Pathological Thrombosis

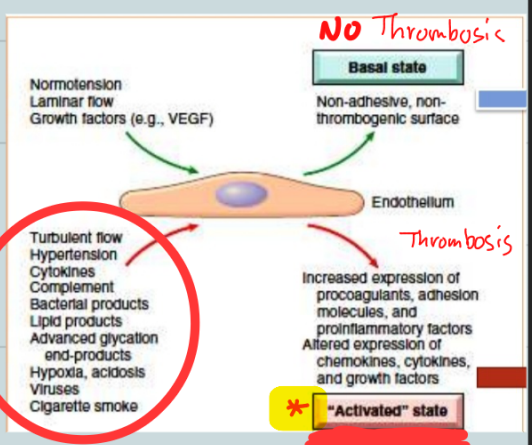
بسم الله الذي لا يضر مع اسمه
شيء في الأرض ولا في السماء
وهو السميع العليم ♥

whenever unnecessary blood clotting is activated

is caused by the presence of at least one of 3 factors (together called **Virchow's triad**)

1. Endothelial Injury (Heart, Arteries)

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



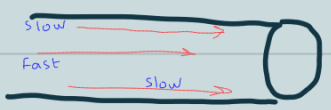
endothelial cell injury
& exposure of subendothelial collagen
↓
Adherence of platelets
↓
Release of Tissue factor
↓
Coagulation event ...

- Injury results in a healing response
- Pathologic effect of vascular healing: Excessive thickening of the intima → luminal stenosis & blockage of vascular flow

2. Stasis / Turbulence (abnormal blood flow)

normal Blood Flow

Laminar Flow



- Stasis is a major factor in venous thrombi

Stasis & Turbulence cause the following ↓

* **Disrupt** normal blood **flow**

* **prevent dilution** of activated clotting factors by fresh flowing blood

توعّر

* **Retard** the inflow of **clotting factor inhibitors**

* Promote **endothelial cell injury**

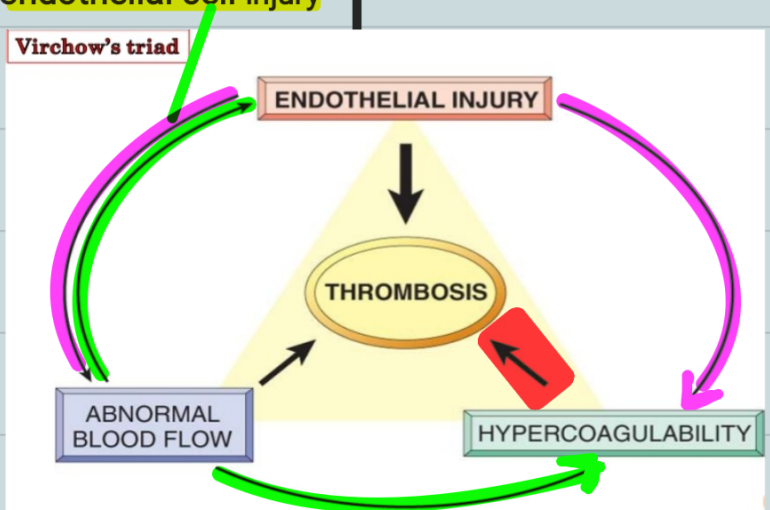
3. Blood Hypercoagulability

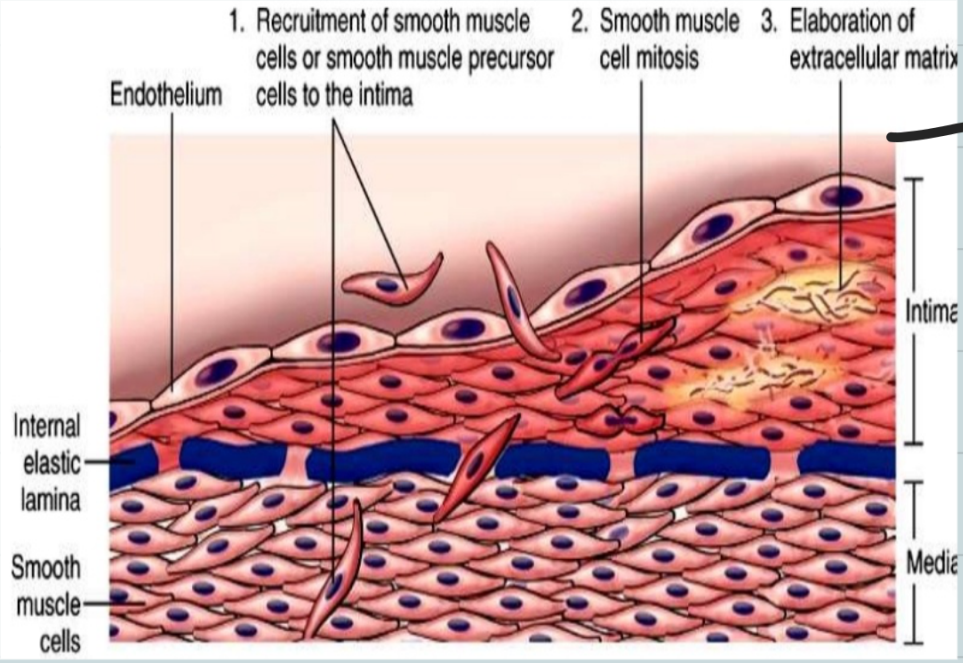
Genitic (primary)

mutations in **factor V gene** and **prothrombin gene**

Acquired (Secondary)

More frequent multifactorial & more complicated





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

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 (Aquired)

Immobilization
MI
AF
surgery
fractures
burns
Cancer
Prosthetic cardiac valves

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.

