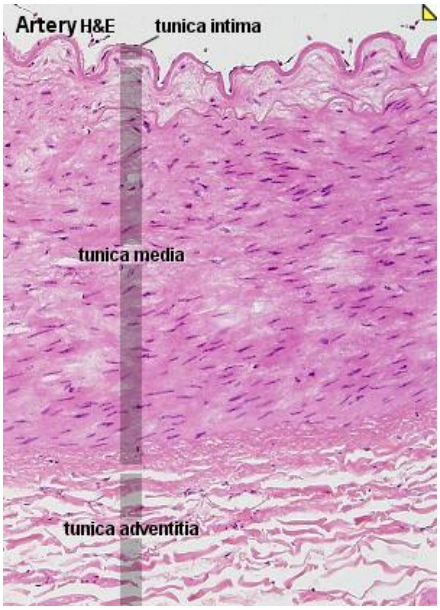
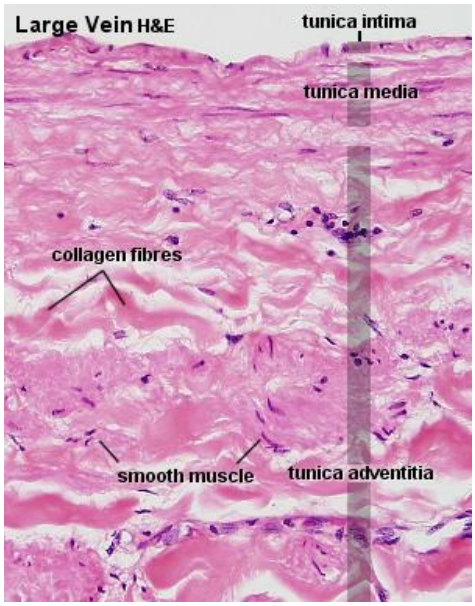




Varicose Veins:

Abnormally dilated tortuous (multawi) veins.

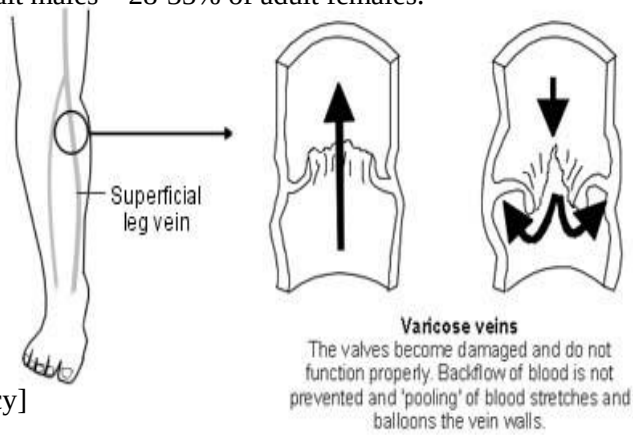
Produced by:

- Prolong standing → Long periods of *Venous Pressure* → prolonged increase in *Intra Laminal Pressure*.
- Loss of vessel-wall *Support Site*.
- Thrombosis (common but not clinically significant)
- Valve incompetence rule
- **Haemorrhoides**
  [The term hemorrhoids refers to a condition in which the veins around the anus or lower rectum are swollen and inflamed]
  esophageal varices
  [abnormally enlarged veins in the lower part of your esophagus – a complication of cirrhoisis]

Common site of involvement: superficial veins of the lower leg 10-20% of adult males – 28-35% of adult females.

Increased Risk when:

- Obesity
- Hereditary
- Proximal thrombus
- legs are dependent for long periods
- Higher incidence in women (pregnancy)
- Cirrhosis of liver --- esophageal varices
- Hemorrhoids --- varicosity of the "hemorrhoidal plexus"



Complications:

- Stasis Dermatitis [chronic eczematous dermatitis due to venous insufficiency]
- Delayed healing
- Stasis ,edema ,tropic skin [stasis: a stoppage/diminution of flow – impairment/cessation of blood flow]
- Varicose ulcers [ulcers of varicose veins]

Thrombophelibitis/ Phelbothrombosis:

Venous thrombosis with inflammation or without

Predisposing Factors:

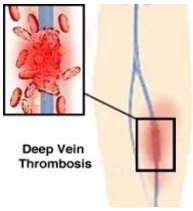
Cardiac Failure – Pregnancy – obesity – immobilization (paralyzed) – genetic hypercoagulability syndrome – neoplasia ..etc.

