institution of intensive antihypertensive therapy, coupled with surgical procedures involving plication of the aorta permits survival of 65% to 75% of patients