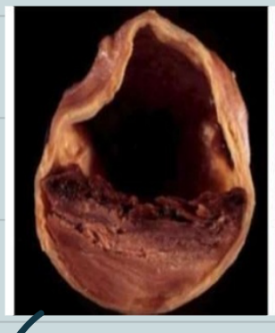
- The propagating portion of a thrombus is poorly attached → fragmentation and embolus formation

Lines of Zahn

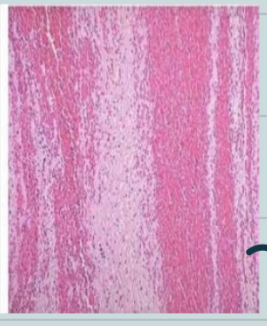
→ lamination (تصفيح)

pale
platelet & fibrin layers

darker
erythrocyte-rich layers



grossly



microscopically

No Lines of Zahn in Postmortem blood clots

Non laminated clots

- **Mural thrombi** → in Heart chambers or Aortic lumen

- **Cardiac Vegetations** → 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

Fates of a Thrombus

Resolution

Dissolution
↓
Thrombi are removed by **fibrinolytic** activity (only in recent thrombi)

propagation

accumulate additional platelets and fibrin, eventually causing **vessel obstruction**

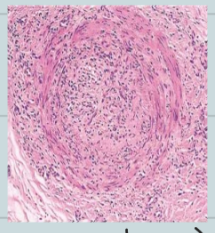
Thromboembolism

Embolization
↓
Thrombi dislodge or **fragment** and are **transported** elsewhere in the **vasculature**

organization 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.



organized Arterial Thrombus

Mycotic aneurysm

) superimposed infection

لا إله إلا أنت سبحانك إني كنت من الظالمين

Embolism

An embolus is a detached intravascular **solid**, **liquid**, or **gaseous** mass that is carried by the blood to a site distant from its point of origin

- Emboli result in partial or complete vascular occlusion.
- consequences of embolism: ischemic necrosis (infarction) of downstream tissue

Types (composition wise)

- Thromboembolism ~1.9a
- Fat embolism
- Air / Nitrogen embdism
- Amniotic fluid embolism

1 Thromboembolism

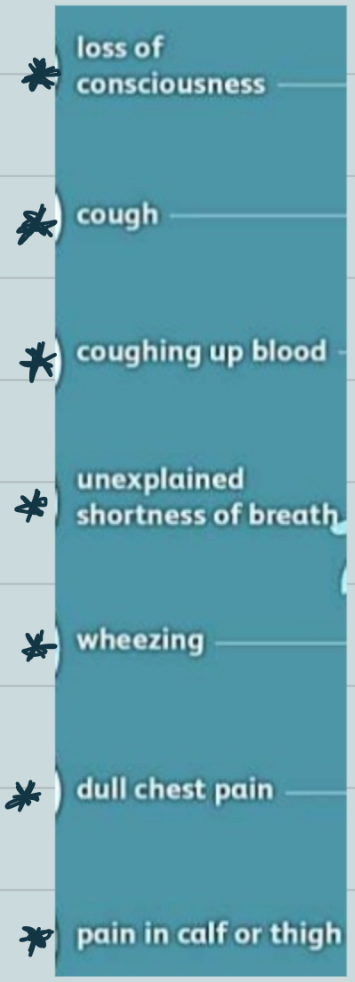
Types (site of origin wise)

- Venous
  - origin: Lower Limbs ~95 Deep veins Thrombi
  - Target: Lungs
- Arterial [systemic emboli]
  - origin: heart chambers
  - Target: Lower limbs 75%

Pulmonary Thromboembolism

- **Asymptomatic** (60%- 80%; small)
- Pulmonary **infarction** (large)
- Pulmonary **hemorrhage**
- Pulmonary **Hypertension** and **right ventricular failure**: (showers of emboli over a long time)
- **Sudden death** (RVF, CV collapse): > 60 % of pulmonary vessels are **obstructed**

Common symptoms ((Non specific))



SADDLE EMBOLUS

Large Embolus Occluding The **Bifurcation** Of **Pulmonary** Artery Trunk (FATAL)

Paradoxical embolus:

Passage of embolus from venous to systemic circulation through **PFO**, **ASD** or **VSD**

from venous → Arterial (systemic)

without → (lungs)

Patent foramen ovale

Atrial sebtal defect

ventricular sebtal defect

Right side of heart ⇒ left side of the heart

2 Arterial (systemic) Thromboembolism

- Emboli traveling within the arterial circulation
- 80% due to **intracardiac mural thrombi (origin)**

- The major **targets** are: cause stroke [سكتة دماغية]
Lower limbs ; Brain ; Intestine; Kidneys; Spleen; etc...
(any organ that has arterial supply!)

causes:
-2/3 Lt. ventricular failure
- ¼ Lt. atrial dilatation
- Ulcerated atherosclerotic plaque
- Aortic aneurysm
- valve vegetation

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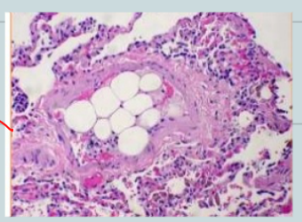
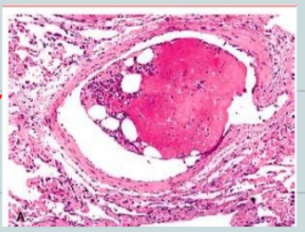
2 Fat embolism

Causes:
1. **Skeletal injury**: (long bones fractures)
2. **Adipose tissue Injury** :(e.g. fat necrosis in **acute pancreatitis**)





Results:
1- Mechanical obstruction of vessels
2- Free fatty acid release: toxic injury to endothelium + systemic immune response
** only 10% or less have clinical findings
= Fat embolism syndrome → Symptoms appear 1-3 days after injury.