Common Sites of Involvment:

Deep leg veins (90% of cases)

Other sites: - The periprostatic venous plexus in males.
- The pelvic venous plexus in females.
- The large veins in the skull and the dural sinuses (especially in the setting of infection or inflammation).
- Portal Tracts (a distinctive arrangement in the liver).
- Popliteal (area behind the knee) ,femoral and iliac veins (continuation of femoral vein).
- Superficial Saphenous Vein (the two principal veins that run the leg) – not clinically significant.

Deep Vein Thrombosis (DVT):



- 50% Clinically silent (asymptomatic) (no symptoms)

Local Manifestations:

- ❖ Distal edema
- ❖ Cyanosis
- ❖ Superficial vein dilation
- ❖ Heat ,tenderness ,redness ,swelling and pain
- ❖ Sometimes , the 1st manifestation of "thrombophlebitis" is a **Pulmonary Embolus** [sudden blockage in lung artery] OUTCOME depending on No. of emboli, it range from "no symptoms at all" to "death"

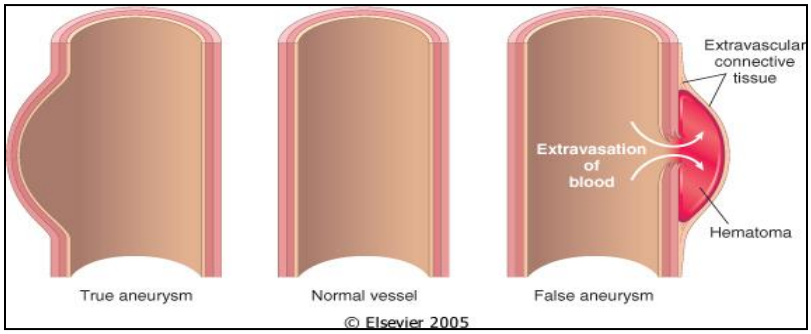
Aneurysms:

Localized abnormal dilation of a blood vessel or the wall of the heart.

- ➢ **True Aneurysm:** when the aneurysm is Bounded by arterial wall components or attenuated wall of the heart

Examples of *True Aneurysms*:
- Atherosclerotic
- Syphilitic (zuhary)
- Congenital vascular aneurysms

- Left ventricular aneurysm that can follow a **myocardial infarction**

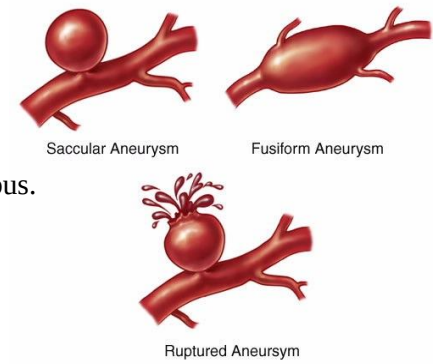


Myocardial Infarction:
gross necrosis of the myocardium ,due to interruption of the blood supply to the area

Hematoma:
localized swelling filled with blood , resulting from a break in a blood vessel.

- ➢ **False Aneurysm (pseudoaneurysm):**
  An extra-vascular **hematoma** that freely communicates with the intravascular space
  Vascular wall have been *Breached* (ma5roo8) - the external wall of the *Aneurysmal Sac* consists of only: outer arterial layers, perivascular tissue or blood clot.
  e.g. Blunt thoracic Trauma [injury in the chest]

- ➢ **Classification:**
  According to macroscopic shape and size:
  - **Saccular Aneurysms**
    essentially *Spherical* (involving only a portion of the cell wall)
    vary in size and diameter (from 5-20 cm.) partially/completely fulled by thrombus.
  - **Fusiform Aneurysms**
    (involving a long segment) vary in diameter and in length.



- ➢ **Most important causes of "Real Aortic Aneurysms":**
  - Atherosclerosis
  - Cystic medial degeneralion (of the arterial media)

Any *vessel* may be affected by *variety of disorders* that weaken arterial walls.
((e.g Congenital defects,fungal infections,syphilis,trauma,immune-mediated diseases etc.))

1. **Atherosclerotic Aneurysm:**
   The most frequent etiology of Aneurysms
   Accords secondary to: destruction of *media* due to the ***Atherosclerotic Plaque*** that forms in the *intima*.