Fat embolus = Fat globules + Hematopoietic cells

- **Pulmonary Insufficiency** (rapid breathing; shortness of breath)
- **Neurologic** symptoms (mental confusion; lethargy; coma)
- **petechial rash** (**pinpoint rash**, found on chest, head, and neck area due to bleeding under skin)
- **Fever**
- **Anemia**
- **Thrombocytopenia**
- Death in 10% of cases

→ Therapy For Fat embolism Syndrome

- *no specific treatment*
- *prevention, early diagnosis, and adequate symptomatic treatment are of paramount importance.*
- ***Supportive care** is the mainstay of therapy*
- *Includes: maintenance of adequate oxygenation and ventilation, stable haemodynamics, blood products as clinically indicated, hydration, prophylaxis of deep venous thrombosis and stress-related gastrointestinal bleeding, and nutrition.*

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3 Air Embolism

- Causes:

1. **Surgical** & obstetric procedures
2. **Traumatic chest wall injury**
3. Decompression sickness: in **Scuba deep-sea divers.**
   ((nitrogen))

لا حول ولا قوة إلا بالله العلي العظيم

Clinical consequences

1. **Painful joints**: rapid formation of gas bubbles within Skeletal Muscles and supporting tissues.

2. **Focal ischemia** in brain and heart

3. **Respiratory** distress (chokes) → Lung edema, hemorrhage, atelectasis, emphysema انتفاخ الرئة

4. **Caisson disease**: in **scuba divers**; gas emboli in the **bones** leads to multiple foci of ischemic necrosis, usually the heads of the femurs, tibias, and humeri تخسس العظام

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4 Amniotic Fluid embolism

[very rare
high mortality rate 20-40 %

infusion of amniotic fluid into **maternal** circulation via tears in placental membranes and rupture of uterine veins.

Microscopic Findings upon autopsy:

fetal squamous cells, *lanugo hair*, *fat*, *mucin* .....etc within the **maternal pulmonary microcirculation**

Through veins → heart → lung

Symptoms:

sudden severe **dyspnea**

cyanosis

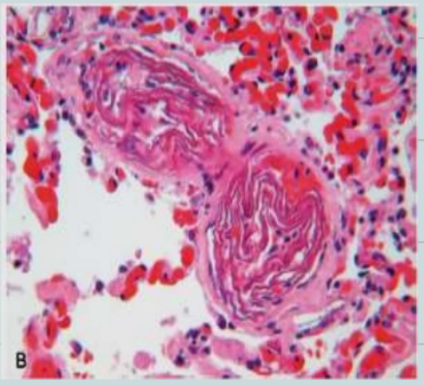
ARDS متلازمة الضائقة التنفسية الحادة

hypotensive shock, followed by seizures, **DIC** and coma

Amniotic Fluid Embolus

= Keratin & fetal squamous cells

(in) pulmonary Arterioles ("maternal")



Infarction

infarct = an area of **ischemic necrosis** caused by occlusion of arterial supply or venous drainage

histologic hallmark :
ischemic coagulative necrosis (ultimately replaced by **scar**)
[note: The *brain is an exception* (liquefactive necrosis)].

- 99% result **from thrombotic/ embolic events**
- other mechanisms: local vasospasm, expansion of atheroma , extrinsic compression of vessel (e.g., by tumor); vessel twisting (e.g. testicular torsion; bowel volvulus); and traumatic vessel rupture

- infarcts may be either red (**hemorrhagic**) or white (**anemic**) and may be either **septic** or **bland**

- **wedge-shaped** (occluded vessel at the apex and periphery of organ forming the base)

- margins of infarcts become defined with time

Factors that influence development of infarcts

- **nature of vascular supply**

- **rate of occlusion development** (collateral circulation)

- **tissue vulnerability to hypoxia** and irreversible damage
  - Neurons → only 3 minutes
  - Myocardial cells → 20 to 30 minutes

- **oxygen content of blood**

RED INFARCTS:

- **occur in any of the following scenarios:**

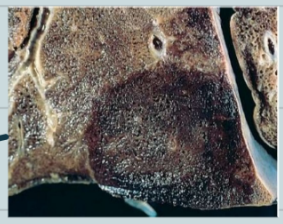
(1) **venous** *occlusions* *(e.g. ovarian torsion)*

(2) **loose** *tissues* *(e.g. lung)*

(3) *tissues with* **dual** *circulations (e.g. lung and small intestine)*

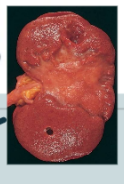
(4) *previously congested tissues because of* **sluggish venous** *outflow*

(5) *when flow is* **re-established** *to a site of previous arterial occlusion and necrosis*



WHITE INFARCTS

- *occur with:* **arterial** *occlusions in* **solid** *organs (such as heart, spleen, and kidney).*



Septic infarctions:

- *occur when infarct is superimposed by infection;*
- *examples:*

1- **infected vegetations**

2- microbes seed an area of necrotic tissue

- infarct is converted into **abscess** *with a greater inflammatory response*

Embolism

Q: If we have an embolus in the pulmonary artery will the embolus be considered of venous or arterial origin and will its final target be the lungs ?

A: Embolism in the pulmonary arteries belongs to venous embolism and the main target is the lung itself .
It is a bit confusing but remember that pulmonary artery carries venous blood from the right side of the heart to the lungs, so it's quite the opposite to what do arteries do.

- *Q: Can pulmonary embolism be of an arterial origin?*
- *A : Yes, the lung can be a target of both venous and arterial embolism types. It depends on the side of the circulation it originated from. So if the embolus is coming from venous circulation and is reaching the lung through the right side of the heart it is a venous embolus. On the other hand, if the embolus is coming out of the left side of the heart and reaching the lung through bronchial circulation, then it is an arterial embolus.*

Patholog of Veins

Varicose Veins

Females > males
30% 10-20%

abnormally dilated, **tortuous veins** produced by prolonged increase in intra-luminal pressure and loss of vessel wall support.

- The **superficial veins of the leg** are most typically involved

Symptoms

venous stasis and **edema** (*simple orthostatic edema*)
+ **cosmetic** effect

Risk Factors

- *Obesity*
- **Female** gender
- *Pregnancy*
- **Familial tendency**
(**premature varicosities** results from imperfect venous wall development)

microscopic morphology

- **Vein wall thinning**
- intimal **fibrosis** in adjacent segments
- spotty medial calcifications (**phlebosclerosis**)
- **Focal intraluminal thrombosis**
- **venous valve deformities** (rolling and shortening)

Complications

- stasis, congestion, edema, pain, and thrombosis
- **chronic varicose ulcers**
- embolism is very rare

& phlebothrombosis Thrombophlebitis

= **Inflammation + thrombosis** of **veins**

- Most common site: **deep leg veins** (90% of all)

Symptoms

distal edema, **cyanosis**, superficial vein dilation, heat, tenderness, redness, swelling, and **pain**

Risk Factors

congestive heart failure, **neoplasia**, *pregnancy*, *obesity*, the **postoperative** state, and **prolonged bed rest** or immobilization

Thrombophlebitis of ***upper limb veins*** are usually associated with local risk factors like:

catheter or canula site; or in some cases can be associated with systemic hypercoagulabilities

Thrombophlebitis

migratory Thrombophlebitis

(**Trousseau sign**):

- **hypercoagulability** occurs as a **paraneoplastic syndrome** related to **tumor elaboration of pro-coagulant factors** (e.g. colon cancer; pancreatic cancer; etc...)

superior vena caval syndrome

caused by neoplasms that **compress** or **invade** the superior vena cava

- Most common is lung cancer

- marked **dilation of veins** of head, neck, and arms with **cyanosis**

inferior vena caval syndrome

- caused by neoplasms **compressing** or **invading** inferior vena cava (m/c: hepatocellular carcinoma and renal cell carcinoma)
- striking tendency to grow within veins

- marked **lower extremity edema**, **distention** of the **superficial** collateral veins of the **lower abdomen** (**medusa**)

اللهم علمنا ما ينفعنا
وانفعنا بما علمتنا
وزدنا علمًا

Pathology of lymphatics

Lymphedema

Swelling & inflammation
Below the Blockage site

- **Primary (congenital)** lymphedema
  Cystic hygroma
  lymphatic **agenesis** or **hypoplasia**.

- **Secondary (obstructive)** lymphedema
  blockage of a previously normal lymphatic
  examples:

  - Malignant **tumors**
  - **Surgical** procedures removing lymph nodes
  - **Post-irradiation**
  - **Fibrosis**
  - **Filariasis**
  - **Postinflammatory thrombosis** and **scarring**

  post treatment effect

Lymphangitis

acute inflammation due to **bacterial infections** spreading into lymphatics

- m/c are **group A β-hemolytic streptococci**.

- lymphatics are **dilated and filled with an exudate** of **neutrophils and monocytes.**

- red, painful subcutaneous streaks (**inflamed lymphatics**), with painful enlargement of the draining lymph nodes (**acute lymphadenitis**).

- Sometimes, subsequent passage into the venous circulation can result in **bacteremia** or **sepsis**.

Chylous

Milky accumulations of lymph in various body cavities

caused by **rupture of dilated lymphatics**, typically obstructed **secondary** to an ***infiltrating tumor mass***

types

- **chylous ascites** (abdomen)

- **Chylothorax** (chest)

- **Chylopericardium** (pericardium)

Arteriosclerosis

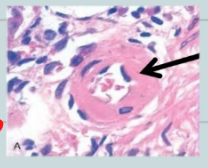
"**hardening** of the arteries"
arterial **wall thickening** and **loss of elasticity**.

Arteriolosclerosis

- affects **small arteries** and **arterioles**
- associated with **hypertension** and/or **diabetes mellitus**

HYPERTENSIVE VASCULAR DISEASE

1 Hyaline Arteriolosclerosis



- Ass. with **benign hypertension**

*elderly patients (normo-tensive)
* diabetis mellitus
- **homogeneous pink hyaline thickening of arteriolar walls**
- luminal narrowing

- leakage of **plasma components** ***across*** injured endothelial cells ***into*** vessel walls
- **increased ECM** production by **smooth muscle cells** in response to ***chronic hemodynamic stress***

Most significant in kidneys
→ ***nephrosclerosis***
(glomerular scarring)

Monckeberg medial calcific sclerosis

calcific deposits in **muscular arteries**

do ***not*** encroach on vessel ***lumen*** and are usually not clinically significant

- **palpable** vessels

typically in persons > age 50

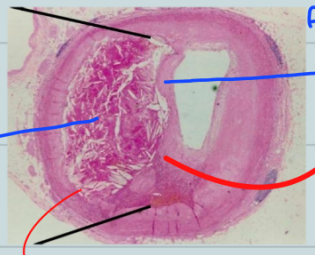
radiographically visible (x-rays, etc...)

Atherosclerosis

characterized by **intimal lesions** =atheromas (a.k.a. atherosclerotic plaques)

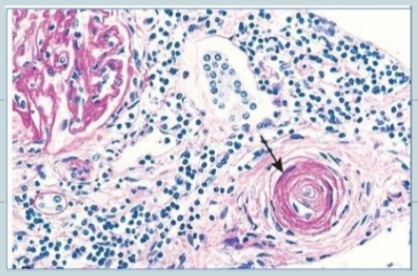
- atheromatous plaque = **raised** lesion with a **core of lipid** (cholesterol and cholesterol esters) **covered** by a **firm, white fibrous cap**

inflammatory process in **endothelial cells** of vessel wall associated with retained low-density lipoprotein (LDL) particles