   Most Frequently in: Abdominal Aorta → **A**bdominal **A**ortic **A**neurysm (AAA).
   Two Variants of AAA: 1) Inflammatory Abdominal A.
   2) Mycotic Abdominal A.

   Clinical Consequences in AAAs:
   - **Rupture** into the peritoneal cavity [the potential space between the parietal and the visceral peritoneum.] or Retroperitoneal tissues *WITH* potentially fatal hemorrhage
   - **Obstruction** of a vessel – particularly of the *Iliac* [ilium: cranial portion of the hip bone] , *Renal ,Mesenteric/Vertebral branches* [that supply the spinal cord] -leading to → ischemic tissue injury.
   - **Embolism (jal6a)** from *Atheroma* [Fatty deposits which lead to the formation of plaques in the blood vessels.] or *Mural thrombus* [thrombus attached to the wall of the endocardium]
   - **Impingement (e9tedam)** on an adjacent structure, such as compression of a ureter / erosion (t2akol) of vertebrae.
   - Presentation as an **Abdominal Mass** (often palpably pulsating) that stimulates a **Tumor**.

2. **Aneurysm due to *Cystic Medial Necorsis:***
   The *Most Frequent* aneurysms of the *Aortic Root*.

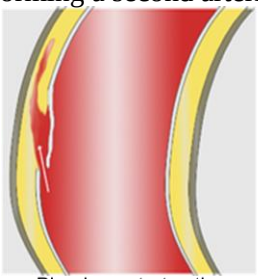
Cystic Medial Necrosis/degenration:
disorder of large arteries.
A degenerative breakdown of collagen, elastin, and smooth muscle caused by aging contribute to weakening of the wall of the artery.

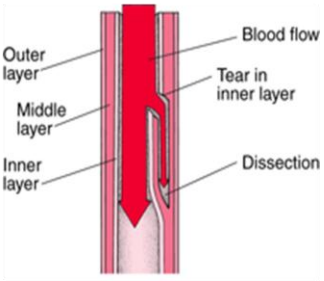
3. **Berry Aneurysm:**
   Small *saccular lesions* most often seen in the *small arteries of the brain* ((especially in the circle of Willis))
   - Aren't present at birth, but develop at sites of *Congenital Medial Weakness* at bifurcations (tasha3ob) of cerebral arteries.
   - Unrelated to *athersclerosis*
   - Often → an association of ***Adult Polycystic Disease*.**
   - **The most frequent cause of *Subarachnoid Hemorrhage*.**

4. **Syphilitic (leutic) Aneurysm:**
   A manifestation of *Teritary Syphilis*.
   Frequency of this complication had been decreased –by → better control and treatment of syphilis in its early stages.
   Casued by: Syphilitic Aortitis.
   Involves: Aortic arch and the ascending aorta (root of aorta).

5. **Aortic Dissection /Dissecting hematoma/ Dissecting aneurysm:**
   the splitting/dissection of an arterial wall by the blood entering through an intimal tear
   A longitudinal tear → usually in ***the wall of the ascending aorta***, forming a second arterial lumen within the media.

   A catastrophic (BIG) illness
   Along the luminar planes of the media
   with the **formation of → intramural blood-filled channel**
   –ruptures → causing sudden hemorrhage → produce death


Blood penetrates the *intima* and enters the *media* layer.



➢ **Morphology:**
Dissection can extend along the aorta
proximally and distally toward the heart
(sometimes)
→all the way into the iliac and femoral arteries.
Ruptures out → causig massive hemorrhage.

(in some instances)
the blood re-ruptures into the lumen of the aorta, -producing→ a second or distal intimal tear & a new vascular channel within the *media* of the aortic wall. (to produce a "double-barreled aorta" with a "false channel")

➢ **Main Characteristics:**

- Clinically present with *Severe Chest Pain*
- Can be confused with heart attach
- Can result in the aortic rupture with bleeding into the pericardial sac, causing hemopericardium and fatal cardiac tamponade.
- It is typically associated with **hypertension** or with cystic medial necrosis (esp. in Marfan syndrome)
- Has no relation to **atherosclerosis**

Hemopericardium:
presence of blood or bloody effusion in pericardial sac

Cardiac Tamponade:
Cardiac compression caused by an accumulation of fluid within the pericardium

Vasculitis:

Mostly immune reaction related:

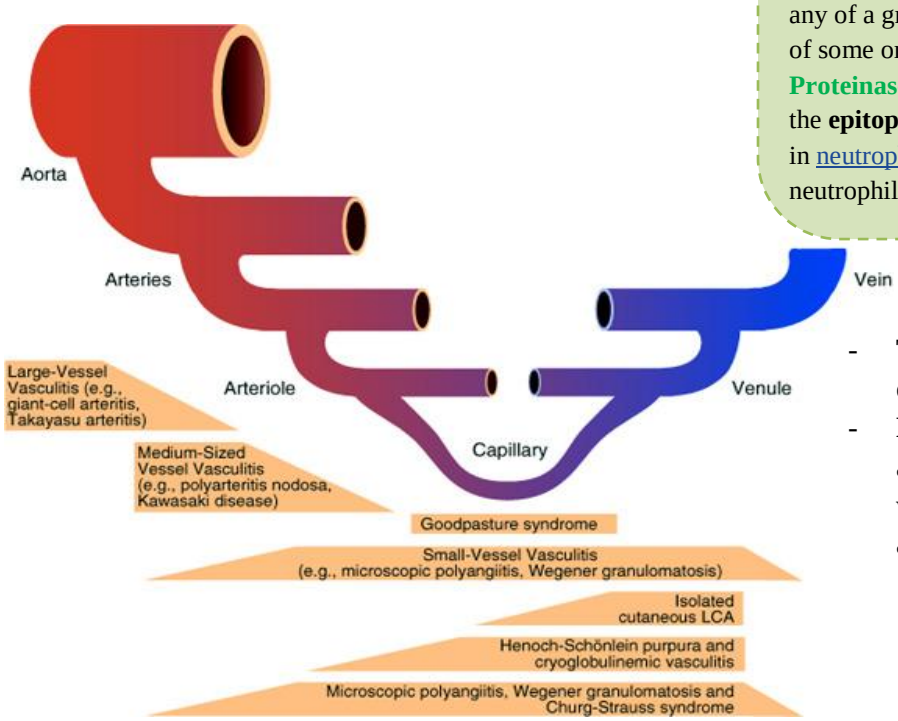
- Immune complexes deposited in the vessel → inflammation of the vessel wall.
- Direct invasion of the vessel wall by *infectious pathogen*.

Immune Complex Mediated:

❖ Infecton ,drug induced ,**S.L.E** …etc
[systemic lupus erythematosus: a chronic ,multifaceted inflammatory disease that can affect any organ system of the body].

❖ Anti-neutrophil cytoplasmic antibodies (ANCAs)
P-ANCAs (perinuclear → myeloperoxidase)
C-ANCAs (cytoplasmic → proteinase 3)

Myeloperoxidase:
a hemoprotein having peroxidase activity, occurring in the primary granules of promyelocytes, myelocytes and neutrophils, and which exhibits bactericidal, fungicidal and virucidal (destructive to viruses) properties.
Peroxidase:
any of a group of iron-porphyrin enzymes that catalyze the oxidation of some organic substrates in the presence of hydrogen peroxide.
Proteinase 3:
the **epitope** of ANCAs .A serine protease enzyme expressed mainly in neutrophil granulocytes. Its exact role in the function of the neutrophil is unknown



- **The widths of the trapezoids** indicate the frequencies of involvement of various portions.
- **Note** that large-, medium-, and small-vessel vasculitis affects arteries, but **only small-vessel vasculitis** involves vessels smaller than arteries. LCA, leukocytoclastic angiitis.

Antineutrophilic Cytoplasmic Antibodies:

serve as useful quantitative diagnostic markers for these conditions, and their levels may reflect the degree of inflammatory activity.
How does it reflect it?? It rise in episodes of recurrence –thus→ are useful in management.

1) Wegner Granulomatosis:

A necrotizing vasculitis, characterized by a triad of:

a) **Acute Necrotizing granulomas** of the upper respiratory tract (ear, nose, sinuse, throat) } or both.
the lower respiratory tract (lung)

b) **Necrotinizing/granulomatous Vasculitis**
affecting small to medium-sized vessels (e.g. capillaries, venules, arterioles and arteries)
most prominent in the lung and upper ways **but** affecting other sites as well.

c) **Renal Disease** in the form of of *Focal Necrotizing*, often *crescentic*, *glomerulitis*.