2 Hyperplastic arteriolosclerosis

- With severe (malignant) hypertension

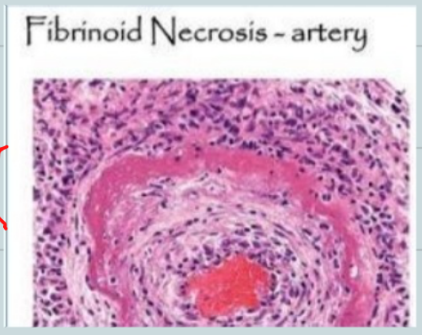


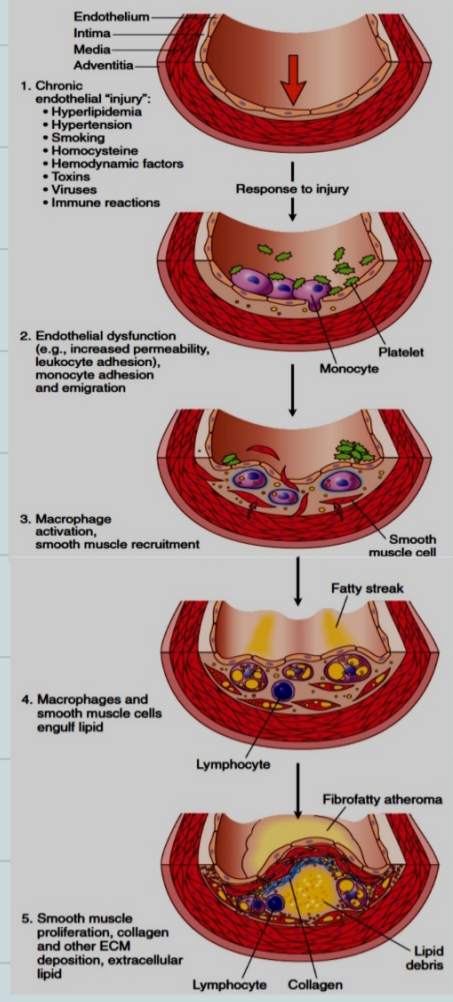
- "onionskin" concentric laminated thickening of arteriolar walls

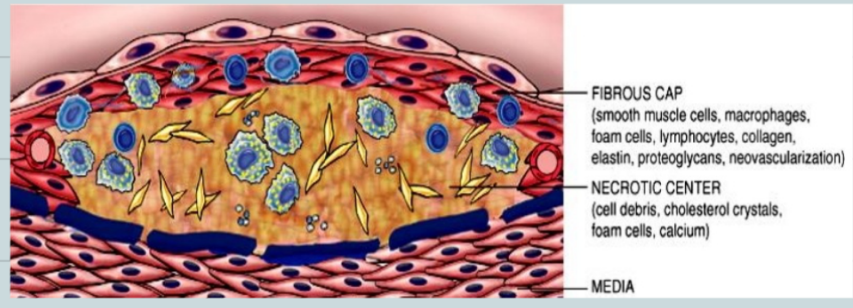
- luminal narrowing

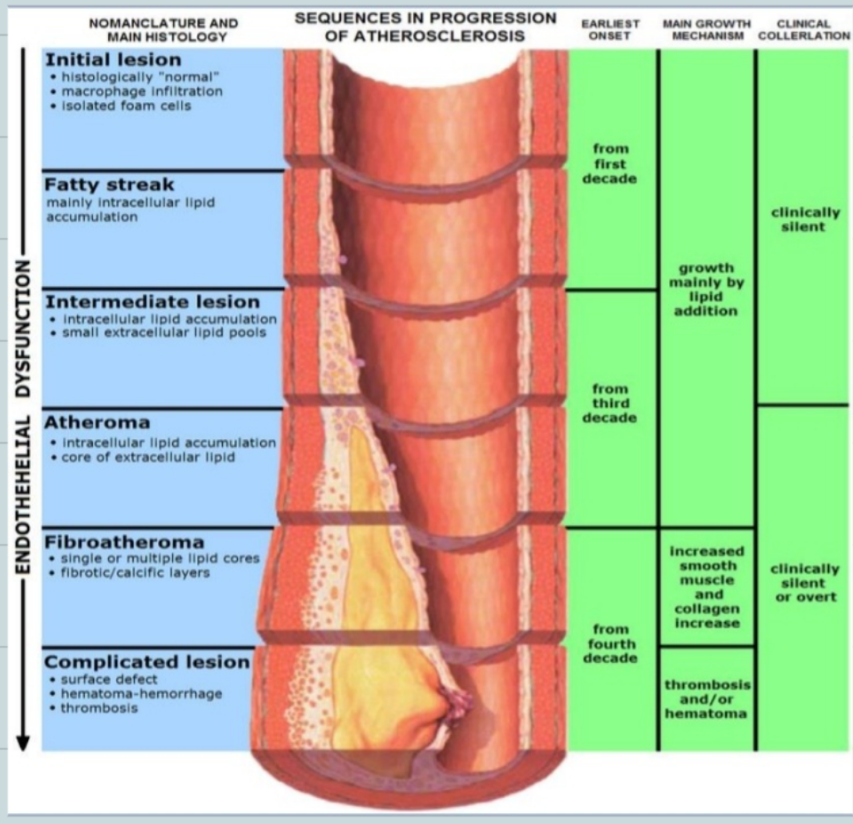
- reduplicated basement membrane

- fibrinoid vessel wall necrosis (necrotizing arteriolitis)

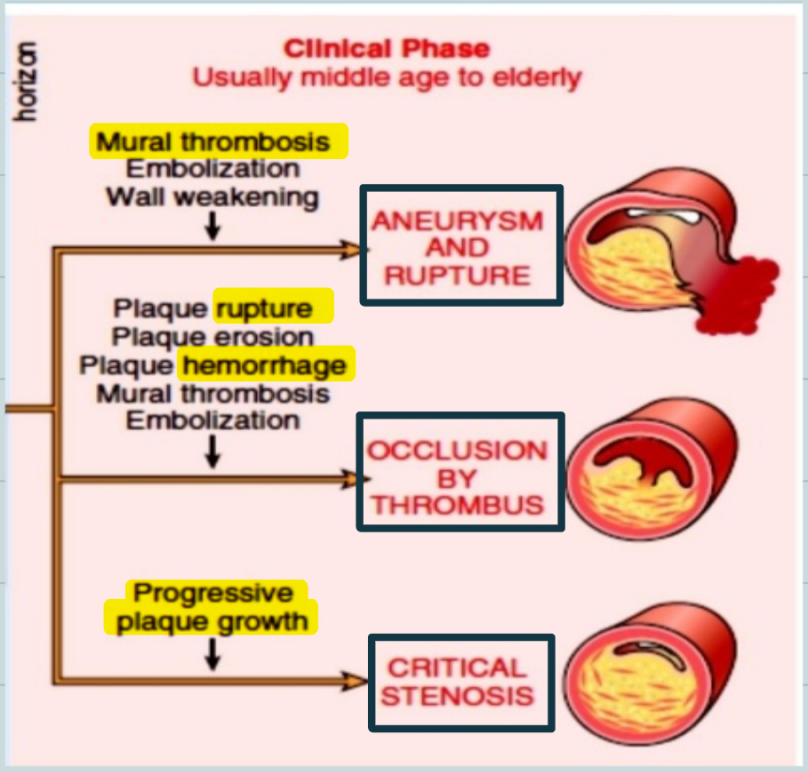








Atherosclerosis progression



Major Risks
Non-modifiable (non-controllable)
Increasing age
Male gender
Family history
Genetic abnormalities
Potentially modifiable (Controllable)
Hyperlipidemia
Hypertension
Cigarette smoking
Diabetes
C-reactive protein (inflammation)

1-age

- ages 40 to 60, incidence of MI in men increases 5 x
- Death rates from IHD rise with each decade

2-Gender

- Premenopausal* → protected against atherosclerosis compared with age-matched men.
- After menopause→ incidence of atherosclerosis-related diseases increases

* unless they are otherwise predisposed by diabetes, hyperlipidemia, or severe hypertension.

3-Genetics

- familial predisposition is multifactorial.
- Either :

1- familial clustering of other risk factors

- e.g. HTN or DM

or :

2- well-defined genetic derangements in lipoprotein metabolism

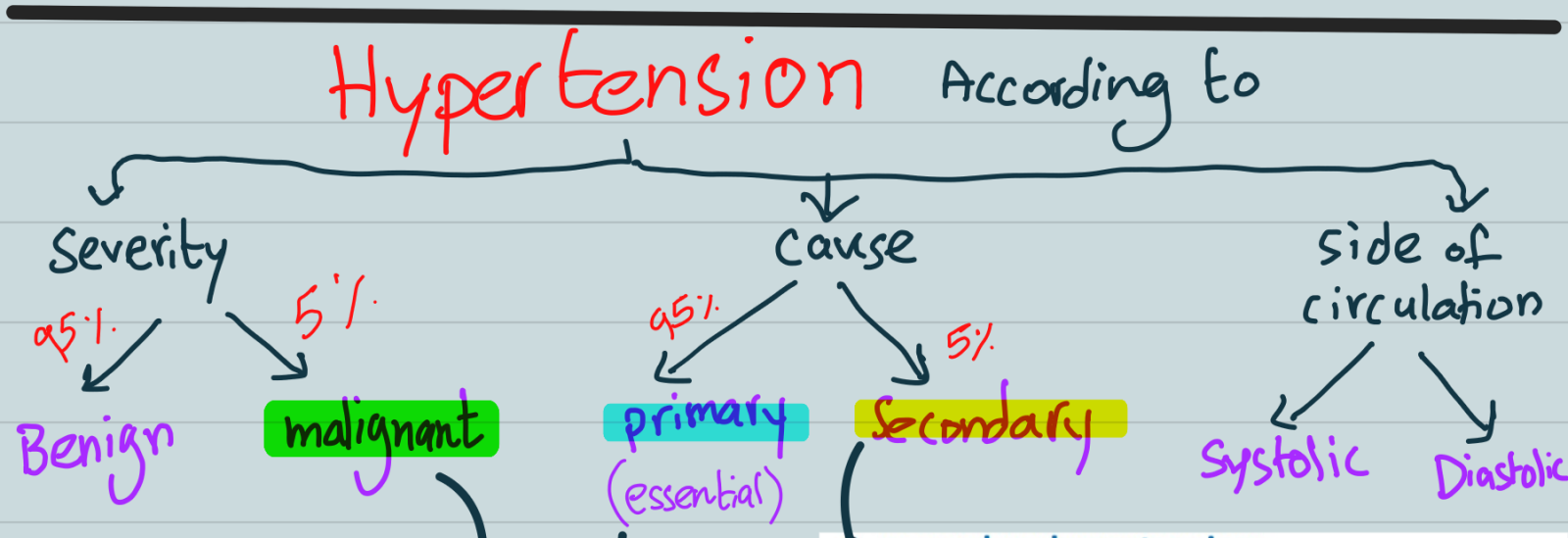
- e.g. familial hypercholesterolemia

Additional Risk Factors for atherosclerosis

- 20% of cardiovascular events occur in the *absence of identifiable risk factors:*

- **Hyperhomocystinemia**
- ***Metabolic syndrome***
- **Lipoprotein a levels**
- **Factors Affecting Hemostasis** (*Elevated levels of procoagulants; Clonal hematopoiesis*)
- **Others:**

-lack of exercise
-competitive, stressful lifestyle ("type A" personality)
-obesity
-High carbohydrate intake



- ***Malignant hypertension***

→ 5% (also known as accelerated HTN)

→ a rapidly rising blood pressure that, if untreated, leads to death within 1 to 2 years

→ **systolic pressures > 200 mm Hg or diastolic pressures > 120 mm Hg**

✓→ renal failure and retinal hemorrhages

✓→ usually superimposed on preexisting benign hypertension (either essential or secondary)

2- secondary hypertension:

Most common: renal disease or renal artery narrowing **(renovascular hypertension)**

Other less common: many other conditions....

- ***Pathogenesis of essential HTN*** (idiopathic)
- **? Genetic factors**

? **familial clustering of hypertension**

● angiotensinogen polymorphisms and angiotensin II receptor variants; polymorphisms of the renin-angiotensin system.