Limited forms/more widespread WG
C-ANCA is present in 95% in the serum of patients.
May lead to death in 2 years in 90% of not treated patients.
Male affected more than females – age average over 40.
Epitheloid cells + giant cells can be seen in it.

Clinical Features:
Untreated – corse is malignant – 80% of patients die in 1st year.

2) Macroscopic Polyangitis/ Microscopic polyarteritis/ lucocytoclastic vasculitis:

A necrotizing vasculitis ,affects "arterioles, capillaries, venules" – all lesions tend to be at the same age

- **Involves:** skin – mucous membranes – lungs – brain – heart – GI – kidney – muscle.
- **Precipitating cause:** immunologic reaction to an antigen
(**Drug**: pencillin) ,(**microorganisms:** strept.), heterolgous proteins and tumor antigens.
- **P-ANCAs** in 70% of patients.
- **Diagnosis:** skin biopsy – histologically there's infiltration of **vessel wall** with **neutrophils**
→ become fragmented (neutrocytoclasia)

3) Polyateritis Nodosa (PAN):

A systemic vasculitis of small/medium sized **muscular arteries** (not arterioles, capillaries or venules)
involving *Renal* and *Visceral* **vessels** – (sparing the pulmonary circulation).
Clinical Manifestaions:
result from *ischemia and infarction* of affected tissues and organs.
Morphology:

- Classic PAN occurs as segmental transmural necrotizing inflammation of **arteries of medium to small size, in any organ** with the possible exception of the lung (most frequently kidney, heart, liver and gastrointestinal tract)
- Individual's lesions may involve **only a portion of the vessel circumference (mu7ee6).**

- ***Histologic Picture During the acute phase***:
Charcterized by: transmural inflammation of the arterial wall **with** neutrophils, esoinophils, mononuclear cells
frequently accompanied by *Fibrinoid Necrosis* (lumen may become thrombosed).
- ***Later*** the acute inflammatory inflitrate (e5tera8)
disappears and replaced by **Fibrous thickening of vessel wall** may extend into the adventitia.

- ***Characeristic of PAN:*** all stages of activity may **COEXIST** in different vessels/within same vessel.

4) Temporal (giant cell, cranial) arteritis:

Segmental Acute & Chronic (often granulomatous) vasculitis
Involving: predominantly (1st) the larger arteries in the head – particularly, the branches of ***Carotid Artery***
[temporal arterial branches of the ophthalmic arteries] --- Overage of 50 – M:F = 1:2
Morphology:

- **Characteristically,** segments of affected arteries develop ***nodular thickenings*** with reduction of the lumen.
- There is **granulomatous inflammation** of the inner half of the media
((with lymphocytes and, multinucleate giant cells and fragmentation of the internal elastic lamina)).
- **The healed stage** reveals *collagenous thickening* of the vessel wall
sometimes the artery is transformed into a fibrous cord.

Clinical Features:

- Most common in older individuals – rare before age 50
- Fever, fatigue, weight loss
- facial pain or headache
  often most intense along the course of the superficial temporal artery, which may be painful to palpation.

- More serious are **ocular symptoms** (associated with involvement of the ophthalmic artery)
  range from diplopia to transient or complete vision loss.
- The **diagnosis** depends on biopsy and histologic confirmation.

5) Thromboangiitis obliterans (Buerger disease):

Inflammation of medium-sized and small arteries, principally the tibial and radial arteries and sometimes secondarily extending to veins and nerves of the extremities.

Begins before age 35 in most cases – cigarette smoking (one of the most consistent aspects of this disorder).

((the lumen is occluded by a thrombus containing two abscesses // The vessel wall is infiltrated with leukocytes)).

Antineutrophil Cytoplasmic Antibodies:

a heterogeneous group of autoantibodies directed against enzymes

Found: within the ***azurophil or primary granules in neutrophils*** -- ***in the lysosomes of monocytes***, -- ***in endothelial cells***.

On what is the description of these autoantibodies is based?

On the **immunofluorescent patterns of staining** of ethanol-fixed neutrophils.

Main Patterns:

- Cytoplasmic Localization of the staining (c-ANCAs)
- Perinuclear staining (p-ANCAs) --- is usually specific for **Myeloperoxidase (MPO).**

Psuedoaneurysms:

false aneurysm, differs from a true aneurysm in:

that its wall does not contain **the components of an artery**, but consists of **fibrous tissue**, which usually continues to enlarge, creating a ***pulsating hematoma.***