● ? Susceptibility genes for essential hypertension: genes that control renal sodium absorption, etc...

- **Environmental factors** modify the impact of genetic determinants

stress, obesity, smoking, physical inactivity, ↑ salt consumption

HTN Potential complications :

- **stroke** (CVD) & multi-infarct dementia
- **atherosclerotic** coronary heart disease
- cardiac **hypertrophy** and heart **failure** (hypertensive heart disease)
- aortic *dissection*
- renal failure
- retinal hemorrhages

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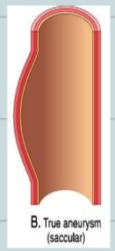
Aneurysm

localized abnormal **dilation** of artery or heart

according to macroscopic shape & size

- **Saccular**
  - **spherical** outpouchings
  - -involving **only a portion** of vessel wall
  - - may contain **thrombi**

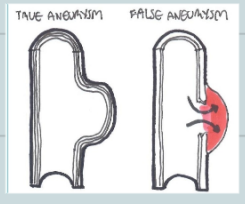


- **fusiform**
  - **diffuse, circumferential** dilation of a **long vascular segment**
  - they vary in diameter and length and can involve **extensive portions** of artery

Note: shape and size are not specific for any disease or clinical manifestations

Types

- **True**
  - **all three layers of arterial wall or heart**
  - e.g.
    - Atherosclerotic, syphilitic, congenital aneurysms,
    - ventricular aneurysms following transmural MI



- **False**
  - **pseudo-aneurysm**
  - **a breach in vascular wall** leading to hematoma communicating with **intravascular space ("pulsating hematoma")**
  - E.g. ventricular **rupture** after MI contained by pericardial adhesion
  - E.g. a leak at the junction of a vascular **graft** with a natural artery.

1 Aortic Aneurysms — The 2 most important causes

1- Atherosclerosis :
- most common cause
→ intimal plaques compress underlying media
→ compromise nutrient and waste diffusion into arterial wall
→ media degeneration and necrosis
→ thinning and weakening of media
→ dilation of vessel

2- Cystic medial degeneration of arterial media
- causes include: trauma; congenital defects (e.g., *berry* aneurysms); hereditary defects in structural components (Marfan); infections (*mycotic* aneurysms); vasculitis.

Abdominal Aortic Aneurysm AAA

- Atherosclerotic aneurysms occur most frequently in **abdominal aorta** (= AAA)

- common iliacs, arch, and descending parts of thoracic aorta can also be involved

- m/c in **men** rarely **< age 50**

- **Atherosclerosis** is a ***major*** cause of AAA
- other contributors include:

Symptoms of Aortic Aneurysm
hoarseness
chest pain
dyspnea
AAA deep abdominal pain or discomfort
pulsating feeling

1- **Hereditary defects in structural components of the aorta**:
(e.g., **Marfan disease** by **defective fibrillin production** affects elastic tissue synthesis) 24

2- An **altered balance of collagen degradation and synthesis** mediated by **local inflammatory** infiltrates and the **destructive proteolytic enzymes**- (e.g. **vasculitis**)

- Usually below renal arteries and above bifurcation of aorta

- can be saccular or fusiform
- may be as large as 15 cm in diameter, and as long as 25 cm

- Microscopically: **atherosclerosis**; ***thinning of media***
- frequently contains a laminated mural thrombus

Complications
* Rupture
* Thrombosis

- Rupture → massive **hemorrhage**. - risk is directly related to size (≥5 cm)

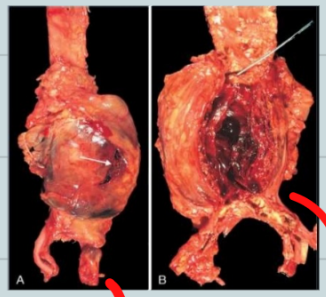
- mortality for unruptured aneurysms =5% - if **rupture** mortality rate **> 50%**

- Obstruction of downstream vessel → ischemic injury

- Embolism
  - mural thrombus

- compression on adjacent structures (e.g. ureter or vertebrae)

- abdominal mass (often pulsating)



rupture

Thrombosis

2 Mycotic Aneurysms

- **Infection** of a **major artery** that weakens its wall is called a mycotic aneurysm
- can originate from:

(1) embolization of a septic **thrombus** (infective endocarditis)

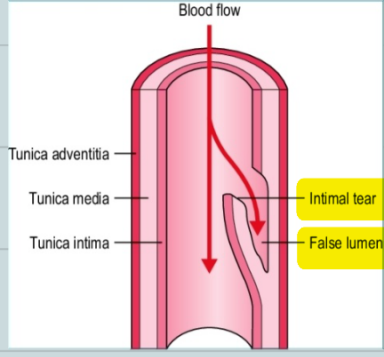
(2) extension of adjacent suppurative process

(3) circulating organisms infecting arterial wall

3 Syphilitic Aneurysm

- Caused by The spirochetes T. pallidum
- A **rare** complication (early recognition and treatment of syphilis)
- **Tertiary stage of syphilis** can cause obliterative endarteritis of **vasa vasorum of aorta**
- ischemic medial injury
- aneurysmal dilation of aorta and aortic annulus
- eventually valvular insufficiency

Arterial Dissection



- **Extravasation** of blood that enters the wall of artery through an **intimal tear**, as a **hematoma** dissecting **between its layers.**

- often but not always aneurysmal

- Both **true** and **false** aneurysms as well as dissections **can rupture**, often ***with catastrophic consequences***

Aortic dissection

- A **catastrophic event** whereby **blood dissects apart** the **media** to form a **blood-filled channel** within aortic wall

- Complications are :

- **massive hemorrhage**

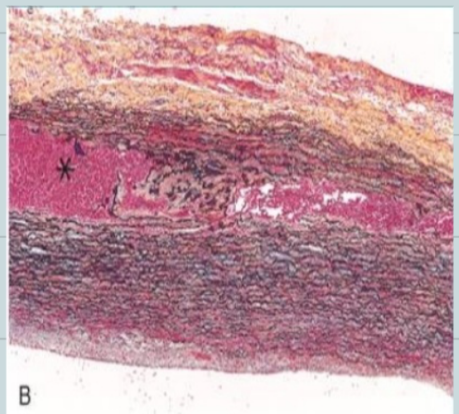
- **cardiac tamponade** (**hemorrhage into the pericardial sac**)

manifestations of aortic dissection
- Sharp chest/ back pain
- **Weak pulses in downstream arteries**

- If ruptures into pericardium → cardiac tamponade

- **Blood pressure difference between Rt & Lt arms**
- ***Hypotension***
- **shock**

Silver stain: display elastic fibers in black color



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** among **inherited** or **acquired** **connective tissue disorders** assosiated with aortic dissection

- Autosomal dominant disease of **fibrillin**, an ECM scaffolding protein required for normal elastic tissue synthesis

- Manifestations include:
- **skeletal abnormalities** (elongated axial bones)

- **ocular findings** (lens subluxation) خلع جزئي

- **cardiovascular** manifestations

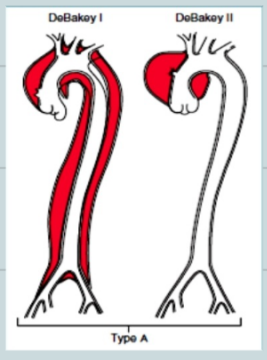
Aortic dissection Types

Type A dissection

- **More common**
- More **dangerous**

- **Proximal** to takeoff of major aortic branches

- involve either **ascending aorta only** or **both ascending and descending aorta** (**types I** and **II** of the ***DeBakey*** **classification**)



Type B dissection

- **Distal** to take off of major aortic branches

- Does **not** involve ~~**ascending**~~ aorta

- usually beginning **distal to subclavian artery**

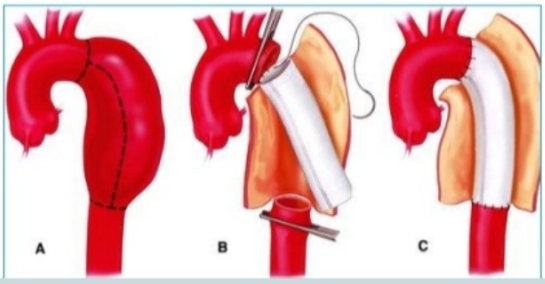
- Also called ***DeBakey type III***

Clinical course

- Previously, aortic dissection was typically fatal, but prognosis has markedly improved Rapid diagnosis and institution of:

1- antihypertensive therapy

2 - surgical procedures involving plication of aorta, wall reconstruction with synthetic graft



all arise From endothelial cells

Vascular Tumors

benign tumors

Contain vascular channels
Lined by normal-appearing endothelial cells

most common
No metastasis
Benign behavior

e.g. hemangioma

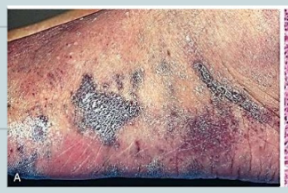
- common
- composed of **blood-filled vessels.**

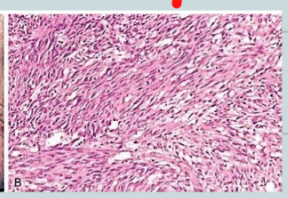
- m/c age: infancy & childhood
- Most are present **from birth**

- many **regress spontaneously** (↓ size)

- m/c location: **head and neck**
- Some in internal organs (1/3 → liver)

Malignant transformation: very rare





Borderline tumors

intermediate between benign and malignant behavior

Rare
No metastasis
Locally aggressive

e.g. kaposi sarcoma

a vascular neoplasm caused by human herpesvirus- 8 = HHV-8

- Several types: classic; endemic; Transplantation-associated; and AIDS-associated;

- **AIDS-associated (epidemic)** KS is an AIDS-defining illness (used as a criterion for diagnosis of AIDS)
- the most common HIV-related malignancy

kaposi sarcoma: **Multiple red-purple skin plaques** or **nodules**, usually on the **distal lower extremities**; *progressively increase in size and number* and **spread proximally**

Malignant tumors

More cellular
Cytologic atypia
Proliferative
Do not form well-organized vessels

rare
metastasis

e.g. angiosarcoma

lesions can occur at **any site,** but most often involve the **skin, soft tissue, breast, and liver.**

- Pathogenesis= ? Carcinogens; ?unknown

- **A latent period between exposure and tumor development**

RISK FACTORS OF ANGIOSARCOMA

- Chemical **carcinogens** → liver angiosarcoma

- **Irradiation**

- Lymphedema → - e.g. ipsilateral upper extremity several years after radical mastectomy with lymph node resection for breast cancer

- **foreign bodies → long-term** (years)

Hemangiomas

Capillary hemangioma

most common type

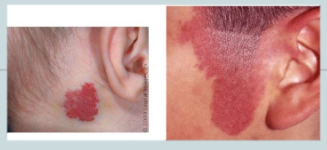
- skin and mucous membranes of oral cavity & lips

Strawberry hemangioma of newborn

(juvenile hemangioma)

- m/c head & neck

- Usually regress with time



Pyogenic granuloma

rapidly growing pedunculated lesions on gingival mucosa

- 1/3 *history* of trauma

Cavernous hemangioma

large, *dilated* vascular channels

- deep organs (liver most common)

- do not spontaneously regress

Cardiac Tumors

- Very rare

- Metastatic Neoplasms are the most common malignancy of heart (5% of patients dying of cancer).

- most common source → lung cancer

- Angiosarcomas → most common primary malignant tumor of heart.

- Benign tumors are also very rare but important for their critical location

CLINICAL FEATURES AND SIGNIFICANCE

1- "ball-valve" obstruction
2- Embolization
3- fever and malaise → tumor elaboration of interleukin-6

- Diagnosis: Echocardiography

- Treatment: surgical resection in benign tumors.

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